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PAPER II · VOLUME FIVE

MD and DNB psychiatry, theory revision

Clinical Psychiatry

Personality, Eating and Sleep

The personality disorders, how each is recognised and told apart from the others. Then disordered eating and disordered sleep, in full.

Dr. Wilfred D'souza · Dr. Niharika Reddy

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WEAVE SPRINT · PAPER II

Clinical Psychiatry

THE WEAVE SPRINT SERIES

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

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

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

ACKNOWLEDGEMENT

A word of thanks

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

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

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

Dr. Wilfred D'souza

Foreword

This fifth volume holds two subjects joined by duration rather than by symptom. Personality disorder is a diagnosis of pattern rather than episode, and it changes how the rest of the paper is read. The eating and sleep-wake disorders follow, both of them conditions in which a psychiatric problem is measured in the body before it is measured anywhere else.

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

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

HOW TO USE THIS BOOK

The colour memory code

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

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

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

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

Contents

FRONT MATTER

Foreword 7

How to use this book 8

PART IX **Personality Disorders** 11

- Framework and classification 12
- The personality disorders by cluster 44
- Aetiology, assessment and treatment 86
- Apply and consolidate 137

PART X **Eating and Sleep-Wake Disorders** 160

- Eating disorders 161
- Sleep-wake disorders 229
- Apply and consolidate 294

BACK MATTER

Subject index 318

About the authors 322

PART IX

IX

Personality Disorders

The personality disorders: how each is recognised, told apart from the others, and understood.

01 Framework and classification

02 The personality disorders by cluster

03 Aetiology, assessment and treatment

04 Apply and consolidate

PAPER II

Personality disorders: framework, types and treatment

Three sections . one complete notes document

This material gathers the personality disorders into one document, first the framework and the classification, then the disorders themselves, then how they are assessed and treated. The first band is **the framework and classification**: the definition and the general diagnostic criteria that must hold before any type is named, the ICD-11 dimensional model that abolished the categorical types for a severity gradient and a set of trait domains, the DSM-5 categorical clusters and their ten types with the alternative hybrid model alongside, and the comparison of the two approaches. The second band is **the personality disorders by cluster**: the cluster A odd and eccentric disorders, borderline personality disorder as the highest-yield type with its features, course and complications, antisocial personality disorder and its forensic significance, the histrionic and narcissistic disorders, the cluster C anxious and fearful disorders, and the borderline-versus-bipolar-II differential that examiners return to. The third band is **aetiology, assessment and treatment**: the genetic, neurobiological and developmental aetiology and the Linehan biosocial model, the assessment instruments and structured interviews, and the treatment block, dialectical behaviour therapy, schema therapy, mentalisation-based and transference-focused psychotherapy, the symptom-targeted pharmacotherapy, and the integrated borderline plan. The examinable skill is the safe, guideline-anchored diagnosis and treatment of each disorder: which criteria gate a personality-disorder diagnosis, how the classification shifted and why exams still test the categorical names, which disorder behind which presentation, the borderline-versus-bipolar-II boundary, which structured psychotherapy for which patient, and the rule that a drug is not the first-line treatment for borderline personality disorder.

HOW TO USE THESE NOTES

Read the three bands as one continuous account of how the personality disorders are classified, recognised and treated. The **framework and classification** band builds the frame: the general criteria that gate every diagnosis, the headline nosology shift where ICD-11 replaced the categorical types with a dimensional model of severity and trait domains, the DSM-5 categorical clusters and their ten types cross-mapped, and why exams still test both. The **personality disorders by cluster** band teaches the disorders themselves, cluster A, borderline personality disorder in full, antisocial personality disorder and its forensic frame, the histrionic and narcissistic disorders, cluster C, and the borderline-versus-bipolar-II differential. The **aetiology, assessment and treatment** band teaches the aetiology and the Linehan biosocial model, the assessment instruments, and the treatment block, where structured psychological therapy is primary and dialectical behaviour therapy, schema therapy, mentalisation-based and transference-focused psychotherapy are owned and fully taught here. Every management recommendation is guideline-anchored, led by NICE with the Indian Psychiatric Society and WFSBP positions stated, on the where-to-treat, target-by-phase, modes-of-treatment spine; every named disorder ends with a **Good to remember** box giving the defining criterion, the discriminator and the first step, and every named therapy ends with one giving the mechanism, the indication and the signature caution. 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. Several boundaries are stated up front and cross-referenced by chapter name: the deep pharmacology of the mood stabilisers, low-dose antipsychotics and serotonin reuptake inhibitors used in symptom-targeted treatment is taught in the Mood disorders: pharmacotherapy notes and the Schizophrenia: management, antipsychotics and adverse effects notes; the bipolar-II side of the differential in the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes; the forensic frame of antisocial personality disorder in the Mental health legislation and forensic psychiatry notes; the complex-PTSD boundary in the Anxiety, OCD and trauma-related disorders notes; the schizotypal-versus-schizophrenia boundary in the Schizophrenia: aetiology, phenomenology, classification and course notes; and the wider psychotherapy models in the Psychotherapies, clinical assessment, MSE and nosology notes. This is doctor-facing revision, so the full technical vocabulary is used, brand names lead with the Indian brand, and every criterion, trait domain, cluster mapping, severity anchor, dose and guideline rule is verified against a source or omitted and flagged.

Framework and classification

1 . Definition, concept and general diagnostic criteria

Why this leads the chapter. Every personality-disorder question turns on one gate that most candidates skip. Before any of the ten named types can be diagnosed, a set of **general diagnostic criteria** must ALL hold: an **enduring pattern** of inner experience and behaviour that is **pervasive and inflexible**, deviates markedly from the person's culture, begins by adolescence, is stable over time, is not better explained by another disorder or a substance, and causes clinically significant impairment or distress. A personality disorder is not diagnosed by pattern-matching a vignette to a type; it is diagnosed by first clearing this general threshold and only then asking

which type fits. Get the gate right and the whole family, however you later classify it, becomes a disciplined exercise; get it wrong and you will pin a lifelong label on a situational mood state.

This fragment sets the **definition of personality disorder**, walks the six-part general threshold criterion by criterion, and isolates the four features an examiner rewards: the **ego-syntonic** quality, the trait-versus-state caution, the cultural reading, and the rule that no type is named until the general criteria are met. The accompanying figure lays the same content out as a general-criteria decision map to be carried as one image. The forward split into the ICD-11 dimensional model and the DSM-5-TR categorical clusters is developed in the topics that follow; the borderline-versus-bipolar-II boundary is cross-referenced to the Mood disorders: classification, depressive & bipolar syndromes, neurobiology notes, the forensic weight of antisocial personality disorder to the Mental health legislation & forensic psychiatry notes, the complex-PTSD boundary to the Anxiety, OCD & trauma-related disorders notes, and the wider technique of the psychological therapies to the Psychotherapies, clinical assessment, MSE & nosology notes.

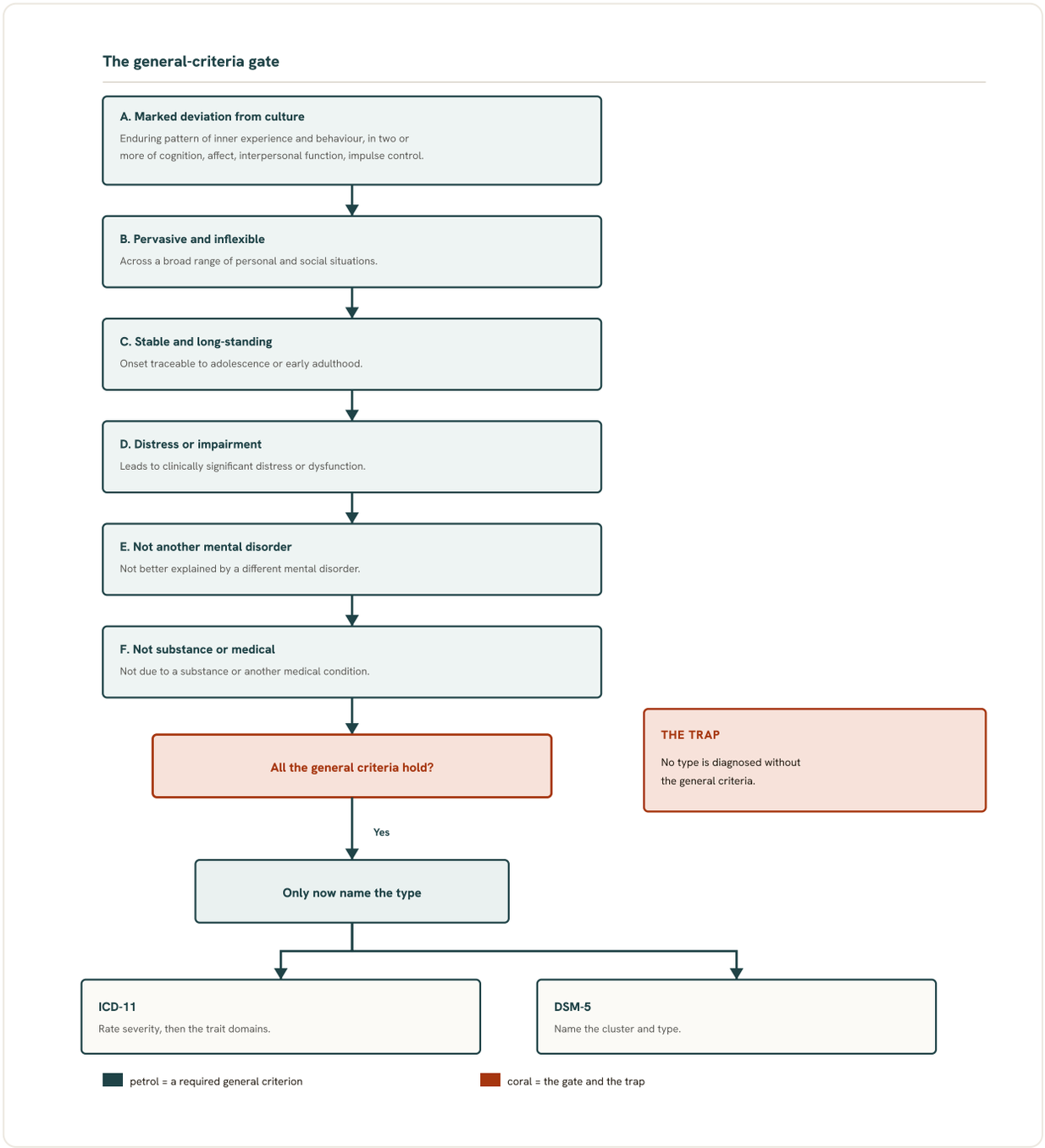


Fig 16.1 The general criteria for a personality disorder are a gate: the pattern must be enduring, pervasive and inflexible, deviate from cultural expectation, begin by adolescence or early adulthood, cause distress or impairment, and not be better explained by another disorder or a substance before any specific type is named.

1.1 Personality, trait, and what makes a disorder

A disorder is the maladaptive extreme of a normal continuum. Personality is the characteristic, deeply ingrained pattern of perceiving, relating to and thinking about oneself and the environment (DSM-5-TR). *Personality traits* are the normal, prominent aspects of that pattern; they become a disorder only when they are inflexible and maladaptive and cause significant functional impairment or subjective distress (DSM-5-TR). That single hinge, from normal-but-prominent to inflexible-and-impairing, is the working **definition of personality disorder**. The concept has a long descriptive spine: Kurt Schneider defined the abnormal (psychopathic) personalities as those who suffer from their abnormality or make society suffer, dissolving the older moral reading into a clinical one. ICD-11 phrases the modern definition as a

marked disturbance in personality functioning, with the central manifestations being impairments in aspects of the *self* (identity, self-worth, capacity for self-direction) and in *interpersonal* functioning (forming and keeping close relationships, understanding others, managing conflict). Hold both halves, self and other, because the dimensional model that follows is built on exactly this pair.

PEARL

The two manuals fight over how to sub-type personality pathology but barely differ on what it *is*: DSM-5-TR and the ICD systems give very similar GENERAL definitions of personality disorder, each locating the core pathology in cognition about self and others, emotional experience and regulation, interpersonal relating and behavioural control (Companion). Learn the general definition once; it survives every classification change.

1.2 The general diagnostic criteria: the six-part gate

All six must hold before any type. The DSM-5-TR general personality disorder criteria are the cleanest checklist to memorise, and the ICD equivalents map onto them almost one to one. They run: an enduring pattern deviating markedly from cultural expectation, shown in at least two areas of functioning (Criterion A); the pattern is inflexible and pervasive across situations (B); it causes clinically significant distress or impairment (C); it is stable and long-standing with onset traceable to adolescence or early adulthood (D); it is not better explained by another mental disorder (E); and it is not due to a substance or another medical condition (F). Reproduce the block as one table; this is the highest-yield single object in the chapter.

CRITERION	WHAT IT REQUIRES
A - Pattern and areas	**An enduring pattern of inner experience and behaviour deviating markedly from the person's culture, shown in two or more of four areas: cognition, affectivity, interpersonal functioning, impulse control**
B - Pervasiveness	The pattern is inflexible and pervasive across a broad range of personal and social situations
C - Impairment	It leads to clinically significant distress or impairment in social, occupational or other important functioning
D - Onset and stability	The pattern is stable and of long duration, with onset traceable back at least to adolescence or early adulthood
E - Not another disorder	Not better explained as a manifestation or consequence of another mental disorder
F - Not organic or substance	Not attributable to the physiological effects of a substance or another medical condition (for example head trauma)

The DSM-5-TR general criteria for a personality disorder (A to F); the ICD-11 essential features map closely onto the same requirements.

RULE **Clear the gate, then name the type.** No specific personality disorder is diagnosed unless every general criterion (enduring, pervasive and inflexible, culturally deviant, onset by adolescence, impairing, and not better explained) is first satisfied. The type is a sub-question that only opens once the gate is passed.

1.3 The four channels of deviation (Criterion A)

Two or more of four must be involved. Criterion A is not satisfied by trouble in one domain of life; the enduring pattern must show up in **two or more of the four** channels below. Learning these four is what lets you defend a diagnosis, because they force you to demonstrate that the disturbance is broad rather than a single symptom.

Cognition

Ways of perceiving and interpreting self, other people and events (for example pervasive suspiciousness, or a chronically fragile self-image).

Affectivity

The range, intensity, lability and appropriateness of emotional response (for example constricted flat affect, or rapidly reactive mood).

Interpersonal functioning

The characteristic pattern of relating (for example detachment and avoidance, or intense and unstable relationships).

Impulse control

The regulation of urges and behaviour (for example recurrent recklessness, aggression or self-damaging impulsivity).

1.4 Enduring, pervasive and inflexible, with onset by adolescence (Criteria B and D)

Trait-like breadth and lifelong reach are what separate a disorder from an episode. The words are load-bearing. *Enduring* and *stable* mean the pattern has run for years, not weeks; ICD-11 asks for a disturbance persisting over an extended period, typically two years or more. *Pervasive and inflexible* mean it holds across a broad range of personal and social situations rather than being triggered by one relationship or setting, and that the person cannot flex out of it when circumstances change. *Onset by adolescence* or early adulthood means the roots are developmental: an **enduring pattern** that first appears in mid or late adult life is a red flag for a personality change due to another medical condition or an unrecognised substance use disorder, not a primary personality disorder (DSM-5-TR). A pattern that is genuinely pervasive and inflexible with onset by adolescence is the developmental signature you are hunting for.

1.5 Deviation from cultural expectation (the cultural criterion)

The yardstick is the person's own culture, not the clinician's. Criterion A demands **deviation from cultural expectation**: the pattern must depart markedly from what the individual's *own* culture expects in cognition, affect, relating and impulse control. DSM-5-TR is explicit that personality disorder must not be confused with problems of acculturation after migration, or with habits, customs, and religious or political values rooted in the person's background; behaviour that looks rigid or dysfunctional may in fact be an adaptive response to real cultural

constraints. This is not a soft caveat but part of the criterion itself, and it is where cross-cultural over-diagnosis happens.

TRAP **Reading personality through one culture's norms.** Scoring deference, family-embeddedness, religiously framed experience or protest that risks the person's own safety as pathological dependence, oddness or callousness, when it is normative in their community, converts a cultural difference into a false diagnosis. Judge deviation against the patient's culture, with an informant if needed.

1.6 Impairment or distress, and the two exclusions (Criteria C, E and F)

Deviance alone is not a disorder. A culturally deviant, enduring, pervasive pattern still is not a personality disorder unless Criterion C is met: it must cause clinically significant **impairment or distress** in social, occupational or other important functioning. Many people have marked, inflexible traits and function well; without the impairment or distress they carry a personality *style*, and ICD-11 formalises this as personality difficulty (QE50.7), a factor influencing health status rather than a mental disorder. The final two criteria are exclusions that protect against mislabelling: the pattern must not be better explained by another mental disorder (E), and must not be attributable to a substance or medical condition such as head trauma (F).

COMMON TRAP

The mirror error to over-diagnosis: reading the enduring interpersonal and cognitive changes of a chronic Axis I illness (long-standing depression, an anxiety disorder, early psychosis, a frontal lesion or ongoing substance use) as a personality disorder. Criteria E and F exist precisely to catch this. If a supposedly lifelong pattern in fact tracks the course of another disorder or a drug, the personality label is wrong.

1.7 The ego-syntonic quality, and trait versus state

The patient rarely complains of the very thing you are diagnosing. Classically the abnormal personality traits are **ego-syntonic**: they are experienced as part of the self and are not felt as problematic, in sharp contrast to the ego-dystonic symptoms of the neurotic disorders, which distress the patient and drive help-seeking. Two consequences follow. First, people with a personality disorder often do not present for the personality disorder itself, but for a comorbid depression, anxiety or crisis, so the diagnosis is easy to miss and easy to make only from collateral history. Second, and the single commonest error, the traits must be distinguished from *states*: features that emerge in response to a specific situational stressor or a transient mental state (a mood episode, an anxiety disorder, or substance intoxication) are not personality (DSM-5-TR). Because the pattern is developmental, in a person under eighteen the features must have been present for at least one year before a personality disorder is diagnosed, and antisocial personality disorder is never diagnosed before eighteen (DSM-5-TR), a rule with direct medico-legal force.

TRAP **Calling a state a trait.** The affective lability, impulsivity and stormy relationships of an acute mood episode, an intoxication or a fresh crisis are not a personality disorder unless the same pattern is enduring, pervasive and predates the current state. Diagnose personality when the patient is at baseline, not mid-storm.

INDIA

Personality disorders are substantially under-recognised in Indian practice, and the ego-syntonic quality is a large part of why: patients present for the comorbid depression, self-harm or family conflict, not for the personality pattern, so it is recorded only when it is actively looked for. Two further Indian angles are genuine. The cultural criterion bites hardest here: deference, joint-family interdependence and religiously framed experience are normative and must not be read as dependent, avoidant or odd pathology. And the disorder-specific psychotherapies are arriving but unevenly available: dialectical behaviour therapy and schema therapy are being delivered and culturally adapted at Indian tertiary and private centres rather than being widely accessible, which shapes what a realistic management plan can offer. The forensic reading of antisocial personality disorder in the Indian court context is carried in the Mental health legislation & forensic psychiatry notes.

1.8 The rule that governs the whole chapter, and the forward map

General criteria first; classification second. With the gate cleared, the chapter splits into two ways of describing what lies beyond it, both cross-mapped in the topics that follow. DSM-5-TR keeps the familiar *categorical* system: the ten types grouped into three clusters (A odd or eccentric, B dramatic or erratic, C anxious or fearful), plus a Section III alternative hybrid model. ICD-11 takes the opposite route, abolishing the categorical types for a *dimensional* model that rates a severity level first and then adds trait-domain qualifiers and an optional borderline pattern. Whichever framework an examiner uses, it is downstream of the same general criteria you have just cleared. The ten categorical types are worth carrying as a single skeleton from the outset.

CLUSTER	TYPE	DESCRIPTOR
A	Paranoid	Odd or eccentric
A	Schizoid	Odd or eccentric
A	**Schizotypal**	Odd or eccentric (schizophrenia-spectrum edge)
B	Antisocial	Dramatic, emotional or erratic
B	**Borderline**	Dramatic, emotional or erratic
B	Histrionic	Dramatic, emotional or erratic
B	Narcissistic	Dramatic, emotional or erratic
C	Avoidant	Anxious or fearful
C	Dependent	Anxious or fearful
C	Anankastic (obsessive-compulsive)	Anxious or fearful

The three DSM-5-TR clusters and ten categorical types; ICD-11 abolishes these types for a dimensional model, developed in the topics that follow.

WORKED EXAMPLE

A woman in her twenties is referred after an overdose following a break-up, described as having "borderline personality disorder". You do not accept the label yet; you run the gate. Is the pattern of stormy relationships, identity instability and impulsivity *enduring* and traceable to adolescence, or does it date from a recent depressive episode (Criterion D, and E)? Is it *pervasive* across settings, or confined to one relationship (B)? Does it cause impairment at baseline, not only in crisis (C)? Only when every general criterion holds do you move to the type-specific criteria. If the pattern proves to be a first depressive episode with situational distress, you have prevented a lifelong misdiagnosis.

GOOD TO REMEMBER

- **Personality disorder (general):** defining requirement is an enduring pattern of inner experience and behaviour, pervasive and inflexible, deviating markedly from cultural expectation in two or more of cognition, affectivity, interpersonal functioning and impulse control, with onset by adolescence, causing impairment or distress, and not better explained by another disorder, a substance or a medical condition. Discriminator from a personality *style*: the impairment or distress (Criterion C); ICD-11 codes the style short of this as personality difficulty. First diagnostic step: clear all the general criteria before naming any type.

1.9 The master hook for the whole chapter

One scaffold to reassemble the day. The chapter has four countable skeletons: the clusters and types, the ICD-11 trait domains, the borderline criterion count, and the core skills therapy. Carry them as one mnemonic now and every later topic hangs off it; the pieces are paid off in full at consolidation.

MNEMONIC

MAD BAD SAD, FIVE DOMAINS, NINE-FOR-FIVE, FOUR TO COPE

MAD BAD SAD are the three clusters holding the ten types: *mad* = Cluster A odd or eccentric (Paranoid, Schizoid, Schizotypal); *bad* = Cluster B dramatic or erratic (Antisocial, Borderline, Histrionic, Narcissistic); *sad* = Cluster C anxious or fearful (Avoidant, Dependent, Anankastic). **FIVE DOMAINS** are the ICD-11 trait qualifiers, hooked as *New Dogs Do Dig Antiques*: Negative affectivity, Detachment, Dissociality, Disinhibition, Anankastia (plus the optional borderline pattern). **NINE-FOR-FIVE** is borderline personality disorder, defined by **five or more of the nine** criteria. **FOUR TO COPE** is dialectical behaviour therapy and its four skills modules (mindfulness, interpersonal effectiveness, emotion regulation, distress tolerance). Four numbers, and the chapter rebuilds from one line.

3	10	5	4
DSM-5-TR CLUSTERS	CATEGORICAL PERSONALITY TYPES	ICD-11 TRAIT DOMAINS	DBT SKILLS MODULES

HOLD THIS

The general diagnostic criteria are a gate, not a description: an enduring, pervasive and inflexible pattern, deviating from cultural expectation, with onset by adolescence, causing impairment or distress, and not better explained otherwise, must all hold before any specific personality-disorder type is diagnosed.

2 · ICD-11 dimensional classification: severity and trait domains

Why this matters. This is the single highest-yield nosology point of the personality-disorders chapter, and the examiner knows it. The **ICD-11 dimensional** system did something no previous classification dared: it **abolished the categorical personality-disorder types altogether**. There is no ICD-11 code for paranoid, schizoid, dissocial, histrionic, anankastic, avoidant or dependent personality disorder. In their place stands a model that first rates **severity** and then, optionally, describes style. Get the architecture of that model straight, the severity gradient that leads and the five trait domain qualifiers that follow, and you can answer almost any framework stem the paper sets.

The governing idea is a reversal of habit. The old question was "which type is this?"; the ICD-11 question is "how severe is the personality dysfunction, and what is its flavour?". Severity is rated **first and leads** because the degree of disturbance in self and interpersonal functioning predicts outcome, service need and risk far better than any type label ever did (ICD-11 CDDR; New Oxford Textbook of Psychiatry). The alternative model of personality disorders in DSM-5-TR Section III (the hybrid AMPD) and the retained DSM-5-TR categorical clusters are cross-mapped here so the two systems can be held side by side, and a legacy ICD-10 box records exactly what was removed and why the categorical names still get tested. The accompanying figure lays the whole model out as one object: the severity gradient down one axis and the trait-domain qualifiers across the other, with the borderline pattern qualifier hung off to the side.

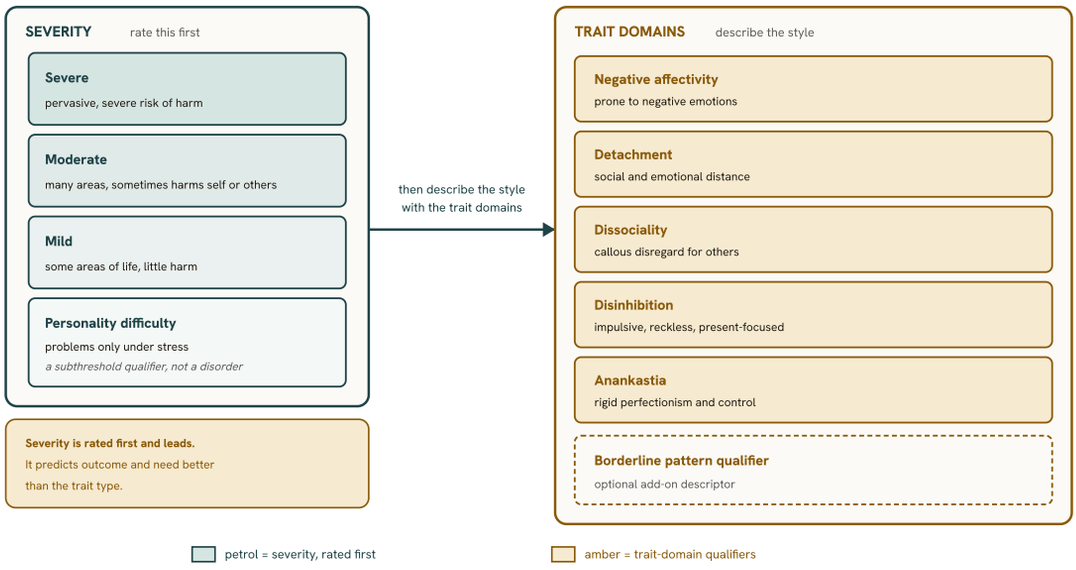


Fig 16.2 The ICD-11 model abolished the categorical types: rate severity first (personality difficulty, then mild, moderate or severe on a harm gradient), then describe the style with the five trait-domain qualifiers, negative affectivity, detachment, dissociality, disinhibition and anankastia, plus the optional borderline pattern qualifier.

2.1 The headline shift: types out, a dimensional model in

The ICD-11 dimensional model replaces the entire list of named types with a single diagnostic pathway (ICD-11 CDDR, grouping 6D10 to 6D11). You confirm the general criteria of a personality disorder (an enduring, pervasive, inflexible pattern of the self and of relationships, deviating from cultural expectation, with onset by adolescence or early adulthood and causing distress or impairment, all taught in the general-framework topic alongside), and then you make three moves in order. **First, rate severity** on a graded scale. **Second, add any of the five trait-domain qualifiers** that best describe the style of the disturbance. **Third, add the borderline pattern qualifier** if that specific picture is present. The type has vanished; the diagnosis is now a severity level plus a set of descriptive qualifiers. This is why the model is called dimensional rather than categorical: personality pathology is placed on continua of severity and trait, not sorted into boxes.

RULE **Severity is rated first and leads.** In the ICD-11 personality classification the degree of self-and-interpersonal dysfunction, not the type, is the primary axis, because severity predicts outcome and need better than any category.

2.2 How severity is defined, and why it carries the weight

Severity is judged on two linked capacities and one consequence. The capacities are **self functioning** (the stability and accuracy of identity, self-worth, self-direction and self-view) and **interpersonal functioning** (the desire and ability to form and sustain relationships, to grasp others' perspectives, and to manage conflict). The consequence is the associated **risk of harm** to self or others together with the breadth of functional impairment (ICD-11 CDDR). The rater asks

how pervasively these are disturbed, across how many domains of life, and with what danger attached. That single graded judgement then does the work the ten separate type-diagnoses used to do. The empirical case, built by Tyrer and the ICD-11 working group, is that the old categories overlapped so heavily that "type" was unreliable, whereas a simple severity rating tracks concurrent and prospective impairment robustly (New Oxford Textbook of Psychiatry).

PEARL

The reason severity leads is not administrative tidiness but prediction. Across longitudinal data the *severity* of personality dysfunction, more than its stylistic type, forecasts self-harm, suicide risk, service use, relationship breakdown and occupational failure. Rate severity well and you have already said the most prognostically useful thing about the patient.

2.3 The severity gradient: personality difficulty, then mild, moderate and severe

The gradient has a subthreshold rung and three disorder rungs. Personality difficulty (coded QE50.7, and deliberately placed not among the mental disorders but in the chapter on factors influencing health status) is **not a diagnosis of disorder**: it flags pronounced personality features that colour presentation or treatment without reaching the threshold. Above it sit mild personality disorder (6D10.0), moderate personality disorder (6D10.1) and severe personality disorder (6D10.2), separated by how many areas of functioning are affected and how much harm attaches. The table is the object to memorise; the highlighted cell in each row is the feature that fixes that level against its neighbours.

LEVEL (ICD-11 CODE)	SELF AND INTERPERSONAL FUNCTIONING	HARM AND IMPAIRMENT
Personality difficulty (QE50.7)	Pronounced features below the disorder threshold; some relevance to health or treatment	**Not a mental disorder; a health-status qualifier, not a diagnosis**
Mild personality disorder (6D10.0)	**Some areas of functioning affected, others spared; many relationships and expected roles still maintained**	Usually not associated with substantial harm to self or others
Moderate personality disorder (6D10.1)	**Marked problems in most relationships and most expected role functions; most areas of functioning affected**	May be associated with harm to self or others
Severe personality disorder (6D10.2)	**Severe disturbance of the sense of self (identity unstable or incoherent) and of nearly all relationships**	High risk of harm to self or others; severe functional impairment

The ICD-11 severity gradient: the subthreshold rung plus three disorder levels, read by degree of self-and-interpersonal dysfunction and attached risk of harm.

2.4 The five trait-domain qualifiers: describing the style once severity is set

Once severity is rated, one or more **trait-domain qualifiers** (postcoordinated codes 6D11.0 to 6D11.4) describe the prominent style of the disturbance. Each trait domain is a broad dimension,

not a type; a patient may carry several. The five, verbatim in construct, are set out below. Note the mnemonic order and hold them as a closed set: a sixth invented domain, or a missing one, is the classic slip.

Negative affectivity (6D11.0)
A tendency to experience a broad range of negative emotions (anxiety, anger, self-loathing, emotional lability, mistrust).

Detachment (6D11.1)
A tendency to maintain interpersonal and emotional distance: social withdrawal, aloofness, limited emotional expression and experience of pleasure.

Dissociality (6D11.2)
Disregard for the rights and feelings of others; self-centredness and lack of empathy, with grandiosity or callousness.

Disinhibition (6D11.3)
Acting rashly and impulsively on immediate stimuli, without consideration of consequences; recklessness and poor planning.

Anankastia (6D11.4)
A rigid focus on one's own and others' standards, commonly shown as perfectionism and emotional and behavioural constraint.

MNEMONIC

"N, then D-D-D, then A"

The five ICD-11 trait domains in order: **N**egative affectivity, then the three D's, **D**etachment, **D**issociality, **D**isinhibition, and finally **A**nankastia. The three D's are easy to muddle, so anchor them: detachment is *distance*, dissociality is *disregard for others*, disinhibition is *daring impulsivity*. The borderline pattern is a sixth token that sits apart, an optional add-on qualifier, not one of the five domains.

2.5 The borderline pattern qualifier: the one pattern that survived

The single named picture the ICD-11 kept is the borderline pattern qualifier (6D11.5), added for clinical utility and continuity with a large treatment literature (ICD-11 CDDR). It is an **additional qualifier**, not a type and not a trait domain, and it typically overlaps the domains (especially negative affectivity and disinhibition). It marks a pervasive pattern of instability of interpersonal relationships, self-image and affect together with marked impulsivity, defined by **five or more of the nine** features: frantic efforts to avoid abandonment; unstable, intense relationships that swing between idealisation and devaluation; identity disturbance; impulsivity that is potentially self-damaging; recurrent self-harm or suicidal behaviour; affective instability; chronic emptiness; inappropriate intense anger; and transient stress-related paranoid ideation or dissociation (Kaplan & Sadock CTP 2024). The threshold and the nine features are the same set the DSM-5-TR uses for borderline personality disorder, which is why the borderline construct crosses cleanly between the systems while the other types do not. The boundary with complex post-traumatic

stress disorder, a distinction ICD-11 sharpened by introducing both, is drawn in the Anxiety, OCD and trauma-related disorders notes.

GOOD TO REMEMBER

- **ICD-11 personality classification:** no types; rate severity first (personality difficulty, then mild, moderate, severe by self-and-interpersonal dysfunction and risk of harm), then add trait qualifiers.
- **The five trait domains:** negative affectivity, detachment, dissociality, disinhibition, anankastia; they describe style, not severity, and several may co-occur.
- **Borderline pattern qualifier (6D11.5):** the one retained pattern; an add-on qualifier defined by five or more of the nine borderline features; overlaps the domains, not a type.

2.6 Assembling a diagnosis: how the pieces combine in practice

A finished ICD-11 personality diagnosis is a severity level with any qualifiers appended, coded by postcoordination. You never stop at a trait: a qualifier without a rated severity is an incomplete diagnosis. The clinical value is that the same language scales from a screening judgement to a detailed formulation, and that severity, the part that drives the management plan and the risk assessment, is stated up front rather than buried inside a type label. The bipolar-II differential that so often shadows an affectively unstable presentation is worked in the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes; the forensic reading of a prominent dissociality trait belongs to the Mental health legislation and forensic psychiatry notes.

WORKED EXAMPLE

A woman in her twenties has an incoherent sense of who she is, stormy relationships that collapse repeatedly, recurrent self-cutting after perceived abandonment, chronic emptiness and stress-related dissociation. Work the model in order. General criteria met. Severity: the self is severely disturbed and almost all relationships are affected with a high risk of self-harm, so this is severe personality disorder (6D10.2). Trait qualifiers: prominent negative affectivity and disinhibition. Pattern: the picture meets five or more of the nine borderline features, so add the borderline pattern qualifier (6D11.5). The diagnosis is "severe personality disorder with negative affectivity, disinhibition and a borderline pattern", not "borderline personality disorder" as a type.

2.7 Cross-map to DSM-5-TR: the categorical clusters and the AMPD hybrid

DSM-5-TR did not follow the ICD-11 all the way. Its main text **retains the categorical system**: three clusters (A the odd or eccentric, B the dramatic or erratic, C the anxious or fearful) holding ten types. In parallel, Section III offers the **alternative model of personality disorders**, a hybrid that mirrors the ICD-11 logic: Criterion A rates self and interpersonal functioning on the Level of Personality Functioning Scale (self as identity and self-direction; interpersonal as empathy and intimacy), and Criterion B specifies five pathological trait domains with 25 facets (Kaplan & Sadock CTP 2024). The two dimensional systems are close cousins but their trait sets are not identical, and the mismatch is examinable. The grid below is the discriminator to hold.

ICD-11 TRAIT DOMAIN	DSM-5-TR AMPD COUNTERPART	NOTE
Negative affectivity	Negative affectivity	Shared construct
Detachment	Detachment	Shared construct
Dissociality	Antagonism	**Same construct, different name**
Disinhibition	Disinhibition	Shared construct
Anankastia	(no AMPD domain)	**ICD-11 only; a constraint pole absent from the AMPD**
(no ICD-11 domain)	Psychoticism	**DSM only; ICD-11 places schizotypal with the psychoses, so it has no psychoticism domain**

The two dimensional trait sets: four align, but ICD-11 carries anankastia and the AMPD carries psychoticism; the highlighted rows are where they part.

TRAP **Do not equate the ICD-11 five with the DSM-5 AMPD five.** Anankastia is ICD-11-only and psychoticism is DSM-only; the schizotypal-versus-schizophrenia placement is set out in the Schizophrenia: aetiology, phenomenology, classification and course notes.

2.8 The legacy ICD-10 box: what changed and why the old names still get tested

The break from ICD-10 is total, which is exactly why the categorical vocabulary refuses to die. Knowing what was removed is itself the exam point.

ICD-10 TO ICD-11: WHAT CHANGED

ICD-10 (F60) **kept the categorical specific types**: paranoid, schizoid, dissocial, emotionally unstable (impulsive and borderline subtypes), histrionic, anankastic, anxious or avoidant, and dependent personality disorder, with schizotypal sitting separately under the schizophrenia grouping. ICD-11 **removed all of these named types** and rebuilt the diagnosis as a severity level plus trait-domain qualifiers, keeping only the borderline pattern as an optional qualifier. Exams still test the categorical names because DSM-5-TR retains them in its main text and clinicians still speak in them, so you must read fluently in both the dimensional and the categorical languages.

COMMON TRAP

Rating a trait qualifier while skipping severity, or naming "borderline personality disorder" as an ICD-11 type. In ICD-11 borderline is a *pattern qualifier* appended to a rated severity, never a standalone type; and no trait domain is a diagnosis on its own.

2.9 India and the cultural frame

Two India-specific cautions attach to this model. The first is **under-recognition**: personality disorders are diagnosed far less often in Indian clinical practice than Western prevalence would predict, an under-detection documented in Indian samples (Gupta & Mattoo, on personality-disorder prevalence and demography). A severity-led system that begins with a graded judgement of self-and-interpersonal dysfunction, rather than demanding a full type match, is well suited to

catching the milder end that Indian services routinely miss. The second is the **cultural expression of pathology**: the general criteria require deviation from what the person's own culture expects, so a self that is deeply embedded in family and community, or a deference and interdependence that would look like dependence through a Western lens, must not be read as disorder. The medico-legal weight of a prominent dissociality trait, which surfaces most sharply in offender assessment, is handled in the Mental health legislation and forensic psychiatry notes.

INDIA

Access to the structured psychotherapies that this severity framing points toward is uneven. Dialectical behaviour therapy and schema therapy are available and being culturally adapted in Indian metros and academic centres but remain scarce elsewhere, so a severe rating that mandates intensive structured therapy can outrun what a given service can deliver. Rate honestly, then match the plan to what is actually accessible, and never read collectivist family-embeddedness as a personality disorder.

5	3	5+	3 / 10
ICD-11 TRAIT DOMAINS	SEVERITY LEVELS (MILD, MODERATE, SEVERE)	OF NINE BORDERLINE-PATTERN FEATURES	DSM-5-TR CLUSTERS / TYPES (RETAINED)

HOLD THIS

ICD-11 has no personality-disorder types: rate severity first (personality difficulty, then mild, moderate, severe by self-and-interpersonal dysfunction and risk of harm), then add any of the five trait domains, negative affectivity, detachment, dissociality, disinhibition, anankastia, and the borderline pattern qualifier.

3 · DSM-5 categorical clusters and the alternative model

Two classifications of personality live side by side inside one manual. **dsm-5-tr** keeps, in its official Section II, the familiar **categorical clusters** and the **ten types** inherited almost unchanged from DSM-IV; and it offers, in its research Section III, an **alternative model**, a dimensional-categorical **hybrid model** (the **ampd**) that scores how badly the personality functions and profiles the traits that colour it. Knowing both, and being able to cross-map them onto the ICD-11 dimensional system, is the single highest-yield task on the personality-disorders paper.

Why it matters, and what this note owns. This note owns the DSM-5-TR side of the nosology: the three **categorical clusters** and their ten types with each type under its correct cluster, and the Section III **alternative model** in full, its Criterion A **level of personality functioning** scale and its Criterion B **pathological trait domains**. The ICD-11 dimensional model (the severity gradient and the five trait qualifiers) is taught forward in its own sibling note and only cross-mapped here; the deeper per-disorder phenomenology of each type lives in the cluster notes, and the general diagnostic criteria that gate any personality-disorder diagnosis are stated here only as the framing rule. The accompanying figure maps the ten types under the three clusters; a second figure alongside it lays the DSM-5 alternative model against the ICD-11 domains.

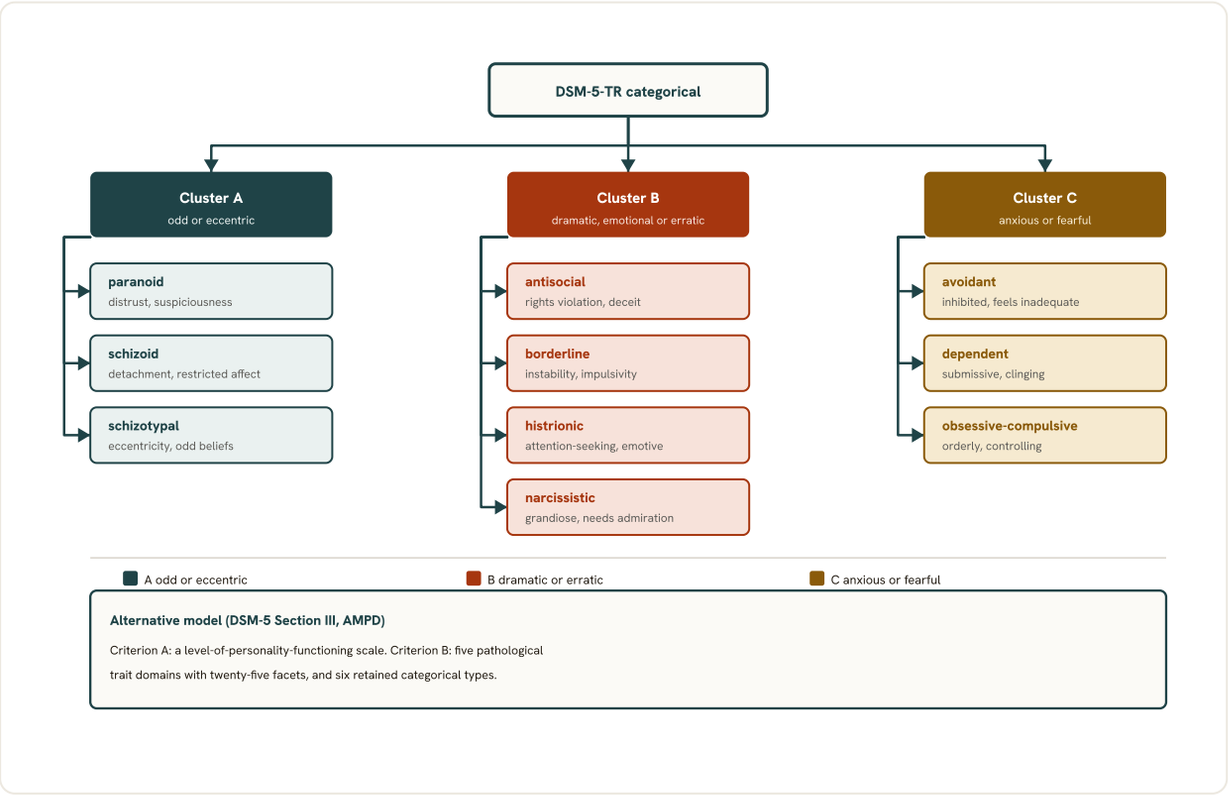


Fig 16.3 The DSM-5-TR keeps the ten categorical types in three clusters, A odd or eccentric, B dramatic or erratic, C anxious or fearful, with the Section III alternative model (a functioning scale plus pathological trait domains) alongside; ICD-10 kept these categories, which is why exams still test them.

3.1 Two systems in one manual: Section II categorical, Section III alternative

The structural fact to state first. dsm-5-tr carries *both* a categorical and a dimensional account of personality disorder. Section II, the official classification for routine clinical use, retains the ten categorical types grouped into three **categorical clusters**, virtually unchanged from DSM-IV-TR (DSM-5-TR 2022). Section III, "Emerging Measures and Models", houses the alternative model (also called the **hybrid model** or **ampd**, the Alternative **dsm-5** Model for Personality Disorders), placed there for further study rather than official use. The APA Board of Trustees kept both deliberately: the categorical set to preserve continuity with clinical practice, and the hybrid model to answer the categorical system's well-known failings (excessive comorbidity between types, and "other specified" being the commonest and least informative diagnosis).

Why the split is examinable. The examiner wants you to be fluent in the official categorical vocabulary (paranoid, borderline, and the rest) *and* able to explain the model that is expected to replace it. That is the through-line of the whole chapter: teach the dimensional direction of travel (ICD-11 has already abolished the categorical types entirely), but master the categorical names because Section II, everyday communication and most examinations still run on them.

GOOD TO REMEMBER

- **DSM-5-TR dual structure:** Section II retains the official categorical system, three clusters and ten types unchanged from DSM-IV-TR; Section III offers the alternative (hybrid AMPD) model for further study; both are printed in the same manual by deliberate APA decision, the categorical for continuity, the dimensional to fix comorbidity and the uninformative "unspecified" default.

3.2 The gate before any label: the general criteria for a personality disorder

The pattern precedes the type. No cluster and no type may be assigned until the general criteria for a personality disorder are met: an enduring, pervasive, inflexible pattern of inner experience and behaviour that *deviates markedly from the expectations of the individual's culture*, is stable over time, traceable to **adolescence or early adulthood**, and leads to distress or impairment, and is not better explained by another mental disorder, a substance, or a medical condition (DSM-5-TR 2022). The same seven general criteria (A to G) sit at the head of the Section III model, where Criterion A is reframed as moderate or greater impairment in personality functioning and Criterion B as one or more pathological traits, but the enduring, pervasive, culturally-deviant, early-onset spine is identical.

Deviation is judged against the person's own culture. The cultural clause is not decorative; the pattern must deviate from the norms of the individual's *own* cultural, ethnic and social reference group, and manifest across cognition, affectivity, interpersonal functioning and impulse control. Reading a migrant's habits, a subculture's mores, or a life-stage's turbulence as personality pathology is the classic misdiagnosis this criterion exists to block.

RULE **The enduring pattern before the label.** Establish the general personality-disorder criteria first, an inflexible, pervasive, culturally-deviant pattern stable since adolescence or early adulthood with distress or impairment, and only then place the cluster and the type; the cluster or the AMPD trait profile is meaningless without that gate.

TRAP **State mistaken for trait.** Do not label situational or episodic features a personality disorder: calling a passing affective lability "borderline" without the enduring identity disturbance and abandonment features, or reading a cultural or developmental norm as pathology, breaks the general criteria and is the commonest error on this topic.

3.3 Cluster A, the odd or eccentric cluster

The cluster and its three types. **cluster a** groups the personality disorders whose sufferers "often appear odd or eccentric" (DSM-5-TR 2022): paranoid personality disorder (a pervasive distrust and suspiciousness, reading others' motives as malevolent), schizoid personality disorder (detachment from social relationships with a restricted, flattened emotional range, the person is a genuine loner who does not want closeness), and schizotypal personality disorder (acute social discomfort with cognitive and perceptual distortions and eccentricities, ideas of reference, odd beliefs and magical thinking, unusual perceptions). The cluster's phenomenology shades toward the schizophrenia spectrum.

The boundary that is tested. Schizotypal is the one to place carefully: its cognitive-perceptual eccentricities are attenuated, not frank psychosis, so it stays a personality disorder in DSM-5-TR (though it is cross-listed with the schizophrenia spectrum), and mislabelling it as schizophrenia is a standard error, developed in the Schizophrenia: aetiology, phenomenology, classification and course notes.

GOOD TO REMEMBER

- **Cluster A (odd or eccentric):** paranoid (pervasive distrust and suspiciousness), schizoid (detachment with restricted emotion, a contented loner), schizotypal (social discomfort plus cognitive-perceptual distortions, ideas of reference, magical thinking); the discriminator from the schizophrenia spectrum is that the distortions are attenuated, not frankly psychotic.

3.4 Cluster B, the dramatic, emotional or erratic cluster

The cluster and its four types. **cluster b** groups those who "often appear dramatic, emotional, or erratic" (DSM-5-TR 2022): antisocial personality disorder (a pervasive disregard for and violation of the rights of others, deceit, impulsivity, aggression and lack of remorse, requiring evidence of conduct disorder before the age of fifteen years), borderline personality disorder (instability of relationships, self-image and affect with marked impulsivity), histrionic personality disorder (pervasive excessive emotionality and attention seeking), and narcissistic personality disorder (grandiosity, need for admiration and lack of empathy). This is the cluster that dominates the clinic and the examination, borderline above all.

Two exam anchors. Antisocial personality disorder is the one type DSM-5-TR will not diagnose before the age of eighteen years and only with a documented childhood conduct disorder, and it carries the forensic weight of the chapter (offender assessment, court reports), developed in the Mental health legislation and forensic psychiatry notes. Borderline is the flagship carried in detail below and across the day.

GOOD TO REMEMBER

- **Cluster B (dramatic, emotional or erratic):** antisocial (disregard for and violation of others' rights; needs conduct disorder before age fifteen and diagnosis only from age eighteen), borderline (instability of relationships, self-image and affect with impulsivity), histrionic (excessive emotionality and attention seeking), narcissistic (grandiosity, need for admiration, lack of empathy); it is the highest-yield cluster.

3.5 Cluster C, the anxious or fearful cluster, and the full ten-type map

The cluster and its three types. **cluster c** groups those who "often appear anxious or fearful" (DSM-5-TR 2022): avoidant personality disorder (social inhibition, feelings of inadequacy and hypersensitivity to negative evaluation, the person wants closeness but is too afraid of rejection to risk it), dependent personality disorder (a pervasive, excessive need to be taken care of, submissive and clinging behaviour, fear of separation), and obsessive-compulsive personality disorder (a preoccupation with orderliness, perfectionism and control at the expense of flexibility and efficiency). Avoidant is separated from social anxiety disorder by its pervasive, trait-level character, and obsessive-compulsive personality disorder is separated from OCD by the absence of true obsessions and compulsions, the confusion of the anankastic character with the anxiety disorder being a stock trap.

The ten under the three. The mapping to reproduce cold, three **categorical clusters** holding **ten types**, is set out in the table and the accompanying figure. DSM-5-TR itself warns that the clustering, though useful for teaching and research, "has serious limitations and has not been

consistently validated": types from different clusters co-occur freely, which is exactly the comorbidity problem the alternative model was built to solve.

CLUSTER	CHARACTER	TYPE	ONE-LINE SIGNATURE
Cluster A	**Odd or eccentric**	Paranoid	Pervasive distrust and suspiciousness; reads motives as malevolent
Cluster A	Odd or eccentric	Schizoid	Detachment from relationships; restricted emotion; a contented loner
Cluster A	Odd or eccentric	Schizotypal	**Social discomfort plus cognitive-perceptual distortions and eccentricity**
Cluster B	**Dramatic, emotional or erratic**	Antisocial	Disregard for and violation of others' rights; deceit, impulsivity, no remorse
Cluster B	Dramatic, emotional or erratic	Borderline	**Instability of relationships, self-image and affect; marked impulsivity**
Cluster B	Dramatic, emotional or erratic	Histrionic	Excessive emotionality and attention seeking
Cluster B	Dramatic, emotional or erratic	Narcissistic	Grandiosity, need for admiration, lack of empathy
Cluster C	**Anxious or fearful**	Avoidant	Social inhibition, feelings of inadequacy, fear of negative evaluation
Cluster C	Anxious or fearful	Dependent	Excessive need to be cared for; submissive, clinging; fear of separation
Cluster C	Anxious or fearful	Obsessive-compulsive	**Preoccupation with order, perfectionism and control; not OCD**

The DSM-5-TR Section II categorical system: three clusters holding ten types; the discriminator cells flag the boundaries most often tested (schizotypal versus schizophrenia, borderline as the flagship, obsessive-compulsive personality versus OCD) (DSM-5-TR 2022).

MNEMONIC

Weird, Wild, Worried

The three clusters by feel. **Cluster A** is **Weird** (odd or eccentric: Paranoid, Schizoid, Schizotypal, the three "P-S-S" types). **Cluster B** is **Wild** (dramatic, emotional or erratic: Antisocial, Borderline, Histrionic, Narcissistic). **Cluster C** is **Worried** (anxious or fearful: Avoidant, Dependent, Obsessive-compulsive). Three, four, three, adding to the ten types.

GOOD TO REMEMBER

- **Cluster C (anxious or fearful):** avoidant (social inhibition and fear of negative evaluation; wants but fears closeness), dependent (excessive need to be cared for, clinging, fear of separation), obsessive-compulsive personality (orderliness, perfectionism and control without true obsessions or compulsions); the whole set is three clusters and ten types, a clustering DSM-5-TR itself flags as weakly validated.

ICD-10 LEGACY: WHAT CHANGED

The old ICD-10 KEPT a categorical list of personality disorders largely paralleling the DSM types (paranoid, schizoid, dissocial for antisocial, emotionally unstable with an impulsive and a borderline type, histrionic, anankastic for obsessive-compulsive, anxious for avoidant, and dependent), with two familiar divergences: narcissistic sat only under "other specific personality disorder", and schizotypal was placed in the schizophrenia and delusional-disorders grouping, not among the personality disorders. ICD-11 then made the sharpest break of all: it *abolished* every one of these discrete categorical types, replacing them with a single personality-disorder diagnosis rated by severity (personality difficulty, then mild, moderate or severe) plus five trait qualifiers and a borderline pattern qualifier. Examinations still test the categorical names because DSM-5-TR Section II retains them, decades of literature and clinical communication use them, and the dimensional transition is recent, so the vocabulary of clusters and types remains the shared currency even where the official classification has moved on.

3.6 Borderline personality disorder: the flagship type and the five-of-nine threshold

The criterion to state cold. borderline personality disorder is "a pervasive pattern of instability of interpersonal relationships, self-image, and affects, and marked impulsivity, beginning by early adulthood", diagnosed when **five or more of the nine** criteria are present (DSM-5-TR 2022). The nine are: frantic efforts to avoid abandonment; a pattern of unstable, intense relationships alternating between idealisation and devaluation; identity disturbance; impulsivity in at least two potentially self-damaging areas; recurrent suicidal or self-mutilating behaviour; affective instability due to marked reactivity of mood; chronic feelings of emptiness; inappropriate, intense anger; and transient, stress-related paranoid ideation or severe dissociative symptoms.

Why the threshold is worth memorising. The **five or more of the nine** rule is the count most likely to be asked, and it is the reason a polythetic categorical system produces so many symptom combinations: two people can each carry the borderline label while sharing only one of the nine features. That combinatorial sprawl is the empirical case for the dimensional models. The borderline-versus-bipolar-II discrimination (do not start a mood stabiliser for personality-based

instability) and the biosocial and psychotherapeutic detail are developed elsewhere in the day and in the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes.

GOOD TO REMEMBER

- **Borderline personality disorder:** a pervasive pattern of instability of relationships, self-image and affect with marked impulsivity from early adulthood; diagnosed on five or more of the nine criteria (abandonment fears, idealisation-devaluation, identity disturbance, impulsivity, recurrent self-harm or suicidality, affective instability, chronic emptiness, intense anger, transient paranoia or dissociation); the first diagnostic step is the enduring pattern, and the five-of-nine polythetic threshold is why the same label covers very different presentations.

3.7 The alternative model (AMPD): a dimensional-categorical hybrid

What the hybrid does. The Section III alternative model for personality disorders redefines a personality disorder as the combination of two things: *impairment in personality functioning* (Criterion A) and one or more *pathological personality traits* (Criterion B) (DSM-5-TR 2022). It is a **hybrid model** because it fuses a dimensional severity axis (how impaired the personality is) with a dimensional trait profile (which traits are elevated), then still allows six named categorical diagnoses to be reconstructed from those dimensions. The general criteria C to G (inflexible and pervasive, stable since adolescence or early adulthood, not better explained by another disorder, a substance or a developmental or cultural norm) carry over unchanged.

The two determinations. Diagnosing under the **ampd** requires two assessments in order: first rate the level of impairment in personality functioning (Criterion A) on the Level of Personality Functioning Scale, then evaluate the pathological personality traits (Criterion B) across the trait domains. A *moderate* or greater impairment in personality functioning is the entry threshold; below it, no personality disorder is diagnosed however the traits fall.

GOOD TO REMEMBER

- **Alternative model (AMPD):** the DSM-5 Section III dimensional-categorical hybrid; a personality disorder is impairment in personality functioning (Criterion A) plus one or more pathological personality traits (Criterion B), retaining general criteria C to G; diagnosis runs A then B, with moderate or greater functional impairment the required entry threshold; it exists to cure the categorical system's comorbidity and its uninformative "unspecified" default.

3.8 Criterion A: the Level of Personality Functioning scale (self and interpersonal)

The self and interpersonal axes. Criterion A grades disturbance in *self* and *interpersonal* functioning on the Level of Personality Functioning Scale (DSM-5-TR 2022). Self functioning has two elements, identity and self-direction; interpersonal functioning has two, empathy and intimacy. The scale differentiates five levels of impairment, from little or no impairment (healthy) through some, moderate and severe to extreme; moderate impairment is the threshold that a personality disorder must reach. The severity of impairment also predicts whether the person has more than one personality disorder or one of the more severe types, which is why the **level of personality functioning** is rated first and leads.

Identity (self)

A stable, bounded sense of self; regulated self-esteem and accurate self-appraisal; capacity to experience and modulate a range of emotion.

Self-direction (self)
Pursuit of coherent short-term and life goals; constructive, prosocial internal standards; the ability to self-reflect productively.

Empathy (interpersonal)
Comprehension of others' experiences and motives; tolerance of differing perspectives; awareness of one's effect on others.

Intimacy (interpersonal)
Depth and duration of connection; the desire and capacity for closeness; mutuality of regard in relationships.

The four elements of personality functioning that anchor Criterion A of the alternative model; two are self elements, two interpersonal, graded across five severity levels with moderate impairment the diagnostic threshold (DSM-5-TR 2022).

GOOD TO REMEMBER

- **Criterion A (Level of Personality Functioning):** the AMPD severity axis, self functioning (identity, self-direction) and interpersonal functioning (empathy, intimacy) across five levels from little-or-no to extreme; moderate or greater impairment is required to diagnose any personality disorder, and severity predicts multiplicity and the more severe types, so it is rated first.

3.9 Criterion B: the five pathological trait domains, and the ICD-11 cross-map

The five domains and their facets. Criterion B profiles the personality across five broad pathological trait domains: **negative affectivity**, **detachment**, **antagonism**, **disinhibition** and **psychoticism**, which subdivide into twenty-five specific trait facets (DSM-5-TR 2022). Each named disorder's B criterion is a defined subset of those facets, for example antisocial personality disorder draws on Antagonism and Disinhibition, avoidant on Negative Affectivity and Detachment. The five domains are the maladaptive-range extensions of the Big Five personality dimensions.

The cross-map to ICD-11, the point examiners love. The ICD-11 dimensional model, taught forward in its own note, uses its own five trait qualifiers, and four broadly correspond to the DSM-5 **pathological trait domains**: negative affectivity, detachment, dissociality (aligning with DSM-5 Antagonism) and disinhibition are shared. The fifth is where they part: DSM-5 has **psychoticism** (which ICD-11 has no equivalent domain for), while ICD-11 has **anankastia** (which DSM-5 folds into low Disinhibition rather than naming). ICD-11 then adds a borderline pattern qualifier on top of its domains, a feature the DSM-5 trait set does not carry.

DSM-5 ALTERNATIVE-MODEL DOMAIN	ICD-11 TRAIT QUALIFIER	WHAT IT CAPTURES
Negative affectivity	Negative affectivity	Frequent, intense negative emotions (anxiety, insecurity, emotional lability)
Detachment	Detachment	Social and emotional withdrawal, avoidance of intimacy, restricted emotion
Antagonism	**Dissociality**	Disregard for others; grandiosity, deceit, callousness, manipulation
Disinhibition	Disinhibition	Impulsivity, irresponsibility, recklessness, distractibility
Psychoticism (no ICD-11 domain)	**Anankastia (no DSM-5 domain)**	DSM-5: odd beliefs, unusual perceptions, eccentricity. ICD-11: rigidity, perfectionism, constraint

Cross-mapping the two dimensional trait systems: four domains broadly correspond, but the fifth differs, DSM-5 names Psychoticism where ICD-11 names Anankastia, and ICD-11 adds a borderline pattern qualifier the DSM-5 set lacks (DSM-5-TR 2022; ICD-11 CDDR).

COMMON TRAP

Do not treat the two dimensional systems as identical five-domain sets. Four domains align (negative affectivity, detachment, antagonism as ICD-11 dissociality, disinhibition), but the fifth is different in each: the DSM-5 alternative model has *psychoticism* and no anankastia domain, whereas ICD-11 has *anankastia* and no psychoticism domain. Naming psychoticism as an ICD-11 domain, or anankastia as a DSM-5 domain, is a straight error.

GOOD TO REMEMBER

- **Criterion B (pathological trait domains):** five domains, negative affectivity, detachment, antagonism, disinhibition and psychoticism, with twenty-five facets, each disorder's B criterion a defined subset; four align with the ICD-11 qualifiers (antagonism maps to dissociality), but DSM-5's fifth is psychoticism while ICD-11's is anankastia, and only ICD-11 adds a borderline pattern qualifier.

3.10 The six retained types and the trait-specified diagnosis

Ten collapse to six, plus a residual. Where the categorical system names ten types, the **alternative model** retains only six that can be diagnosed by their own criteria sets: antisocial, avoidant, borderline, narcissistic, obsessive-compulsive and schizotypal personality disorders (DSM-5-TR 2022). Each is defined by a characteristic Criterion A functioning impairment plus a specified Criterion B trait subset. The four categorical types dropped from the named list (paranoid, schizoid, histrionic, dependent) are not lost: when a personality disorder is present but no specific set is met, it is diagnosed as *personality disorder, trait specified* (PD-TS), recording the impairment level and the elevated trait domains directly.

How a diagnosis is built. To diagnose, say, avoidant personality disorder under the hybrid model, the clinician confirms moderate or greater impairment across the relevant functioning elements (Criterion A) and the specified maladaptive traits, here in Negative Affectivity and Detachment (Criterion B), then checks the general criteria C to G. The system's promise is that the profile itself, the severity level and the domain scores, carries the clinical information even when no tidy category fits, which is precisely the case in most real patients.

GOOD TO REMEMBER

- **The six AMPD types and PD-TS:** the alternative model diagnoses six specific types, antisocial, avoidant, borderline, narcissistic, obsessive-compulsive and schizotypal, each an A-functioning impairment plus a B-trait subset; the other cases (including the dropped paranoid, schizoid, histrionic and dependent) become personality disorder, trait specified, recorded by severity level and trait domains.

3.11 The India lens: under-recognition, cultural expression and the medico-legal frame

An under-recognised group read through the wrong culture. Personality disorders are markedly under-recognised in routine Indian practice: they present through their comorbidities (a mood or anxiety disorder, a substance problem, repeated self-harm) rather than as a personality diagnosis, they are under-coded, and the personality label is often avoided for fear of stigma. The cultural clause in the general criteria matters acutely here, deviation must be judged against the patient's own cultural, familial and social norms, so that collectivist family embeddedness is not misread as dependent personality, culturally sanctioned religious or ritual practice is not misread as schizotypal eccentricity, and gendered social scripts are not misread as histrionic or avoidant traits. The cultural expression of personality pathology is genuine and must be weighed before any type is fixed.

Where the label carries force. Antisocial personality disorder is the type with the sharpest medico-legal edge in Indian practice, surfacing in offender assessment, fitness and court reports, where a careful, non-pejorative formulation matters, and it is developed in the Mental health legislation and forensic psychiatry notes. On the treatment side, the structured psychotherapies that the borderline diagnosis calls for, dialectical behaviour therapy and schema therapy, are available only patchily outside the metros and are being culturally adapted for Indian settings; that availability gap, not the classification, is usually what limits care once the diagnosis is correctly made.

INDIA

Personality disorders are under-recognised in Indian practice, surfacing through comorbid mood, anxiety, substance or self-harm presentations and often left uncoded for fear of stigma. The general criteria's cultural clause is the safeguard: judge deviation against the patient's own cultural and social norms, so family embeddedness is not scored as dependency, religious practice not as schizotypal eccentricity, and social scripts not as histrionic or avoidant pathology. Antisocial personality disorder carries the medico-legal weight (offender and court work, cross-referenced to the forensic notes), and the structured psychotherapies borderline needs, dialectical behaviour therapy and schema therapy, are unevenly available and still being culturally adapted for India.

3	10	5	five
CATEGORICAL CLUSTERS (A ODD, B DRAMATIC, C ANXIOUS)	CATEGORICAL PERSONALITY-DISORDER TYPES ACROSS THE CLUSTERS	PATHOLOGICAL TRAIT DOMAINS IN THE DSM-5 ALTERNATIVE MODEL	OF THE NINE CRITERIA NEEDED TO DIAGNOSE BORDERLINE PERSONALITY DISORDER

HOLD THIS

DSM-5-TR keeps the categorical clusters in Section II (Cluster A odd or eccentric: paranoid, schizoid, schizotypal; Cluster B dramatic, emotional or erratic: antisocial, borderline, histrionic, narcissistic; Cluster C anxious or fearful: avoidant, dependent, obsessive-compulsive, ten types in all) and offers, in Section III, the alternative hybrid model (AMPD): Criterion A the Level of Personality Functioning scale (self: identity, self-direction; interpersonal: empathy, intimacy) plus Criterion B the five pathological trait domains (negative affectivity, detachment, antagonism, disinhibition, psychoticism), diagnosing six specific types; ICD-11 has abolished the categorical types for a severity-led dimensional model whose fifth domain is anankastia, not psychoticism, yet the categorical names remain the examinable vocabulary.

4 · Categorical versus dimensional approaches

Why this matters. This is the conceptual hinge of the whole personality-disorders chapter, and it is the stem an examiner reaches for when a single question has to test understanding rather than recall. The sibling notes teach the two live classifications in full, the DSM-5-TR categorical clusters and the ICD-11 dimensional model; this note owns the argument *between* them. The **categorical versus dimensional** debate asks a prior question: is a personality disorder a distinct kind of thing that you either have or do not, or a position on a set of continua that shade imperceptibly into ordinary personality? Answer that, and you can explain every failing of the old system and every design choice of the new one.

Held tightly, the **comparison of approaches** is a small object: two definitions, a short list of **strengths and weaknesses** on each side, and the reason the evidence tilted one way. The categorical approach trades validity for familiarity; the dimensional approach trades familiarity for validity and coverage; and the modern manuals split the difference, ICD-11 committing fully to dimensions while DSM-5-TR keeps the categories in front and files the dimensional model behind them for study. The accompanying figure lays the two approaches out as one grid, discrete boxes on one side against graded continua on the other, and it is the single image to carry for this

topic. The detailed severity gradient, the trait domains and the cluster map are developed in the sibling framework notes and only invoked here as evidence.



Fig 16.4 Categorical classification names discrete types (familiar, but with excessive comorbidity, arbitrary thresholds and a large residual group), while dimensional classification grades severity and traits on continua (better validity and coverage of the subthreshold majority); the field moved dimensional, though the categories are still examined.

4.1 Two ways to carve personality: kinds versus degrees

The distinction to state first. The categorical approach holds that personality disorders are *qualitatively distinct clinical syndromes*, discrete kinds that a person either meets or does not, sorted into named types (DSM-5-TR 2022). The dimensional approach holds the opposite: that personality pathology represents *maladaptive variants of personality traits that merge imperceptibly into normality and into one another*, so that difference from health is one of degree, not of kind (DSM-5-TR 2022). One view draws boundaries and puts patients in boxes; the other places them on continua of severity and trait and reads their position. Everything else in the debate follows from this single fork, kinds against degrees.

Why the fork is not merely philosophical. The choice determines what the diagnosis can say. A category returns a name; a dimension returns a profile. Where a categorical system must decide a threshold above which a person "has" the disorder, a dimensional system can describe exactly how far along each continuum the person sits, and can keep describing the many people who fall below any categorical cut-off. The five-factor model of normal personality supplies the trait scaffolding the dimensional side builds on, its maladaptive poles becoming the pathological trait domains of the modern models (New Oxford Textbook of Psychiatry).

RULE **Kinds versus degrees is the whole debate.** The categorical approach treats a personality disorder as a discrete type you have or lack; the dimensional approach treats it as a position on continua of severity and trait that grade smoothly into normal personality. Every strength and weakness on either side is a consequence of this one choice.

4.2 The categorical approach: where it is strong

Familiarity and communicability are its real assets. The categorical approach is the default of clinical medicine: a diagnosis is a name, and a name is fast to assign, easy to record, and instantly communicable to another clinician in a single phrase (Tasman Psychiatry). Saying "borderline personality disorder" transmits a rich prototype in two words; there is a century of description, a large treatment literature and an examination vocabulary all built on the named types. The explicit operational criteria introduced from DSM-III onward gave the categories a further, genuine payoff: they let the field *document* substantial community and clinical prevalence, high rates of social and occupational impairment, and a more chronic course for co-occurring disorders (New Oxford Textbook of Psychiatry). These are not trivial gains, and they are why the categories have survived every attempt to retire them.

The prototype is the mechanism. Clinicians reason by pattern-matching a patient to a remembered exemplar, and the categorical types are ready-made prototypes. That cognitive fit, not evidence of natural boundaries, is the categorical system's durable strength: it maps onto how doctors actually think and talk.

PEARL

The categorical system persists because of its *communicative* value, not its scientific validity. The single most defensible thing to say for it is that a type name carries a whole clinical picture in one phrase, which is exactly what a busy clinic and a shared record need. Hold that as its strength, and its weaknesses become easier to name honestly.

4.3 The categorical approach: where it fails

Four failings, and they are examinable as a set. The problems are the mirror image of the discrete-kinds assumption, and the New Oxford Textbook lists them cleanly: *extensive co-occurrence of personality disorders* (excessive comorbidity, patients routinely meeting several type criteria at once), *considerable heterogeneity within categories* (two people can share a diagnosis while sharing almost none of its features), *arbitrary diagnostic thresholds without empirical bases* (the cut-off between disorder and no-disorder is a convention, not a natural join), and *incomplete coverage of the full range of personality pathology* (the many patients whose difficulty is real but does not match any single type). Temporal instability of the categories and their limited validity and clinical utility round out the indictment (New Oxford Textbook of Psychiatry).

Borderline is the worked case of heterogeneity. Because borderline personality disorder is polythetic, diagnosed on **five or more of the nine** criteria, two patients can each carry the label while overlapping on only one feature. That combinatorial sprawl is the categorical system's within-category heterogeneity made concrete, and it is the empirical case the dimensional models

were built to answer. The unstable, affectively reactive presentation also blurs into bipolar-II, a boundary drawn in the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes.

The residual category tells the story. The clearest sign of incomplete coverage is that the residual not-otherwise-specified diagnosis (personality disorder not otherwise specified in DSM-IV, and the "other specified" and "unspecified" categories in DSM-5-TR) has been among the most frequently assigned of all the personality-disorder diagnoses, and is also the least informative one (DSM-5-TR 2022; New Oxford Textbook of Psychiatry). A system whose commonest verdict is "a personality disorder, but none of the ones we named" is telling you its named types do not cover the territory.

TRAP **Reading the types as non-overlapping natural kinds.** The categorical model implies the types are distinct and separable, but they co-occur freely and blur at every edge; treating a tidy single-type label as the natural truth of a patient, rather than a convenient summary, is the error the dimensional case exists to correct.

COMMON TRAP

Mistaking the frequency of the not-otherwise-specified diagnosis for clinician laziness. It is a structural signal, not a shortcut: the residual category is common *because* most real personality pathology is subthreshold or mixed and falls between the named types. That is the coverage problem, not a documentation problem.

4.4 The dimensional approach: its strengths

It fixes what the categories broke. By placing severity and traits on continua, the dimensional approach retains the information a category discards: a profile records how impaired the personality is and which traits are elevated, so two patients who would share one categorical label are told apart, and a patient who matches no type is still fully described (Tasman Psychiatry). This is why the empirical structure favours it: a meta-analytic review of many latent-structure studies found little persuasive evidence for discrete taxa underlying the personality disorders, with schizotypal the only possible partial exception (New Oxford Textbook of Psychiatry). If the underlying reality is continuous, a continuous model has better *validity* and, crucially, covers the subthreshold majority that the categorical cut-offs leave out.

Severity leads because severity predicts. The single most useful dimension is overall severity of personality dysfunction, which forecasts self-harm, service use, relationship breakdown and occupational failure better than any type label; rating it first is the design principle both modern dimensional systems adopt. The schizotypal exception, the one construct with some categorical support, sits at the schizophrenia-spectrum edge and is developed in the Schizophrenia: aetiology, phenomenology, classification and course notes.

4.5 The dimensional approach: its price

Validity is bought with unfamiliarity. The dimensional model's weaknesses are the reciprocal of its strengths. It is *less familiar* and harder to communicate: a severity level plus a set of trait

qualifiers does not compress into a single memorable phrase the way "narcissistic personality disorder" does, and clinicians trained on the types find the profiles unintuitive at first (New Oxford Textbook of Psychiatry). There has also been decades of *controversy over the best dimensional representation*, how many domains, which ones, how to set the severity anchors, though considerable consensus has now settled on a small set of core trait domains. Notably, once clinicians use the dimensional models they tend to find them *more* clinically useful than the categories, so the unfamiliarity is a transitional cost rather than a permanent defect (New Oxford Textbook of Psychiatry).

GOOD TO REMEMBER

- **Categorical approach:** personality disorders as qualitatively distinct kinds sorted into named types; strengths are clinical familiarity and one-phrase communicability plus documented prevalence, impairment and course; weaknesses are excessive comorbidity, within-category heterogeneity, arbitrary thresholds, temporal instability, limited validity, and incomplete coverage of the subthreshold majority (a large not-otherwise-specified group).
- **Dimensional approach:** personality pathology as maladaptive variants of traits on continua of severity and trait that merge into normality; strengths are better validity, retention of individual profile information and coverage of the subthreshold majority; weaknesses are unfamiliarity, harder single-phrase communication, and past controversy over the best representation.
- **The discriminator to state:** kinds versus degrees; every strength and weakness on each side follows from that one assumption.

4.6 The comparison of approaches, side by side

The object to reproduce cold. The **comparison of approaches** is best held as a two-column ledger of matched trade-offs; the highlighted cells are the points most often tested. Read across each row and the reciprocal structure is obvious: what one approach gains, the other loses.

DIMENSION OF COMPARISON	CATEGORICAL APPROACH	DIMENSIONAL APPROACH
Core claim	Distinct kinds; a disorder you have or lack	**Degrees; maladaptive variants merging into normality**
Diagnosis returns	A type name	A severity level plus a trait profile
Communicability	**High; one phrase carries a prototype**	Lower; a profile is harder to state briefly
Clinical familiarity	**High; the default of medicine, a century of use**	Low; newer and initially unintuitive
Comorbidity	**Excessive; types co-occur freely**	Dissolved; overlap is expected on shared traits
Within-category variation	Large; polythetic criteria yield heterogeneous cases	**Captured; the profile separates similar-looking patients**
Thresholds	Arbitrary, without empirical basis	Graded; the cut is a chosen point on a continuum
Coverage of subthreshold pathology	Poor; a large not-otherwise-specified residual	**Good; the subthreshold majority is still described**

DIMENSION OF COMPARISON	CATEGORICAL APPROACH	DIMENSIONAL APPROACH
Empirical validity	Limited; little evidence for discrete taxa	**Better; matches the continuous latent structure**

The categorical and dimensional approaches as matched trade-offs; the highlighted cells mark each side's decisive advantage, and every row is a consequence of the kinds-versus-degrees choice.

MNEMONIC

Categories COMMUNICATE, dimensions VALIDATE

The one-line split. The **categorical** approach wins on **C**ommunicability and **C**linical familiarity but is **C**omorbidity-ridden and **C**rudely-thresholded. The **dimensional** approach wins on **V**alidity, **V**ariation captured and coverage, but is unfamiliar and hard to state in a phrase. Communicate versus validate is the trade-off, and the field has decided validity should lead.

4.7 Why the field moved to dimensional

The evidence tilted, then the manuals followed. The reason for the shift is not fashion but data. Latent-structure research found little support for discrete personality-disorder taxa, and by a 2007 survey a large majority of personality-disorder experts, some **eighty-seven per cent**, judged that personality pathology was dimensional in nature (New Oxford Textbook of Psychiatry). The verdict in the literature is that the classification of personality pathology has been "slowly but inexorably moving from a categorical to a dimensional model", because a dimensional model better reflects the continuous phenomena and better describes both the distance from normal personality and the degree of severity. That, in a phrase, is **why the field moved to dimensional**: the discrete kinds the categories assume do not appear to exist, and continua describe the reality and predict the outcomes better.

Two manuals, two speeds of change. ICD-11 made the full commitment: it abolished the categorical types outright and rebuilt the diagnosis as a severity rating (personality difficulty, then mild, moderate or severe) followed by the trait-domain qualifiers and an optional borderline pattern, the model taught forward in the ICD-11 dimensional-classification note. DSM-5-TR was more cautious, and its caution is itself the examinable compromise: it kept the familiar categorical clusters and ten types in Section II for everyday use, and placed the alternative model of personality disorders (the AMPD, a dimensional-categorical hybrid built on the Level of Personality Functioning Scale plus five trait domains) in Section III for further study, developed in the DSM-5 categorical-clusters note. The hybrid is the evidence-driven middle path: it scores personality functioning and traits dimensionally, yet still lets a handful of named types be reconstructed, so the field can move toward dimensions without discarding a century of clinical vocabulary at once.

ICD-10 TO ICD-11: WHAT CHANGED

ICD-10 (F60) **kept the categorical types**, paranoid, schizoid, dissocial, emotionally unstable, histrionic, anankastic, anxious and dependent, in step with the DSM tradition. ICD-11 **removed every one of them** and replaced the list with a severity-led dimensional diagnosis plus trait qualifiers and a borderline pattern qualifier. This is the field moving to dimensional in its purest form. Examinations still test the categorical names because DSM-5-TR Section II retains them, because decades of literature and clinical communication run on them, and because the transition is recent, so a candidate must read fluently in both the categorical and the dimensional languages.

WORKED EXAMPLE

Take the same patient through both approaches. A man in his thirties has rigid perfectionism and interpersonal coldness, plus marked suspiciousness, but does not fully meet any single type. The *categorical approach* stalls: he has features of obsessive-compulsive, paranoid and schizoid personality disorder, meets none cleanly, and is filed as a personality disorder, unspecified, a label that says almost nothing. The *dimensional approach* succeeds: rate moderate personality dysfunction and note prominent anankastia and detachment with some negative affectivity, and the profile describes him precisely and guides the plan. The contrast is the whole argument in one case: the category returns a shrug, the dimensions return a description.

4.8 The India lens and the cultural frame

Under-recognition meets a severity-led model. Personality disorders are markedly under-recognised in routine Indian practice: they surface through comorbid mood, anxiety, substance or self-harm presentations rather than as a personality diagnosis, and the personality label is often avoided for fear of stigma (Gupta and Mattoo, on Indian personality-disorder prevalence and demography). A severity-led dimensional system, which begins with a graded judgement of self-and-interpersonal dysfunction rather than demanding a full type match, is well suited to catching the milder and mixed presentations that categorical thresholds cause Indian services to miss. The categorical versus dimensional choice is therefore not only academic here; the dimensional route may materially improve detection.



Fig 16.5 The legacy DSM categorical model groups personality disorders into Cluster A, odd or eccentric; Cluster B, dramatic, emotional or erratic; and Cluster C, anxious or fearful. ICD-11 instead prioritises severity and trait domains.

The cultural expression of pathology cuts across both. Whichever approach is used, the general criteria require deviation from what the person's *own* culture expects, so a self deeply embedded in family and community, or a deference and interdependence that would read as dependence through a Western lens, must not be scored as disorder; personality pathology must not be read through a single culture's norms. On the treatment side, the structured psychotherapies a dimensional severity rating points toward, dialectical behaviour therapy and schema therapy, are available and being culturally adapted in Indian metros and academic centres but remain scarce elsewhere, so a severe rating can outrun what a service can deliver. Antisocial personality disorder carries the sharpest medico-legal edge in Indian practice, and its offender and court framing is handled in the Mental health legislation and forensic psychiatry notes; the wider technique of the psychological therapies belongs to the Psychotherapies, clinical assessment, MSE and nosology notes.

INDIA

Personality disorders are under-recognised in Indian practice, presenting through their comorbidities and often left uncoded for fear of stigma; a dimensional, severity-first reading is better at catching the subthreshold and mixed cases that categorical cut-offs miss. Judge deviation against the patient's own cultural and social norms, so collectivist family-embeddedness is not scored as dependency and religiously framed experience not as schizotypal oddness. The structured psychotherapies that the dimensional model points to, dialectical behaviour therapy and schema therapy, are available and being culturally adapted in the metros and academic centres but unevenly accessible elsewhere; and antisocial personality disorder carries the medico-legal weight, cross-referenced to the forensic notes.

3 / 10	5	5+	87%
DSM-5-TR CATEGORICAL CLUSTERS / TYPES	DIMENSIONAL TRAIT DOMAINS (ICD-11 AND AMPD)	OF NINE BORDERLINE CRITERIA (THE POLYTHETIC THRESHOLD)	OF PD EXPERTS JUDGED PATHOLOGY DIMENSIONAL (2007 SURVEY)

HOLD THIS

The categorical approach treats a personality disorder as a discrete type (familiar and communicable, but comorbid, heterogeneous, arbitrarily thresholded and poor at covering the subthreshold majority); the dimensional approach treats it as severity plus traits on continua (better validity and coverage, but less familiar); the field moved to dimensional because the discrete kinds do not appear to exist, and ICD-11 abolished the types while DSM-5-TR keeps them in front with the AMPD hybrid behind as the evidence-driven compromise.

The personality disorders by cluster

5 . Cluster A: paranoid, schizoid and schizotypal

Why this cluster is worth its marks. **cluster a** is the **odd or eccentric** group, and it rewards a candidate who can do two things: give the one-line essential feature of each of its three types cold, and defend the boundaries that separate them from one another and from frank psychosis. The three are paranoid personality disorder (a pervasive distrust and suspiciousness in which others' motives are read as malevolent), schizoid personality disorder (detachment from social relationships with a restricted range of emotional expression), and schizotypal personality disorder (acute discomfort in close relationships together with cognitive or perceptual distortions and eccentricities of behaviour) (DSM-5-TR). The thread that ties them is a retreat from, or a distortion of, ordinary social relatedness, and at the schizotypal end that thread runs into the **schizophrenia spectrum**.

This fragment first frames the cluster on one grid, then takes each type through its defining criterion and the discriminator an examiner rewards, and finally isolates the single highest-yield boundary of the group, schizotypal versus schizophrenia, with the nosological twist that schizotypal is the one member that both manuals place, wholly or partly, among the psychotic disorders rather than the personality disorders. Two prior cautions carry over from the

framework topics of this chapter and are assumed throughout: the general personality-disorder criteria (an enduring, pervasive, inflexible pattern, culturally deviant, with onset by adolescence, causing impairment, and not better explained otherwise) must be cleared before any of these types is named; and ICD-11 has abolished the categorical types, so "paranoid" and "schizoid" as labels survive only in DSM-5-TR and in clinical shorthand. The deep pharmacology sits in the Schizophrenia: management, antipsychotics and adverse effects notes, the schizophrenia boundary in the Schizophrenia: aetiology, phenomenology, classification and course notes, and the wider psychotherapy technique in the Psychotherapies, clinical assessment, MSE and nosology notes.

5.1 The cluster on one frame

Three types, one thread, graded by how far from reality they drift. DSM-5-TR groups the three types whose sufferers "often appear odd or eccentric" (DSM-5-TR). Read the cluster as a gradient of departure from normal relatedness: paranoid personality disorder distrusts other people but keeps a firm hold on reality; schizoid personality disorder withdraws from other people without distorting them; schizotypal personality disorder adds to that withdrawal a layer of cognitive and perceptual oddness that shades toward, but never reaches, frank psychosis. Learning the three essential features and their thresholds as one block is the fastest route to every cluster A question.

TYPE	ESSENTIAL FEATURE (DSM-5-TR)	THRESHOLD	CORE DISCRIMINATOR
Paranoid	A pervasive distrust and suspiciousness such that others' motives are interpreted as malevolent	Four or more of the seven features	**Suspiciousness with reality intact; no cognitive-perceptual distortion**
Schizoid	Detachment from social relationships with a restricted range of emotional expression	Four or more of the seven features	**A contented loner: no desire for closeness at all**
Schizotypal	Acute discomfort in close relationships, plus cognitive or perceptual distortions and eccentricities of behaviour	Five or more of the nine features	**Odd cognition and perception; the schizophrenia-spectrum edge**

The three DSM-5-TR Cluster A types with their essential features, criterion thresholds and core discriminators; the accompanying figure lays the same three on a distrust-to-distortion gradient.

PEARL

A one-word tag per type keeps them apart under pressure: paranoid is *distrust*, schizoid is *detachment*, and schizotypal is *distortion* laid on top of detachment. The suspiciousness of paranoid and the aloofness of schizoid can both appear in schizotypal, but only schizotypal carries the odd beliefs, perceptions and speech; that added layer is the whole diagnosis.

5.2 Paranoid personality disorder

Malevolent motives read into a neutral world. The essential feature of paranoid personality disorder is a pervasive distrust and suspiciousness of others such that their motives are interpreted as malevolent, beginning by early adulthood and present across contexts, shown by **four or more of the seven** features (DSM-5-TR). The seven are: unjustified suspicion that others are exploiting, harming or deceiving them; preoccupation with doubts about the loyalty or trustworthiness of associates; reluctance to confide for fear the information will be used against them; reading hidden demeaning or threatening meaning into benign remarks or events; persistently bearing grudges; perceiving attacks on their character that are not apparent to others, with a quick angry counterattack; and recurrent unjustified suspicions about a partner's fidelity. The interpersonal signature is a person who scrutinises every action for hostile intent and cannot accept loyalty when it is shown. Crucially the beliefs are held with suspicion, not with delusional conviction: the patient can, at least in principle, acknowledge an alternative reading, which is exactly what separates this from a persecutory delusion (Companion).

GOOD TO REMEMBER

- **Paranoid personality disorder:** defining criterion is a pervasive distrust and suspiciousness in which others' motives are read as malevolent, four or more of the seven features present, from early adulthood. Discriminator upward from psychosis: the suspicions are not of delusional intensity and alternatives can be acknowledged (from delusional disorder and schizophrenia). Discriminator within the cluster: no cognitive-perceptual distortion or eccentricity, which would move it to schizotypal. First step: confirm the general personality-disorder criteria, then that the suspiciousness is pervasive and reality-based, not a fixed delusion.

5.3 Schizoid personality disorder

The genuine loner, not the frightened one. The essential feature of schizoid personality disorder is a pervasive pattern of detachment from social relationships and a restricted range of emotional expression in interpersonal settings, from early adulthood, shown by **four or more of the seven** features (DSM-5-TR). The seven are: neither desiring nor enjoying close relationships, including being part of a family; almost always choosing solitary activities; little interest in sexual experiences with another person; taking pleasure in few activities; lacking close friends or confidants other than first-degree relatives; indifference to praise or criticism; and emotional coldness, detachment or flattened affect. The person is a contented loner who does not want closeness, presenting a bland exterior with little visible emotional reactivity. The examiner's favourite contrast is with avoidant personality disorder: the avoidant person longs for relationships but is held back by fear of rejection, whereas the schizoid person has no desire for them in the first place. That single question, "do they want closeness?", separates the two.

GOOD TO REMEMBER

- **Schizoid personality disorder:** defining criterion is detachment from social relationships with a restricted, flattened range of emotional expression, four or more of the seven features, from early adulthood. Discriminator from avoidant: absence of any desire for closeness (avoidant desires it but fears rejection). Discriminator from schizotypal: detachment without cognitive-perceptual distortion or eccentricity. First step: establish that solitariness is preferred and pervasive, not a secondary retreat from anxiety, depression or psychosis.

5.4 Schizotypal personality disorder

Attenuated oddness, not frank psychosis. The essential feature of schizotypal personality disorder is a pervasive pattern of social and interpersonal deficits marked by acute discomfort with, and reduced capacity for, close relationships, together with cognitive or perceptual distortions and eccentricities of behaviour, shown by **five or more of the nine** features (DSM-5-TR). The nine are: ideas of reference (incorrect readings of casual events as having special personal meaning, but short of delusional conviction); odd beliefs or magical thinking influencing behaviour (superstition, belief in clairvoyance, telepathy or a "sixth sense"); unusual perceptual experiences including bodily illusions; odd thinking and speech (vague, digressive or over-elaborate, but without derailment or incoherence); suspiciousness or paranoid ideation; inappropriate or constricted affect; behaviour or appearance that is odd, eccentric or peculiar; lack of close friends other than first-degree relatives; and excessive social anxiety that does not ease with familiarity and is tied to paranoid fears rather than to negative self-judgement. Two distinctions matter. Ideas of reference are not delusions of reference: the difference is conviction. And the social anxiety, unlike the avoidant type's, does not settle as the person gets to know the setting, because it is driven by suspiciousness. Under stress these patients may have transient psychotic-like episodes lasting minutes to hours, usually too brief to meet the threshold for a brief psychotic disorder.

GOOD TO REMEMBER

- **Schizotypal personality disorder:** defining criterion is social and interpersonal deficits plus cognitive or perceptual distortions and eccentricity, five or more of the nine features, from early adulthood. Discriminator within the cluster: it is paranoid plus schizoid plus the distortion layer (odd beliefs, unusual perceptions, odd speech, eccentricity), which neither of the others has. Discriminator upward: the distortions are attenuated, ideas of reference not delusions, no sustained frank psychosis. First step: demonstrate the cognitive-perceptual oddness is enduring and trait-level, then that it never crosses into frank, sustained psychosis.

5.5 Schizotypal versus schizophrenia, and the schizophrenia-spectrum placement

The one member that belongs to the psychosis family. Schizotypal is the boundary the day is built to test. It is characterised by an enduring pattern of unusual speech, perceptions, beliefs and behaviours that resemble *attenuated* forms of the defining symptoms of schizophrenia (ICD-11 CDDR). The line from schizophrenia is drawn on intensity alone: schizophrenia is diagnosed when the symptoms are severe and sustained enough to meet its own threshold, and until then the presentation stays eccentric or peculiar rather than overtly psychotic. The familial evidence backs the kinship: schizotypal aggregates in the first-degree biological relatives of people with

schizophrenia and is regarded as part of the schizophrenia-related spectrum of psychopathology. This is why the classification treats it as a half-member of the personality-disorder family. DSM-5-TR lists it in the personality-disorders chapter but cross-lists it in the Schizophrenia Spectrum and Other Psychotic Disorders chapter and codes it F21. ICD-11 goes further and places schizotypal disorder (6A22) squarely within the schizophrenia and other primary psychotic disorders grouping, not among the personality disorders at all, continuing what ICD-10 did with F21. So while ICD-11 abolished paranoid and schizoid as categories, it kept schizotypal, but moved it out of the personality chapter entirely.

BOUNDARY	CLUSTER A SIDE	THE OTHER SIDE	DISCRIMINATOR
Schizotypal vs schizophrenia	Attenuated distortions; eccentric or peculiar; transient stress-related psychosis of minutes to hours	Frank, sustained delusions or hallucinations meeting threshold	**Intensity alone: attenuation versus frank sustained psychosis**
Schizotypal vs paranoid or schizoid	Cognitive-perceptual distortions and marked eccentricity present	Suspiciousness alone (paranoid) or detachment alone (schizoid)	Presence of the distortion and eccentricity layer
Schizoid vs avoidant	No desire for relationships; content in solitude	Desires relationships but fears rejection	Whether closeness is wanted at all
Paranoid vs delusional disorder	Suspicion not of delusional intensity; alternatives can be acknowledged	Fixed persecutory delusion held with conviction	Delusional conviction and intensity

The four boundaries most often tested around Cluster A; the highlighted cell is the single discriminator that decides schizotypal versus schizophrenia. The schizophrenia side is developed in the Schizophrenia: aetiology, phenomenology, classification and course notes.

RULE **Schizotypal is schizophrenia-spectrum, and the line is intensity.** ICD-11 codes schizotypal disorder 6A22 with the primary psychotic disorders, not the personality disorders; it is separated from schizophrenia by the attenuation of its symptoms alone, not by a different set of symptoms.

TRAP **Calling attenuated schizotypal features schizophrenia.** Ideas of reference, magical thinking, odd speech and eccentricity that never reach frank, sustained delusions or hallucinations are schizotypal, not schizophrenia; upgrading them to a psychotic diagnosis on their oddness alone is the classic error.

WORKED EXAMPLE

A man in his late twenties is referred as "possible schizophrenia". He has always been a fringe figure: he believes he can sense events before they happen, dresses in a way that does not quite fit together, speaks in a vague, over-elaborate way, and keeps to himself because he suspects colleagues mean him harm. There are no sustained delusions and no hallucinations; under stress he has had brief spells of feeling watched that pass within an hour or two. This is schizotypal personality disorder (or ICD-11 schizotypal disorder), not schizophrenia: the cognitive-perceptual features are attenuated and enduring, never crossing into frank, sustained psychosis. The label you avoid saves him a lifelong psychotic diagnosis and its treatment.

5.6 Management in outline, and the cultural reading

Engagement is the treatment; drugs are narrow and symptom-targeted. Cluster A is managed, not cured, and the deep detail is cross-referenced. No drug is disorder-specific for any of the three. The working mode is a structured, supportive psychotherapy delivered with a reliable, low-intensity, non-intrusive stance: for the paranoid and schizoid patient the whole difficulty is engagement, because suspiciousness or a genuine preference for solitude makes them slow to enter therapy and quick to leave it, and over-warmth or forced intimacy can rupture the alliance. For schizotypal personality disorder, a low-dose second-generation antipsychotic (for example, risperidone) can be used in a symptom-targeted, adjunctive way for the cognitive-perceptual features, with the dosing and adverse-effect detail carried in the Schizophrenia: management, antipsychotics and adverse effects notes rather than re-taught here. The broader psychotherapy technique sits in the Psychotherapies, clinical assessment, MSE and nosology notes.

GOOD TO REMEMBER

- **Structured supportive psychotherapy (Cluster A):** mechanism is a reliable, low-intensity working alliance that reduces isolation and reality-tests suspicion without forcing intimacy; indication is all three Cluster A types, chosen because they rarely tolerate intensive, emotionally demanding therapy; signature caution is that engagement itself is the obstacle, so over-familiarity or a pushed pace ruptures the alliance in the paranoid and schizoid patient. For schizotypal, a low-dose second-generation antipsychotic is adjunctive and symptom-targeted only, never disorder-specific.

INDIA

Two genuine India angles converge on Cluster A. First, under-recognition: these are the quietest personality disorders, ego-syntonic and rarely self-referred, so the paranoid and schizoid patient reaches services only through a comorbidity or a family crisis, and the diagnosis is made only when it is actively sought. Second, and more testable, the cultural reading of schizotypal features. Both DSM-5-TR and the ICD-11 CDDR warn explicitly that culturally sanctioned beliefs and practices, belief in the evil eye, a sixth sense, spirit visitation of a deceased relative, shamanic or trance experience, speaking in tongues, magical beliefs about health and illness, can look schizotypal to an uninformed clinician and must not be scored as cognitive-perceptual distortion when they are normative for the person's community. The cultural expression of personality pathology has to be weighed against the patient's own milieu, with an informant where needed, before any Cluster A type is fixed. Structured psychotherapy is available unevenly and mostly at tertiary and private centres.

5.7 The countable skeleton

One line rebuilds the cluster. Three types, two thresholds and one spectrum placement are the whole of Cluster A worth carrying into the hall. Hook them once and each type unpacks from the tag.

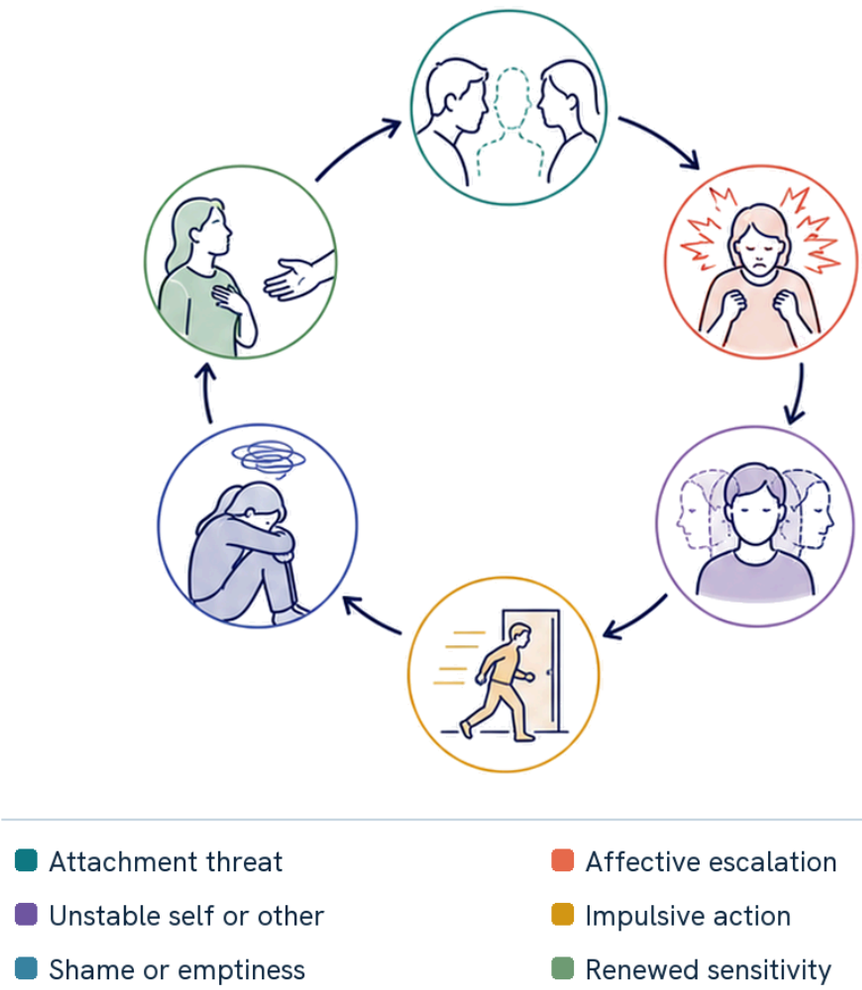


Fig 16.6 A common borderline personality dynamic links perceived attachment threat to rapid affective escalation, unstable self-other representations, impulsive action, shame or emptiness and renewed interpersonal sensitivity.

MNEMONIC

DISTRUST, DETACH, DISTORT

The three Cluster A types by their one-word core. **DISTRUST** is paranoid personality disorder (suspiciousness, motives read as malevolent, four or more of the seven, reality intact). **DETACH** is schizoid personality disorder (a contented loner, restricted emotion, four or more of the seven, no desire for closeness). **DISTORT** is schizotypal personality disorder (detachment plus cognitive-perceptual oddness and eccentricity, five or more of the nine, the schizophrenia-spectrum edge). Distrust keeps reality; detachment leaves it alone; distortion bends it, but never breaks it.

3 CLUSTER A TYPES (ODD OR ECCENTRIC)	**4** OF SEVEN FEATURES: PARANOID OR SCHIZOID THRESHOLD	**5** OF NINE FEATURES: SCHIZOTYPAL THRESHOLD	**6A22** ICD-11 SCHIZOTYPAL, IN THE SCHIZOPHRENIA SPECTRUM

HOLD THIS

Cluster A is odd or eccentric: paranoid is distrust with reality intact (four or more of the seven), schizoid is detachment with no wish for closeness (four or more of the seven), schizotypal is both plus cognitive-perceptual distortion (five or more of the nine); and schizotypal alone sits on the schizophrenia spectrum (ICD-11 schizotypal disorder 6A22), separated from schizophrenia by attenuation, not by a different symptom.

6 . Borderline personality disorder

The type every examiner returns to. Of the ten categorical types, borderline personality disorder (BPD) is the single highest-yield entity of the chapter: it carries as many criteria as any type, the most differentials, and the only personality-disorder treatment literature dense enough to examine on its own. The whole picture reduces to one **pervasive pattern of instability**, of interpersonal relationships, of self-image and of affect, welded to marked impulsivity, beginning by early adulthood and present across a variety of contexts. Learn it as a polythetic set of nine features from which **five or more of the nine criteria** must be present, and you can both make the diagnosis and defend it against the two presentations that most often masquerade as it, bipolar II disorder and complex post-traumatic stress disorder.

This fragment walks the nine criteria one by one, spells the threshold, teaches the **course** and the **complications**, and closes on the ICD-11 borderline pattern qualifier as the dimensional equivalent of the same construct. The **affective instability** boundary with the bipolar-II side is cross-referenced to the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes; the trauma boundary to the Anxiety, OCD and trauma-related disorders notes; and the depth of the disorder-specific psychotherapies (dialectical behaviour therapy, schema therapy, mentalisation-based treatment) to their own topics in this chapter. Everything here sits downstream of the general personality-disorder criteria, which must be cleared before the count begins.

6.1 The essential feature: instability across three domains plus impulsivity

Instability is the signature, in relationships, in the self and in affect. The essential feature of borderline personality disorder is a pervasive pattern of instability of interpersonal relationships, self-image and affects, together with marked **impulsivity**, that begins by early adulthood and is present in a variety of contexts (DSM-5-TR). Three domains are unstable, relationships, self and mood, and a fourth behavioural strand, impulsivity, cuts across all of them; the nine diagnostic criteria are simply these four strands unpacked. Holding the four-strand skeleton first is what stops BPD collapsing into a list to be memorised blind: abandonment and idealisation-devaluation are the *relationship* strand; identity disturbance and chronic emptiness are the *self* strand; reactive mood and inappropriate anger are the *affect* strand; and self-damaging impulsivity, recurrent self-harm and transient stress-related paranoia or dissociation are the *behaviour-under-load* strand. The instability is not situational turbulence but a lifelong, ego-syntonic pattern that the general criteria have already certified as enduring, pervasive and inflexible.

6.2 The nine criteria in full

Reproduce all nine; the count depends on it. The DSM-5-TR criteria (F60.3) are the cleanest form of the construct, and the ICD-11 borderline pattern list mirrors them almost word for word. Carry the block as one table, because a defensible diagnosis names *which* criteria are met, not merely that the patient "seems borderline".

NO.	CRITERION	WHAT IT REQUIRES
1	Abandonment	Frantic efforts to avoid real or imagined **abandonment** (self-harm and suicidal acts are counted separately, under criterion five)
2	Unstable relationships	A pattern of unstable and intense interpersonal relationships alternating between extremes of idealisation and devaluation
3	Identity disturbance	**Identity disturbance**: a markedly and persistently unstable self-image or sense of self
4	Impulsivity	Impulsivity in at least two areas that are potentially self-damaging (spending, sex, substance use, reckless driving, binge eating)
5	Self-harm / suicidality	Recurrent suicidal behaviour, gestures or threats, or self-mutilating behaviour
6	Affective instability	**Affective instability due to a marked reactivity of mood (episodic dysphoria, irritability or anxiety usually lasting a few hours and only rarely more than a few days)**
7	Chronic emptiness	Chronic feelings of emptiness
8	Inappropriate anger	Inappropriate, intense anger or difficulty controlling anger (frequent temper, constant anger, recurrent physical fights)
9	Paranoia / dissociation	Transient, stress-related paranoid ideation or severe dissociative symptoms

The nine DSM-5-TR borderline personality disorder criteria; the highlighted reactivity clause of criterion six is the pivot on which the bipolar-II discrimination turns. The ICD-11 borderline pattern list is materially identical.

6.3 The threshold, and why the disorder is heterogeneous

Five or more of the nine, in any combination. Borderline personality disorder is diagnosed when a pervasive pattern of instability and impulsivity is present, as shown by **five or more of**

the nine criteria. Because any five will do, the set is polythetic: two patients can both carry the diagnosis while sharing only one criterion between them, which is why the presentation ranges so widely, from the quietly empty and self-harming to the floridly angry and quasi-psychotic. The count is not the first step, though; the general personality-disorder criteria (enduring, pervasive and inflexible, onset by adolescence, not better explained by another disorder or a substance) are cleared first, and only then are the nine tallied. This is also why heterogeneity is examinable: naming the five you have counted forces the discipline that a pattern-matched label lacks.

MNEMONIC

AM SUICIDE

The nine criteria, one letter each: **A**bandonment fears; **M**ood instability (reactive affect); **S**uicidal or self-mutilating behaviour; **U**nstable, intense relationships (idealisation to devaluation); **I**mpulsivity in two or more self-damaging areas; **C**ontrol of anger poor (inappropriate intense anger); **I**dentity disturbance; **D**issociation or paranoia that is transient and stress-related; **E**mptiness (chronic). Nine letters, nine criteria, threshold five.

9	5	2
BORDERLINE CRITERIA (DSM-5-TR)	MINIMUM MET, OF THE NINE, TO DIAGNOSE	SELF-DAMAGING AREAS FOR THE IMPULSIVITY CRITERION

WORKED EXAMPLE

A woman in her early twenties presents after an overdose following a break-up. On history she describes terror of being left (one), relationships that swing from "he is perfect" to "he is cruel" within days (two), never knowing who she really is (three), recurrent cutting and this overdose (five), moods that flip within an afternoon in response to a text going unanswered (six), and a background sense of emptiness (seven). That is five of the nine met with room to spare, on an enduring pattern predating the current crisis. The diagnosis is defensible not because she "looks borderline" but because you can name the criteria and show they are trait, not a fresh state.

6.4 Abandonment, and the idealisation-devaluation of relationships

The relationship strand is driven by intolerance of aloneness. The frantic efforts to avoid real or imagined **abandonment** (criterion one) are cued by the mere perception of impending separation or the loss of external structure, so that a clinician ending a session on time or arriving a few minutes late can trigger panic or fury out of all proportion. The same sensitivity produces the unstable, intense relationships of criterion two: the patient idealises a new caregiver or lover at the first meeting, demands closeness, then switches abruptly to devaluation when the other is felt not to give or care enough. These sudden reversals in the view of another person, from all-good to all-bad, are the surface of the classical defence of splitting, in which contradictory images of self and other cannot be held together, so they alternate instead.

PEARL

Splitting is the mechanism the object-relations tradition (Otto Kernberg) places at the centre of the borderline structure: the inability to integrate good and bad representations of the same person, so that idealisation and devaluation swing back and forth rather than blending into a whole, ambivalent view. Naming splitting explains criterion two and the "you are the only one who understands me / you are just like all the others" whiplash that clinicians feel in the room.

6.5 Identity disturbance and chronic emptiness

The self strand is a self that will not hold still. Identity disturbance (criterion three) is a markedly and persistently unstable self-image or sense of self: values, goals, career direction, sexual identity and even the felt sense of existing can shift suddenly, and the person may report at times a feeling that they do not exist at all. It is worst in unstructured settings, where there is no external scaffold to borrow an identity from, which is why performance can collapse in open-ended work or study while holding up under close direction. Running underneath it is criterion seven, chronic feelings of emptiness, a persistent hollowness distinct from the sadness of depression: not "I feel low" but "there is nothing inside". The two together are the self-strand that the disorder-specific psychotherapies target most directly, and they are also the features most easily overlooked when attention fixes on the dramatic behaviours.

6.6 Affective instability and inappropriate anger, and the bipolar-II line

The affect is reactive, fast and interpersonally cued. Affective instability (criterion six) is defined by a marked reactivity of mood: episodic dysphoria, irritability or anxiety that is triggered, most often by an interpersonal event, and that usually lasts a few hours and only rarely more than a few days. Criterion eight, inappropriate and intense anger, belongs to the same strand: disproportionate temper, sustained anger, sometimes physical fights, frequently aimed at the very person whose closeness is most wanted. This is the criterion pair that collides with bipolar II disorder, and the collision is the highest-yield discrimination on the topic. Bipolar mood runs as sustained, autonomous episodes lasting days to weeks, carrying change in sleep, energy and goal-directed activity; borderline mood is reactive, brief and settles when the trigger resolves. The full grid sits on the bipolar-II topic; here, hold the discriminator.

RULE **Read the clock and the trigger on the mood.** Borderline affective instability is reactive, interpersonally cued and settles in hours; a mood that runs autonomously for days to weeks with change in sleep, energy and goal-direction is a bipolar episode, not a personality trait.

TRAP **Starting a mood stabiliser for personality-based instability.** Missing the borderline-versus-bipolar-II distinction and reading reactive, hours-long lability as bipolar II disorder medicalises a pattern whose primary treatment is structured psychotherapy; the drug then follows the misdiagnosis rather than the illness.

6.7 Impulsivity, recurrent self-harm and suicidality

The behaviour-under-load strand is where the risk lives. Criterion four requires **impulsivity** in at least two potentially self-damaging areas, spending, sex, substance use, reckless driving or

binge eating, and it is counted separately from the self-directed acts. Those acts are criterion five: **recurrent self-harm** and **suicidality**, which is to say recurrent suicidal behaviour, gestures or threats, or self-mutilating behaviour. Self-harm in borderline personality disorder frequently functions to regulate unbearable affect or to end dissociation rather than to end life, but the two intentions coexist and the mortality is real, so the presence of a regulatory function never downgrades the risk assessment. Chronic recurrent self-harm and acute suicidality must be held apart in the moment: the enduring pattern is expected, while a change from baseline, a new plan or a fresh stressor, is what escalates management.

6.8 Transient stress-related paranoia and dissociation

The quasi-psychotic criterion is transient and stress-bound. Criterion nine is **transient stress-related paranoia** (paranoid ideation) or severe **dissociation**, arising in states of high affective arousal, most often under the threat of abandonment, and resolving as the arousal subsides. The qualifiers are load-bearing: *transient* and *stress-related* separate these micro-psychotic experiences from the sustained, autonomous psychosis of a schizophrenia-spectrum disorder, and from the enduring cognitive-perceptual oddness of schizotypal personality. They are brief, reactive, and the patient usually retains some distance from them once calm. This criterion is also the one that overlaps most with the dissociative and hyperarousal features of complex post-traumatic stress disorder, which is the second great mimic of the syndrome.

> **COMMON TRAP**
>
> Collapsing complex post-traumatic stress disorder into borderline personality disorder. Both follow early adversity and share affect dysregulation, dissociation and unstable relationships, but complex post-traumatic stress disorder is built on the trauma triad (re-experiencing, avoidance, hyperarousal) plus disturbances in self-organisation, and its instability is trauma-cued rather than the enduring, abandonment-driven identity pathology of borderline personality disorder. The boundary and its therapeutic consequences are developed in the Anxiety, OCD and trauma-related disorders notes.

6.9 The course: waxing and waning, remission that outpaces recovery

The symptoms remit long before function returns. The **course** of borderline personality disorder is a waxing and waning one that trends, over years, toward **symptomatic remission**: in long-term prospective follow-up (the McLean Study of Adult Development led by Zanarini, and the Collaborative Longitudinal Personality Disorders Study), the majority of patients no longer meet full criteria after several years, and the acute, impulsive features, self-harm, quasi-psychotic episodes, stormy relationships, tend to attenuate earliest. The catch is that *functional* recovery, stable work and durable relationships, lags well behind symptomatic remission; a patient can stop meeting criteria yet remain vocationally and interpersonally disabled. This dissociation between symptom remission and functional recovery is the examinable heart of the prognosis, and it reframes the disorder as more remitting than its acute presentation suggests, while cautioning against calling a symptomatic remission a cure.

6.10 The complications, and the management orientation

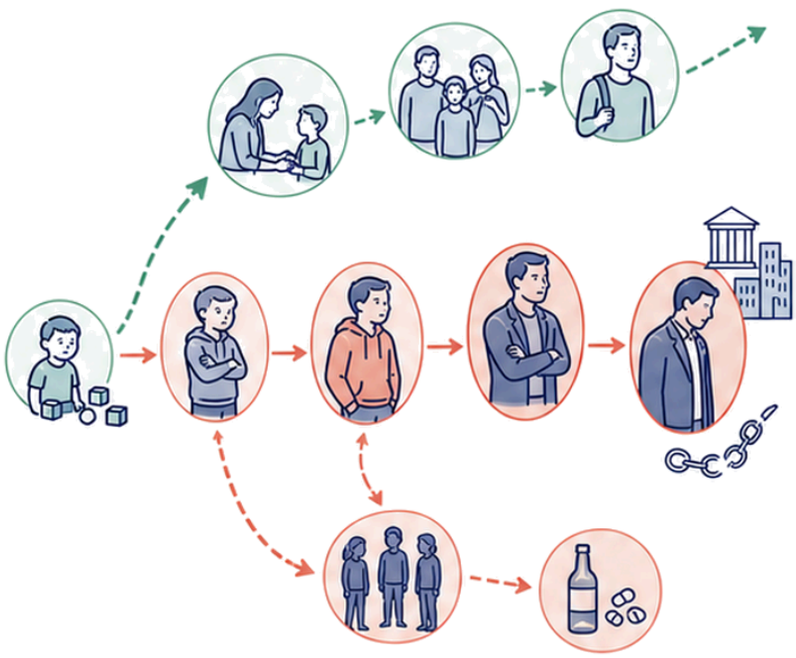
The morbidity clusters in four places. The **complications** of borderline personality disorder are, first, repeated self-harm; second, completed suicide, whose lifetime rate is substantial and highest in the early acute years; third, comorbid substance use, mood and eating disorders that both worsen impulsivity and confound the diagnosis; and fourth, the corrosive impact on relationships and on work, which is the burden that outlasts the symptoms. The management orientation follows directly from the course: structured psychological therapy is the primary treatment, the disorder-specific models (dialectical behaviour therapy, schema therapy, mentalisation-based treatment) are taught on their own topics, and pharmacotherapy is symptom-targeted, adjunctive and time-limited rather than a treatment for the disorder itself; the deep drug detail sits in the Mood disorders: pharmacotherapy notes. Crucially, a diagnosis of borderline personality disorder is never grounds to exclude a patient from care.

INDIA

Borderline personality disorder is markedly under-recognised in Indian practice. Patients reach services through the self-harm or the overdose, which is coded as the attempt rather than the pattern, so the underlying disorder is captured only when it is deliberately sought. Two adaptations matter. First, cultural expression: transient dissociative and possession-trance presentations are normative idioms of distress in many Indian settings and must not be over-read as the criterion-nine dissociation, just as family interdependence must not be read as pathological abandonment fear; judge every criterion against the person's own culture. Second, access: dialectical behaviour therapy and schema therapy are being delivered and culturally adapted at Indian tertiary and private centres rather than being widely available, which is what a realistic management plan must reckon with.

6.11 The ICD-11 borderline pattern qualifier: the dimensional equivalent

The same construct, carried forward into the dimensional model. ICD-11 abolished the categorical types, but it did not abolish borderline; it preserved the construct as an optional *borderline pattern* specifier (6D11.5), applied on top of a rated severity level and the trait-domain qualifiers. A complete ICD-11 formulation reads, for example, "moderate personality disorder with negative affectivity, dissociality and disinhibition, borderline pattern": severity first, trait domains next, borderline pattern last. The specifier is defined by the same list of features, a pervasive pattern of instability of relationships, self-image and affects with marked impulsivity, and the CDDR applies the *same* formal threshold as DSM-5-TR, five or more of nine features, so the count is not a DSM-only device; it overlaps most with the negative affectivity, dissociality and disinhibition domains. It was retained for a frankly pragmatic reason, which examiners like: to flag the patients who respond to the established borderline-specific psychotherapies. The categorical name therefore survives inside the dimensional system, which is why both are testable.



Early conduct problems
Deceit or aggression
Intervention and resilience
Persistent rule violation
Adult consequences

Fig 16.7 Antisocial personality disorder is developmentally anchored in earlier conduct problems, but progression is not inevitable; social context, comorbidity, intervention and resilience shape the adult pathway.

GOOD TO REMEMBER

- **Borderline personality disorder:** defining requirement is a pervasive pattern of instability of relationships, self-image and affect with marked impulsivity, meeting five or more of the nine criteria (abandonment, unstable idealising-devaluing relationships, identity disturbance, self-damaging impulsivity in at least two areas, recurrent self-harm or suicidality, reactive affective instability, chronic emptiness, inappropriate anger, transient stress-related paranoia or dissociation). Discriminator: the affect is rapidly reactive and interpersonally triggered and lasts hours, versus the sustained autonomous episodes of bipolar II disorder. First diagnostic step: clear the general personality-disorder criteria, then count the nine. ICD-11 keeps the construct as the optional borderline pattern qualifier on the dimensional model.

HOLD THIS

Borderline personality disorder is five or more of the nine, on a pervasive base of unstable relationships, unstable identity and reactive affect with impulsivity; the affect swings in hours and tracks interpersonal triggers, which is exactly what separates it from the sustained, autonomous episodes of bipolar II disorder.

7 . Antisocial personality disorder and forensic implications

The type the courts ask about. antisocial personality disorder (**aspd**; dissocial personality disorder in the older ICD terms) is the Cluster B type defined by a pervasive **disregard for and violation of the rights of others**, and it is the one personality disorder the examination reliably pushes into the medico-legal arena. Two features make it examinable beyond the criteria list. First, it is the only personality disorder with a hard developmental gate: it may not be diagnosed without evidence of conduct disorder before adolescence and it may not be diagnosed before adulthood, so the **age criterion** is part of the definition rather than a footnote. Second, it carries the **forensic implications** of the chapter, offending, the assessment of dangerousness, and the court report, and it forces the candidate to hold apart three things that are constantly conflated: the disorder, the construct of psychopathy, and criminality itself.

This fragment states the essential feature and walks the seven adult criteria, fixes the conduct-disorder precursor and the two ages, separates **aspd** from psychopathy and its **pcl-r** construct, cross-maps the categorical name onto the ICD-11 dimensional model, and then develops the two forensic tasks the psychiatrist is actually asked to perform, the structured assessment of dangerousness and the court report. The deep offender-management and legislative detail is carried in the Mental health legislation and forensic psychiatry notes; the general psychotherapy technique in the Psychotherapies, clinical assessment, MSE and nosology notes; and any symptom-targeted pharmacology in the Mood disorders: pharmacotherapy notes and the Schizophrenia: management, antipsychotics and adverse effects notes. Everything here sits downstream of the general personality-disorder criteria, which must be cleared before the count begins.

7.1 The essential feature: a pervasive disregard for the rights of others

The pattern violates the rights of others, and it began in childhood. The essential feature of antisocial personality disorder is a pervasive pattern of disregard for, and violation of, the rights of others, occurring since the age of fifteen years and continuing into adulthood (DSM-5-TR, F60.2). The manual notes in the same breath that this pattern has also been called **psychopathy**, sociopathy or dyssocial personality disorder, which is precisely the overlap the topic exists to disentangle. The behaviour is not a run of bad choices but an enduring, ego-syntonic trait structure: callous unconcern for others' feelings, a readiness to deceive and exploit, impulsive action without forethought, irritable aggression, reckless indifference to safety, chronic irresponsibility, and an absence of guilt. Because deceit and manipulation are central to the presentation, DSM-5-TR advises explicitly that the clinical account be corroborated from collateral and documentary sources rather than taken on the patient's word, a caution that matters even more when the assessment is destined for a court.

7.2 The seven adult criteria, and the three-of-seven threshold

Three or more of the seven, on the enduring pattern. Criterion A is met by **three or more of the seven** features below, present since the age of fifteen years (DSM-5-TR). Carry the block as one table, because a defensible antisocial diagnosis names which features are met, and the

affective item, **lack of remorse**, is the one that later separates the disorder from the narrower construct of psychopathy.

NO.	CRITERION (DSM-5-TR)	WHAT IT REQUIRES
1	Failure to conform to law	Failure to conform to social norms with respect to lawful behaviour, repeatedly performing acts that are grounds for arrest (whether or not arrest follows)
2	Deceitfulness	**deceitfulness**: repeated lying, use of aliases, or conning others for personal profit or pleasure
3	Impulsivity	Impulsivity or failure to plan ahead; decisions made on the spur of the moment without regard for consequences
4	Irritability and aggressiveness	Irritability and aggressiveness, indicated by repeated physical fights or assaults (acts in self-defence do not count)
5	Reckless disregard for safety	Reckless disregard for the safety of self or others (dangerous driving, high-risk behaviour, endangering a dependent)
6	Consistent irresponsibility	Consistent **irresponsibility**: repeated failure to sustain work or to honour financial obligations
7	Lack of remorse	**lack of remorse: being indifferent to, or rationalising, having hurt, mistreated or stolen from another**

The seven DSM-5-TR antisocial personality disorder criteria, of which three or more must be present; the highlighted affective item, lack of remorse, is the feature that most nearly touches the psychopathy construct and is the least likely to be present in ordinary offending.

MNEMONIC

CORRUPT

The seven criteria, one letter each. **C**onformity to law lacking (acts that are grounds for arrest); **O**bligations ignored (consistent irresponsibility, work and money); **R**eckless disregard for safety; **R**emorselessness (lack of remorse); **U**nderhandedness (deceitfulness, lying and conning); **P**lanning absent (impulsivity, failure to plan ahead); **T**emper (irritability and aggressiveness, repeated fights). Seven letters, seven criteria, threshold three.

Two exclusions close the definition. Two further clauses complete Criterion A's scaffolding. The individual must be at least eighteen years old (Criterion B) and must have shown evidence of conduct disorder with onset before fifteen years (Criterion C); and the antisocial behaviour must

not occur exclusively during the course of schizophrenia or a bipolar disorder (Criterion D), so that antisociality driven by mania or by psychosis is excluded from the personality diagnosis.

GOOD TO REMEMBER

- **Antisocial personality disorder:** defining requirement is a pervasive pattern of disregard for and violation of the rights of others since age fifteen, meeting three or more of the seven criteria (failure to conform to law, deceitfulness, impulsivity, irritability and aggressiveness, reckless disregard for safety, consistent irresponsibility, lack of remorse), with the individual at least eighteen and with documented conduct disorder before fifteen, and the behaviour not confined to schizophrenia or bipolar disorder. Discriminator: it is a trait pattern with a childhood-conduct-disorder precursor, not a synonym for a criminal record. First diagnostic step: clear the general personality-disorder criteria, confirm the conduct-disorder history and the age, then count the seven.

7.3 The conduct-disorder precursor and the age criterion

The developmental spine: oppositional to conduct to antisocial. The **age criterion** is what makes this a developmental diagnosis. The disorder cannot be given before eighteen years, and it cannot be given at all without evidence of conduct disorder with onset before fifteen years (DSM-5-TR). Conduct disorder is itself a repetitive, persistent violation of the basic rights of others or of major age-appropriate norms, and its behaviours fall into four groups, aggression to people and animals, destruction of property, deceitfulness or theft, and serious violation of rules; oppositional defiant disorder is a frequent earlier precursor of the childhood-onset type. The line runs, in the minority who progress, from oppositional through conduct disorder into the adult antisocial pattern, and the childhood-onset conduct disorder carries the worse prognosis. Most children with conduct disorder do not become antisocial adults, but essentially every antisocial adult has a conduct-disordered childhood, which is exactly why the history is a mandatory gate and not an optional descriptor. The developmental trajectory is laid out in the accompanying figure.

RULE **No conduct disorder before fifteen, no antisocial personality disorder.** Evidence of childhood conduct disorder and an age of at least eighteen are hard gates in the criteria; the antisocial diagnosis is a developmental one and cannot be built from an adult offending history alone.

TRAP **Reading a criminal record as antisocial personality disorder.** Offending, gang membership or survival-driven rule-breaking is not the disorder without the enduring, pervasive trait pattern and the conduct-disorder precursor; and antisociality seen only during mania or psychosis is excluded by Criterion D. Crime is neither necessary nor sufficient for the diagnosis.

7.4 Antisocial personality disorder versus psychopathy: the affective-interpersonal core and the PCL-R

Psychopathy is the narrower, affect-defined construct. psychopathy overlaps with antisocial personality disorder but is not the same thing, and treating them as interchangeable is the classic error. The categorical diagnosis is defined largely by observable antisocial *behaviour*; psychopathy is defined by an affective-interpersonal *core*, the glib superficial charm, grandiosity, pathological lying, callousness and, above all, the absence of guilt, remorse or empathy that Hervey Cleckley

described in *The Mask of Sanity* (1941). The construct has been dropped from the formal classifications but survives in forensic psychology and the prison service, where it is assessed with the Hare Psychopathy Checklist-Revised (the **pcl-r**; Robert Hare, 1991), a rater-scored instrument of twenty items, each scored zero to two, to a maximum of forty. The items load onto two factors: an interpersonal-affective factor (the charm, callousness and exploitation, the true psychopathic core) and a lifestyle-antisocial factor (the impulsive, irresponsible and violent behaviour that overlaps heavily with the antisocial criteria). Most people who meet the antisocial criteria do *not* reach a high psychopathy score; psychopathy is the smaller, affect-defined subset carrying the interpersonal-affective factor, and it is that factor, not the criminal record, that predicts the most instrumental and remorseless offending. The overlap and the nesting are shown in the figure alongside.

PEARL

Karpman, paralleling Cleckley's clinical description, distinguished *primary* from *secondary* psychopathy: the primary psychopath acts without anxiety, guilt or autonomic arousal, the mark of the affective deficit, while the secondary psychopath reports guilt and fear but offends through poor impulse control and emotional lability. The distinction maps loosely onto the two PCL-R factors, and it is why "psychopathy" is best reserved for the affective-interpersonal core and, as the general-psychiatry texts advise, largely avoided as a loose synonym for antisocial personality disorder.

GOOD TO REMEMBER

- **Psychopathy and the PCL-R:** the defining feature is the affective-interpersonal core, callousness and the absence of guilt, remorse and empathy (Cleckley), which the behaviour-based antisocial criteria do not require; the instrument is the Hare Psychopathy Checklist-Revised, twenty items scored zero to two to a maximum of forty, with an interpersonal-affective factor and a lifestyle-antisocial factor. Discriminator from antisocial personality disorder: psychopathy is the smaller, affect-defined subset, so most antisocial patients are not psychopathic; the two terms are not interchangeable, and psychopathy is not a diagnostic category in DSM-5-TR or ICD-11.

7.5 The classification frame: DSM-5-TR categorical, ICD-10 dissocial, ICD-11 dissociality

The same construct across three systems. In DSM-5-TR Section II the disorder is the antisocial type in Cluster B (the dramatic, emotional or erratic cluster). ICD-10 kept an almost identical categorical entity under the name dissocial personality disorder, requiring at least three of six traits, callous unconcern for others' feelings, gross and persistent irresponsibility and disregard for social norms, incapacity to maintain enduring relationships, very low frustration tolerance with a low threshold for aggression, incapacity to experience guilt or to profit from experience or punishment, and a marked proneness to blame others, with a childhood conduct-disorder history offered as supporting evidence. ICD-11 then abolished the categorical types altogether: there is now a single personality-disorder diagnosis rated first by severity (personality difficulty, then mild, moderate or severe) and qualified by the five trait domains. The antisocial construct is captured dimensionally by prominent **dissociality** (the ICD-11 domain 6D11.2, whose core feature is a disregard for the rights and feelings of others, self-centredness and lack of empathy),

usually together with **disinhibition** (impulsivity, irresponsibility, recklessness), at a moderate or severe level. The forward teaching of the ICD-11 dimensional model, its severity gradient and its five domains, is developed on the framework topics; here, hold the cross-map. The categorical names still dominate the examination and the courtroom because DSM-5-TR retains them and the case-law is written in them, which is why "antisocial" and "dissocial" remain testable even after ICD-11 has moved on.

7.6 Forensic implications: offending and the assessment of dangerousness

The task is a structured, defensible estimate of future risk. The **forensic implications** begin with offending: antisocial personality disorder is far commoner in men and is markedly over-represented in prison populations, and it frequently co-occurs with substance misuse, which amplifies impulsive aggression. The recurring clinical demand is the **assessment of dangerousness**, an estimate of the risk of future violence. Three approaches exist, and the examinable point is which is preferred. Unstructured clinical judgement is variable and biased; purely actuarial scoring is reliable but ignores the individual's own risk and protective factors; the recommended middle path is **structured professional judgement**, in which the clinician rates empirically derived risk factors systematically and then integrates them with the individual case (Clinical Methods in Psychiatry, AIIMS 2024). The most researched such instrument is the HCR-20, a twenty-item scheme across three scales, ten Historical (static) factors including past violence, young age at first violence, psychopathy and personality disorder, five Clinical (dynamic) factors, and five Risk-Management (dynamic) factors; each item is scored absent, partial or present, and the clinician then makes a summary rating of low, moderate or high, which is deliberately *not* a mechanical sum of ticks. A high PCL-R psychopathy score is itself one of the strongest single predictors of violent recidivism, which is why the two instruments are used together. Whenever risk is judged to be directed at an identifiable person, the duty to protect can override confidentiality. The three approaches are contrasted in the accompanying figure.

RULE **Structure the risk estimate, then judge it; never tick and total.** Dangerousness is assessed by structured professional judgement, empirically derived factors rated systematically and then weighed for the individual, ending in a reasoned low, moderate or high rating, not by the raw count on any checklist.

7.7 Forensic implications: court reports and the medico-legal frame

Antisocial personality disorder is a matter of disposal, not of exculpation. The second forensic task is the court report, and its central caution is that the antisocial diagnosis rarely excuses a crime. The insanity defence turns on a defect of reason from unsoundness of mind such that the accused did not know the nature of the act or that it was wrong, and the courts distinguish this *legal* insanity sharply from any *medical* diagnosis; a personality disorder, being a stable trait pattern rather than a disorder that clouds the knowledge of nature or wrongfulness, seldom meets that test. Psychiatric evidence is treated as relevant, not determinative. So the forensic weight of antisocial personality disorder falls on the assessment of dangerousness, on fitness to stand trial, and on disposal and sentencing, and on the psychiatric prediction of future dangerousness that the law can demand before an insanity-acquitted person is discharged, rather

than on culpability itself. In the report the clinician is an expert assisting the court: the opinion must be evidence-based, non-pejorative, carefully recorded (what is not written is treated as not done), and explicit about the limits of prediction. This medico-legal frame, the statutes, the fitness assessment, the disposal pathways and the report structure, is developed in the Mental health legislation and forensic psychiatry notes.

Criminal responsibility (insanity defence)
Whether unsoundness of mind prevented the accused knowing the nature of the act or that it was wrong; a legal test, judged against medical evidence, that antisocial personality disorder rarely satisfies.

Fitness to stand trial
Whether the accused can understand the charge and proceedings and instruct a defence; assessed before culpability is considered.

Assessment of dangerousness
A structured, professionally judged estimate of the risk of future violence, informing bail, disposal, discharge and management, not guilt.

The expert witness / court report
The psychiatrist assists the court with an impartial, evidence-based, well-documented opinion; relevant to the court's decision but never determinative of it.

The four medico-legal points where antisocial personality disorder meets the court; note that all four concern process, risk and disposal, not the excusing of the offence, whose statutory detail sits in the Mental health legislation and forensic psychiatry notes.

WORKED EXAMPLE

A man in his thirties is remanded after a violent robbery and referred for a court report and a "diagnosis of psychopathy". The history shows truanting, cruelty and theft from before the age of twelve, expulsions, repeated adult convictions, a chaotic work record, unpaid debts, and a flat indifference to the people he has hurt. He meets the antisocial criteria comfortably, with conduct disorder well before fifteen. The report says three things and no more than it can defend: the diagnosis is antisocial personality disorder (not, on this assessment, formally psychopathy, which would need a rated PCL-R); the disorder does not amount to the unsoundness of mind that founds an insanity defence, so it does not reduce his responsibility for the act; and his risk of future violence, assessed by structured professional judgement, is high and should guide disposal. The value of the report lies in what it declines to claim as much as in what it asserts.

RULE **Antisocial personality disorder is not an insanity defence.** As a stable trait pattern it seldom meets the legal test of unsoundness of mind; its forensic relevance is to dangerousness, fitness and disposal, not to exculpation, and the psychiatric opinion is relevant to the court, never determinative.

7.8 Management: guideline-anchored, psychological-first, and drug-sparing

Structured psychological work leads; no drug treats the personality. Management is modest and guideline-anchored, and the deep detail is cross-referenced. NICE recommends offering people with antisocial personality disorder group-based cognitive and behavioural interventions, involving the person in choosing their duration and intensity, and delivered where relevant in

criminal-justice as well as health settings, and it stresses a structured clinical assessment to make the diagnosis in the first place (NICE 2015). No drug is recommended or licensed for the antisocial personality itself; pharmacotherapy is reserved for a comorbid mental disorder, and where impulsive aggression is a treatment target it is addressed adjunctively and off-licence, for example with a mood stabiliser such as divalproex (Valprol, Encorate) or a second-generation antipsychotic, with the dosing and adverse-effect detail carried in the Mood disorders: pharmacotherapy notes and the Schizophrenia: management, antipsychotics and adverse effects notes rather than re-taught here. Engagement is the perennial obstacle: the condition is ego-syntonic, motivation is external, and manipulation of the therapeutic frame is expected, so treatment leans on structure, clear limits and realistic goals. Mentalisation-based treatment, adapted for antisocial personality disorder (Bateman and Fonagy), is an emerging structured option under trial; its general technique sits in the Psychotherapies, clinical assessment, MSE and nosology notes.

GOOD TO REMEMBER

- **Group cognitive-behavioural therapy for antisocial personality disorder:** mechanism is a structured, skills-based group intervention targeting the cognitions and behaviours that drive offending, delivered with firm limits; indication is antisocial personality disorder, especially with an offending history, and it is the NICE-recommended mainstay (NICE 2015), the person sharing the choice of duration and intensity; signature caution is that no drug is recommended for the personality itself, that medication is only for a comorbid disorder or, adjunctively and off-licence, for impulsive aggression, and that poor engagement and manipulation of the frame are the rule, not the exception.

INDIA

Three genuine India angles converge on antisocial personality disorder. First, under-recognition: personality disorders are markedly under-recognised in routine Indian practice, and the antisocial pattern in particular surfaces through its comorbidities, a substance-use disorder, repeated violence, or a legal referral, rather than as a personality diagnosis, so it is captured only when actively sought and a corroborated developmental history is taken. Second, the medico-legal frame is where the label carries the most force in India: the insanity defence rests on the unsoundness-of-mind test derived into the Indian Penal Code, which antisocial personality disorder rarely satisfies, so the psychiatrist's work is court reports, fitness assessment and the prediction of dangerousness rather than exculpation, and the dedicated forensic and secure-service infrastructure to support it is thin, developed in the Mental health legislation and forensic psychiatry notes. Third, the cultural expression of personality pathology: deviation must be judged against the person's own cultural, social and economic milieu, so that survival-driven or subcultural offending, or antisociality confined to a substance-use context, is not scored as trait pathology, and the diagnosis is not read through a single culture's norms. Structured group and mentalisation-based programmes for the disorder are scarcely available outside a few tertiary and forensic centres.

COMMON TRAP

Collapsing psychopathy into antisocial personality disorder and treating the two as one label. The antisocial criteria are behaviour-based and are met by a large, heterogeneous group, most of whom are not psychopathic; psychopathy is the narrower affective-interpersonal construct measured by the PCL-R, and it, not the criminal record, drives the most instrumental and remorseless offending. Writing "psychopath" where the assessment supports only antisocial personality disorder, or vice versa, is a straight error, and in a court report it is a consequential one.

3	15	18	20
OF THE SEVEN CRITERIA TO DIAGNOSE ANTISOCIAL PERSONALITY DISORDER	AGE (YEARS) BEFORE WHICH CONDUCT DISORDER MUST BE EVIDENT	MINIMUM AGE (YEARS) FOR THE ANTISOCIAL DIAGNOSIS	ITEMS ON THE HARE PSYCHOPATHY CHECKLIST-REVISED (PCL-R)

HOLD THIS

Antisocial personality disorder is a pervasive disregard for the rights of others, three or more of the seven criteria (failure to conform to law, deceitfulness, impulsivity, irritability and aggressiveness, reckless disregard for safety, consistent irresponsibility, lack of remorse), gated by conduct disorder before fifteen and an age of at least eighteen; it is not a synonym for psychopathy (the narrower affective-interpersonal PCL-R construct) nor for criminality, and its forensic weight is in the structured assessment of dangerousness and the court report, not in the insanity defence.

8 . Histrionic and narcissistic personality disorders

Why these two share a page. Both sit in **cluster b**, the dramatic, emotional or erratic group, and both are built around a hunger for the regard of others, so the examiner tests them together and rewards the candidate who can separate them cleanly. histrionic personality disorder is a pervasive pattern of excessive emotionality and **attention-seeking** (DSM-5-TR), and narcissistic personality disorder is a pervasive pattern of grandiosity, a **need for admiration** and a lack of empathy (DSM-5-TR). They look alike from a distance, two people who must be regarded, but the object of the wanting differs: the histrionic person needs to be the centre of attention and will take any role to get there, while the narcissistic person needs admiration for a superiority they believe is real.

This fragment frames the pair on one grid, takes each through its essential feature and criterion threshold, then isolates the single modern high-yield point, the split within narcissism between **grandiose narcissism** and **vulnerable narcissism**, the overt entitled presentation and the covert, hypersensitive, shame-prone one. Two cautions carry over and are assumed throughout: the general personality-disorder criteria (an enduring, pervasive, inflexible pattern, deviant from the person's own culture, with onset by adolescence or early adulthood, causing impairment, and not better explained otherwise) must be cleared before either type is named; and ICD-11 has abolished both as categories, so histrionic and narcissistic survive as labels only in DSM-5-TR and in clinical shorthand. The fully taught structured psychotherapies of this chapter and the general technique sit in the Psychotherapies, clinical assessment, MSE and nosology notes; the

borderline neighbour that both are most often confused with is developed elsewhere in this chapter.

8.1 The two on one frame

Both crave regard; the object of the craving divides them. Read the pair as a single question asked two ways. The histrionic patient organises a whole personality around being noticed, with emotion as the currency; the narcissistic patient organises it around being admired as superior, with a fragile self-esteem underneath. Learn the essential feature, the threshold and the one discriminator for each as a block, and most stems fall out of it.

TYPE	ESSENTIAL FEATURE (DSM-5-TR)	THRESHOLD	CORE DISCRIMINATOR
Histrionic	Pervasive and excessive emotionality and attention-seeking; discomfort when not the centre of attention	Five or more of the eight features	**Wants attention itself; will be fragile, seductive or dependent, whatever draws the eye**
Narcissistic	Grandiosity (in fantasy or behaviour), need for admiration, and lack of empathy	Five or more of the nine features	**Wants admiration for a believed superiority; entitlement and empathy failure, over a fragile self-esteem**

The two Cluster B attention-oriented types with their essential features, criterion thresholds and the single dividing discriminator; the accompanying figure sets attention-seeking against need for admiration on one axis.

> **PEARL**
>
> One question separates them at the bedside: *what does the attention buy?* For histrionic personality disorder attention is the goal in itself, and the person will happily be seen as fragile, ill or dependent if that is what turns heads. For narcissistic personality disorder attention must come as admiration of superiority; being pitied or seen as needy is intolerable, because it threatens the grandiose self.

8.2 Histrionic personality disorder

Emotion as performance, staged to be watched. The essential feature of histrionic personality disorder is a pervasive pattern of excessive emotionality and attention-seeking behaviour, beginning by early adulthood and present across contexts, shown by **five or more of the eight** features (DSM-5-TR). The eight are: discomfort in situations where they are not the centre of attention; interaction often marked by inappropriate sexually seductive or provocative behaviour; rapidly shifting and shallow expression of emotions; consistent use of physical appearance to draw attention to the self; a style of speech that is excessively impressionistic and lacking in detail; self-dramatisation, theatricality and exaggerated expression of emotion; suggestibility, being easily influenced by others or by circumstances; and considering relationships to be more intimate than they actually are. The signature is a person who is lively, charming and flirtatious on first meeting but whose emotions seem turned on and off too quickly to be deeply felt, whose strong opinions come with dramatic flair but no supporting detail, and

who becomes uncomfortable or aggrieved the moment the spotlight moves. Impairment tends to be milder than in the other Cluster B types and is chiefly interpersonal, with romantic and same-sex friendships strained by the demand for constant attention and the provocative style.

GOOD TO REMEMBER

- **Histrionic personality disorder:** defining criterion is pervasive excessive emotionality and attention-seeking, five or more of the eight features, from early adulthood, with discomfort when not the centre of attention as the anchoring item. Discriminator from narcissistic: it wants attention as such and will accept a fragile or dependent role to get it, where narcissistic wants admiration for superiority. Discriminator from borderline: no chronic emptiness, identity disturbance, self-destructiveness or angry abandonment ruptures. Discriminator from dependent: the flamboyant, theatrical, exaggerated emotionality that dependent lacks. First step: confirm the general personality-disorder criteria, then that the emotionality and attention-seeking are trait-level and pervasive, not a phase or a state.

8.3 Narcissistic personality disorder

Grandiosity above, fragility below. The essential feature of narcissistic personality disorder is a pervasive pattern of grandiosity in fantasy or behaviour, a need for admiration and a lack of empathy, beginning by early adulthood and present across contexts, shown by **five or more of the nine** features (DSM-5-TR). The nine are: a grandiose sense of self-importance; preoccupation with fantasies of unlimited success, power, brilliance, beauty or ideal love; a belief in being special and unique, understood only by, or fit to associate only with, other special or high-status people or institutions; a requirement for excessive admiration; a sense of entitlement, meaning unreasonable expectations of especially favourable treatment or automatic compliance; interpersonal exploitativeness, taking advantage of others to reach one's own ends; a lack of empathy, an unwillingness to recognise or identify with the feelings and needs of others; envy of others or a belief that others envy oneself; and arrogant, haughty behaviours or attitudes. The clinically important addition, spelled out in the DSM-5-TR text, is that the self-esteem beneath the grandiosity is almost always fragile: criticism or defeat can leave the person feeling ashamed, humiliated, hollow and empty, reacting with disdain, rage, or a withdrawal that masks the grandiosity rather than removing it. Empathy is typically cognitive but not emotional, they can read another's perspective yet do not feel with them.

GOOD TO REMEMBER

- **Narcissistic personality disorder:** defining criterion is grandiosity, a need for admiration and a lack of empathy, five or more of the nine features, from early adulthood. Discriminator from histrionic: admiration must be for a believed superiority, and a needy or fragile role is intolerable. Discriminator from antisocial: narcissistic lacks the impulsivity, aggression, deceit and childhood conduct-disorder history, and exploits to enhance the self rather than for material gain. Discriminator from borderline: relatively stable self-image and far less physical self-destructiveness and abandonment terror. First step: confirm the general criteria, then look past the grandiose surface for the fragile self-esteem that the presentation is defending.

8.4 Grandiose versus vulnerable narcissism

One disorder, two faces. The highest-yield modern point is that narcissism presents along two phenotypes rather than one. grandiose narcissism is the overt, textbook picture: openly entitled,

arrogant, exhibitionistic, admiration-demanding, thick-skinned. vulnerable narcissism is the covert picture: hypersensitive, shame-prone, anxiously self-conscious, socially withdrawn, quietly nursing the same grandiose fantasies but defending them behind apparent humility or complaint. DSM-5-TR anchors both in its own text and in its alternative model: it describes narcissistic self-esteem as "variable and vulnerable," regulated through approval-seeking, with grandiosity that may be "either overt or covert," and it records the more vulnerable presentations by adding negative-affectivity traits such as depressivity and anxiousness. The two are not different disorders but different surfaces over a shared core of fragile self-worth; many patients oscillate between them, showing grandiosity when supplied with admiration and collapsing into the vulnerable state when it is withheld. The examiner's trap is to recognise only the loud, grandiose form and miss the quiet, shame-driven one, which is easily mistaken at face value for depression or social anxiety.

FACET	GRANDIOSE (OVERT)	VULNERABLE (COVERT)
Surface presentation	Entitled, arrogant, exhibitionistic, self-assured	**Hypersensitive, shy, self-effacing, complaining**
Self-esteem	Openly inflated, thick-skinned to criticism	Fragile, shame-prone, easily wounded
Response to a slight	Disdain, rage, counterattack	Withdrawal, humiliation, resentment, rumination
Grandiosity	Displayed openly in behaviour	Kept in private fantasy, masked by humility
Common misread	Simple arrogance or a difficult personality	**Depression or social-anxiety disorder**

Grandiose and vulnerable narcissism as two surfaces over one fragile core; the highlighted cells are the covert features most often missed and the diagnosis they are mistaken for.

WORKED EXAMPLE

A man in his thirties presents with low mood, social withdrawal and a sense of being chronically slighted. On the surface this reads as depression. Listening closer, the withdrawal follows a series of perceived humiliations, each one a failure of the world to recognise a talent he privately believes is exceptional; he is exquisitely sensitive to criticism, envious of colleagues he considers his inferiors, and empty when admiration is not forthcoming. There is no overt boastfulness. This is vulnerable narcissism, the covert face of narcissistic personality disorder, not a primary depressive disorder, and treating only the mood while missing the fragile grandiose core underneath is why the presentation keeps returning.

RULE **Attention itself versus admiration for superiority.** The histrionic person wants to be the centre of attention and will take any role, even a fragile or dependent one, to get it; the narcissistic person wants admiration specifically for a superiority they believe is real, and a needy role is intolerable. That is the dividing line.

TRAP **Seeing only the loud narcissist.** Expecting only the grandiose, arrogant presentation and missing vulnerable narcissism, whose covert shame, hypersensitivity and withdrawal are read at face value as depression or social anxiety, leaves the diagnosis and the fragile grandiose core underneath untouched.

8.5 Telling them apart from their neighbours

The confusions the paper actually sets. Both types share features with their Cluster B neighbours, and the marks are in the discriminators, drawn straight from the DSM-5-TR differential text. Hold the grid below and each boundary is a one-line answer.

BOUNDARY	SHARED FEATURE	DISCRIMINATOR
Histrionic vs narcissistic	Both crave attention from others	**Histrionic wants attention as such and accepts a fragile role; narcissistic wants praise for superiority and emphasises status**
Histrionic vs borderline	Attention-seeking, manipulative behaviour, rapidly shifting emotions	Borderline adds self-destructiveness, angry relationship ruptures, chronic emptiness and identity disturbance
Histrionic vs antisocial	Impulsive, superficial, seductive, manipulative	Histrionic manipulates for nurturance and is more exaggerated in emotion; antisocial manipulates for material gain
Histrionic vs dependent	Reliance on others for reassurance	Dependent lacks the flamboyant, exaggerated, theatrical emotionality of histrionic
Narcissistic vs antisocial	Exploitative, lacking empathy	Narcissistic lacks impulsivity, aggression, deceit and a conduct-disorder history; exploits to enhance the self, not for profit
Narcissistic vs borderline	Anger to minor slights	Narcissistic keeps a relatively stable self-image with little physical self-destructiveness or abandonment terror

The six boundaries most often tested around the two types; the highlighted cell is the single discriminator that decides histrionic from narcissistic. The borderline side is developed elsewhere in this chapter.

8.6 The classification shift: ICD-11 and the alternative model

Both categories are gone, but the vocabulary survives. This is where the day's headline nosology point lands on these two types. ICD-11 abolished the categorical personality-disorder types entirely: there is no ICD-11 code for histrionic or narcissistic personality disorder. A histrionic-like presentation is now captured by rating **severity** first (personality difficulty, then mild, moderate or severe) and adding the trait qualifier of negative affectivity for the dramatic, labile emotionality, with the attention-seeking element falling under dissociality. A narcissistic-like presentation is captured by severity plus dissociality, whose text explicitly names self-

centredness, a sense of entitlement, expectation of others' admiration and attention-seeking to remain the centre of focus, with the vulnerable, shame-prone features added under negative affectivity (ICD-11 CDDR). The DSM-5-TR alternative hybrid model treats the two unequally: narcissistic personality disorder is one of the six specific types it retains, defined by impairment plus the antagonism traits of grandiosity and attention-seeking, whereas histrionic is one of the four categorical types it drops, becoming personality disorder, trait specified when the general criteria are met without a named set.

ICD-10 TO ICD-11: WHAT CHANGED

ICD-10 (F60) kept histrionic personality disorder (F60.4) as a named categorical type, but narcissistic never had its own ICD-10 category at all, sitting only under "other specific personality disorder" (F60.8). ICD-11 then removed every categorical type, rebuilding the diagnosis as a severity level plus trait-domain qualifiers, so both histrionic and narcissistic now exist only as descriptive patterns within that dimensional frame. Exams still test the categorical names because DSM-5-TR retains them in its main text and clinicians still speak in them, so read fluently in both languages.

8.7 Management in outline, and the cultural reading

Psychotherapy is the frame; drugs are for comorbidity only. Neither type has a disorder-specific drug, and neither is a receptor problem to be medicated. The working approach is a structured, long-term psychotherapy, with the transference-focused and schema-based models the ones with the most traction for narcissistic pathology (their mechanism and indication are taught in full in their own topics of this chapter). The narcissistic patient tests the alliance hardest, because the very grandiosity that brings collapse also resists the therapist's help, and the vulnerable, shame-prone presentation must be handled without either colluding with the grandiosity or shaming the fragile self beneath it. Medication is reserved for a comorbid depressive or anxiety disorder, or a short crisis, never for the personality itself, and the deep pharmacology is cross-referenced to the Mood disorders: pharmacotherapy notes and the Schizophrenia: management, antipsychotics and adverse effects notes rather than re-taught here.

INDIA

Three genuine India angles converge here. First, under-recognition: personality disorders are diagnosed far less often in routine Indian practice than Western prevalence would predict, and these two surface through their comorbidities, a mood or anxiety disorder, a relational crisis, rather than as a personality label that clinicians often avoid for fear of stigma. Second, the cultural expression of pathology: the general criteria require deviation from the person's *own* culture, so narcissistic-looking traits inflate in individualistic settings and warrant less clinical weight where they are normative, while a warm, expressive, family-embedded emotional style must not be scored as histrionic through a colder cultural lens, and the historical over-diagnosis of histrionic personality disorder in women along gendered social scripts is a bias to guard against actively. Third, availability: the structured psychotherapies these patients need, transference-focused and schema therapy, are unevenly available and still being culturally adapted for India, concentrated at tertiary and private centres. The medico-legal weight of personality pathology attaches to antisocial personality disorder, handled in the Mental health legislation and forensic psychiatry notes, not to these two.

8.8 The countable skeleton

Two essential features, two thresholds, one split. Everything worth carrying into the hall about these two types hooks onto a handful of counts and one distinction. Anchor the mnemonics and each criterion set unpacks.

MNEMONIC

PRAISE ME · GRANDIOSE

PRAISE ME for the eight histrionic criteria: **P**rovocative or seductive; **R**elationships thought more intimate than they are; **A**ttention (uncomfortable when not its centre); **I**nfluenced easily (suggestible); **S**tyle of speech impressionistic and vague; **E**motions shallow and rapidly shifting; **M**ade-up appearance used to draw the eye; **E**xaggerated, theatrical emotional display.
GRANDIOSE for the nine narcissistic criteria: **G**randiose self-importance; **R**equires excessive admiration; **A**rrogant and haughty; **N**eeds to be seen as special and unique; **D**reams of unlimited success, power, brilliance, beauty or ideal love; **I**nterpersonally exploitative; **O**btuse to others' feelings (lacks empathy); **S**ense of entitlement; **E**nvious of others or believes others envy them.

8	9	4	5
HISTRIONIC CRITERIA; FIVE OR MORE NEEDED	NARCISSISTIC CRITERIA; FIVE OR MORE NEEDED	CLUSTER B DRAMATIC TYPES	ICD-11 TRAIT DOMAINS; BOTH TYPES ABOLISHED

HOLD THIS

Histrionic personality disorder is excessive emotionality and attention-seeking (five or more of the eight, uncomfortable when not the centre of attention); narcissistic personality disorder is grandiosity, a need for admiration and a lack of empathy (five or more of the nine); and grandiose and vulnerable narcissism are one disorder's two faces, the overt entitled and the covert shame-prone, over a shared core of fragile self-esteem.

9 . Cluster C: avoidant, dependent and anankastic

Why the anxious cluster earns its marks. **cluster c** is the **anxious or fearful** group, and it rewards the candidate who can give the one-line essential feature of each type cold and then defend the two boundaries an examiner reaches for first: avoidant personality disorder against a genuine loner and against social anxiety disorder, and anankastic personality disorder against obsessive-compulsive disorder. The three types are avoidant personality disorder (social inhibition, feelings of inadequacy and hypersensitivity to negative evaluation, with avoidance of activities involving interpersonal contact), dependent personality disorder (a pervasive and excessive need to be taken care of leading to submissive and clinging behaviour and fears of separation), and obsessive-compulsive personality disorder, the anankastic type (a preoccupation with orderliness, perfectionism and mental and interpersonal control at the expense of flexibility, openness and efficiency) (DSM-5-TR 2022). The thread that ties them is that fear, not oddness and not drama, is the organising affect: fear of rejection, fear of being left, and fear of losing control.

This fragment frames the cluster on one grid, takes each type through its defining criterion and the discriminator that decides it, then isolates the single highest-yield boundary of the group, the anankastic character against the anxiety disorder that shares its name. Two cautions from the framework topics of this chapter are assumed throughout: the general personality-disorder criteria (an enduring, pervasive, inflexible pattern, from early adulthood, causing impairment and not better explained otherwise) must be cleared before any type is named; and ICD-11 has abolished the categorical types, so "avoidant", "dependent" and "anankastic" survive as labels only in the legacy classifications and in clinical shorthand, with the dimensional model taught on the framework topics. The OCD side sits in the Anxiety, OCD and trauma-related disorders notes and the wider psychotherapy technique in the Psychotherapies, clinical assessment, MSE and nosology notes.

9.1 The cluster on one frame

Three types, one affect, three fears. DSM-5-TR groups the three types whose sufferers "often appear anxious or fearful" (DSM-5-TR 2022). Read the cluster by what each type is afraid of and what it does about the fear: the avoidant person wants closeness but withdraws from it for fear of criticism and rejection; the dependent person clings to a caretaker for fear of being left to cope alone; the anankastic person controls and orders the world for fear of anything going wrong. Learning the three essential features with their exact thresholds as one block is the fastest route to every cluster C question, because the thresholds differ across the three and are a favourite trip-wire.

TYPE	ESSENTIAL FEATURE (DSM-5-TR)	THRESHOLD	CORE DISCRIMINATOR
Avoidant	Social inhibition, feelings of inadequacy and hypersensitivity to negative evaluation, with avoidance of interpersonal contact	Four or more of the seven features	**Wants closeness but fears rejection (vs schizoid, who has no desire)**
Dependent	A pervasive, excessive need to be taken care of, leading to submissive, clinging behaviour and fears of separation	Five or more of the eight features	**Appeasement and a replacement caretaker, not rage (vs borderline)**
Anankastic (obsessive-compulsive)	Preoccupation with orderliness, perfectionism and mental and interpersonal control, at the expense of flexibility, openness and efficiency	Four or more of the eight features	**Ego-syntonic traits; no true obsessions or compulsions (vs OCD)**

The three DSM-5-TR Cluster C types with their essential features, criterion thresholds and core discriminators; the accompanying figure lays the same three on a fear-and-response map (fear of rejection, of separation, of loss of control).

PEARL

Tag each type by the fear that drives it: avoidant fears *rejection*, dependent fears *separation*, anankastic fears *loss of control*. Then hold the three thresholds apart, because they are not the same number: avoidant is **four or more of the seven**, dependent is **five or more of the eight**, and anankastic is **four or more of the eight**. Dependent is the odd one out that needs five.

9.2 Avoidant personality disorder

The person who longs for closeness and flees it. The essential feature of avoidant personality disorder is a pervasive pattern of social inhibition, feelings of inadequacy and hypersensitivity to negative evaluation, beginning by early adulthood and present across contexts, shown by **four or more of the seven** features (DSM-5-TR 2022). The seven are: avoiding occupational activities that involve significant interpersonal contact for fear of criticism, disapproval or rejection; being unwilling to get involved with people unless certain of being liked; showing restraint within intimate relationships for fear of being shamed or ridiculed; being preoccupied with being criticised or rejected in social situations; being inhibited in new interpersonal situations because of feelings of inadequacy; viewing the self as socially inept, unappealing or inferior; and being unusually reluctant to take personal risks or try new activities for fear of embarrassment. The signature is a person who deeply wants relationships and feels the loneliness of their absence, but assumes others are critical until stringently proven otherwise. Two contrasts do most of the work. Against schizoid personality disorder, the single question is whether closeness is wanted at all: the avoidant person wants it and fears rejection, the schizoid person has no desire for it. Against social anxiety disorder, the difference is pervasiveness and self-concept: avoidant personality disorder is a trait-level pattern with an entrenched sense of inferiority, present even without the

anxiety disorder, whereas social anxiety may be more situational, though the two overlap heavily and some regard avoidant personality disorder as a more severe form.

GOOD TO REMEMBER

- **Avoidant personality disorder:** defining criterion is pervasive social inhibition, feelings of inadequacy and hypersensitivity to negative evaluation, four or more of the seven features, from early adulthood. Discriminator from schizoid: closeness is wanted but feared (schizoid does not want it). Discriminator from social anxiety disorder: trait-level and pervasive with an entrenched inferiority, present even without the anxiety disorder. First step: confirm the general personality-disorder criteria, then that the avoidance is pervasive and character-level, not confined to circumscribed performance situations.

9.3 Dependent personality disorder

The excessive need to be cared for. The essential feature of dependent personality disorder is a pervasive and excessive need to be taken care of that leads to submissive and clinging behaviour and fears of separation, from early adulthood, shown by **five or more of the eight** features (DSM-5-TR 2022). The eight are: difficulty making everyday decisions without excessive advice and reassurance; needing others to assume responsibility for most major areas of life; difficulty expressing disagreement for fear of losing support or approval; difficulty initiating projects or doing things alone from a lack of self-confidence; going to excessive lengths to obtain nurturance and support, even volunteering for unpleasant tasks; feeling uncomfortable or helpless when alone from exaggerated fears of being unable to cope; urgently seeking another relationship as a source of care when a close one ends; and being unrealistically preoccupied with fears of being left to care for oneself. The behaviours are designed to elicit caregiving and arise from a self-perception of being unable to function alone. The examiner's favoured contrast is with borderline personality disorder: both fear abandonment, but the person with dependent personality disorder responds with increasing appeasement and submissiveness and urgently seeks a replacement caretaker, whereas the person with borderline personality disorder responds with rage, emptiness and demands, against a background of unstable and intense relationships. From avoidant personality disorder it is separated by direction: the dependent person seeks proximity, the avoidant person avoids it, though both share feelings of inadequacy.

GOOD TO REMEMBER

- **Dependent personality disorder:** defining criterion is a pervasive, excessive need to be taken care of leading to submissive, clinging behaviour and fears of separation, five or more of the eight features (the only cluster C type that needs five), from early adulthood. Discriminator from borderline: appeasement and a replacement caretaker, not rage and emptiness; relationships stable-clinging, not unstable-intense. Discriminator from avoidant: proximity-seeking, not proximity-avoiding. First step: confirm the general criteria, then that the dependence is excessive and unrealistic beyond situational and cultural norms, not normative interdependence.

9.4 Anankastic (obsessive-compulsive) personality disorder

Control at the cost of flexibility. The essential feature of obsessive-compulsive personality disorder, the anankastic type, is a preoccupation with orderliness, perfectionism and mental and interpersonal control, at the expense of flexibility, openness and efficiency, from early adulthood,

shown by **four or more of the eight** features (DSM-5-TR 2022). The eight are: preoccupation with details, rules, lists, order, organisation or schedules to the point that the major purpose is lost; perfectionism that interferes with task completion; excessive devotion to work and productivity to the exclusion of leisure and friendships, not explained by economic necessity; over-conscientiousness, scrupulousness and inflexibility about matters of morality, ethics or values, not explained by culture or religion; inability to discard worn-out or worthless objects even when they have no sentimental value; reluctance to delegate unless others submit to exactly one's own way; a miserly spending style, money hoarded against future catastrophe; and rigidity and stubbornness. The core is control: the perfectionism is so complete that projects are never finished and time is poorly allocated, and the rigidity holds even when compromise would clearly serve the person's own interest. The whole pattern is **ego-syntonic**, experienced as reasonable and correct, which is exactly what sets up the marquee trap of the topic.

GOOD TO REMEMBER

- **Anankastic (obsessive-compulsive) personality disorder:** defining criterion is a pervasive preoccupation with orderliness, perfectionism and mental and interpersonal control at the expense of flexibility, openness and efficiency, four or more of the eight features, from early adulthood. Discriminator from OCD: the traits are ego-syntonic character, with no true obsessions and no compulsions. First step: confirm the general criteria and that the perfectionism and control are a pervasive, ego-syntonic trait, then search for (and either exclude or separately co-diagnose) the true obsessions and compulsions of OCD.

9.5 Anankastic versus OCD: ego-syntonic character against ego-dystonic illness

Similar names, different animals. This is the boundary the topic is built to test. Despite the shared name, obsessive-compulsive personality disorder and obsessive-compulsive disorder are clinically quite different (DSM-5-TR 2022). The anankastic personality is not characterised by intrusive thoughts, images or urges, nor by repetitive behaviours performed in response to them; it is an enduring, pervasive maladaptive pattern of excessive perfectionism and rigid control, and it is **ego-syntonic**, the person sees the traits as sensible and right and is not distressed by them, though the people around them often are. Obsessive-compulsive disorder is defined by true **obsessions** (intrusive, unwanted thoughts, images or urges) and **compulsions** (repetitive acts performed to neutralise the distress), and it is **ego-dystonic**, the symptoms are experienced as alien, unwanted and resisted. The presence or absence of true obsessions and compulsions is the line. The two are not mutually exclusive: when the criteria for both are met, both are diagnosed, and comorbid anankastic personality is found in roughly a quarter to a third of people with OCD in longitudinal cohorts. The obsessive-compulsive disorder side, its phenomenology and its treatment, is developed in the Anxiety, OCD and trauma-related disorders notes.

FEATURE	ANANKASTIC (OBSESSIVE-COMPULSIVE) PERSONALITY DISORDER	OBSESSIVE-COMPULSIVE DISORDER (OCD)
Relation to self	Ego-syntonic: traits seen as reasonable, right and desirable	Ego-dystonic: symptoms alien, unwanted, resisted
Core content	A pervasive character style of perfectionism, orderliness and rigid control	Discrete intrusive symptoms sitting on the background personality
Obsessions and compulsions	**Absent: no true obsessions and no compulsions**	Present and defining
Distress	Little distress about the traits themselves; others carry the frustration	Marked distress and active resistance to the symptoms
If both present	When the criteria for both are met, both diagnoses are recorded	

The anankastic-versus-OCD discrimination; the highlighted cell is the single feature that decides it, the absence of true obsessions and compulsions in the personality disorder. The OCD side is developed in the Anxiety, OCD and trauma-related disorders notes.

RULE **The line is true obsessions and compulsions, and ego-tone.** Anankastic personality is an ego-syntonic character of perfectionism and control with no true obsessions or compulsions; OCD is defined by ego-dystonic, resisted obsessions and compulsions. Their presence or absence, not the shared name, decides the diagnosis.

TRAP **Calling a rigid perfectionist "OCD".** Orderliness, perfectionism, hoarding of worthless items and inability to delegate, experienced by the person as simply being thorough and correct, are anankastic personality, not OCD, unless there are genuine intrusive obsessions and neutralising compulsions the person resists.

WORKED EXAMPLE

An accountant in his forties is referred as "OCD". He is a relentless perfectionist who cannot discard years of worn-out paperwork, refuses to delegate, and misses deadlines because he re-checks his work endlessly; he regards all of this as ordinary diligence and is untroubled by it, though his family and colleagues are worn down. He describes no intrusive unwanted thoughts and performs no rituals to neutralise anxiety. This is obsessive-compulsive personality disorder, the anankastic type, not OCD: the pattern is ego-syntonic character without true obsessions or compulsions. Had he also described intrusive contamination fears and washing rituals he resisted, both diagnoses would stand together.

9.6 How cluster C maps onto the dimensional and legacy systems

The categorical names decoded into traits. The framework topics of this chapter teach the dimensional model in full; three cluster-C-specific facts are worth carrying. First, the ICD-11 anankastia trait domain, a narrow focus on a rigid standard of perfection and on controlling oneself, others and situations to enforce it, is essentially the OCPD phenotype re-cast as a dimension, and it is the domain with no counterpart in the DSM-5 alternative model. Second, the avoidant and dependent phenotypes load mainly on negative affectivity with detachment, the

anxious-fearful traits. Third, and high-yield, the DSM-5-TR alternative model of personality disorders retains only six categorical types, antisocial, avoidant, borderline, narcissistic, obsessive-compulsive and schizotypal: avoidant and obsessive-compulsive survive the cut, but dependent personality disorder is one of the four types dropped. In the legacy ICD-10, the same phenotypes carried different names, anankastic for obsessive-compulsive and anxious for avoidant, with dependent kept as it is; exams still test these categorical names because DSM-5-TR retains them and the literature is written in them.

PEARL

Two cluster C facts examiners like: the ICD-11 fifth domain is *anankastia* (the OCPD phenotype), where the DSM-5 alternative model instead has psychoticism; and the alternative model keeps avoidant and obsessive-compulsive but drops dependent, so "which cluster C type is not in the DSM-5 alternative model?" has a clean answer, dependent.

9.7 Management in outline, and the cultural reading

Psychological therapy leads; drugs treat comorbidity only. Cluster C is managed, not cured, and no drug is disorder-specific for any of the three. The primary mode is structured psychological therapy: graded exposure to the avoided or feared situation with cognitive restructuring of the beliefs of inadequacy for avoidant personality disorder (the technique overlaps with the treatment of social anxiety disorder), assertiveness and autonomy-building for dependent personality disorder, and work on rigidity and control for the anankastic patient. The schema-mode and dialectical approaches, and the deeper psychotherapy technique, are developed on the psychotherapy topics of this chapter and in the Psychotherapies, clinical assessment, MSE and nosology notes. Pharmacotherapy stays narrow: where avoidant personality disorder carries a comorbid social anxiety disorder, a serotonin reuptake inhibitor may help that comorbidity, with the agents, Indian brands and dosing carried in the Anxiety, OCD and trauma-related disorders notes rather than re-taught here.

GOOD TO REMEMBER

- **Structured cognitive-behavioural therapy (Cluster C):** mechanism is graded exposure to avoided or feared situations plus cognitive restructuring of the core beliefs (inadequacy in avoidant, helplessness in dependent, the need for control in anankastic), with assertiveness and autonomy skills; indication is all three cluster C types, and it engages fastest in avoidant and dependent personality disorder, where the anxious behaviour is distressing to the person; signature caution is the alliance and pace, shame drives drop-out in the avoidant patient, the anankastic patient resists surrendering control, and the dependent patient can transfer their dependence onto the therapist. Medication is for a comorbidity only, never disorder-specific.

INDIA

Two genuine India angles meet on cluster C. First, under-recognition: the anankastic patient is ego-syntonic and almost never self-refers, reaching services only through burnout, a comorbid depression or a family complaint, while the avoidant and dependent patient usually presents for a comorbid anxiety or depressive disorder rather than for the personality itself, so the diagnosis is made only when it is actively sought. Second, and the more testable, is the cultural reading of dependent personality disorder. In collectivist Indian family life, interdependence, deference to elders, and decisions made jointly or by the family (about marriage, work or money) are normative, not pathological; scoring them as dependent personality disorder through a single culture's autonomy norms is a straight error, and DSM-5-TR itself requires that dependence be judged excessive relative to the person's own cultural and situational context, weighed with an informant. The same caution applies to over-conscientiousness read as anankastic when it reflects religious or cultural observance. Structured psychotherapy for these disorders is available unevenly and mostly at tertiary and private centres.

9.8 The countable skeleton

One line rebuilds the cluster. Three types, three fears, three thresholds and one marquee boundary are the whole of cluster C worth carrying into the hall. Hook them once and each type unpacks from its fear.

MNEMONIC

FLEE, CLING, CONTROL

The three cluster C types by the fear they answer. **FLEE** is avoidant personality disorder (wants closeness, flees it for fear of rejection, four or more of the seven). **CLING** is dependent personality disorder (needs to be cared for, clings for fear of separation, five or more of the eight). **CONTROL** is anankastic or obsessive-compulsive personality disorder (orders the world for fear of anything going wrong, ego-syntonic, no true obsessions or compulsions, four or more of the eight). Flee from people, cling to a person, control the world.

3	5	6
CLUSTER C TYPES (ANXIOUS OR FEARFUL)	ICD-11 TRAIT DOMAINS (ANANKASTIA IS THE CLUSTER C ONE)	DSM-5 ALTERNATIVE-MODEL TYPES KEPT (DEPENDENT DROPPED)

HOLD THIS

Cluster C is anxious or fearful: avoidant wants closeness but fears rejection (four or more of the seven), dependent needs to be cared for and clings for fear of separation (five or more of the eight), anankastic controls and perfects the world (four or more of the eight); and the marquee line is anankastic personality (ego-syntonic character, no true obsessions or compulsions) against OCD (ego-dystonic, resisted obsessions and compulsions).

10 · Borderline personality disorder versus bipolar II

The discriminator the whole chapter builds toward. Two conditions share a surface, mood that will not hold still and behaviour that runs ahead of judgement, and the examiner exploits the overlap: is this borderline personality disorder or is this bipolar II disorder? Getting **borderline**

versus bipolar right is not a fine academic point but the fork on which the management plan turns, because one answer sends the patient to a structured psychotherapy and the other to a mood stabiliser. This note owns that **discrimination**. The bipolar-II side of the picture, the hypomanic and depressive syndromes and their neurobiology, is developed in the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes; here the two are held side by side and pulled apart.

The trick is to resist matching on the shared symptom and instead read **one discriminator** at a time, each of which points reliably to one diagnosis. The **bipolar ii differential** reduces to a small grid of these discriminators, the tempo and trigger of the mood change, the duration of a shift, the presence of an unstable identity and chronic emptiness, the character of the self-harm and impulsivity, the family history, and the response to a mood stabiliser. Read across the grid and the answer usually declares itself. The accompanying figure lays the same discriminators out as a two-column contrast, and it is the single image to carry for this topic.

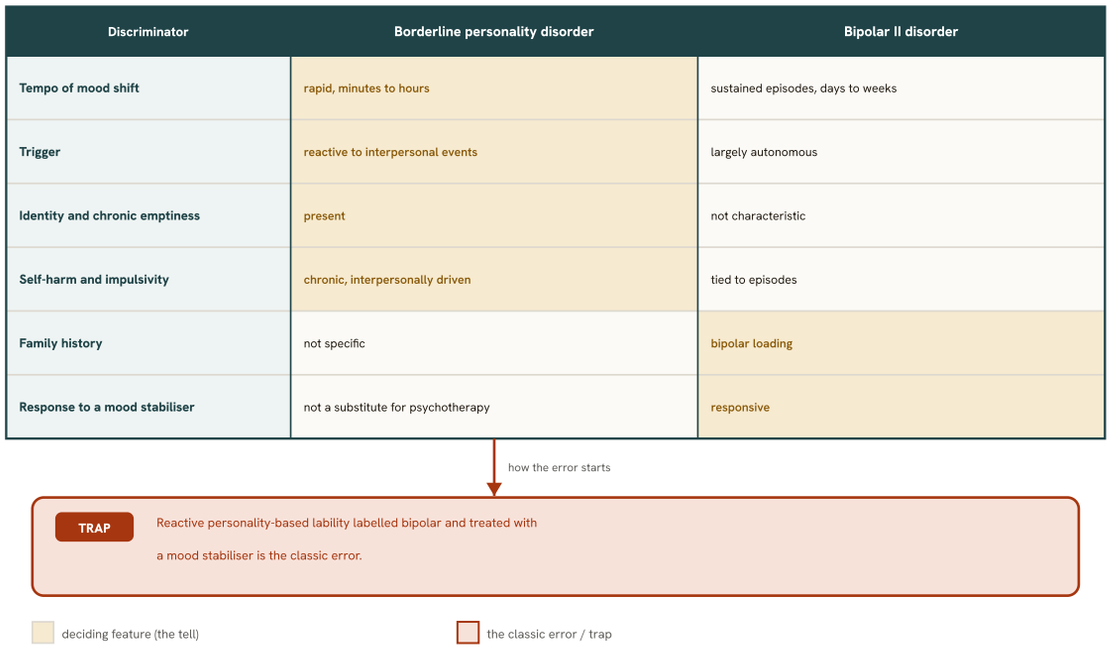


Fig 16.8 Borderline mood instability is rapid and reactive to interpersonal triggers, with identity disturbance and chronic emptiness; bipolar II episodes are sustained and autonomous with a bipolar family history and a mood-stabiliser response. The bipolar syndromes are taught in the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes.

10.1 The shared surface, and why the misdiagnosis is easy

Both present with mood lability and impulsivity, so the overlap is real. A patient who describes moods that flip, relationships that combust and impulsive acts they regret can fit either label on first telling, and the temptation is to hear "unstable mood" and reach for bipolar II disorder. The overlap is genuine: affective instability and impulsivity are cardinal in borderline personality disorder and are also the texture of the hypomanic and depressive swings of bipolar II. But the two instabilities have a different *form*, and the discipline of this topic is to describe the

mood change rather than name it, then let its tempo, its trigger and its duration decide. The discrimination fails only when the clinician pattern-matches on the shared word and stops looking.

RULE **Read the mood, do not name it.** Discriminate borderline from bipolar II on the tempo, the trigger and the duration of the affective change: reactive, interpersonally cued shifts over minutes to hours are borderline; autonomous, sustained episodes over days to weeks are bipolar.

10.2 The discriminator grid: one discriminator per row

The object to reproduce cold. Hold the differential as a grid in which each row turns on a single discriminator, and each discriminator points to one side. The highlighted cell in every row is the one that settles that row; read the whole column on either side and the diagnosis emerges as a pattern, not a single sign. This is the same grid drawn in the figure alongside.

DISCRIMINATOR	BORDERLINE PERSONALITY DISORDER	BIPOLAR II DISORDER
Tempo of affective instability	**Rapid shifts over minutes to hours, often several within one day**	Sustained episodes evolving over days to weeks
Trigger of the mood change	**Reactive, cued by an interpersonal event (rejection, criticism, a threat of abandonment)**	Largely autonomous; episodes arise and remit on their own timetable, frequently unprovoked
Duration of a mood shift	Dysphoria, irritability or anxiety usually lasting a few hours and only rarely more than a few days	**Hypomania at least four consecutive days; a depressive episode at least two weeks**
Baseline between shifts	Chronic dysphoria and emptiness; rarely a settled euthymic baseline	**Return to a euthymic baseline between discrete episodes**
Identity and emptiness	**Identity disturbance and chronic emptiness are core and enduring**	Identity is stable; emptiness is not a defining feature
Self-harm and impulsivity	**Self-harm is recurrent and affect-regulating, interpersonally cued; impulsivity is a pervasive lifelong trait**	Risk-taking and impulsivity are state-bound to the hypomanic phase; self-harm clusters in depressive episodes
Family history	Loads toward cluster B traits, trauma, mood and substance problems	**A strong loading for bipolar disorder points toward bipolar**
Response to a mood stabiliser	Modest and symptom-targeted at best; never a substitute for structured psychotherapy	**Often clearly effective; the mainstay of treatment**

The borderline-versus-bipolar-II discriminator grid; each row turns on one discriminator, and the highlighted cell is the one that decides it. No single row is definitive alone; the diagnosis is the whole pattern read across the columns.

5+	4	5
OF NINE BORDERLINE CRITERIA TO DIAGNOSE	DBT SKILLS MODULES (THE THERAPY BORDERLINE RESPONDS TO)	ICD-11 TRAIT DOMAINS (THE DIMENSIONAL CROSS-MAP)

10.3 The affective instability tempo, and the duration of mood shifts

The clock is the first and best discriminator. The **affective instability tempo** in borderline personality disorder is fast: mood swings over minutes to hours, sometimes several times within an afternoon, and it settles as quickly as it flared. The DSM-5-TR criterion says as much, episodic dysphoria, irritability or anxiety usually lasting a few hours and only rarely more than a few days. The **duration of mood shifts** in bipolar II disorder is categorically longer: hypomania requires a distinct period of persistently elevated or irritable mood lasting **at least four consecutive days**, and a major depressive episode lasts **at least two weeks**. Between those episodes the bipolar patient typically returns to a euthymic baseline, whereas the borderline patient rarely does, sitting instead on a floor of chronic dysphoria. So the two questions to ask are: how long does a low or an irritable spell last, and where does the mood return to when it passes.

10.4 The trigger: reactivity to interpersonal triggers versus autonomy

Ask what set the mood off. The second discriminator is the **reactivity to interpersonal triggers**. Borderline affective instability is characteristically reactive: a text left unanswered, a perceived slight, a looming separation, and the mood collapses or the anger flares, then eases when the relationship is repaired. Bipolar mood is largely autonomous; a hypomanic or depressive episode arrives and departs on its own timetable, often with no proportionate external cause, and it does not switch off the moment an interpersonal rupture is mended. This is why the history should chase the antecedent of every mood change: a mood that tracks the state of a relationship minute by minute is borderline reactivity, whereas a mood that runs for a week regardless of what is happening around the person is a bipolar episode. Rejection sensitivity, the exquisite reactivity to perceived abandonment, is the borderline signature that has no bipolar counterpart.

WORKED EXAMPLE

A woman in her twenties is referred as "query bipolar II, mood swings". On history the swings last two to three hours, always follow a fight with her partner or a fear he is pulling away, and lift the moment he reassures her; she describes never knowing who she is and a constant hollow emptiness, and she cuts when the distress peaks. Nothing here is a sustained, autonomous, days-long episode with a euthymic return. Read one discriminator at a time, the hours-long tempo, the interpersonal trigger, the identity disturbance and emptiness, and the picture is borderline personality disorder, not bipolar II. The label matters: it points to a structured psychotherapy, not to a mood stabiliser prescribed for a bipolarity she does not have.

10.5 Identity, chronic emptiness, and the character of self-harm and impulsivity

Look for the features bipolar II simply does not carry. The **identity and self-harm** row is decisive because these features belong to borderline personality disorder and are not part of bipolar II at all. Identity disturbance, a markedly and persistently unstable self-image, and chronic emptiness, a persistent hollowness distinct from depressive sadness, are core borderline criteria

with no bipolar equivalent; a genuinely stable sense of self between episodes argues strongly against borderline. The *character* of the self-harm and impulsivity discriminates too. In borderline personality disorder self-harm is recurrent, interpersonally cued and typically functions to regulate unbearable affect or to end dissociation, and the impulsivity is a pervasive, lifelong trait present between crises. In bipolar II the risk-taking and impulsivity are state-bound, confined to the hypomanic phase and absent when euthymic, and the self-harm clusters within depressive episodes. So the same behaviour, cutting or reckless spending, discriminates by *when* it happens: pervasive and trigger-linked in borderline, phasic and episode-linked in bipolar.

PEARL

Impulsivity that is present *between* episodes is a personality trait; impulsivity that appears only *during* an elevated mood and vanishes when the mood settles is a state. Trait impulsivity, chronic emptiness and an unstable identity together pull hard toward borderline; state-bound impulsivity on a euthymic baseline pulls toward bipolar II. The discriminator is not the behaviour but its timing against the mood.

10.6 Family history, and the response to a mood stabiliser

Two corroborating discriminators, held as support rather than proof. The **family history** is a genuine pointer because bipolar disorder is among the most heritable of psychiatric conditions: a strong, well-documented bipolar loading in first-degree relatives raises the prior for bipolar II, whereas a family picture weighted toward trauma, cluster B traits, mood and substance problems fits borderline better. Treat it as corroboration, not verdict, since both loadings can coexist. The **response to a mood stabiliser** is the other corroborating discriminator, and it is where the error becomes consequential. In bipolar II a mood stabiliser (lithium, valproate, or lamotrigine (Lamitor, Lametec) for the depressive pole) is foundational and often clearly effective; the deep pharmacology sits in the Mood disorders: pharmacotherapy notes. In borderline personality disorder the same drugs have at best a modest, symptom-targeted effect and are never a substitute for structured psychotherapy, which is the primary treatment. A patient whose "mood swings" do not respond to an adequate mood-stabiliser trial was very likely never bipolar; the non-response is itself a discriminator.

GOOD TO REMEMBER

- **Borderline personality disorder:** a pervasive pattern of instability of relationships, self-image and affect with marked impulsivity, five or more of the nine criteria; the affect is rapidly reactive, interpersonally triggered and lasts hours, on a floor of chronic emptiness and an unstable identity. First discriminating step in this differential: read the tempo, the trigger and the baseline of the mood change.
- **Bipolar II disorder:** at least one hypomanic episode (elevated or irritable mood, at least four consecutive days) plus at least one major depressive episode (at least two weeks), with a euthymic baseline between discrete, largely autonomous episodes and no full manic episode. Discriminator: sustained, autonomous, days-to-weeks episodes, not minutes-to-hours interpersonal reactivity.
- **Dialectical behaviour therapy:** the structured psychotherapy the borderline side responds to; it targets emotion dysregulation and self-harm through four skills modules (mindfulness, distress tolerance, emotion regulation, interpersonal effectiveness). Indication is recurrent self-harm and affective instability in borderline personality disorder, not the mood episodes of bipolar II. Caution: it treats the personality pathology, not a coexisting bipolar illness, which still needs its mood stabiliser.

10.7 The classic trap, and holding both diagnoses when they co-occur

The error and its correction. The classic trap is to hear reactive, hours-long, interpersonally driven lability, label it bipolar II, and start a mood stabiliser, medicalising a personality-based instability whose primary treatment is a structured psychotherapy. The drug then chases a misdiagnosis rather than an illness, the patient does not improve, and the failure is read as treatment resistance rather than as the wrong target. The correction is the grid: apply the discriminators before the label. Equally, the two are not mutually exclusive, and a comorbid bipolar II disorder is not rare in patients with borderline personality disorder; when a genuine, sustained, autonomous episode with a euthymic baseline is documented alongside the pervasive reactive pattern, both diagnoses stand and both treatments are needed, the mood stabiliser for the episodes and the structured psychotherapy for the personality pathology. The boundary with complex post-traumatic stress disorder, the other great mimic of borderline instability, is drawn in the Anxiety, OCD and trauma-related disorders notes.

TRAP **Labelling reactive instability "bipolar" and starting a mood stabiliser.** Reading minutes-to-hours, interpersonally cued lability as bipolar II and prescribing a mood stabiliser treats a personality-based instability with the wrong tool and delays the structured psychotherapy that is its actual primary treatment.

COMMON TRAP

Assuming the diagnosis is either-or. A strong bipolar family history or a documented autonomous episode does not exclude borderline personality disorder, and a clear borderline pattern does not exclude a genuine comorbid bipolar II. Diagnose each on its own evidence: the pervasive reactive pattern for borderline, a discrete sustained autonomous episode with a euthymic return for bipolar II. Where both are truly present, treat both.

10.8 The India lens: under-recognition, cultural expression, and access to therapy

The trap is amplified in Indian practice. Borderline personality disorder is markedly under-recognised in India: patients reach services through the overdose or the self-harm, which is coded as the act rather than the pattern, so the reactive lability underneath is repeatedly mislabelled as a mood disorder and medicated as bipolar. Two adaptations matter for this discrimination. First, cultural expression: affective display, transient dissociative or possession-trance presentations, and intense family-embedded reactions are normative idioms of distress in many Indian settings and must be judged against the person's own cultural and social norms before they are read as either borderline reactivity or a bipolar episode; personality pathology must not be read through a single culture's norms. Second, access: the structured psychotherapies the borderline side points to, dialectical behaviour therapy and schema therapy, are being delivered and culturally adapted at Indian tertiary and private centres rather than being widely available, so recognising that a patient needs psychotherapy rather than a mood stabiliser is only the first step, and a realistic plan must reckon with what the local service can actually deliver.

INDIA

Because borderline personality disorder is under-recognised in Indian practice, reactive interpersonal lability is disproportionately mislabelled bipolar and treated with a mood stabiliser, the exact trap of this topic. Judge every mood change against the patient's own cultural norms before scoring it, so that culturally sanctioned emotional expression and dissociative or possession-form idioms are not read as either a borderline criterion or a hypomanic episode. And recognise that catching the borderline diagnosis only helps if dialectical behaviour therapy or schema therapy is reachable; both are available and being adapted in the metros and academic centres but scarce elsewhere, which the plan must accommodate.

MNEMONIC

REACTIVE and RAPID versus AUTONOMOUS and SUSTAINED

The one-line split. Borderline instability is **REACTIVE** (interpersonally triggered) and **RAPID** (minutes to hours, on a floor of emptiness with an unstable identity); bipolar II mood is **AUTONOMOUS** (arises unprovoked) and **SUSTAINED** (days to weeks, with a euthymic baseline). Trigger and tempo first, then the corroborating family history and mood-stabiliser response.

HOLD THIS

Borderline affective instability is reactive, interpersonally triggered and lasts minutes to hours on a floor of chronic emptiness and unstable identity; bipolar II mood is autonomous and sustained over days to weeks (hypomania at least four consecutive days, depression at least two weeks) with a euthymic baseline; the mood stabiliser helps bipolar, while structured psychotherapy is primary for borderline, and mislabelling the first as the second is the classic, consequential error.

Aetiology, assessment and treatment

11 . Aetiology of personality disorders

Ask the question at three levels, then name one model. The **aetiology of personality disorders** is examined the same way every time: at the **genetic** level (what is heritable), the **neurobiological** level (the circuitry and neurochemistry that carry the trait), and the **developmental** level (attachment, trauma, temperament and the environment that shapes the trait into a disorder), all knitted together by a **gene-environment** transaction rather than a single cause. What is inherited is never a diagnostic category; it is a set of continuous personality trait dimensions whose extreme configurations, transacting with a formative environment, become the inflexible patterns the classification then labels.

This fragment walks the three levels in turn, states the integrating gene-environment principle, and then teaches the one disorder-specific aetiological model examiners expect by name, the Marsha Linehan biosocial theory of borderline personality disorder: a biologically heightened **emotional vulnerability** transacting reciprocally, across development, with a pervasively **invalidating environment**. The accompanying figure lays the transaction out schematically. The bipolar-II side of the affective-instability spectrum is cross-referenced to the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes; the complex-PTSD boundary to the Anxiety, OCD and trauma-related disorders notes; the deep serotonergic and mood-stabiliser pharmacology to the Mood disorders: pharmacotherapy notes; and attachment-based technique to the Psychotherapies, clinical assessment, MSE and nosology notes.

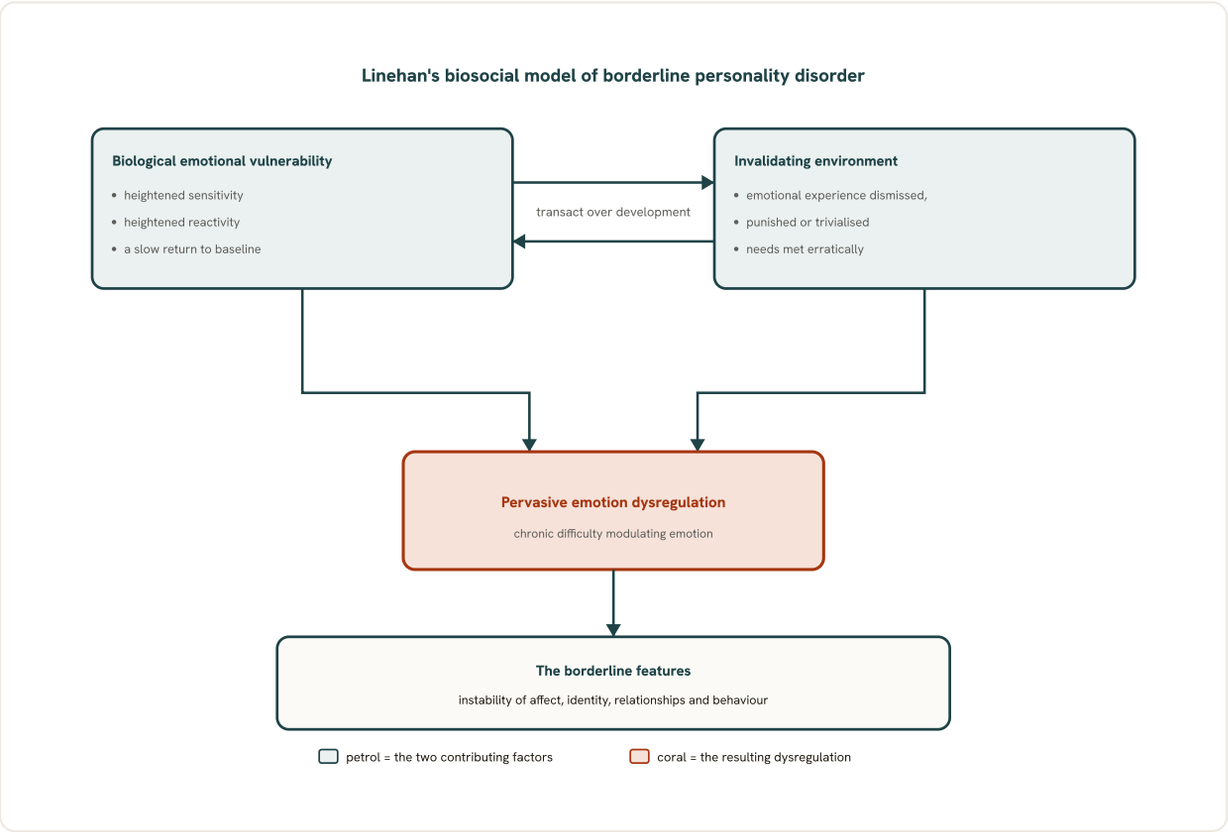


Fig 16.9 Linehan's biosocial model holds that a biological emotional vulnerability (heightened sensitivity and reactivity with a slow return to baseline) transacts reciprocally over development with a pervasively invalidating environment, and the transaction produces the pervasive emotion dysregulation that underlies the borderline features.

11.1 The three-level frame and the diathesis-stress spine

No personality disorder has a single cause. The stable finding across every type is that a heritable trait diathesis is necessary but not sufficient; the disorder emerges only when that diathesis meets a formative environment, and the two do not simply add but transact. Hold the three levels as a scaffold, the **genetic** (heritable trait dimensions), the **neurobiological** (the amygdala-prefrontal emotion circuitry and the serotonergic substrate of impulsive aggression) and the **developmental** (attachment disruption, childhood maltreatment, temperament), and read them not as competing explanations but as three windows on one process. This diathesis-stress spine, sharpened into a reciprocal transaction, is what the Linehan model makes concrete for borderline personality disorder, and it is the frame an examiner is testing when the question simply says "discuss the aetiology".

11.2 The genetic level: heritable dimensions, not heritable categories

Personality disorder is moderately heritable, and inherited as continuous traits. Twin, family and adoption studies agree that liability to personality pathology varies quantitatively and is moderately heritable, with estimates for normal and abnormal personality traits clustering in the roughly 40 to 60 per cent band. Crucially, personality disorders are not transmitted as discrete entities; what is inherited is a spread of trait dimensions, and the disorders are unusual configurations of the same traits that vary across the general population. The heritable loading is shared across spectra rather than tidy per-category packages: an **impulsivity and externalising** spectrum and an **affective-instability** spectrum load onto borderline and antisocial pathology

together, which is why comorbidity among the types is the rule. Most of the heritability sits in complex gene-gene interaction (epistasis) rather than in the average effect of single variants, so that individual common genes explain very little variance even in samples of tens of thousands, the so-called missing-heritability problem; the variants that are found tend to regulate neural development and neuroplasticity.

ENTITY	HERITABILITY ESTIMATE	WHAT IT TELLS YOU
Personality traits (normal + abnormal)	Moderate, roughly 40 to 60 per cent	Inherited as continuous dimensions, not discrete categories; mostly via epistasis (gene-gene interaction)
Any personality disorder	**Broad heritability about 44 per cent (Torgersen twins: MZ concordance 58, DZ 36 per cent)**	The anchor twin figure; DZ correlations well under half of MZ point to gene-environment interaction
Clusters A, B and C	Moderate, 40 to 60 per cent each	Separate heritable influences further differentiate the three clusters
Antisocial personality (symptom count)	The most heritable type, around 38 per cent	Best-studied; adoption and twin data converge; an unstable, hostile home acts synergistically
Paranoid personality (symptom count)	The least, around 28 per cent	Weak-to-moderate range across the ten categorical types

Heritability of personality pathology (Torgersen SCID-II twin data and DSM symptom-count studies). The highlighted any-disorder figure is the number to carry; the message is moderate, dimensional, and gene-environment dependent.

11.3 The neurobiological level: an amygdala-prefrontal imbalance and a serotonergic floor

Emotion dysregulation and impulsive aggression have a shared circuitry. At the **neurobiological** level the recurring finding is an imbalance in the **amygdala-prefrontal** emotion-regulation circuit: a hyperreactive amygdala that responds fast and hard to emotionally salient and socially threatening cues, paired with hypofunction of the prefrontal regions (orbitofrontal cortex and anterior cingulate) that would normally exert top-down inhibitory control. The net effect is intense, poorly braked affect, exactly the reactivity that shows up clinically as affective instability and as anger that outruns its trigger; imaging of individuals with impulsive aggression (Emil Coccaro and colleagues) shows just this exaggerated amygdala and blunted orbitofrontal response to social threat. Beneath the circuit sits a neurochemical dimension: reduced central **serotonergic** function correlates with **impulsive aggression** and with self-directed violence, the endophenotype Larry Siever placed at the centre of the borderline and antisocial impulsive-aggressive spectrum. That serotonergic floor is the rationale, cross-referenced to the Mood disorders: pharmacotherapy notes, for the symptom-targeted use of serotonin reuptake inhibitors against impulsive aggression, and it is a dimensional trait marker, not a disorder-specific lesion.

11.4 The developmental level: attachment, trauma and temperament

Early relationships and adversity shape the trait, but do not simply cause the disorder. Three **developmental** strands recur. First, **attachment disruption**: disorganised early attachment and a caregiving environment that fails to help the child read and regulate internal states leave a fragile capacity to mentalise (to hold minds in mind), which Peter Fonagy places at the heart of the borderline vulnerability. Second, **childhood trauma and maltreatment**: histories of sexual and physical abuse, neglect and pervasive family adversity are over-represented, and childhood sexual abuse is reported by a large minority to a majority of borderline patients (roughly 40 to 70 per cent). Third, **temperament**: early-emerging, procedurally learned dispositions (high negative emotionality, effortful-control deficits, novelty seeking) are the behavioural face of the heritable diathesis and are visible long before a disorder can be diagnosed. The developmental level is the environment that the genetic diathesis meets, and it is where the therapeutic leverage lies; the complex-PTSD-versus-borderline boundary that this trauma strand raises is developed in the Anxiety, OCD and trauma-related disorders notes.

TRAP **Reading childhood abuse as the cause of borderline personality disorder.** Childhood sexual abuse is reported by roughly 40 to 70 per cent of borderline patients, yet the association is only moderate (a meta-analytic effect size of about r = 0.28) and is neither necessary nor sufficient; trauma is a powerful risk factor that transacts with the diathesis, not the single cause.

11.5 The integrating principle: the gene-environment transaction

Diathesis and environment do not add, they transact. The three levels are bound by **gene-environment** interplay operating in both directions. Genes moderate how strongly a child reacts to adversity (gene-environment interaction), and a child's heritable temperament also shapes and selects the very environment it meets: an irritable, emotionally intense infant evokes harsher, more inconsistent caregiving (evocative gene-environment correlation) and later seeks out environments that amplify the trait (active correlation). The consequence is a feedback loop rather than a sum, in which a heritable diathesis and a formative environment escalate one another over years. This is precisely why the same adversity produces a personality disorder in one child and resilience in another, and why heritability estimates that ignore this transaction over-read the genetic share. The transactional reading is the modern successor to plain diathesis-stress, and it is the exact logic the Linehan model applies to borderline personality disorder.

RULE **A heritable trait becomes a disorder only in transaction with development.** Inherited trait dimensions set the diathesis; the disorder emerges through their reciprocal gene-environment transaction with attachment, trauma and the family environment, never from heredity or adversity alone.

11.6 The Linehan biosocial model: the disorder-specific synthesis

Borderline personality disorder is fundamentally a disorder of the emotion-regulation system. The Linehan **biosocial model** is the aetiological theory to name for borderline personality disorder, and it is simply the gene-environment transaction made concrete. In the **Linehan biosocial** account, the pervasive emotion dysregulation that underlies the borderline features results from continuous, reciprocal transactions among three components: a biological

emotional vulnerability, a pervasively **invalidating environment**, and the emotion dysregulation the two of them generate. None of the three is the cause on its own; the causal force is the transaction between them, running back and forth across development, and the figure alongside sets out that reciprocal loop. Because the model locates borderline pathology in emotion regulation rather than in character or manipulation, it is also non-pejorative and directly treatment-guiding, which is why it, and not the older object-relations account alone, is the model expected in an examination answer.

3	3	5	44
COMPONENTS OF THE LINEHAN BIOSOCIAL TRANSACTION	FEATURES OF THE EMOTIONAL VULNERABILITY	ICD-11 DIMENSIONAL TRAIT DOMAINS	BROAD HERITABILITY OF ANY PERSONALITY DISORDER, PER CENT (TORGERSEN TWINS)

11.7 The biological emotional vulnerability

Fast up, hard hit, slow down. The first arm of the model is a biologically mediated **emotional vulnerability**: a predisposition to affective instability arising from genetic, intrauterine and temperamental factors, and defined by three properties. The person is emotionally over-sensitive (a low threshold, so a wide range of ordinary stimuli triggers a response), over-reactive (a high magnitude of physiological arousal once triggered), and slow to return to baseline. The third property is the engine of the disorder: because arousal decays slowly, each subsequent event lands on top of incompletely settled arousal, making extreme responses ever more likely, the emotional equivalent of a burn victim feeling the wind. This vulnerability is the biological diathesis of the transaction, and it is what a treatment such as dialectical behaviour therapy targets with emotion-regulation and distress-tolerance skills.

Heightened sensitivity

A low threshold for emotional response; a wide range of everyday cues sets emotion off.

Heightened reactivity

A high magnitude of physiological arousal once a response is triggered.

Slow return to baseline

Arousal decays slowly, so successive events compound and escalate; the property that drives the instability.

11.8 The pervasively invalidating environment

An environment that teaches the child not to trust its own experience. The second arm is the **invalidating environment**: one that pervasively criticises, minimises, trivialises, punishes or only erratically reinforces the communication of internal experience, and that over-simplifies how easy problems are to solve ("you're not really upset", "you don't know what you want"). Two consequences follow. The child learns that its own thoughts and feelings are wrong, unimportant or shameful, and so comes to rely excessively on the social surround for cues on how to feel, producing the interpersonal hypersensitivity that marks the disorder. And because ordinary emotional displays are dismissed while extreme ones are intermittently attended to, the environment shapes escalation: the child who is ignored until it threatens self-harm, and then receives soothing, is taught that only extreme distress works. Childhood sexual and physical

abuse are the prototypical severe instances, but the invalidation need not be abusive; a well-meaning but poorly matched caregiver of a highly vulnerable child can invalidate inadvertently.

PEARL

The most examinable mechanism inside the invalidating environment is *intermittent reinforcement of extreme emotional displays*. When calm requests are ignored and only crisis behaviour (self-harm, threats) draws a caring response, the environment inadvertently trains the very high-amplitude behaviour it then finds intolerable. Naming this loop explains why borderline self-harm is so resistant to simple reassurance and why dialectical behaviour therapy works to break the contingency rather than to scold the behaviour.

11.9 The transaction, its product, and the treatment it points to

The two arms amplify each other; emotion dysregulation is the product. The force of the model is reciprocal. A highly emotionally vulnerable child is harder to soothe and so makes an environment more invalidating, even when caregivers mean well; and a more invalidating environment, by punishing and confusing emotional learning, further worsens the vulnerability. Round the loop turns, and its cumulative product is a pervasive **emotion dysregulation** that expresses itself across the borderline domains, the unstable relationships, the identity disturbance, the reactive affect, the impulsivity and the self-harm, all of them re-read as failures of emotion regulation rather than as separate symptoms. Because the model names a mechanism, it names a treatment: dialectical behaviour therapy was built directly on it to teach the emotion-regulation skills the transaction never allowed to develop, and schema therapy targets the same developmental deficits through the maladaptive schema modes; both are developed on their own topics in this chapter.

INDIA

Three India-specific points are genuine here. First, under-recognition: personality disorders are markedly under-diagnosed in Indian practice because patients reach services through the self-harm, the overdose or a comorbid depression, which is coded as the event rather than the enduring pattern, so the aetiological formulation is rarely made. Second, cultural expression: judge every trait and every "invalidation" against the person's own cultural and family norms, because collectivist family interdependence must not be misread as pathological dependence or as an invalidating environment, and possession-trance and dissociative idioms are normative in many settings. Third, access and forensic framing: dialectical behaviour therapy and schema therapy are being delivered and culturally adapted at Indian tertiary and private centres rather than being widely available, and antisocial personality disorder in particular carries a medico-legal frame (offender assessment, court reports) developed in the Mental health legislation and forensic psychiatry notes.

GOOD TO REMEMBER

- **Borderline personality disorder (aetiology):** the model to name is the Linehan biosocial theory, a biologically heightened emotional vulnerability (heightened sensitivity, heightened reactivity, slow return to baseline) transacting reciprocally over development with a pervasively invalidating environment, the transaction producing the pervasive emotion dysregulation that underlies the borderline features. Discriminator from a purely genetic or purely trauma account: the cause is the reciprocal transaction, not either arm alone. The diagnosis still rests on five or more of the nine criteria, developed on its own topic; the first aetiological step is to think transaction, not single cause.

GOOD TO REMEMBER

- **Dialectical behaviour therapy (from the model):** mechanism is to correct the emotion dysregulation the biosocial model places at the core, teaching skills across mindfulness, distress tolerance, emotion regulation and interpersonal effectiveness, delivered through its four treatment modes (individual therapy, the skills group, intersession phone coaching, and the therapist consultation team). Indication is borderline personality disorder, especially where reducing recurrent self-harm is the priority (NICE, Indian Psychiatric Society). Signature caution: it is a comprehensive structured programme, not a brief "DBT-informed" add-on, and delivering truncated fragments is not delivering dialectical behaviour therapy.

HOLD THIS

Borderline personality disorder is neither purely inborn temperament nor purely childhood damage: it is the product of a biologically heightened emotional vulnerability transacting reciprocally, across development, with a pervasively invalidating environment, and it is that gene-environment transaction, not either half alone, that generates the pervasive emotion dysregulation underlying the borderline features.

12 · Assessment of personality

Why this matters. The **assessment of personality** is the step that turns a clinical impression into a defensible diagnosis, and it is examined as a set of named instruments plus two governing principles. The instruments split cleanly into two families: clinician-administered **structured interviews** that confirm a diagnosis against the general criteria, and self-report questionnaires that screen for it. The two principles are the ones a stem is built to catch: a self-report screen identifies, an interview confirms, and personality is a trait, not a state, so it must never be diagnosed from the affect of an acute episode. Learn the instruments as their structure, not their item wording, and hold the two principles as the reasoning frame.



Fig 16.10 Personality assessment integrates longitudinal history, clinical interview, collateral information, dimensional trait and severity measures, developmental context and the effects of the current mental state.

This note typesets the working toolkit: the International Personality Disorder Examination (IPDE) and the Structured Clinical Interview for DSM-5 Personality Disorders (SCID-5-PD) as the categorical confirming interviews; the Personality Inventory for DSM-5 (PID-5) as the dimensional self-report of the trait model; the Zanarini Rating Scale for Borderline Personality Disorder (ZAN-BPD) as a symptom-severity and change measure; and the **ICD-11 severity anchors** as the graded frame that a modern, severity-led assessment records first. The general diagnostic criteria that open the chapter are assumed here; this note owns the measurement, the screen-then-confirm logic, and the state-versus-trait caution. The wider technique of clinical assessment and the mental state examination belong to the Psychotherapies, clinical assessment, MSE and nosology notes.

12.1 Two families of instrument: interviews that confirm, questionnaires that screen

Map the toolkit before the details. Every personality instrument is either an interview or a self-report, and either categorical (does the patient meet the criteria of a named type) or dimensional (how severe and along which trait continua). The four workhorses populate the grid: the IPDE

and the SCID-5-PD are clinician interviews that yield categorical diagnoses; the PID-5 is a self-report that yields a dimensional trait profile; the ZAN-BPD is a clinician scale that quantifies borderline severity for tracking change. Sitting over all of them is the ICD-11 severity judgement, which is not a questionnaire at all but a clinical grading of impairment recorded first. Fix that two-by-two picture and each instrument slots into place.

The prior gate still governs. No instrument replaces the general threshold: an enduring, pervasive, inflexible, culturally deviant pattern with onset by adolescence and clinically significant impairment, not better explained by another disorder. The instruments operationalise that judgement; they do not bypass it. A structured interview is, in effect, the general criteria and the type criteria administered as a disciplined, replicable schedule.

12.2 Self-report versus interview: screen wide, confirm narrow

The single most testable design principle. The **self-report versus interview** distinction is the logic that ties the instruments together. Self-report questionnaires (the IPDE screening questionnaire, the SCID-5 Personality Questionnaire, the PID-5) are quick, patient-completed and deliberately over-inclusive: they are sensitive but not specific, so they *screen* and over-identify. The clinician-administered **structured interview** (the IPDE, the SCID-5-PD) then probes only what screened positive and *confirms* the diagnosis, distinguishing a genuine enduring trait from an endorsed item. A positive screen is a prompt to interview, never a diagnosis in itself. This screen-then-confirm architecture is why the interview instruments each ship with a matched self-report front end.

RULE **Screen wide, confirm narrow.** A self-report questionnaire (the IPDE-SQ, the 106-item SCID-5-SPQ, the PID-5) screens and over-identifies; the structured interview (the IPDE, the SCID-5-PD) confirms against the criteria. A screen that is positive earns an interview; it does not earn a label.

PEARL

Read the self-report and interview as a sensitivity-then-specificity pair. The questionnaire is tuned to miss few cases (high sensitivity, low specificity), so its job is to decide which criteria are worth a clinician's time; the interview is tuned to confirm (specificity), so its job is to decide whether the endorsed pattern is real, enduring and impairing. Getting the order wrong, confirming from a questionnaire, is the commonest measurement error in the personality field.

12.3 The instruments, typeset

The object to hold cold. The instruments are learned as their structure and scoring with a citation, not as their items. The table gathers the four workhorse instruments and their matched screeners; the highlighted cells are the structural facts an examiner reaches for.

INSTRUMENT	ADMINISTRATION	STRUCTURE AND COVERAGE	SCORING	SOURCE
IPDE	Clinician semi-structured interview	**Two modules, an ICD-10 and a DSM-IV module, usable alone or together; questions grouped by six life areas (work; self; interpersonal relationships; affects; reality testing; impulse control), not by disorder**	Each relevant criterion rated 0 absent, 1 subthreshold, 2 criterion level; trait present at least the past five years, one criterion before the age of twenty-five; output both dimensional and categorical (definite or probable)	Loranger, Janca, Sartorius; WHO / Cambridge UP (Loranger et al. 1994)
IPDE-SQ	Self-report screener (true or false)	First-pass filter flagging which disorders to probe in the full interview	A positive screen prompts the interview; not diagnostic	IPDE screening questionnaire
SCID-5-PD	Clinician structured interview	Covers the ten DSM-5 (Section II) personality disorders plus other-specified personality disorder; the DSM-5 successor to the SCID-II	**Each criterion rated 1 absent, 2 subthreshold, 3 threshold; the DSM-5 required criterion counts then decide each diagnosis**	First, Williams, Benjamin, Spitzer 2016 (APPI)
SCID-5-SPQ	Self-report screener (yes or no)	A 106-item questionnaire the patient completes first, so the interview probes only criteria that screened positive	Screening only; not itself diagnostic	SCID-5 Personality Questionnaire
PID-5 (and PID-5-BF)	Self-report dimensional trait measure	**Five higher-order trait domains over twenty-five lower-order facets; full form 220 items, Brief Form 25 items (domain level); operationalises the DSM-5 Section III alternative model Criterion B**	Items scored 0 to 3; domain and facet scores as item means (higher is more maladaptive); no single categorical cut-off	Krueger, Derringer, Markon, Watson, Skodol 2012 (APA)

INSTRUMENT	ADMINISTRATION	STRUCTURE AND COVERAGE	SCORING	SOURCE
ZAN-BPD	Clinician-rated severity and change scale	Nine items, one per DSM borderline criterion, spanning the affective, cognitive, impulsive and interpersonal domains; rated over the prior week	**Each item 0 to 4; total on the 0 to 36 scale; higher is greater severity; tracks change, not a diagnostic cut-off**	Zanarini et al. 2003

The four workhorse personality instruments and their matched self-report screeners, typeset as structure and scoring with a citation; the highlighted cells mark the single fact most often tested for each. Item wording is not reproduced.

GOOD TO REMEMBER

- **IPDE:** a WHO semi-structured interview in two modules (ICD-10 and DSM-IV) organised by six life areas, criteria scored 0, 1 or 2, giving both a dimensional and a categorical output, paired with the IPDE-SQ self-report screener.
- **SCID-5-PD:** the DSM-5 structured interview over the ten Section II personality disorders, criteria rated 1, 2 or 3, fronted by the 106-item SCID-5-SPQ self-report screener; the successor to the SCID-II.
- **PID-5:** the self-report dimensional measure of the five trait domains and twenty-five facets (220-item full form, 25-item brief), the alternative-model Criterion B instrument, with no categorical cut-off.
- **ZAN-BPD:** nine clinician-rated items, one per borderline criterion, each 0 to 4 for a total on the 0 to 36 scale; a severity and change measure, not a diagnostic test.
- **ICD-11 severity anchors:** personality difficulty, mild, moderate and severe on a harm gradient; severity is graded first, before any trait qualifier.

12.4 The categorical confirming interviews: IPDE and SCID-5-PD

Two interviews, one job: confirm a named type against the criteria. The IPDE, developed by Armand Loranger out of the earlier Personality Disorder Examination and adopted under the WHO joint programme, is the international standard. Its distinctive move is to organise questions by life area rather than by disorder, walking the patient through work, self, interpersonal relationships, affects, reality testing and impulse control, so the clinician rates the underlying criteria as they surface across the whole life rather than interrogating one type at a time. Each criterion is scored 0, 1 or 2, and the IPDE builds in a temporal guard against late-onset mimics: a criterion counts only if the trait has run for at least the past five years, with at least one criterion satisfied before the age of twenty-five. It returns both a dimensional score per disorder and a categorical verdict (definite, or probable when one criterion short).

The SCID-5-PD is the DSM-native equivalent. The SCID-5-PD covers the ten DSM-5 Section II personality disorders and rates each criterion on the standard SCID convention of 1 (absent), 2 (subthreshold) or 3 (threshold), with the DSM-5 criterion counts then deciding each diagnosis. Its 106-item self-report questionnaire, the SCID-5-SPQ, is completed first so the interview need only probe the criteria that screened positive, the screen-then-confirm logic in a single package.

Both instruments are copyright and are learned as structure and scoring, not as reproduced items.

GOOD TO REMEMBER

- **IPDE (the discriminator):** the interview organised by six life areas, not by disorder; criteria scored 0, 1 or 2 with the five-year and pre-age-twenty-five temporal guard, yielding a dimensional plus categorical result.
- **SCID-5-PD (the discriminator):** the DSM-5 structured interview over the ten Section II types, criteria rated 1, 2 or 3 to the DSM criterion counts, with the 106-item SCID-5-SPQ as its screen.

12.5 The PID-5 and the dimensional trait map

The self-report that measures the trait model directly. The PID-5 is the self-report operationalisation of the DSM-5 Section III alternative model of personality disorders, specifically its Criterion B maladaptive-trait model. It measures five higher-order trait domains, negative affectivity, detachment, antagonism, disinhibition and psychoticism, resolved into twenty-five lower-order facets. The full form is 220 items on a four-point scale from 0 to 3, scored at domain and facet level; the 25-item Brief Form (PID-5-BF), five items per domain, gives a domain-level profile only. Being dimensional, it has no categorical cut-off: an elevated domain or facet mean flags maladaptive trait expression, to be read alongside the Criterion A judgement of how impaired personality functioning is.

The trait domains almost, but not exactly, match ICD-11. The high-yield trap is that the two dimensional systems share four domains and differ on the fifth. Read the table across: the DSM-5 alternative model and ICD-11 both carry negative affectivity, detachment, a callous-antagonistic domain and disinhibition, but ICD-11's fifth domain is anankastia (rigid over-control), whereas the alternative model's fifth is psychoticism (odd, eccentric cognition). ICD-11 also names its antagonistic domain dissociality and adds a borderline pattern qualifier.

TRAIT-DOMAIN THEME	ICD-11 QUALIFIER	DSM-5 ALTERNATIVE MODEL / PID-5 DOMAIN
Negative emotionality	Negative affectivity	Negative affectivity
Social withdrawal	Detachment	Detachment
Callous, antagonistic	Dissociality	Antagonism
Under-control, impulsivity	Disinhibition	Disinhibition
The differing fifth domain	**Anankastia (rigid over-control)**	**Psychoticism (odd, eccentric)**
Optional pattern specifier	Borderline pattern qualifier	A borderline type is reconstructed from the traits

The two dimensional trait systems share four domains; the fifth differs, ICD-11 adding anankastia while the DSM-5 alternative model adds psychoticism. Cross from one to the other by swapping anankastia for psychoticism (and dissociality for antagonism).

MNEMONIC

Three D's, plus N and A

The five ICD-11 trait domains: the three D's, **D**etachment, **D**issociality and **D**isinhibition, plus **N**egative affectivity and **A**nankastia. To convert to the DSM-5 alternative model, keep negative affectivity, detachment and disinhibition, rename dissociality as antagonism, and swap anankastia for psychoticism.

COMMON TRAP

Reading a high PID-5 domain score as a diagnosis. The PID-5 is dimensional and has no categorical cut-off; an elevated negative-affectivity or detachment mean describes trait expression, it does not by itself declare a personality disorder. The diagnosis still needs the level-of-functioning judgement (Criterion A) and the general threshold. A trait score is a profile, not a verdict.

12.6 The ICD-11 severity anchors

Severity is graded first, and it leads the diagnosis. The **ICD-11 severity anchors** are the frame a modern assessment records before anything else: rather than matching a type, the clinician grades how impaired personality functioning is, on a continuum from personality difficulty through mild, moderate and severe. The judgement rests on impairment of the *self* (identity, self-worth, self-direction) and of *interpersonal* functioning (relationships and roles), together with the risk of harm to self or others. There is no numeric cut-off; the harm gradient is the quick discriminator, running from typically none in mild, through sometimes in moderate, to often in severe.

SEVERITY LEVEL (CODE)	SELF AND INTERPERSONAL FUNCTIONING	HARM TO SELF OR OTHERS
Personality difficulty (QE50.7)	Longstanding pronounced traits shown only intermittently (for example under stress) or at low intensity; some but not notable disruption; a health-status factor, not a mental disorder	Minimal
Mild (6D10.0)	Some areas of self-functioning affected, or all areas mildly; problems in many relationships and roles, but some are maintained	Typically none
Moderate (6D10.1)	Multiple areas of self-functioning affected at moderate severity; marked problems in most relationships and most roles compromised; may show mild dissociative or psychotic-like states under stress	**Sometimes**
Severe (6D10.2)	Severe disturbance in the sense of self; virtually all relationships affected and role performance severely compromised or absent; impairment across nearly all of life	**Often**

The ICD-11 severity anchors, graded on self and interpersonal impairment plus harm risk; the harm gradient (minimal, typically none, sometimes, often) is the quick discriminator, and personality difficulty is a health-status factor rather than a disorder. Trait-domain qualifiers may be added at any severity level.

12.7 The ZAN-BPD: a measure of change, not a diagnostic test

Quantify severity, then track it. The ZAN-BPD, derived by Mary Zanarini from the borderline criteria and the revised diagnostic interview for borderlines, is a nine-item clinician-rated scale in which each item maps onto one of the nine DSM borderline criteria, from frantic efforts to avoid abandonment through to transient stress-related paranoia or dissociation. Each item is rated 0 to 4 over the prior week, for a total on the 0 to 36 scale, with higher scores meaning greater severity. Its purpose is to be sensitive to change: it is the instrument used to measure whether borderline symptoms are responding in a treatment trial or over follow-up, not to make the diagnosis. That confirming diagnosis is the job of the general criteria and a structured interview; the ZAN-BPD then meters the severity over time.

GOOD TO REMEMBER

- **ZAN-BPD (the discriminator):** nine clinician-rated items, one per borderline criterion, each 0 to 4 for a 0 to 36 total; a severity and change measure sensitive to treatment response, explicitly not a diagnostic instrument.

12.8 State versus trait: assess personality at baseline, not mid-storm

The caution that governs the whole assessment. The **state versus trait** rule is the reason a personality diagnosis is timed as much as it is measured: a personality disorder is an enduring, pervasive pattern, so it must not be diagnosed from the affect, impulsivity or relational chaos of an acute episode of another disorder. The lability of a mood episode, the disinhibition of intoxication and the reactivity of a fresh crisis all mimic personality pathology while they last. The safeguards are to assess when the patient is at baseline rather than in the acute state, to require that the same pattern predates and outlasts the episode, and to lean on collateral and longitudinal history rather than a single cross-sectional interview. Situational affective lability without the identity disturbance and abandonment fear is not borderline personality; and the reactive, hours-long mood swings of borderline must be separated from the sustained, autonomous episodes drawn out in the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes.

TRAP **Diagnosing a personality disorder mid-episode.** Affective instability, impulsivity and stormy relationships during an acute mood episode, an intoxication or a fresh crisis are state, not trait. Diagnose personality at baseline, against a pattern that is enduring, pervasive and pre-existing, or you will pin a lifelong label on a passing storm.

WORKED EXAMPLE

A woman in her twenties is admitted after self-harm during a depressive relapse, floridly labile and rejecting. The temptation is to record borderline personality disorder from the presentation. The disciplined route is the reverse: quantify the acute picture (a ZAN-BPD can meter current borderline symptoms, but does not diagnose), treat the mood episode, and defer the personality assessment. Reassessed at baseline some weeks later with collateral, a structured interview (the SCID-5-PD or the IPDE) then establishes whether an enduring, pervasive pattern truly predated the episode. Screen if useful, confirm by interview, and grade ICD-11 severity, but do it when the storm has passed.

12.9 The India lens and the cultural frame

Under-recognition makes systematic assessment worth more, not less. Personality disorders are substantially under-recognised in routine Indian practice, surfacing through comorbid mood, substance, anxiety or self-harm presentations rather than as a personality diagnosis, and often left uncoded for fear of stigma. A brief self-report screen embedded in a busy clinic is exactly the low-cost step that raises detection of the mixed and subthreshold cases that a purely impressionistic assessment misses, provided a structured interview then confirms. A severity-led ICD-11 reading, which begins with a graded judgement of dysfunction rather than a full type match, suits this reality well.

Read pathology against the patient's own culture, not a single norm. Every instrument still requires deviation from what the person's *own* culture expects, so joint-family interdependence, deference and religiously framed experience must not be scored as dependent, avoidant or odd pathology; personality must not be read through one culture's norms. On the treatment side, the

structured psychotherapies a severity rating points toward, dialectical behaviour therapy and schema therapy, are available and being culturally adapted at Indian tertiary and private centres but scarce elsewhere, so a severe rating can outrun what a service can deliver; the wider technique of these therapies belongs to the Psychotherapies, clinical assessment, MSE and nosology notes. Antisocial personality disorder carries the sharpest medico-legal weight in the Indian context, where the structured assessment feeds offender and court framing, cross-referenced to the Mental health legislation and forensic psychiatry notes.

INDIA

Personality disorders are under-recognised in Indian practice and present through their comorbidities, so a brief self-report screen followed by a confirming structured interview genuinely raises detection of the subthreshold and mixed cases. Judge deviation against the patient's own cultural norms, so collectivist family-embeddedness is not miscoded as dependency and religiously framed experience not as schizotypal oddness. The structured psychotherapies that a severity rating points to, dialectical behaviour therapy and schema therapy, are available and being culturally adapted in the metros and academic centres but unevenly accessible elsewhere; and antisocial personality disorder carries the medico-legal weight, where the assessment feeds the forensic notes.

5 ICD-11 AND ALTERNATIVE-MODEL TRAIT DOMAINS	**25** PID-5 LOWER-ORDER TRAIT FACETS (220-ITEM FULL FORM)	**0 to 36** ZAN-BPD TOTAL SCORE RANGE (NINE ITEMS)	**4** ICD-11 SEVERITY ANCHORS (DIFFICULTY TO SEVERE)

HOLD THIS

Assessment of personality runs screen-then-confirm: self-report questionnaires (the IPDE-SQ, the 106-item SCID-5-SPQ, the PID-5) screen and over-identify, and a structured interview (the IPDE, organised by six life areas; the SCID-5-PD, over the ten DSM-5 types) confirms against the criteria; grade the ICD-11 severity anchors first (difficulty, mild, moderate, severe on a harm gradient), map the five trait domains and twenty-five PID-5 facets, meter borderline change with the ZAN-BPD, and never fix the diagnosis during an acute episode, because personality is a trait, not a state.

13 . Dialectical behaviour therapy for borderline personality disorder

The first treatment that worked, and the template for all the rest. Dialectical behaviour therapy (DBT) is the psychotherapy that changed the outlook for borderline personality disorder: developed by Marsha Linehan out of failed attempts to treat chronically suicidal women with standard behaviour therapy, it was the first treatment of any kind shown in a randomised trial to reduce self-harm in a group that clinicians had come to regard as untreatable. Examiners return to it because it is comprehensive and highly structured, so it can be dismantled into named parts: a theory of the disorder, an organising philosophy, four skills modules, four treatment modes, a hierarchy of what to treat first, and staged goals across the whole arc of recovery. Learn those parts as a scaffold and the topic answers itself.

This fragment builds DBT from its foundations upward. It sets out the **biosocial theory** that DBT rests on, the central **dialectic** of acceptance and change from which the treatment takes its name, the four skills modules with **mindfulness** as their core, the four modes that together make a comprehensive programme, the target hierarchy that decides what is worked on in any given session, the stages of treatment, and the evidence for the reduction in self-harm and hospitalisation that earned DBT its place at the front of the guidelines. The disorder itself is taught on its own topic; here the pharmacology stays deliberately light and adjunctive, and the deep drug detail is cross-referenced to the Mood disorders: pharmacotherapy notes.

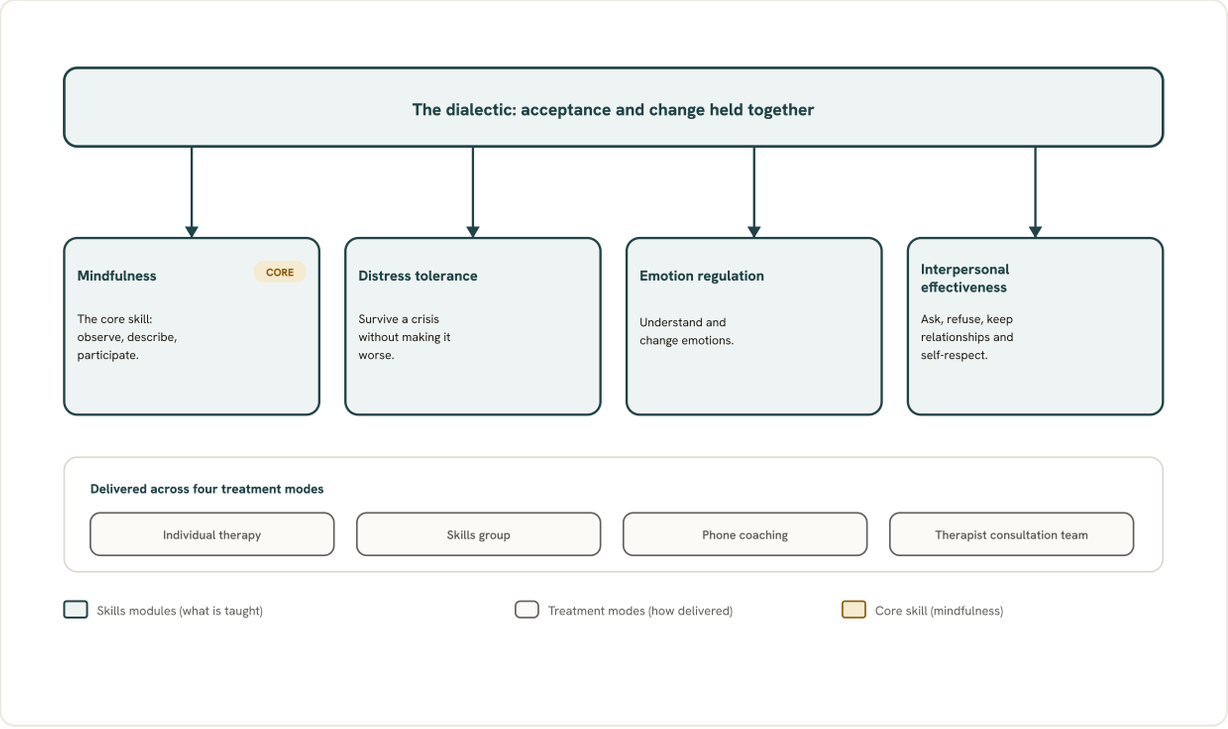


Fig 16.11 Dialectical behaviour therapy holds acceptance and change in dialectic and teaches four skills modules, mindfulness at the core, distress tolerance, emotion regulation and interpersonal effectiveness, delivered across four modes: individual therapy, the skills group, phone coaching and the therapist consultation team.

13.1 Origins and the core proposition

A behaviour therapy rebuilt around emotion dysregulation. Dialectical behaviour therapy grew in the early nineteen-eighties from work on repetitive suicidal and self-harm behaviour. Standard cognitive and behavioural change strategies, applied to chronically suicidal borderline patients, met a wall: pushing for change alone was experienced as invalidating, provoked drop-out and sometimes worsened the very behaviours being targeted. Linehan's solution was to place emotion dysregulation, rather than any single symptom, at the centre of the disorder, and to hold change strategies in constant balance with an equally strong stance of acceptance. The first controlled trial (Linehan and colleagues, published in the Archives of General Psychiatry) tested the new treatment in chronically parasuicidal women and made DBT the first psychotherapy to demonstrate efficacy in borderline personality disorder, opening the era of therapeutic optimism from which schema therapy, mentalisation-based treatment and transference-focused psychotherapy all followed.

4	4	3
SKILLS TRAINING MODULES	TREATMENT MODES IN COMPREHENSIVE DBT	STAGE-ONE TARGET TIERS (LIFE-THREATENING FIRST)

13.2 The biosocial theory of emotion dysregulation

Biology transacting with an invalidating environment. The biosocial theory is DBT's model of how borderline personality disorder arises, and it is the mechanism the whole treatment is built to address. A biological **emotional vulnerability**, meaning heightened sensitivity to emotional stimuli, greater intensity of response, and a slow return to baseline, transacts over development with a pervasively **invalidating environment**, one that dismisses, punishes or responds erratically to the child's inner experience. The transaction is reciprocal: the environment's invalidating responses amplify the biological sensitivity, and the resulting dysregulated behaviour draws further invalidation. The end state is pervasive **emotion dysregulation**, and DBT reads the criterion behaviours of the disorder, self-harm, impulsivity, stormy relationships and identity disturbance, as either the direct consequence of that dysregulation or the person's own attempts to re-regulate an unbearable affective state. Framing self-harm as attempted problem-solving, rather than manipulation, is what makes the theory both compassionate and clinically directive.

> **PEARL**
>
> The biosocial theory is why DBT never treats self-harm as a thing to be simply forbidden. If the behaviour is an attempt to solve the problem of unbearable emotion, then removing it without teaching a replacement skill leaves the person defenceless. Every act on the target list is therefore met with a behavioural chain analysis (what preceded it, what it achieved) and a substitute skill, not merely a safety contract.

13.3 The central dialectic: acceptance and change

Two technologies held in tension. The name of the treatment comes from **dialectics**: the philosophical stance that every position (thesis) calls up its opposite (antithesis) and that truth is found by synthesising the valid kernel of each. The single overarching dialectic in DBT is **acceptance and change**. The therapist must simultaneously accept the patient exactly as they are and relentlessly push for the changes that will build a life worth living, moving fluidly between the two poles rather than settling at either. This resolves the impasse that defeated ordinary behaviour therapy. Practically, DBT fuses two technologies: **behaviourism**, the technology of change (chain analysis, contingency management, skills training, exposure), and mindfulness derived from Zen, the technology of acceptance (validation, and ultimately radical acceptance of reality as it is). The constant motion between accepting and pushing is what gives DBT its characteristic speed and flow, and failures of the treatment are typically dialectical failures, a therapist stuck rigidly at one pole.

RULE **Structured psychotherapy is primary; DBT is the best-evidenced option for self-harm.** In borderline personality disorder the primary treatment is a structured, theory-based psychological therapy, and DBT is the intervention guidelines name first where reducing recurrent self-harm is the priority (NICE 2009, IPS 2023); medication is only adjunctive, symptom-targeted and time-limited.

13.4 The four skills modules, and mindfulness as the core

Four modules, built around a mindfulness centre. DBT teaches concrete behavioural skills in four modules, and Kaplan and Sadock name them exactly: mindfulness, distress tolerance, emotion regulation and interpersonal effectiveness. Mindfulness is the *core*: it is not one module among equals but the foundation, taught first and re-taught recurrently at the start of each of the other three modules. Its skills are the observing, describing and participating "what" skills, and the non-judgmental, one-mindful and effective "how" skills, converging on *wise mind*, the synthesis of emotion mind and reasonable mind. An elegant way to hold the set, shown in the accompanying figure, is that two modules serve *acceptance* (mindfulness and distress tolerance) and two serve *change* (emotion regulation and interpersonal effectiveness), so the four modules themselves mirror the central acceptance-change dialectic. A wrong module is the classic slip; verify the four against this list.

MODULE	POLE	WHAT IT TRAINS	SIGNATURE SKILLS
Mindfulness	**Acceptance (the CORE, re-taught before each module)**	Present-moment, non-judgmental awareness; the ground for all other skills	Observe, describe, participate; non-judgmentally, one-mindfully, effectively; wise mind
Distress tolerance	Acceptance	Surviving a crisis without making it worse; accepting what cannot be changed now	TIP, distraction (ACCEPTS), self-soothe, IMPROVE the moment, radical acceptance
Emotion regulation	Change	Understanding, naming and reducing the intensity of emotions; lowering vulnerability	Check the facts, opposite action, PLEASE (reduce vulnerability), accumulate positives
Interpersonal effectiveness	Change	Asking, refusing and keeping relationships and self-respect intact	DEAR MAN (objectives), GIVE (relationship), FAST (self-respect)

The four DBT skills modules; mindfulness is the core set, taught first and revisited before each of the other three. Two modules serve acceptance, two serve change.

MNEMONIC

DEAR MAN

The interpersonal-effectiveness skill for getting an objective met: **D**escribe the situation, **E**xpress feelings, **A**ssert the ask or the no, **R**einforce (name the benefit to the other), stay **M**indful (broken-record, ignore attacks), **A**ppear confident, **N**egotiate. Its two companions are GIVE (Gentle, Interested, Validate, Easy manner) for keeping the relationship, and FAST (Fair, no Apologies, Stick to values, Truthful) for keeping self-respect.

13.5 Distress tolerance, emotion regulation and interpersonal effectiveness

The three modules that complete the set. **Distress tolerance** is the acceptance-side crisis kit: skills to get through an urge to self-harm or an overwhelming affect without acting on it, from physiological down-regulation (the TIP skills) to distraction, self-soothing and, at its deepest, radical acceptance of a painful reality that cannot be changed in the moment. **Emotion regulation** is the change-side workhorse: it teaches the person to identify and label emotions, to check whether an emotion fits the facts, to act opposite to an unjustified emotion's urge, and to reduce baseline vulnerability by attending to sleep, eating, exercise and mastery. **Interpersonal effectiveness** supplies scripts for asking effectively, refusing without rupture, and preserving both the relationship and self-respect, which directly targets the abandonment sensitivity and the idealisation-devaluation swings of the disorder. The three change-and-tolerance modules are where the biosocial deficits are actively repaired.

TRAP **Calling a stand-alone skills class "DBT".** A weekly skills group on its own is skills training, not comprehensive DBT; the full treatment requires all four modes working together (individual therapy, skills group, phone coaching and a consultation team). Over-relying on distress tolerance while neglecting emotion regulation is the parallel error, since it teaches survival without teaching change.

13.6 The four treatment modes

Four modes, five functions. Comprehensive DBT is delivered through four **modes**, each serving a treatment function. The two that carry the standard trial format are **individual therapy plus skills group**: weekly **individual therapy** (which enhances motivation, uses the diary card and chain analysis, and where the individual therapist bears ultimate responsibility for the target behaviours) and the weekly **skills training group** (which enhances capabilities by teaching the four modules in a psychoeducational, class-like format). Alongside them run between-session phone coaching (which ensures the generalisation of skills into real life, coaching the patient through a crisis in the moment rather than in the next session) and the therapist consultation team (the "therapy for the therapists", which enhances the clinicians' own capability and motivation, guards treatment fidelity, and protects against burnout and dialectical failure). Between them these modes deliver DBT's five functions: enhance capabilities, improve motivation, ensure generalisation, structure the environment, and enhance the therapist. A programme missing any mode is not comprehensive DBT.

MODE	FUNCTION IT SERVES	WHAT HAPPENS IN IT
Individual therapy (weekly)	Improve motivation	One-to-one; diary card review and behavioural chain analysis; the individual therapist holds ultimate responsibility for the targets
Skills training group (weekly)	Enhance capabilities	Class-format teaching of the four modules with homework; a leader and co-leader; opens with a mindfulness exercise
Phone coaching (between sessions)	Ensure generalisation	Brief in-the-moment coaching to apply a skill during a crisis; commonly structured by a rule limiting calls after self-harm
Consultation team	**Enhance the therapist (fidelity and morale)**	Weekly clinician meeting; "therapy for the therapists"; sustains adherence and prevents dialectical failure and burnout

The four modes of comprehensive DBT and the functions they serve. The consultation team, exported into the treatment itself from the trials that use teams to control adherence, is the uniquely DBT mode examiners test.

13.7 The hierarchy of treatment targets

What gets worked on first is fixed, not chosen ad hoc. Because borderline patients typically present with many simultaneous problems, DBT imposes a strict order on what any given individual session addresses. Within the first stage of treatment the targets are ranked: **life-threatening behaviours** first (suicidal and self-harm acts, and credible threats), then **therapy-interfering behaviours** (of the patient or the therapist, such as missing sessions, arriving intoxicated, or a therapist's own drift), and finally **quality-of-life-interfering behaviours** (the substance use, disordered eating, unemployment and relational chaos that seriously destabilise a life). Only once those are in hand does the work turn to increasing behavioural skills. The diary card is scanned against this hierarchy at the top of every session, so a fresh episode of self-harm always pulls the agenda upward regardless of what patient or therapist would rather discuss. This ordering is one of the most reliably examined facts of the topic.

WORKED EXAMPLE

A young woman in comprehensive DBT arrives wanting to talk about a job interview. Her diary card, however, shows a cutting episode three evenings earlier. The target hierarchy decides the agenda: the therapist addresses the life-threatening behaviour first, running a chain analysis of the cutting and rehearsing the distress-tolerance skill that could have replaced it, before any time goes to the interview (a quality-of-life target). The ordering is not coldness; it is the treatment refusing to let the highest-risk behaviour be talked past.

13.8 The DBT stages of treatment

Staged goals across the whole arc of recovery. The **DBT stages** organise the treatment over time, beginning with a distinct pre-treatment phase of orientation and commitment, in which

patient and therapist agree the goals, the mode structure and the working agreements before therapy proper starts. The staged plan then runs from severe behavioural dyscontrol toward a full life, and the target hierarchy above sits inside the first stage.

STAGE	PRESENTING LEVEL	GOAL OF THE STAGE
Pre-treatment	Not yet committed or oriented	Orientation, commitment, agreeing goals, modes and working agreements
Stage one	Severe behavioural dyscontrol	**Behavioural control and stability (the life-threatening, therapy-interfering, quality-of-life target hierarchy)**
Stage two	Quiet desperation; controlled behaviour but emotional suffering	Non-anguished emotional experiencing; processing the past, including trauma and post-traumatic stress
Stage three	Problems in living	Ordinary happiness and unhappiness; self-respect and individual life goals
Stage four	Sense of incompleteness	Capacity for sustained joy, freedom and connection

Pre-treatment plus the four DBT stages. Most suicidal borderline patients enter at stage one, where behavioural control is the priority; trauma processing waits for stage two, after stability is achieved.

13.9 The evidence base: self-harm and hospitalisation

The reductions that earned it the guidelines. The foundational trial (Linehan and colleagues, in chronically parasuicidal women with borderline personality disorder) showed that, compared with treatment as usual, DBT reduced the frequency and medical severity of self-harm, produced fewer psychiatric hospitalisation days, and markedly improved treatment retention by lowering drop-out, even where it did not initially separate on depression or hopelessness. Those two signals, less **self-harm** and less **hospitalisation**, have been the durable core of the DBT evidence base ever since, replicated across later trials and reviews, and DBT remains the most extensively researched and evaluated of all treatments for the disorder (the basis for its Cochrane and guideline standing). Both NICE and the Indian Psychiatric Society recommend DBT specifically to reduce recurrent self-harm and emotion dysregulation, which is exactly the outcome the original trial demonstrated.

13.10 Place among the borderline psychotherapies, and the guideline anchor

One of several structured therapies, first among equals for self-harm. DBT sits alongside schema therapy, mentalisation-based treatment and transference-focused psychotherapy as the evidence-based structured psychotherapies for borderline personality disorder, each with its own mechanism and their own topics in this chapter; DBT is distinguished by its behavioural roots, its skills curriculum, and the strongest evidence base for reducing self-harm. The guideline position is consistent and worth carrying cleanly: structured psychological therapy is the primary treatment, DBT (or an equally comprehensive programme) is offered where recurrent self-harm is

the priority, and pharmacotherapy is not a treatment for the disorder itself but a symptom-targeted, time-limited adjunct for a comorbidity or a crisis. A programme is only DBT if it keeps the biosocial theory, the acceptance-change dialectic, all four modes and the target hierarchy; strip those out and what remains is skills training under a borrowed name. The wider technique of the structured psychotherapies is developed in the Psychotherapies, clinical assessment, MSE and nosology notes.

INDIA

Personality disorders are markedly under-recognised in Indian practice, so the borderline patient usually reaches services coded by the self-harm episode rather than by the pattern, and the structured therapy that would help is rarely offered. Two things temper a realistic plan. First, availability: dialectical behaviour therapy and schema therapy are being delivered and culturally adapted at Indian tertiary and private centres rather than being widely accessible, and few settings can staff a full four-mode programme with a trained consultation team, so pragmatic, skills-focused adaptations are common. Second, cultural fit: the mindfulness and acceptance core of DBT sits naturally with Indian contemplative traditions, an asset when framed in the patient's own idiom, while the interpersonal-effectiveness scripts must be calibrated to family interdependence rather than imported wholesale, since assertiveness norms and the very expression of personality pathology differ by culture and must not be read through a single culture's lens.

GOOD TO REMEMBER

- **Dialectical behaviour therapy (DBT):** core mechanism is the biosocial theory, an emotional vulnerability transacting with an invalidating environment to produce pervasive emotion dysregulation, treated by fusing behavioural change with mindfulness-based acceptance (the acceptance-change dialectic). Structure: four skills modules (mindfulness as the core, plus distress tolerance, emotion regulation and interpersonal effectiveness) delivered through four modes (individual therapy, skills group, phone coaching and consultation team), with a target hierarchy of life-threatening, then therapy-interfering, then quality-of-life-interfering behaviours across staged treatment. Indication: borderline personality disorder with recurrent self-harm, suicidality and chronic emotion dysregulation; it is the first-line structured psychotherapy for self-harm (NICE, IPS). Signature caution: it must be comprehensive and delivered with fidelity, it is resource-intensive, and trauma processing waits for stage two, only after stage-one behavioural control.

HOLD THIS

DBT rests on the biosocial theory of emotion dysregulation and the acceptance-change dialectic, and is delivered as four skills modules (mindfulness at the core, with distress tolerance, emotion regulation and interpersonal effectiveness) through four modes (individual therapy, skills group, phone coaching, consultation team), targeting life-threatening before therapy-interfering before quality-of-life behaviours; its landmark evidence is the reduction in self-harm and hospitalisation.

14 . Schema therapy for personality disorders

The integrative psychotherapy built for the difficult patient. Schema therapy is Jeffrey Young's integrative model, weaving cognitive-behavioural technique together with attachment theory, object relations and the experiential (gestalt) tradition, and designed expressly for the chronic, characterological presentations on which ordinary short-term cognitive-behavioural therapy

stalls. It matters in the examination because it is one of the small handful of structured psychotherapies with a genuine borderline personality disorder evidence base, and because its vocabulary, **early maladaptive schemas**, coping styles, **schema modes** and **limited reparenting**, is asked directly. Learn it as a four-part architecture: the schemas (the enduring self-defeating patterns), the domains that group them, the coping styles that keep them alive, and the modes framework that carries the work with the labile borderline patient.

This fragment teaches the model forward: what a **schema** is and where it comes from, the **five schema domains** holding the **eighteen early maladaptive schemas**, the **three coping styles** (surrender, avoidance, overcompensation), and the mode map that reduces the borderline patient's shifting states to a workable set of parts. It then teaches the two signature techniques, limited reparenting and experiential **mode work**, and closes on the trial evidence and the Indian picture. The wider landscape of psychotherapy models and technique sits in the Psychotherapies, clinical assessment, MSE and nosology notes; the borderline construct itself, and the sibling therapies dialectical behaviour therapy and mentalisation-based treatment, are developed on their own topics in this chapter.

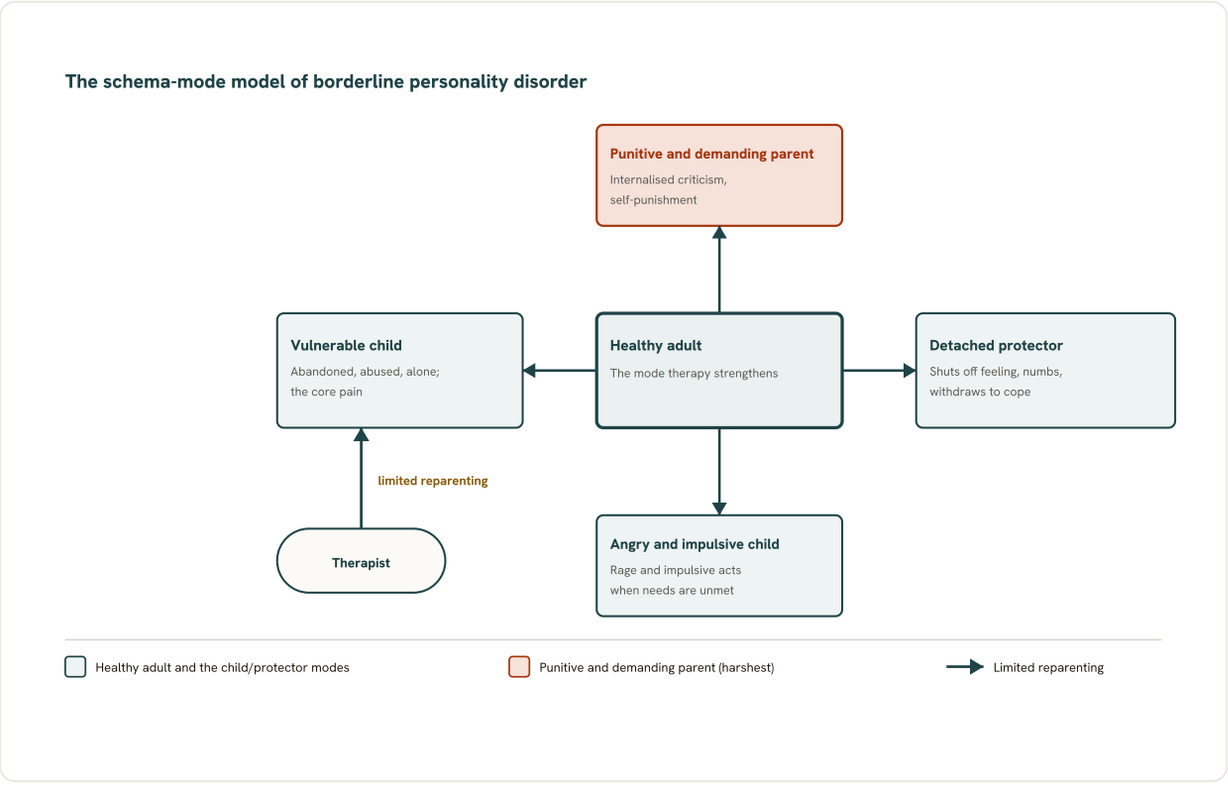


Fig 16.12 Schema therapy formulates borderline personality disorder as shifts between modes, the vulnerable child, the angry and impulsive child, the detached protector and the punitive and demanding parent, and uses limited reparenting to meet the vulnerable child's needs and build up the healthy adult mode.

14.1 What schema therapy is, and who it is for

An integrative model for ego-syntonic, characterological pathology. Standard cognitive-behavioural therapy assumes a patient who forms an alliance quickly, has a stable identity and clear goals, can access and verbalise affect, and can change through logic and homework. The personality-disordered patient breaks every one of those assumptions: the pathology is rigid, ego-syntonic and interpersonal, and it defeats a symptom-focused approach. Schema therapy was

constructed by Young for exactly this population. Its organising idea is that maladaptive personality patterns grow from **unmet core emotional needs** in childhood, five of them, secure attachment, autonomy and competence and a sense of identity, freedom to express valid needs and emotions, spontaneity and play, and realistic limits and self-control. Where these needs go unmet, through aversive experience interacting with temperament, the child lays down self-defeating patterns that harden into personality. The therapy aims to meet those needs, in a bounded therapeutic form, and so to heal the patterns rather than merely manage the symptoms.

14.2 Early maladaptive schemas: the enduring self-defeating pattern

Broad, pervasive, self-perpetuating themes about self and relationships. An early maladaptive schema is a broad, pervasive theme about oneself and one's relationships, comprising memories, emotions, cognitions and bodily sensations, laid down in childhood or adolescence and elaborated across the lifespan, dysfunctional to a significant degree. The examples make it concrete: abandonment, mistrust and abuse, emotional deprivation, defectiveness and shame. A schema begins as a reasonably accurate reading of a harmful early environment and becomes inaccurate as the person grows, yet it fights for survival: the patient assimilates new experience to fit the schema and discounts what would disconfirm it, which is schema perpetuation. This is what makes personality pathology so treatment-resistant, and it is why the schema, not the presenting symptom, is the unit of the work. Schemas are traits: present as a vulnerability, they may or may not be activated at a given moment, and when a schema is triggered the person is flooded with the original affect.

18	5	3	4
EARLY MALADAPTIVE SCHEMAS (YOUNG)	SCHEMA DOMAINS GROUPING THEM	MALADAPTIVE COPING STYLES	DYSFUNCTIONAL BORDERLINE MODES

14.3 The five schema domains and the eighteen schemas

Each domain answers one thwarted childhood need. The **eighteen early maladaptive schemas** are grouped into **five schema domains**, and the domain is the more examinable unit because each maps cleanly onto one of the unmet core emotional needs. Hold the five domains and one or two schemas under each, rather than reciting all eighteen; the domain structure is what the questions test.

DOMAIN	THWARTED CORE NEED	REPRESENTATIVE SCHEMAS WITHIN IT
Disconnection and Rejection	**Secure attachment: safety, stability, nurturance, acceptance**	**Abandonment/Instability; Mistrust/Abuse; Emotional Deprivation; Defectiveness/Shame; Social Isolation**
Impaired Autonomy and Performance	Autonomy, competence, a sense of identity	Dependence/Incompetence; Vulnerability to Harm; Enmeshment/Undeveloped Self; Failure
Impaired Limits	Realistic limits and self-control	Entitlement/Grandiosity; Insufficient Self-Control/Self-Discipline
Other-Directedness	Freedom to express valid needs and emotions	Subjugation; Self-Sacrifice; Approval/Recognition-Seeking
Overvigilance and Inhibition	Spontaneity and play	Negativity/Pessimism; Emotional Inhibition; Unrelenting Standards; Punitiveness

The five schema domains, each anchored to the childhood emotional need it reflects; the highlighted Disconnection and Rejection domain is the one most central to borderline pathology and the main target of limited reparenting.

14.4 The three maladaptive coping styles: surrender, avoidance, overcompensation

How the patient responds when a schema is triggered. A schema is the wound; the coping style is what the person does when it is touched. Young identifies **three coping styles**, and they map onto the innate threat responses. In surrender, the patient yields to the schema, accepts it as true and lives it out, choosing partners and situations that confirm it. In avoidance, the patient arranges life so the schema is never activated, blocking the thoughts and numbing the affect, often through detachment, substance use or workaholism. In overcompensation, the patient fights the schema by acting as though its opposite were true, so a defectiveness schema can drive grandiose perfectionism. All three are adaptive survival responses in childhood that become maladaptive later, because each perpetuates the very schema it was meant to escape.

COPING STYLE	INNATE ANALOGUE	WHAT THE PATIENT DOES
Surrender	Freeze	Yields to the schema, accepts it as true, repeats the schema-driven pattern and confirms it
Avoidance	Flight	Keeps the schema from firing at all: blocks thought and affect, withdraws, self-medicates, disengages from therapy
Overcompensation	**Fight**	**Behaves as though the opposite of the schema were true; counter-attacks, and overshoots into grandiosity or control**

The three maladaptive coping styles and their fight, flight, freeze analogues; a single patient uses different styles with different schemas and at different times.

MNEMONIC

FIGHT, FLIGHT, FREEZE

The three coping styles as the three threat responses: **Fight** is overcompensation (act as if the opposite is true), **Flight** is avoidance (keep the schema from firing), **Freeze** is surrender (yield to it and live it out). Three responses, three coping styles, one schema underneath.

14.5 The schema modes framework: from traits to states

Modes are the workhorse for the labile borderline patient. The original schema model is a trait model, and it fits the more stable personality disorders. It struggles with borderline personality disorder, where patients score high on almost every schema at once and flip between extreme states within minutes, angry, then terrified, then detached, then self-hating. Young answered this with the schema modes concept: a mode is the moment-to-moment state, the cluster of schemas and coping responses that is active right now, in contrast to the schema, which is the enduring trait. Reframing a chaotic patient as a small set of parts that alternate, rather than dozens of simultaneously active schemas, is what makes borderline pathology workable in the room, and it is why the modes framework, not the schema list, is the tool used with these patients. The mode map is shown in the accompanying figure.

RULE **Trait for the stable, state for the labile.** Use the schema (trait) model for the more stable personality disorders and the schema modes (state) model for the borderline patient who shifts between extreme states, because that patient has too many schemas active at once to work with one at a time.

14.6 The borderline modes: the four that shift, and the one that heals

Four dysfunctional modes, alternating around an underdeveloped Healthy Adult. The borderline patient characteristically moves between **four dysfunctional modes**. The **vulnerable child** (the Abandoned Child) carries the raw pain of the schemas, fragile, frightened, alone; the Angry and Impulsive Child erupts in rage or acts out impulsively when needs go unmet; the

detached protector is a coping mode of avoidance, shutting off all feeling and connection to keep the pain out; and the **punitive parent** (the internalised critical, sometimes demanding, parent) attacks the child with self-hatred and punishment, and drives much of the self-harm. Against these stands the fifth mode, the Healthy Adult, which in the borderline patient is weak and which the therapy exists to strengthen: the goal of treatment is to build a Healthy Adult that can nurture the vulnerable child, set limits on the angry child, bypass the detached protector and banish the punitive parent.

WORKED EXAMPLE

A woman in her twenties arrives numb and monosyllabic, saying she feels nothing (the detached protector is in charge). As the therapist gently makes contact, she begins to cry about being unlovable and terrified of being left (the vulnerable child breaks through). Asked about the previous night's cutting, she says flatly that she deserved it and is disgusting (the punitive parent speaks). Minutes later she is furious that the session must end (the angry child). The formulation is not "unstable and manipulative" but a rapid cycling through four modes, each of which is addressed differently: soothe the child, validate but contain the anger, get past the protector, and fight the punitive parent.

14.7 Limited reparenting: the relational engine

The therapist partially meets the unmet childhood needs, within bounds. Limited reparenting is the central relational stance of the therapy and its main active ingredient. Within the ethical and professional limits of the therapeutic frame, the therapist deliberately provides an approximation of the emotional experiences the patient missed in childhood, warmth, stability, containment and, where needed, firm limit-setting, so as to directly address the core emotional needs behind the schemas, above all those in the Disconnection and Rejection domain. It is *limited* precisely because the therapist does not become the parent and does not regress the patient into dependency; the more severe the early deprivation, and the more childlike the borderline patient's needs, the more central this reparenting becomes. It runs alongside empathic confrontation, in which the therapist holds warmth and honest challenge together, confronting the schema-driven behaviour while staying firmly on the patient's side.

PEARL

Limited reparenting is what most sharply distinguishes schema therapy from ordinary cognitive-behavioural therapy, in which the relationship is a vehicle for technique rather than an active ingredient. Here the relationship *is* a treatment: the therapist is a stable, caring figure who models the Healthy Adult the patient will internalise. The word "limited" is load-bearing, and marks the difference between therapeutic reparenting and a boundary violation.

14.8 Experiential mode work: imagery rescripting and chair work

Change is driven at the level of affect, not just cognition. Schema therapy works across four channels, cognitive (testing and reframing the schema), behavioural (pattern-breaking, replacing the coping responses), the therapy relationship (limited reparenting and empathic confrontation), and, most distinctively, the experiential. Experiential **mode work** engages the modes emotionally

rather than by argument. In imagery rescripting, the patient re-enters a painful childhood image; the therapist, and later the patient's own Healthy Adult, enters the scene to protect and comfort the vulnerable child and to confront the offending figure, so the memory is re-scripted with the need met. In chair work, the modes are externalised onto chairs and put into dialogue, so the Healthy Adult can, for instance, be given a voice to answer and expel the punitive parent. These techniques are the engine of schema change, and treating the therapy as cognitive disputation alone forfeits them.

TRAP **Running schema therapy as ordinary CBT.** Reducing it to cognitive disputation and homework, skipping the limited reparenting and the experiential imagery and chair work, strips out its two signature ingredients and leaves the schemas, which are held in affect and memory, untouched.

14.9 The evidence, in borderline and other personality disorders

A real trial base, strongest in borderline personality disorder. The landmark trial is Giesen-Bloo and colleagues' multicentre randomised study in the Netherlands (2006), which ran schema therapy against transference-focused psychotherapy over roughly three years in borderline personality disorder and found schema therapy superior, with a higher recovery rate (about double) and notably lower attrition. Farrell and colleagues (2009) showed a large added benefit from a group form of schema therapy, and Bamelis and colleagues (2014) extended the evidence beyond borderline, with a randomised trial supporting schema therapy across a range of personality disorders (including avoidant, dependent, obsessive-compulsive, paranoid, histrionic and narcissistic types), for which specific mode models have since been mapped. On this base, schema therapy sits, with dialectical behaviour therapy and mentalisation-based treatment, among the structured psychological therapies that are the primary treatment for borderline personality disorder; drug treatment is not first-line, and the deep pharmacology is cross-referenced to the Mood disorders: pharmacotherapy notes.

14.10 Place in management, and the Indian picture

A structured, longer-term psychotherapy delivered by trained, supervised therapists. Schema therapy is a structured, theory-based, longer-term treatment, typically delivered over a couple of years, moving from assessment and schema education, through the experiential and cognitive change work built on the reparenting relationship, to behavioural pattern-breaking and relapse prevention. It is not a brief intervention and, like the other borderline-specific therapies, it should not be delivered outside a supervised, structured service. Where availability is thin, its ideas, the mode formulation and limited reparenting, still usefully inform a general management plan even when the full protocol is out of reach.

INDIA

Personality disorders are markedly under-recognised in Indian practice, so the patients who would benefit from schema therapy are often reached only through a self-harm presentation and never formulated. Access is the second constraint: schema therapy, like dialectical behaviour therapy, is being delivered and adapted at a small number of Indian tertiary and private centres rather than being widely available, and trained, supervised schema therapists are scarce. Cultural calibration matters throughout, both because the schema content is read against family and cultural norms (interdependence and duty to family are not, in themselves, a Subjugation or Self-Sacrifice schema) and because the reparenting relationship must respect local expectations of the therapeutic bond; personality pathology must never be judged through a single culture's norms.

GOOD TO REMEMBER

- **Schema therapy (Young):** mechanism is healing early maladaptive schemas, the enduring self-defeating patterns laid down by unmet core emotional needs, through limited reparenting (the therapist partially and within bounds meets the missed needs) plus experiential mode work (imagery rescripting, chair work) alongside cognitive and behavioural change; the eighteen schemas sit in five domains, the three coping styles are surrender, avoidance and overcompensation, and the borderline patient is worked through the schema modes (vulnerable child, angry and impulsive child, detached protector, punitive parent) toward a strengthened Healthy Adult. Indication is chronic, characterological personality pathology, with the strongest evidence in borderline personality disorder (Giesen-Bloo 2006, superior to transference-focused psychotherapy) and support across other personality disorders (Bamelis 2014). Signature caution: it is a structured, longer-term therapy needing trained, supervised delivery, and stripping out the reparenting and the experiential work turns it into ordinary CBT that leaves the schemas untouched.

HOLD THIS

Schema therapy heals early maladaptive schemas through limited reparenting and experiential mode work; for the borderline patient the workhorse is the schema modes map, four dysfunctional modes (vulnerable child, angry and impulsive child, detached protector, punitive parent) cycling around a Healthy Adult that the therapy exists to strengthen.

15 . Mentalisation-based therapy and transference-focused psychotherapy

Two more borderline-specific therapies, two different engines. Alongside dialectical behaviour therapy and schema therapy, the borderline literature carries two further structured, manualised, evidence-based models, and the examiner wants the *mechanism* of each, not a vague label. Mentalisation-based therapy (**MBT**, also called mentalisation-based treatment, developed by Bateman and Fonagy) reads borderline pathology as a collapse of mentalising, the capacity to understand behaviour in terms of underlying mental states, that occurs under attachment stress; its whole technique is to restore that capacity through a particular therapeutic posture. Transference-focused psychotherapy (**TFP**, developed by Clarkin and Kernberg) reads the same pathology through object relations theory as a set of split, unintegrated internal representations of self and other, and works to integrate them in the live, here-and-now transference.

The high-yield frame is the contrast: both work in the here and now and both use the therapeutic relationship as the instrument, but MBT deliberately does *not* interpret the unconscious (it takes a curious, not-knowing **mentalising stance** to get the patient thinking about minds again), whereas TFP *does* interpret, using the sequence of clarification, confrontation and interpretation to fuse the idealised and persecutory images the patient cannot hold together. This fragment teaches each engine, its indications and its evidence, and closes by placing both alongside dialectical behaviour therapy and schema therapy; those two are taught on their own topics in this chapter, and the wider psychodynamic and general models are cross-referenced to the Psychotherapies, clinical assessment, MSE and nosology notes.

15.1 Mentalising: holding mind in mind

Mentalising is reading behaviour, one's own and others', in terms of mental states.

Mentalising is the social-cognitive ability to make sense of actions, of the self and of other people, in terms of the mental states behind them, thoughts, feelings, wishes, beliefs and desires; in plain language, it is attentiveness to thinking and feeling in oneself and in others. It is an ordinary human capability that underpins everyday interaction, and it runs across four dimensions that can each become unbalanced: automatic versus controlled, self versus other, cognitive versus affective, and internal versus external. In personality disorder these capacities are reduced, and reduced most sharply at moments of interpersonal stress, which is exactly when they are most needed. Bateman and Fonagy argue that many borderline symptoms emerge from this distortion or loss of mentalising: the person loses the felt distinction between their own perspective and external reality, cannot easily coordinate their mind with another's, and is left isolated, since the joining of minds (the so-called we-mode) is the ingredient of ordinary connection that the loss of mentalising obstructs.

15.2 The collapse under attachment stress, and the nonmentalising modes

Rising arousal switches mentalising off, and older, prementalising modes reappear.

Mentalising develops within attachment relationships and is vulnerable within them: high arousal, generated by attachment hyperactivation and disorganised attachment strategies, disrupts the less well-practised, higher cognitive capacity to reflect. As the accompanying figure shows, when interpersonal arousal climbs past a threshold, balanced mentalising drops away and three developmentally earlier, nonmentalising modes take over. The examinable point is that borderline phenomena are not random: each mode is what you get when one dimension of mentalising dominates and the others fail.

NONMENTALISING MODE	EXPERIENCE OF THE MIND	CLINICAL FACE
Psychic equivalence	**Inner states are felt as literally, unquestionably real; thought equals reality, so no alternative perspective is possible ("concreteness of thought")**	Overwhelming certainty ("the therapist hates me", "I am wicked"), the drama and terror that make self-harm feel comprehensible
Teleological	Mental states are believed only if their outcome is physically observable; affection is real only with a touch, care real only with an action	Demands for concrete proof of caring; self-harm or action as the only "real" expression of a state; the automatic pole dominates
Pretend mode	Thoughts and feelings become severed from reality; much is said about mental states with little genuine connection to them	Hypermentalising or pseudo-mentalising, long inconsequential talk about feelings; taken to an extreme, derealisation and dissociation

The three nonmentalising (prementalising) modes that reappear when mentalising collapses under arousal; the highlighted psychic-equivalence cell is the one that makes borderline self-harm and quasi-psychotic certainty intelligible.

MNEMONIC

TOO REAL · ONLY IF SEEN · NOT REAL AT ALL

The three nonmentalising modes in three tags. **Too real** = psychic equivalence (thought is experienced as reality). **Only if seen** = teleological (a state counts only if physically demonstrated). **Not real at all** = pretend mode (talk about feelings cut loose from any real feeling; hypermentalising).

15.3 The mentalising stance: curious, not-knowing, restoring the capacity to think

The stance is the treatment: inquisitive, not-knowing, aimed at reviving mentalising rather than delivering insight. The bedrock clinical principle of MBT is an authentic **mentalising stance**, an inquisitive, "not-knowing" posture that respects the opacity of minds: minds can never be truly known, so the clinician's task is to inquire, to be genuinely curious, and to be willing to be surprised by the patient's answer, with the aim of raising the patient's own awareness of their internal states through a shared process. The work is in the here and now and stays close to preconscious, working-memory experience rather than pursuing unconscious meaning through free association; a session typically settles onto a single focus within the first ten to fifteen minutes and then works around it. Crucially, MBT does not push interpretation while the patient is non-mentalising and highly aroused; the first job is to lower arousal and re-open thinking, because interpreting a mind that has collapsed into psychic equivalence only confirms the concreteness. The secure-base relationship to the therapist is what makes it safe to explore one's own mind and find it reflected in another's.

WORKED EXAMPLE

A patient arrives certain that "you were going to cancel on me, I could tell". A not-knowing MBT response is not a reassurance and not an interpretation of transference; it is a curious inquiry: "I did not know that was in your mind, help me understand what told you that, and what it was like to sit with it." The clinician marks their own not-knowing, slows the moment, and invites the patient back into thinking about the mental states involved, theirs and the clinician's, rather than settling the "fact". The same opening moment in TFP would be handled differently (see below), and that difference is the whole point of the topic.

RULE **Structured, borderline-specific psychotherapy is the treatment; the model is chosen by fit, not by a proven winner.** MBT, TFP, dialectical behaviour therapy and schema therapy are all evidence-based for borderline personality disorder, and head-to-head trials show no single model is reliably superior; choose by patient fit, therapist training and local availability, and deliver it as a structured programme, not as eclectic support.

15.4 MBT: structure, indications and evidence

A structured, time-limited programme of individual plus group work, first proven in a partial-hospitalisation trial. MBT is delivered as a combination of individual and group sessions within a structured, time-limited programme (the original model ran in a day-hospital and partial-hospitalisation setting, with later intensive-outpatient versions), all built around the mentalising stance. Its indications have widened from borderline personality disorder (the primary and best-evidenced target) to antisocial personality disorder (MBT-ASPD), to adolescents with emerging borderline features and self-harm (MBT-A), and to other conditions marked by mentalising failure. The founding evidence is Bateman and Fonagy's randomised trial of partial-hospitalisation MBT against treatment as usual: the MBT group showed significant reductions in depressive symptoms, in suicidal and self-injurious behaviour and in inpatient days, with better social and interpersonal functioning; the gains began to appear after about six months and were maintained after treatment ended, with long-term follow-up showing durable benefit. That "benefit emerges over months, then holds" shape is itself examinable.

PEARL

MBT reframes what a borderline crisis *is*: not primarily a behaviour to be managed but a moment when mentalising has gone offline. This is why the intervention is a stance rather than a technique menu, and why "the therapist not knowing what to say" is treated as useful data, an accurate read of the patient's own collapsed mentalising, rather than a therapist failure.

GOOD TO REMEMBER

- **Mentalisation-based therapy (MBT), Bateman and Fonagy:** mechanism, borderline symptoms arise from a loss of mentalising (understanding behaviour in terms of mental states) under attachment-stress arousal, so the therapy restores mentalising through a curious, not-knowing mentalising stance in the here and now, working close to conscious experience rather than interpreting the unconscious. Indication, borderline personality disorder first-line among structured therapies, extended to antisocial personality disorder and to adolescents; evidence from a partial-hospitalisation RCT versus treatment as usual with gains maintained after termination. Signature caution, do not push interpretation while the patient is non-mentalising and highly aroused, lower arousal and re-open thinking first.

15.5 TFP and the object-relations frame: split, unintegrated representations of self and other

The borderline mind is built of split object dyads that idealise and persecute but never integrate. Transference-focused psychotherapy rests on an ego-psychological and **object relations** framework derived from Melanie Klein and elaborated by Otto Kernberg. Its unit of analysis is the internal *object dyad*: a single mental representation of the self linked to a representation of another person by an affect (for example, a weak, needy self bound to an exploitative but needed other by fear). In health these representations integrate into whole, ambivalent images; in borderline personality organisation (BPO) they do not. Instead the patient relies on splitting, keeping idealised and persecutory representations apart so that the same person is experienced as all-good or all-bad and flips between the two. TFP therefore diagnoses along a *level of personality organisation* axis rather than by categorical criteria, assessing the interrelated domains of identity, object relations, primitive defences, reality testing, aggression and moral values in a structural interview, which the Structured Interview of Personality Organization (STIPO-R) helps to operationalise. The core therapeutic target is **identity diffusion**, the lack of an integrated sense of self, and the goal is identity consolidation.

COMMON TRAP

Reading TFP as "diagnose the DSM type, then treat it". TFP works on borderline personality organisation, a broader structural construct than DSM borderline personality disorder that cuts across many categorical personality diagnoses and also covers narcissistic pathology. Two patients with different DSM labels can share the same borderline-level organisation and the same split object relations, which is exactly what TFP targets; the categorical label is not the treatment axis.

15.6 The TFP technique: integrating the dyads in the here-and-now transference

Contract first, then channel the split into the transference and interpret it into integration. TFP is an individual therapy, tested as a twice-weekly treatment of at least one to two years' duration, and it begins with explicit contracting, goal-setting and limit-setting. The wager is that the patient's split, unintegrated representations of self and other will inevitably be enacted in the relationship with the therapist, the *transference*, where they become available in the room in the here and now rather than only in history. Work follows a priority hierarchy (suicidal and self-destructive behaviour first, then threats to the continuity of the treatment, then the dominant transference theme) and a technical sequence of clarification, then confrontation of contradictions, then interpretation. By naming, for example, that the patient is treating the

therapist as the persecutor of the moment while a needy, idealising self is split off, TFP brings the two poles into contact so they can begin to integrate; as the split dyads fuse into whole-object relations, identity consolidates. Reflective functioning and integration, not symptom management alone, are the intended fruits.

TRAP **Deep transference interpretation into a mind that has already collapsed.** Interpretation only works on a patient who can still mentalise; delivering a Kernberg-style transference interpretation while the patient is in psychic equivalence and high arousal tends to confirm the concreteness and destabilise, which is precisely the failure MBT was built to avoid. Restore mentalising and lower arousal first; interpret when there is a mind available to think with.

3	4	6	2
MBT NONMENTALISING MODES (PSYCHIC EQUIVALENCE, TELEOLOGICAL, PRETEND)	DIMENSIONS OF MENTALISING IN MBT	TFP STRUCTURAL-INTERVIEW DOMAINS OF PERSONALITY ORGANISATION	TFP SESSIONS A WEEK (TWICE WEEKLY, OVER ONE TO TWO YEARS)

15.7 TFP indications and evidence

For borderline and narcissistic pathology in a patient who can tolerate interpretation, with a solid RCT base. TFP is indicated for borderline and narcissistic personality disorders and, more broadly, for patients at a borderline level of organisation who can commit to a long, twice-weekly, interpretation-based treatment and tolerate the affect it raises; the containing contract and limit-setting are what make it usable in a high-risk population. The evidence base is genuinely randomised. Clarkin and colleagues' three-arm trial compared TFP, dialectical behaviour therapy and supportive psychodynamic therapy and found all three efficacious, with TFP producing broad change across domains; Doering and colleagues found TFP superior to treatment by experienced community psychotherapists on borderline symptoms, suicide attempts and dropout; and the Giesen-Bloo multicentre trial comparing schema therapy and TFP showed both producing significant improvement, with schema therapy holding an advantage on recovery and retention. Mechanism-consistent findings (Levy and colleagues) show TFP increasing reflective functioning and shifting attachment toward security, which is the change the object-relations model predicts.

GOOD TO REMEMBER

- **Transference-focused psychotherapy (TFP), Clarkin and Kernberg:** mechanism, an object-relations therapy that treats borderline pathology as split, unintegrated internal representations of self and other (object dyads) held apart by splitting, and integrates them by working the here-and-now transference through clarification, confrontation and interpretation, with identity diffusion as the core target and identity consolidation the goal. Indication, borderline and narcissistic personality disorders (borderline personality organisation) in a patient able to tolerate a long, twice-weekly, interpretive treatment; evidence from several RCTs. Signature caution, it is interpretation-heavy, so it needs a mind that can still mentalise and a firm contract, or the interpretation destabilises.

15.8 Where MBT and TFP sit alongside dialectical behaviour therapy and schema therapy

Four structured models, four different theories of the same disorder. The four borderline-specific psychotherapies are best held as one grid: each names a different core mechanism and a

different signature technique, and no head-to-head trial has crowned one as reliably best, which is why the selection RULE above governs. The two owned on their own topics in this chapter (dialectical behaviour therapy and schema therapy) are set here beside MBT and TFP so the discriminators are visible at a glance.

THERAPY	THEORETICAL MODEL	CORE MECHANISM / TARGET	SIGNATURE TECHNIQUE
Dialectical behaviour therapy (Linehan)	Biosocial: emotional vulnerability plus an invalidating environment	Emotion dysregulation and behavioural dyscontrol	Skills training (mindfulness, distress tolerance, emotion regulation, interpersonal effectiveness) held in dialectic of acceptance and change
Mentalisation-based therapy (Bateman and Fonagy)	Attachment and mentalising deficit	Loss of mentalising under arousal	**Curious, not-knowing mentalising stance that restores thinking about mental states in the here and now**
Transference-focused psychotherapy (Clarkin and Kernberg)	Ego-psychology and object relations (Klein, Kernberg)	Identity diffusion; split, unintegrated self and other representations	**Here-and-now transference interpretation (clarification, confrontation, interpretation) to integrate the split dyads**
Schema therapy (Young)	Early maladaptive schemas and schema modes	Maladaptive schema modes (vulnerable child, detached protector, punitive parent)	Limited reparenting and mode work

The four structured, evidence-based borderline psychotherapies; the two highlighted cells are the discriminator of this topic, MBT restores mentalising through a stance while TFP interprets the transference to integrate split object relations.

INDIA

Personality disorders are markedly under-recognised in Indian practice, and the specialist psychotherapies are scarcer still. Dialectical behaviour therapy has the widest, if uneven, adaptation and delivery at Indian tertiary and private centres; mentalisation-based therapy and transference-focused psychotherapy are far less available, concentrated in a few academic and psychodynamically trained settings, and the binding constraint is the training and supervision pipeline rather than patient demand. Two cautions travel with the models. First, mentalising norms are cultural: the we-mode, joint attention and the expression of affect are read differently across Indian family systems, and interdependence must not be mistaken for a mentalising failure. Second, the object-relations reading of a case must be held against the patient's own cultural idiom, so that a culturally normative deference or a shared family self is not over-interpreted as split or diffuse identity. Judge the mind against its own context, not a single culture's template.

HOLD THIS

MBT (Bateman and Fonagy) restores lost mentalising through a curious, not-knowing stance and does not interpret; TFP (Clarkin and Kernberg) integrates split object-relations dyads by interpreting the here-and-now transference, and does; both work in the present through the therapeutic relationship, and both sit as evidence-based options beside dialectical behaviour therapy and schema therapy.

16 . Pharmacotherapy in personality disorders

The most trapped question on the day: which drug? The examinable truth about **pharmacotherapy in personality disorders** is almost entirely a truth about restraint. There is no medication registered to treat any personality disorder, structured psychological therapy is the primary treatment, and every drug that is used is used **symptom-targeted** and adjunctive, aimed at a defined target symptom or a comorbidity, never at the disorder itself. The single fact worth more than any dose is the NICE position on borderline personality disorder: drug treatment is not first-line, and the candidate who reaches for a mood stabiliser or an antipsychotic as the answer has walked into the classic trap.

This fragment leads with the guideline law, states the NICE rule as both a rule and a trap, then reconciles it with what the wider bodies (the Indian Psychiatric Society CPG and WFSBP) permit at symptom-domain level. It teaches the three psychobiological symptom domains and the agent class attached to each, keeps the pharmacology deliberately light, and cross-references the deep drug detail to the Mood disorders: pharmacotherapy notes and the Schizophrenia: management, antipsychotics and adverse effects notes. The bipolar-II differential that so often triggers a wrong prescription sits in the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes, and the forensic frame for antisocial personality disorder in the Mental health legislation and forensic psychiatry notes.

16.1 The governing principle: adjunctive, symptom-targeted, never disorder-specific

No drug licensed. The organising principle of **pharmacotherapy in personality disorders** is that it is never a disorder-specific registered treatment. **No drug licensed** for any personality disorder exists in any major formulary, and every guideline body, however permissive, agrees that psychotherapy is primary and medication is at most an adjunct. What medication can legitimately do is act on a discrete **symptom-targeted** problem, an aggressive outburst, a run of cognitive-perceptual disturbance, a depressive or anxious swing, or a comorbid Axis-I disorder sitting alongside the personality pathology. It follows that a drug is prescribed against a named, measurable target with a defined review point, and is stopped if that target does not move. The diagnosis itself must be secure before any of this begins: an enduring, pervasive, inflexible pattern with onset by adolescence, not a situational state, and for borderline personality disorder that means the full pattern (five or more of the nine criteria), not a single stormy fortnight.

PEARL

Read every personality-disorder drug question as a question about a target symptom, not about the disorder. "What is the drug treatment for borderline personality disorder?" has no correct drug answer, the correct answer is that structured psychological therapy is the treatment and medication is only a time-limited, symptom-targeted adjunct. Reframing the question this way is what protects you from the trap the stem is usually setting.

16.2 The NICE rule: drug not first-line for borderline

The most tested management fact of the day. NICE (2009, CG78) states that drug treatment should not be used specifically for borderline personality disorder or for the individual symptoms and behaviours associated with it (repeated self-harm, marked emotional instability, risk-taking, transient psychotic symptoms). Structured psychological therapy is primary. Medication is legitimate in only two situations: to treat a diagnosed comorbid condition, or short-term during a crisis (a sedative, usually for no longer than one week). NICE guidance is explicit that antipsychotic drugs should not be used for the medium- and long-term treatment of the disorder. This is the highest-yield line of the topic, and a mis-statement of it is a critical error.

RULE **Drug not first-line for borderline.** In borderline personality disorder, structured psychological therapy is primary; any medication is symptom-targeted, adjunctive and time-limited, for a comorbidity or a crisis only, and antipsychotics are not for medium- or long-term use (NICE 2009).

TRAP **Reaching for a drug first.** Starting a mood stabiliser or antipsychotic as the primary treatment for borderline personality disorder, or for its self-harm and lability directly, is the classic error and contradicts NICE outright; there is no licensed disorder-specific drug to reach for.

0	3	1
DRUGS LICENSED FOR BORDERLINE PERSONALITY DISORDER	SYMPTOM-TARGET DOMAINS FOR ADJUNCTIVE DRUGS	WEEK, USUAL MAXIMUM FOR CRISIS SEDATION (NICE)

16.3 Reconciling the guidelines: NICE, the Indian Psychiatric Society, WFSBP

Same primacy of psychotherapy, differing latitude on adjuncts. The bodies agree that psychological therapy is the primary treatment and that no drug is a registered disorder-specific treatment; they differ in how much symptom-targeted prescribing they permit. NICE is the most restrictive (no drug for the disorder or its symptoms; a crisis sedative or comorbidity treatment only). The Indian Psychiatric Society CPG (IPS 2023) makes psychotherapy central but allows that, in some patients, antipsychotics may reduce hostility, suspiciousness, affect dysregulation and cognitive-perceptual distortions, and that mood stabilisers may reduce impulsivity and aggressiveness. WFSBP (2007) supplies the fullest symptom-domain framework for adjunctive agents. Hold NICE as the safe default answer and the domain framework as the "where is there any evidence" detail.

GUIDELINE (YEAR)	POSITION ON DRUG TREATMENT	WHAT IT PERMITS
NICE, CG78 (2009)	**Do NOT use drugs specifically for borderline personality disorder or its symptoms; no antipsychotic for medium- or long-term use**	Treat a comorbidity; a crisis sedative for usually no longer than one week; review and deprescribe
NICE, personality disorders (2015)	No drug for the personality itself; structured clinical assessment to diagnose	Antipsychotic or sedative only for short-term crisis or a comorbid condition; group cognitive-behavioural therapy for antisocial personality disorder
the Indian Psychiatric Society CPG (2023)	Psychotherapy is central; DBT recommended early	Symptom-targeted antipsychotics and mood stabilisers in some patients; avoid benzodiazepines
WFSBP (2007)	Adjunctive, symptom-targeted only, not a registered treatment	Agent chosen by symptom domain; trial for at least three months against defined targets
APA (2024)	Structured psychotherapy primary; medication adjunctive	Time-limited, single measurable target symptom; reconcile and review medications at least every six months

The guideline hierarchy for these notes: NICE leads and is the safest default, the Indian Psychiatric Society and WFSBP add the symptom-domain latitude, APA supplies the core prescribing principles.

16.4 The three symptom domains and the agent attached to each

The Soloff-style psychobiological domains organise all adjunctive prescribing. When medication is used at all, it is targeted at one of three symptom domains rather than at the disorder, a framework running from Soloff's psychobiological model through the APA and WFSBP schemes. The **cognitive-perceptual** domain (suspiciousness, ideas of reference, transient quasi-psychotic or dissociative experiences) attracts a low-dose antipsychotic; the **impulsive-behavioural dyscontrol** domain (impulsive aggression, self-damaging impulsivity, anger) attracts a mood stabiliser, with a low-dose antipsychotic as an alternative where aggression dominates; and the **affective instability domain** (affective dysregulation, depressive mood swings, anxiety) attracts an SSRI. The mapping is the examinable core, shown in the accompanying figure.

SYMPTOM DOMAIN	TARGET FEATURES	AGENT CLASS WITH ADJUNCTIVE EVIDENCE
Cognitive-perceptual	Suspiciousness, ideas of reference, transient quasi-psychotic and dissociative symptoms	**Low-dose antipsychotic (second-generation)**
Impulsive-behavioural dyscontrol	Impulsive aggression, self-damaging impulsivity, anger and behavioural discontrol	**Mood stabiliser (a low-dose antipsychotic where aggression dominates)**
Affective instability and anxiety	Affective dysregulation, depressive mood swings, rejection sensitivity, anxiety	**SSRI**

The three psychobiological symptom domains and the drug class attached to each; this is the framework the wider guidelines use, always as an adjunct to primary psychotherapy, never as a treatment for the disorder.

16.5 Low-dose antipsychotics: cognitive-perceptual and impulsive domains

Low, short, and for a named target only. A **low-dose antipsychotic**, a second-generation agent such as quetiapine (Qutipin, Seroquel), olanzapine (Oleanz, Zyprexa) or aripiprazole (Arip, Abilify), carries moderate adjunctive evidence in the **cognitive-perceptual** domain and, where impulsivity and aggression dominate, in **impulsive-behavioural dyscontrol** (WFSBP 2007; IPS 2023). The doses used are low and the course is meant to be short and target-defined; the deep pharmacology, receptor profiles and adverse effects belong to the Schizophrenia: management, antipsychotics and adverse effects notes and are not re-taught here. The overriding constraint is the NICE one: an antipsychotic is legitimate short-term, but must not be continued for the medium- and long-term treatment of borderline personality disorder, so a crisis prescription is reviewed and stopped once the crisis resolves.

16.6 Mood stabilisers: impulsive aggression

For impulsive aggression, not for a personality-based "mood swing". A **mood stabiliser**, valproate or divalproex (Valprol, Encorate), lamotrigine (Lamitor, Lametec) or topiramate, has adjunctive evidence for reducing impulsivity and impulsive aggression that antipsychotics have not settled (IPS 2023; WFSBP 2007). The trap this sets is the borderline-versus-bipolar-II confusion: the rapid, reactive affective instability of borderline personality disorder, minutes to hours and triggered by interpersonal events, is not the sustained syndromic mood episode of bipolar II, and starting a mood stabiliser for personality-based lability, mislabelled as bipolarity, is a common and consequential error. That differential is developed in the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes, and the deep drug detail in the Mood disorders: pharmacotherapy notes; here the mood stabiliser stays a targeted adjunct for impulsive aggression.

TRAP **Treating borderline lability as bipolar II.** Rapid, reactive, interpersonally cued affective instability is a borderline feature, not a bipolar mood episode; reflexively starting a mood stabiliser for it medicalises a personality target and misses the primary psychotherapy.

16.7 SSRIs: the affective instability and anxiety domain

Best-shown class for affective dysregulation and anxiety, still adjunctive. Of the drug classes, the SSRI has the clearest evidence for the **affective instability domain**, the depressive mood, mood swings and anxiety of the disorder (WFSBP 2007), and an SSRI such as fluoxetine (Fludac, Prozac) or sertraline (Serlift, Zoloft) is a reasonable adjunct where that domain predominates or where a comorbid depressive or anxiety disorder is present. It also carries the practical advantage of relative safety in overdose, which matters in a population with recurrent self-harm. As always, this is symptom-targeted and adjunctive to psychotherapy, and where an SSRI is used for a comorbidity it is that comorbidity, not the personality disorder, that is being treated; the deep detail sits in the Mood disorders: pharmacotherapy notes.

16.8 The cautions: avoid polypharmacy, deprescribe, and what never to use

The prohibitions are as examinable as the indications. Several cautions run across the guidelines. Avoid benzodiazepines: the Indian Psychiatric Society CPG advises against them in borderline personality disorder because of dependence risk and disinhibition that can worsen impulsive behaviour. Avoid drugs dangerous in overdose: WFSBP counsels against irreversible MAOIs and lithium despite some efficacy, precisely because these patients need agents safe in parasuicidal acts. Above all, **avoid polypharmacy** and long-term prescribing without a clear indication: NICE guidance is to review repeat prescriptions regularly, to discourage polypharmacy, and to withdraw drugs started in a crisis once it resolves, aiming to reduce and stop unnecessary treatment. The maintenance rule that antipsychotics are not for medium- or long-term use in borderline personality disorder belongs here too.

> **COMMON TRAP**
>
> A borderline patient accumulates a low-dose antipsychotic, a mood stabiliser, an SSRI and a "when required" benzodiazepine over successive crises, none reviewed. This is exactly the polypharmacy NICE tells you to unwind: benzodiazepines should be off entirely, the crisis-era drugs deprescribed once the crisis has passed, and the whole regimen reconciled against a single measurable target. Deprescribing is an active clinical act, not neglect.

16.9 Crisis, comorbidity, and antisocial personality disorder

Where a drug is genuinely indicated, and where none is. Two legitimate windows remain. In a crisis, short-term cautious sedation (an antihistamine such as promethazine, or a low-dose antipsychotic) may be considered as part of an overall plan, usually for no longer than one week, alongside risk assessment and a collaborative crisis plan (NICE 2009). For a diagnosed comorbid depression, post-traumatic stress or anxiety disorder, that comorbidity is treated on its own terms within the structured programme. Antisocial personality disorder is the instructive contrast: there is no drug treatment for the antisocial personality itself, the mainstay is a group cognitive-behavioural intervention (often in criminal-justice or health settings), and pharmacotherapy is reserved strictly for a comorbid mental disorder (NICE 2015). The offender-assessment and court-report frame is cross-referenced to the Mental health legislation and forensic psychiatry notes.

WORKED EXAMPLE

A woman in her twenties with borderline personality disorder presents in crisis after an argument, with rising urges to self-harm and transient paranoid ideas. The correct plan is not to "start" a drug for the disorder: assess risk, enhance coping and reflective capacity, agree a crisis plan, and, if sedation is needed, offer it cautiously and briefly (usually no longer than one week), reviewing to stop it as the crisis settles. The definitive treatment is referral to a structured psychological therapy such as dialectical behaviour therapy; any antipsychotic used for the cognitive-perceptual symptoms is short-term and target-defined, never a maintenance prescription.

16.10 Place in the plan, and the Indian picture

Medication sits inside a psychotherapy-led, stepped plan. The management spine holds: outpatient care by default with admission avoided and, when unavoidable, brief and purposeful; short-term focus on crisis and safety, mid-term on the core features through structured psychological therapy, long-term on function and relationships. Within that, pharmacotherapy is a symptom-targeted, time-limited adjunct, layered onto (never in place of) dialectical behaviour therapy, schema therapy, mentalisation-based treatment or transference-focused psychotherapy by availability and fit. The candidate who leads with a drug has inverted the plan.

INDIA

Personality disorders are markedly under-recognised in Indian practice, so many patients reach services only through a self-harm or crisis presentation and are prescribed a symptom drug without ever being formulated or offered the structured therapy that is the actual treatment. Two realities shape a sensible plan. Access: dialectical behaviour therapy and schema therapy are being delivered and culturally adapted at a small number of Indian tertiary and private centres rather than being widely available, so the psychotherapy-first rule can be hard to enact and makes disciplined, minimal prescribing (and active deprescribing) all the more important. Culture: personality pathology must never be read through a single culture's norms, its expression, and the antisocial and medico-legal framing in particular, differs by setting, and the forensic use of an antisocial diagnosis carries weight that belongs with the forensic notes, not with a prescription pad.

GOOD TO REMEMBER

- **Pharmacotherapy overall:** mechanism is symptom-targeted and adjunctive (never disorder-specific; no drug licensed); indication is a defined target symptom, a comorbidity, or short-term crisis sedation; signature caution is that it is not first-line for borderline personality disorder, psychotherapy is primary, and antipsychotics are not for medium- or long-term use (NICE 2009).
- **Low-dose antipsychotic:** targets the cognitive-perceptual domain (and impulsive aggression where it dominates); short-term and low-dose; caution is no medium- or long-term use in borderline personality disorder.
- **Mood stabiliser:** targets impulsive-behavioural dyscontrol and impulsive aggression; caution is not to mistake reactive borderline lability for bipolar II and medicalise a personality target.
- **SSRI:** targets the affective instability and anxiety domain and any comorbid depression or anxiety; advantage is relative safety in overdose; caution is it treats the symptom or comorbidity, not the disorder.
- **Never:** avoid benzodiazepines (dependence, disinhibition), avoid drugs dangerous in overdose (irreversible MAOIs, lithium), and avoid polypharmacy; review and deprescribe.

HOLD THIS

No drug is licensed for a personality disorder; structured psychological therapy is primary and NICE says drug treatment is not first-line for borderline personality disorder, so all pharmacotherapy is symptom-targeted and adjunctive, a low-dose antipsychotic for the cognitive-perceptual domain, a mood stabiliser for impulsive-behavioural dyscontrol, an SSRI for the affective instability and anxiety domain, for a comorbidity or a crisis only, while you avoid benzodiazepines, avoid polypharmacy, and deprescribe.

17 . The integrated management of borderline personality disorder

The treatment block, assembled into one plan. The separate topics teach the disorder and each therapy on its own; this one is the synthesis, the single algorithm an examiner wants when the question is simply "how would you manage this patient". The integrated management of borderline personality disorder is *integrated* in two senses: it braids the strands of care (crisis work, structured psychotherapy, selective medication, care planning and psychoeducation) into one coherent plan, and it reconciles the guideline bodies (NICE, the Indian Psychiatric Society and WFSBP) into one defensible line. The plan is built on a three-part spine, **LOCUS** (where care happens), **FOCUS** (what is targeted, and when) and **MODUS** (by what means), and it is anchored, above everything, on one rule that examiners test relentlessly: a drug is not the first-line treatment for this disorder.

This fragment walks the spine end to end. It fixes the **locus** of care as outpatient and community by default, sets the **focus** by phase from short-term crisis and risk through the mid-term core-feature work to long-term function and remission, and lays out the **modus**, with structured psychological therapy as the primary mode and medication kept symptom-targeted, time-limited and adjunctive. Throughout, the guideline order is NICE first (CG78), then the Indian Psychiatric Society CPG and WFSBP, and the pharmacology stays deliberately light, with the deep drug detail cross-referenced to the Mood disorders: pharmacotherapy notes and the Schizophrenia: management, antipsychotics and adverse effects notes. The integrated algorithm is drawn in the accompanying figure.

4	0	1 wk
STRUCTURED PSYCHOTHERAPIES FOR THE DISORDER (DBT, SCHEMA, MBT, TFP)	DRUGS LICENSED FOR BORDERLINE PERSONALITY DISORDER (NICE)	USUAL CEILING FOR A CRISIS SEDATIVE (NICE)

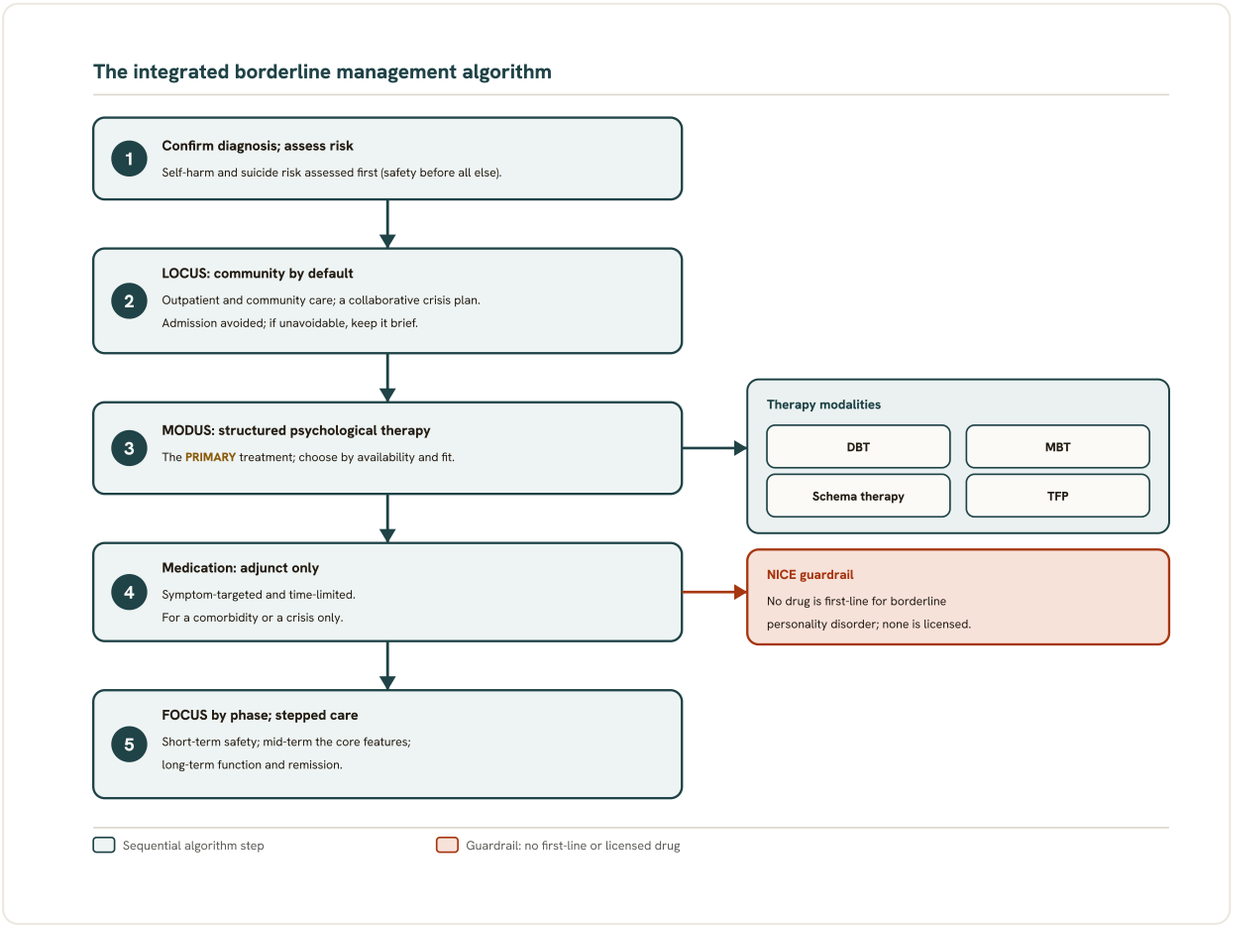


Fig 16.13 The integrated plan treats borderline personality disorder mostly in the community with a crisis plan, makes structured psychological therapy (dialectical behaviour therapy, mentalisation-based therapy, schema therapy or transference-focused psychotherapy) the primary treatment, and uses medication only, symptom-targeted and time-limited, for a comorbidity or a crisis, because no drug is first-line or licensed for the disorder.

17.1 The integrated frame, and the one rule that governs it

One plan, one governing rule. Before the spine, fix the principle that shapes every branch of it. NICE (2009, CG78) states that drug treatment should not be used specifically for borderline personality disorder or for its individual symptoms (repeated self-harm, marked emotional instability, risk-taking, transient psychotic symptoms), that no drug is licensed for the disorder, and that structured psychological therapy is the primary treatment; medication is reserved for a comorbid condition or short-term use in a crisis. The Indian Psychiatric Society (IPS 2023) agrees, making psychotherapeutic methods the central treatment and permitting only symptom-targeted adjuncts; WFSBP (2007) frames any drug as adjunctive and symptom-domain-targeted rather than disorder-specific. The three bodies differ only in how much room they leave for adjunctive medication, NICE the most restrictive, IPS and WFSBP allowing symptom-targeted agents, and they are unanimous that psychotherapy is primary. The whole integrated plan is therefore a psychotherapy-led plan into which crisis care, selective medication and care planning are fitted.

RULE **Structured psychological therapy is primary; any medication is adjunctive.** In borderline personality disorder the first-line treatment is a comprehensive, structured, theory-based psychotherapy delivered by trained supervised practitioners; medication is only ever symptom-targeted, time-limited and reserved for a comorbidity or a crisis (NICE 2009, IPS 2023, WFSBP 2007).

TRAP **Reaching for a drug first-line for the disorder itself.** Starting an antipsychotic or a mood stabiliser as the primary treatment for borderline instability, or continuing antipsychotics for medium- and long-term management, runs directly against NICE (which recommends no drug for the disorder and no antipsychotic for its longer-term treatment). A wrong first-line for borderline personality disorder is a critical error.

17.2 LOCUS, where the care happens

Outpatient and community by default; admission avoided, and when unavoidable, brief and purposeful. The **locus** of care is the community. Borderline personality disorder is managed in outpatient and community settings within a stepped care framework, matching intensity to need and stepping up only as required. Inpatient admission is actively avoided, because prolonged admission tends to entrench regression, reinforce crisis behaviour and disrupt the very relationships and roles that recovery depends on; NICE is explicit that admission should be avoided unless there is a serious risk to self or others that cannot be managed any other way, and that when it does happen it should be brief and for an agreed, specific purpose (typically the containment of acute risk), not open-ended asylum. The **service frame** matters as much as the setting: people with the disorder must not be excluded from any health or social care service because of the diagnosis or because they have self-harmed, and any planned change or withdrawal of a service is agreed with the person through a structured, phased plan rather than an abrupt discharge. Community continuity, not the ward, is the therapeutic base.

COMMON TRAP

Admitting the recurrently self-harming borderline patient to a general ward for safety, and keeping them. Beyond the brief, purposeful containment of an unmanageable acute risk, prolonged admission is not protective; it interrupts the outpatient therapy that actually reduces self-harm, models the crisis as the route to care, and is where iatrogenic regression sets in. The default is to manage the crisis in the community and return the person to their structured programme.

17.3 FOCUS, the target shifts by phase

Three phases, three different jobs. The **focus** of the work is not static; it moves as the patient stabilises. In the *short term* the target is safety, the job is crisis management and **risk** (containing self-harm and suicidality, and building a collaboratively agreed crisis plan). In the *mid term* the target is the core disorder itself, the emotion dysregulation, identity disturbance, abandonment sensitivity and unstable relationships, addressed through a sustained course of structured psychological therapy. In the *long term* the target is life beyond symptoms, function, relationships, work or study, and consolidated remission, which for many patients is a realistic goal given that borderline personality disorder has a substantially remitting long-term course. Sequencing

matters: trauma processing and deep character work wait until behavioural safety is established, so a plan that opens with intensive trauma exposure while the patient is still cutting weekly has the phases in the wrong order.

PHASE	PRIMARY TARGET (THE FOCUS)	THE MAIN WORK
Short-term	**Safety, risk and the acute crisis**	Risk assessment, crisis management, a collaboratively agreed crisis plan with identified triggers; brief, purposeful containment only if risk is otherwise unmanageable
Mid-term	The core disorder features	A sustained course of structured psychological therapy (the emotion dysregulation, identity, abandonment and relational instability)
Long-term	Function, relationships and remission	Rehabilitation into role and relationship, relapse prevention, and consolidation of gains toward remission

The focus of the integrated plan shifts by phase; safety is secured before core-feature therapy, and function follows.

17.4 The short-term focus, crisis management, risk and self-harm

Assess the risk, hold the person, agree a plan, avoid the ward. When a patient presents in crisis, the sequence is concrete. Assess the current **risk** to self and others; ask what has happened before and what helped then; help the person manage the surge of anxiety by enhancing coping and reflective capacity rather than simply removing them from their life; and avoid hospital admission unless there is a serious risk that cannot be managed otherwise (NICE 2009). The durable output of a crisis contact is a crisis plan, agreed collaboratively and documented, that names the person's individual triggers, the early-warning signs, the self-management steps and skills to use first, and exactly whom to contact and how, so that the next crisis has a rehearsed pathway that is not the emergency department by default (IPS 2023 makes the collaboratively agreed crisis plan central to reducing hospitalisation in acutely suicidal patients). Self-harm is managed within, not outside, this frame: it is understood, taught and responded to inside the structured therapy, and the person is never excluded from care because of it. Where a sedative is genuinely needed to get through an acute crisis, its use is short-term and time-limited (see the medication topic below), and benzodiazepines are avoided.

WORKED EXAMPLE

A woman known to the community team presents at night after superficial cutting following a relationship rupture, distressed but not acutely suicidal on assessment. The integrated response is not admission. The clinician assesses current risk, reviews what helped in previous episodes, helps her down-regulate the immediate arousal, and works with her on the crisis plan, updating the trigger list, rehearsing the distress-tolerance skill to use first, and confirming the daytime contact route back into her structured therapy. If night-time distress is extreme, a short-term sedative for a few days may be considered as part of the plan, not a new standing prescription, and she returns to her outpatient programme rather than to a ward.

17.5 MODUS, structured psychological therapy as the primary mode

The primary means is a structured, theory-based psychotherapy, delivered whole. The **modus** of the plan is led by structured psychological therapy. The question is rarely whether to offer psychotherapy but *which structured psychotherapy*, chosen by availability, patient fit and the dominant problem: dialectical behaviour therapy (DBT, Marsha Linehan), the best-evidenced option where reducing recurrent self-harm is the priority; schema therapy (Jeffrey Young), where enduring maladaptive patterns and identity are central; mentalisation-based treatment (MBT, Bateman and Fonagy), where the loss of mentalising under attachment stress dominates; and transference-focused psychotherapy (TFP, Clarkin and Kernberg), an object-relations treatment for the patient who can tolerate a long interpretive therapy. What matters more than the brand is that whichever is chosen is comprehensive, theory-based, of adequate duration and delivered by trained, supervised practitioners; NICE warns specifically against brief psychological interventions of less than three months offered outside a structured, supervised service. Each modality has its own topic; the wider models and technique are developed in the Psychotherapies, clinical assessment, MSE and nosology notes.

STRUCTURED THERAPY	CORE MECHANISM (WHAT IT TARGETS)	BEST-FIT INDICATION
Dialectical behaviour therapy (DBT)	**Emotion dysregulation from a biosocial deficit; skills plus acceptance-change balance**	Recurrent self-harm and suicidality the priority (the first-line, best-evidenced choice for self-harm)
Schema therapy	Early maladaptive schemas and schema modes; limited reparenting	Pervasive lifelong patterns and identity work; where relational-pattern change is central
Mentalisation-based treatment (MBT)	Restoring mentalising that collapses under attachment-stress arousal	Marked mentalising failures and attachment-driven relational storms
Transference-focused psychotherapy (TFP)	Integrating split self-other representations through the here-and-now transference	Identity diffusion in a patient able to tolerate a long, interpretive treatment

The four structured psychotherapies of the modus; the choice is by availability and fit, and all four have randomised support. Delivery must be comprehensive and of adequate duration.

MNEMONIC

LOCUS, FOCUS, MODUS

The integrated plan on one spine. **LOCUS**, where: outpatient and community, stepped care, admission avoided (and brief and purposeful if unavoidable). **FOCUS**, what and when: short-term safety and risk, mid-term the core disorder via structured therapy, long-term function and remission. **MODUS**, by what means: structured psychological therapy primary, medication symptom-targeted and time-limited, plus multidisciplinary care planning and psychoeducation. The three read as a single sentence, care in the community, targeted by phase, delivered chiefly by psychotherapy.

17.6 Schema therapy done properly, the mode framework

The therapy that reads the disorder as shifting modes, and reparents them. Because it is the lens many Indian trainees will use, schema therapy deserves its structure stated cleanly. It understands borderline personality disorder not as one steady state but as rapid flips between *schema modes*, moment-to-moment states each schema activates. In the borderline patient the recurring modes are the **vulnerable child** (the abandoned, abused, frightened core the whole treatment aims to protect and heal), the **angry and impulsive child** (raw rage and impulsive self-harm when needs go unmet), the **detached protector** (the numbing, dissociative, disconnected withdrawal that shields the child from feeling), the **punitive and demanding parent** (the internalised critical voice that attacks the self and drives self-harm and guilt), and the **healthy adult** (the mode the therapy builds up to meet needs adaptively). The signature mechanism is *limited reparenting*: within professional bounds the therapist provides, in a corrective form, the safety, validation and limits the patient's developmental environment failed to supply, and through mode work (including chair work) weakens the maladaptive modes and strengthens the healthy adult. It is delivered as a longer-term treatment, and its randomised evidence in borderline personality disorder is a genuine strength.

GOOD TO REMEMBER

- **Structured psychological therapy (the primary mode):** mechanism, whichever of DBT (biosocial emotion dysregulation, skills plus the acceptance-change dialectic), schema therapy (schema modes healed by limited reparenting), MBT (restoring mentalising under attachment stress) or TFP (integrating split object representations in the transference) fits; all four are comprehensive, theory-based and of adequate duration. Indication, borderline personality disorder as the first-line treatment; the choice is by availability and fit, with DBT the best-evidenced where recurrent self-harm is the priority. Signature caution, a brief or fragmentary intervention of less than three months outside a structured supervised service is not adequate treatment, and behavioural safety precedes deep trauma or character work.

17.7 MODUS, the place of medication, symptom-targeted and time-limited

Not for the disorder; for a comorbidity or a crisis, by symptom domain. Medication is the adjunctive, not the primary, arm of the modus, and its rules follow the guideline order. NICE (2009) is the most restrictive: no drug for the disorder or its symptoms, no antipsychotics for medium- and long-term treatment, and any drug started in a crisis reviewed and stopped once

the crisis resolves, with regular review of repeat prescriptions and active discouragement of polypharmacy. Two legitimate windows remain. The first is **medication for comorbidity**: a co-occurring depressive, anxiety, post-traumatic or other disorder is treated on its own merits, within the structured borderline programme. The second is the short-term crisis: a sedative (for example a sedating antihistamine, or a low-dose antipsychotic) may be used cautiously as part of the plan, usually for no longer than one week. Where IPS and WFSBP permit symptom-targeted adjuncts, they are matched to the dominant symptom domain, and Indian brand names are given first with the pharmacology kept light and cross-referenced. Two agents to avoid: benzodiazepines (dependence and disinhibition, IPS), and drugs unsafe in overdose such as irreversible MAOIs and lithium as a first choice (WFSBP), because these patients must be given drugs safe in parasuicidal acts.

SYMPTOM DOMAIN	ADJUNCTIVE CLASS (INDIAN BRAND FIRST; LIGHT)	GUIDELINE NOTE
Cognitive-perceptual (suspiciousness, transient quasi-psychotic symptoms)	**Low-dose atypical antipsychotic, quetiapine (Qutipin) or olanzapine (Oleanz)**	WFSBP first among drugs here; NICE forbids medium- and long-term antipsychotic use, so short, targeted only
Affective dysregulation (depressed mood, anxiety, mood swings)	SSRI, sertraline (Serlift) or fluoxetine (Fludac)	WFSBP best-shown class for this domain; treat true comorbid depression or anxiety on its merits
Impulsive-behavioural dyscontrol and aggression	Low-dose atypical antipsychotic; mood stabiliser, valproate or divalproex (Valprol, Encorate), if antipsychotic insufficient	IPS and WFSBP symptom-targeted option; not a disorder-specific treatment
Acute crisis (short-term only)	A sedative, a sedating antihistamine or low-dose antipsychotic	NICE, cautious, part of the plan, usually no longer than one week; review and stop

Adjunctive medication is symptom-domain-matched, time-limited and reserved for a comorbidity or a crisis; the deep dosing sits in the pharmacotherapy notes. Doses are not re-taught here.

PEARL

The most useful prescribing question in a borderline patient already on three or four psychotropics is not "what to add" but "what to stop". NICE asks you to review people prescribed drugs without a diagnosed comorbidity, aim to reduce and discontinue treatments started in past crises, and discourage polypharmacy. A careful deprescribing review is often the single most guideline-concordant medication act you can perform, and it protects a group who are, by the nature of the disorder, at high overdose risk.

17.8 Multidisciplinary care planning, psychoeducation and the service frame

The connective tissue that holds the plan together. Around the therapy and the selective medication sit the elements that make the plan durable. A comprehensive **multidisciplinary care plan** is developed with the person (and carers where appropriate), documenting the agreed goals, the roles of each team member, the crisis plan with its identified triggers, and the long-term aims for education and employment; it names who does what and is the reference point every

team member works from, which is also how a patient prone to splitting is held by a consistent, communicating team rather than played between its members. **Psychoeducation** is therapeutic in its own right: a collaborative, de-stigmatising explanation of the condition, its typically remitting long-term course, and the rationale for a psychotherapy-led plan improves engagement and reduces the hopelessness the diagnosis can carry. The **service frame** closes the loop, no exclusion from services because of the diagnosis or self-harm, agreed supervision for the staff who do this demanding work, and a structured, phased handover whenever care is transferred, so that the person never falls through the gap between services.

17.9 The guideline synthesis, NICE then IPS and WFSBP reconciled

One line the three bodies will all sign. The examiner-safe synthesis is easy to hold once the hierarchy is clear. All three agree that structured psychotherapy is primary and that no drug is a registered treatment for the disorder; they differ only in how much adjunctive medication they permit. NICE (CG78, 2009) leads and is the most restrictive, no drug for the disorder, no long-term antipsychotic, drugs only for a comorbidity or briefly in a crisis. The Indian Psychiatric Society (IPS 2023) makes psychotherapy central, names DBT for the early reduction of emotion dysregulation and self-harm, builds the plan around a collaboratively agreed crisis plan, and permits symptom-targeted antipsychotics or mood stabilisers in some patients while forbidding benzodiazepines. WFSBP (2007) supplies the symptom-domain map for those adjuncts and insists any drug be trialled against clear targets for at least three months and stopped if it does not help. Read together (the guideline stack, nice ips wfsbp), the message is one: lead with structured psychological therapy, use medication sparingly and by symptom, and review it to stop.

GUIDELINE	POSITION ON PSYCHOTHERAPY	POSITION ON MEDICATION
NICE 2009 (CG78), the home guideline	**Structured, theory-based psychotherapy is primary; DBT or MBT where self-harm reduction is the priority**	No drug for the disorder; no medium- or long-term antipsychotic; only for a comorbidity or short-term in a crisis (a sedative, usually up to one week)
Indian Psychiatric Society 2023	Psychotherapy central; DBT early for emotion dysregulation and self-harm; a collaboratively agreed crisis plan	Symptom-targeted antipsychotics or mood stabilisers in some patients; benzodiazepines avoided
WFSBP 2007	Psychotherapy primary; pharmacotherapy adjunctive only	Symptom-domain-matched (antipsychotic for cognitive-perceptual and impulsive, SSRI for affective); avoid MAOIs and lithium first; trial at least three months

The three guideline bodies reconciled; they converge on psychotherapy-primary and diverge only on the room left for symptom-targeted adjuncts, NICE most restrictive.

17.10 The integrated plan in Indian practice

Where the algorithm meets the ground here. The integrated plan is drawn as an algorithm in the accompanying figure; four India realities shape how it runs. First, *under-recognition*,

personality disorders are markedly under-recognised in routine Indian practice, and borderline patients often reach services labelled only by their crises, self-harm, an overdose, a mood or substance comorbidity, so the disorder is captured, and the psychotherapy-led plan started, only when it is actively looked for. Second, *availability and cultural adaptation*, the structured psychotherapies the plan depends on are concentrated in a few tertiary and metropolitan centres; DBT and schema therapy are being trained and culturally adapted in India (attending to family involvement, idioms of distress and the collectivist framing of autonomy and relationship), but access is uneven, and a realistic plan often blends available structured elements with skilled generic clinical management rather than assuming a full programme. Third, the *medico-legal frame*, most forcefully around antisocial rather than borderline personality disorder, is where the diagnosis carries legal weight, and offender assessment, court reports and the prediction of dangerousness are developed in the Mental health legislation and forensic psychiatry notes. Fourth, the *cultural expression of personality pathology*, deviation is judged against the person's own cultural, social and economic milieu, so that culturally sanctioned dependence, help-seeking idioms or situational distress are not misread as trait pathology, and the disorder is not read through a single culture's norms.

INDIA

A pragmatic Indian version of the integrated plan holds the spine and adapts the resourcing. The locus stays outpatient (admission is even less desirable where beds are scarce and asylum-style regression easy to induce), and the crisis plan is built around the family and, for many patients, WhatsApp-reachable daytime contact rather than a formal crisis line. The modus leads with whatever structured therapy is genuinely available, DBT-informed skills work most often, with schema therapy where a trained therapist exists, delivered with cultural adaptation; medication stays symptom-targeted and minimal, with Indian brands (quetiapine as Qutipin, sertraline as Serlift) named first but kept light and, crucially, reviewed to stop. The single most common local error is the mirror image of the guideline: a borderline patient carried for years on rotating polypharmacy with no structured psychotherapy ever started. Distinguishing borderline instability from bipolar II before reaching for a mood stabiliser is developed in the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes, and the complex-PTSD boundary in the Anxiety, OCD and trauma-related disorders notes.

GOOD TO REMEMBER

- **Borderline personality disorder (the disorder being managed):** defining criterion set, a pervasive pattern of instability of affect, self-image and relationships with marked impulsivity, diagnosed on five or more of the nine DSM-5-TR criteria (and captured as the borderline pattern qualifier on the ICD-11 dimensional model). First management step, establish safety and a collaboratively agreed crisis plan, then start structured psychological therapy as the primary treatment. The discriminator that changes management, it is an enduring, pervasive, inflexible pattern with onset by adolescence, not a situational affective lability and not bipolar II, so the answer is a psychotherapy-led plan, not a mood stabiliser started first-line.

HOLD THIS

Borderline personality disorder is managed community-first on the LOCUS, FOCUS, MODUS spine, with structured psychological therapy as the primary treatment and medication only symptom-targeted, time-limited and for a comorbidity or a crisis: NICE says no drug is first-line for the disorder, and starting one is the trap.

Apply and consolidate

18 . Worked cases

The arc closer, where the chapter is put to work. A personality-disorders question is rarely won on the criteria list alone; it is won on the **formulation**, the reasoning that carries a presentation through the general criteria, to a named diagnosis, past its mimics, into a model of why the person is the way they are, and out to a defensible first-line plan. Long cases and vivas test exactly this movement, so this fragment reasons four worked cases end to end rather than restating the facts the earlier topics have already fixed. Each case is a **worked example** in the strict sense: the presentation is given, and then the discriminators, the model and the management are argued out loud.

The four are chosen to exercise the four highest-yield skills of the chapter. The first is a borderline personality disorder case formulated on both the Linehan biosocial and the schema-modes models and treated with structured psychotherapy, with medication kept symptom-targeted; the second works the borderline-versus-bipolar-II discrimination through the discriminator grid to the correct diagnosis and the correct first-line treatment; the third is an antisocial forensic vignette, with its conduct-disorder history, its structured risk assessment and its medico-legal frame; and the fourth is a cluster-C (anankastic) case. Read across them and one spine repeats, laid out in the figure alongside: clear the general criteria, count and type, discriminate from the mimic, formulate, and treat with psychotherapy first.

3	5	9	4
DSM-5 CLUSTERS (A, B AND C)	ICD-11 TRAIT DOMAINS	BORDERLINE CRITERIA, THRESHOLD FIVE	DBT SKILLS MODULES

18.1 Working a personality-disorder case: the shared spine

Every case runs the same five-step spine. Before any single case, hold the sequence that all four share, because a case answer that jumps straight to a label has skipped the marks. First, clear the **general personality-disorder criteria**: an enduring, pervasive, inflexible and maladaptive pattern of inner experience and behaviour, deviating from the person's cultural expectation, stable and of long duration with onset traceable to adolescence or early adulthood, causing distress or impairment, and not better explained by another mental disorder, a substance or a medical condition. Second, **count and type** against the criteria of the suspected disorder. Third, **discriminate** from the presentations that mimic it, above all the borderline-versus-bipolar-II line. Fourth, **formulate**, on a disorder-specific model where one exists. Fifth, **treat**, with structured psychotherapy primary and any drug symptom-targeted, adjunctive and time-limited.

The spine is the same whether the type survives as a DSM-5-TR category or is re-expressed as an ICD-11 severity level with trait qualifiers; the categorical name is a shorthand for a point on that dimensional map.

RULE **Formulate before you medicate.** Clear the general criteria, count and type, discriminate from the mimic, build the formulation, and only then treat, with structured psychotherapy primary and any drug symptom-targeted, adjunctive and time-limited; the label is the start of the reasoning, not the end of it.

18.2 case: the borderline patient, presentation and the general criteria

case: A woman in her early twenties is referred after an overdose that followed a break-up. On history she describes a terror of being left that predates this relationship, a lifelong pattern of relationships that swing from "he is perfect" to "he is cruel" within a single afternoon, a persistent sense of never knowing who she really is, recurrent cutting between crises, moods that flip within a couple of hours when a text goes unanswered, a background hollow emptiness, and disproportionate anger she cannot hold down. The presentation is dramatic, but the discipline is to work the spine, not to pattern-match. The general criteria are cleared first: this is an enduring, pervasive pattern present across relationships and settings, traceable to her adolescence, ego-syntonic and inflexible, not confined to the current crisis, and not better explained by a primary mood disorder or a substance. Only now is the count taken, and it is comfortably met: abandonment fear, unstable idealising-devaluing relationships, identity disturbance, recurrent self-harm, reactive affective instability, chronic emptiness and inappropriate anger give **five or more of the nine criteria** with room to spare. The diagnosis is defensible because the criteria can be named and shown to be trait, not a fresh state.

18.3 case: the borderline patient, the dual formulation

Two models, read together, not as rivals. The formulation borrows from both disorder-specific frameworks, because each answers a different question. Marsha Linehan's biosocial model answers *why*: a constitutional **emotional vulnerability**, heightened sensitivity, intense responses and a slow return to baseline, has transacted over her development with a pervasively **invalidating environment** that dismissed or punished her inner experience, and the two have amplified one another into a pervasive **emotion dysregulation**; her overdose and her cutting are read as attempts to solve the problem of unbearable affect, not as manipulation. Jeffrey Young's schema therapy answers *what is in the room*: because she scores high on almost every early maladaptive schema at once and flips between extreme states within minutes, she is formulated not as a list of schemas but through the **schema modes**, the small set of parts that alternate, cycling through the vulnerable (abandoned) child, the angry and impulsive child, the detached protector and the punitive parent, around a Healthy Adult that is weak and must be strengthened. The dual formulation is drawn in the accompanying figure, the biosocial pathway feeding the mode map.

PEARL

The two models are complementary, not competing. The Linehan biosocial theory gives the *mechanism* (emotion dysregulation) and the technology of change (concrete skills); the schema-modes map gives the *parts* (which mode is speaking now) and the relational and experiential engine (limited reparenting, chair work). A single clinician can formulate one patient on both, which is exactly why dialectical behaviour therapy and schema therapy are best held as siblings in one evidence-based family rather than as a forced choice.

WORKED EXAMPLE

Re-expressing the same patient in the ICD-11 dimensional system, forward-taught on the framework topics, gives a formulation of the form "moderate personality disorder, with prominent negative affectivity and disinhibition and some dissociality, plus the borderline pattern qualifier": the severity level is rated first and leads, the trait domains qualify it, and the borderline pattern is appended last. The categorical name "borderline personality disorder" and this dimensional string describe the same woman; the ICD-11 borderline pattern qualifier was retained precisely to flag the patients who respond to the borderline-specific psychotherapies, which is what makes both worth carrying.

18.4 case: the borderline patient, the structured-therapy plan

Structured psychotherapy is primary; the drug is a time-limited adjunct. The plan follows the formulation and the guidelines exactly. Structured psychological therapy is the primary treatment (NICE 2009, IPS 2023): where reducing recurrent self-harm is the priority, comprehensive dialectical behaviour therapy is the option named first, delivering its **four skills modules** (mindfulness at the core, with distress tolerance, emotion regulation and interpersonal effectiveness) through its four modes, with the target hierarchy putting the life-threatening behaviours ahead of everything else. The schema layer runs alongside or in sequence: limited reparenting and experiential mode work address the parts the skills alone do not reach, and the two layers map neatly onto her modes, as below. Medication stays symptom-targeted, adjunctive and time-limited: no drug is licensed for the disorder, so a short course of a sedative or a low-dose antipsychotic such as quetiapine (Qutipin, Seroquel) may be considered cautiously in a crisis, usually for no longer than a week, and a serotonin reuptake inhibitor such as fluoxetine (Fludac, Prozac) is added only if a comorbid depressive disorder is present, with the dosing carried in the Mood disorders: pharmacotherapy notes rather than re-taught here. The complex-PTSD boundary, which her trauma history raises, is drawn in the Anxiety, OCD and trauma-related disorders notes.

BORDERLINE MODE	HOW IT PRESENTS IN THE ROOM	THE MATCHED MOVE (DBT AND SCHEMA)
Vulnerable (abandoned) child	Raw terror of being left; fragile, frightened, alone	**Limited reparenting and validation; soothe and meet the need within bounds**
Angry and impulsive child	Rage or impulsive acting-out when a need goes unmet	Validate the feeling and contain the behaviour; distress-tolerance and emotion-regulation skills
Detached protector	Numbness, "I feel nothing", connection shut off	Gently bypass it to reach the child; empathic confrontation of the avoidance
Punitive parent	Self-hatred, "I deserved it", driving the self-harm	Fight and expel it through chair work; build the Healthy Adult and self-compassion

The four borderline schema modes matched to the therapeutic move, showing how the DBT skills (distress tolerance, emotion regulation) and the schema-therapy engines (limited reparenting, chair work) act on different modes of the same patient.

GOOD TO REMEMBER

- **Borderline personality disorder:** defining requirement is a pervasive pattern of instability of relationships, self-image and affect with marked impulsivity, meeting five or more of the nine criteria, on a base already cleared against the general personality-disorder criteria. First diagnostic step in the case: clear the general criteria, then count the nine; the affect is rapidly reactive and interpersonally cued and lasts hours, which is what separates it from bipolar II.
- **Dialectical behaviour therapy:** mechanism is the biosocial theory (emotional vulnerability transacting with an invalidating environment to give emotion dysregulation), treated by fusing behavioural change with mindfulness-based acceptance across four skills modules and four modes with a life-threatening-first target hierarchy; indication is borderline personality disorder with recurrent self-harm, the first-line structured therapy for self-harm (NICE, IPS); caution is that it must be comprehensive and delivered with fidelity, and trauma work waits for stage two.
- **Schema therapy:** mechanism is healing early maladaptive schemas through limited reparenting plus experiential mode work (imagery rescripting, chair work), with the labile borderline patient worked through the schema modes toward a strengthened Healthy Adult; indication is chronic characterological pathology, strongest-evidenced in borderline personality disorder; caution is that stripping out the reparenting and the experiential work leaves it as ordinary CBT that never touches the schemas.

18.5 case: borderline versus bipolar II, working the grid to the diagnosis and the treatment

case: A woman in her mid-twenties is referred as "query bipolar II disorder, mood swings, please start a mood stabiliser". The temptation is to hear "unstable mood" and reach for the drug, so the whole task is to read the mood rather than name it, working one discriminator at a time. On history the swings last two to three hours, always follow a fight with her partner or a fear that he is pulling away, and lift the moment he reassures her; between them she sits not on a settled euthymic baseline but on a floor of chronic emptiness; she describes never knowing who she is; and she cuts when the distress peaks. Read across the discriminator grid, the tempo is minutes to hours rather than the sustained days-to-weeks of a bipolar episode; the trigger is reactive and interpersonal rather than autonomous; the baseline is chronic dysphoria rather than a euthymic

return; and identity disturbance, chronic emptiness and affect-regulating self-harm are present, none of which belongs to bipolar II. Nothing here is a distinct hypomanic period of **at least four consecutive days** or a major depressive episode of **at least two weeks**. The diagnosis is borderline personality disorder, not bipolar II, and the first-line treatment follows the diagnosis: a structured psychotherapy, not a mood stabiliser prescribed for a bipolarity she does not have. The bipolar-II syndromes themselves are developed in the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes.

TRAP **Reaching for a drug first, on a pattern-matched label.** Reading reactive, hours-long, interpersonally cued lability as bipolar II and starting a mood stabiliser first-line, or drugging borderline personality disorder itself against NICE, treats a personality-based instability with the wrong tool and delays the structured psychotherapy that is its actual primary treatment.

GOOD TO REMEMBER

- **Bipolar II disorder:** defining requirement is at least one hypomanic episode (elevated or irritable mood lasting at least four consecutive days, with change in sleep, energy and goal-directed activity) plus at least one major depressive episode (at least two weeks), with a euthymic baseline between discrete, largely autonomous episodes and no full manic episode. Discriminator from borderline: the mood runs sustained, autonomous and days-to-weeks long, not the minutes-to-hours interpersonal reactivity of the personality disorder; the mood stabiliser is foundational here, whereas in borderline it is at best a symptom-targeted adjunct. When a genuine autonomous episode is documented alongside the pervasive reactive pattern, both diagnoses stand and both treatments are needed.

18.6 case: the antisocial forensic vignette, the history and the risk assessment

case: A man in his thirties is remanded after a violent robbery and referred for a court report with a request to "diagnose psychopathy". The vignette turns on the developmental gate and on the structure of the risk estimate. Collateral and documentary sources, which the manual insists on because deceit is central and the account cannot be taken on his word, show truanting, cruelty to animals and theft from well before the age of twelve, expulsions, a chaotic work record, unpaid debts, repeated adult convictions, and a flat indifference to the people he has hurt. The conduct disorder before fifteen and the age of at least eighteen are both satisfied, and he meets **three or more of the seven** adult criteria comfortably, so the diagnosis is antisocial personality disorder. The demand for a "diagnosis of psychopathy" is answered carefully: psychopathy is the narrower affective-interpersonal construct measured on the Hare Psychopathy Checklist-Revised, and it is not asserted without a rated instrument. The risk of future violence is then estimated by **structured professional judgement**, not by clinical impression and not by a raw checklist total: empirically derived factors are rated systematically, for instance on the HCR-20 across its historical, clinical and risk-management scales, and then weighed for this individual to a reasoned low, moderate or high rating.

RULE **No conduct disorder before fifteen, no antisocial personality disorder; and structure the risk estimate, do not total it.** The antisocial diagnosis is developmental and needs a documented childhood conduct disorder, and dangerousness is judged by structured professional judgement, factors rated systematically and then weighed for the case, never by the raw count on a checklist.

18.7 case: the antisocial forensic vignette, the medico-legal frame and the mainstay

The report claims only what it can defend, and the treatment is psychological. The medico-legal frame is where the diagnosis carries its weight, and its central caution is that antisocial personality disorder is a matter of disposal, not of exculpation. The insanity defence turns on an unsoundness of mind such that the accused did not know the nature of the act or that it was wrong, and a stable trait pattern rarely meets that legal test; the report therefore states three things and no more, that the diagnosis is antisocial personality disorder (not, on this assessment, formally psychopathy, which would need the rated instrument), that the disorder does not amount to the unsoundness of mind that founds an insanity defence and so does not reduce his responsibility, and that his risk of future violence, structured and then judged, is high and should guide disposal. The value of the report lies as much in what it declines to claim as in what it asserts, and the statutes, the fitness assessment and the disposal pathways sit in the Mental health legislation and forensic psychiatry notes. Management is modest and psychological-first: NICE recommends group-based cognitive and behavioural interventions, with the person sharing the choice of duration and intensity (NICE 2015), delivered where relevant in criminal-justice settings; no drug treats the personality, and where impulsive aggression is a target it is addressed adjunctively and off-licence, for example with divalproex (Valprol, Encorate), the detail cross-referenced to the Mood disorders: pharmacotherapy notes. Mentalisation-based treatment adapted for the disorder is an emerging structured option under trial.

GOOD TO REMEMBER

- **Antisocial personality disorder:** defining requirement is a pervasive pattern of disregard for and violation of the rights of others since fifteen, three or more of the seven criteria, gated by documented conduct disorder before fifteen and an age of at least eighteen, and not confined to schizophrenia or a bipolar illness. Discriminator: it is a trait pattern with a childhood conduct-disorder precursor, not a synonym for a criminal record or for the narrower affective-interpersonal construct of psychopathy; its forensic weight is in the structured assessment of dangerousness and the court report, not in the insanity defence.
- **Group cognitive-behavioural therapy (for antisocial personality disorder):** mechanism is a structured, skills-based group intervention targeting the cognitions and behaviours that drive offending, delivered with firm limits and shared choice of intensity; indication is antisocial personality disorder, especially with an offending history, and it is the NICE-recommended mainstay (NICE 2015); signature caution is that no drug is recommended for the personality itself (medication only for a comorbidity or, adjunctively and off-licence, for impulsive aggression) and that poor engagement and manipulation of the therapeutic frame are the rule, since the condition is ego-syntonic.

18.8 case: the cluster-C (anankastic) patient, formulation and management

case: An accountant in his forties is referred as "OCD, please treat". He is a relentless perfectionist who cannot discard years of worn-out paperwork, refuses to delegate unless colleagues submit to

exactly his method, and misses deadlines because he re-checks his work endlessly; he regards all of this as ordinary diligence and is untroubled by it, though his family and his team are worn down. Crucially, he describes no intrusive unwanted thoughts and performs no rituals to neutralise anxiety. The spine settles the diagnosis: the general criteria are cleared (an enduring, pervasive, ego-syntonic pattern from early adulthood, causing impairment and not better explained otherwise), and he meets **four or more of the eight** features of obsessive-compulsive personality disorder, the anankastic type, a preoccupation with orderliness, perfectionism and mental and interpersonal control at the expense of flexibility, openness and efficiency. The single discriminator that decides the case is the absence of true obsessions and compulsions and the ego-syntonic tone: this is character, not the ego-dystonic, resisted symptom pattern of obsessive-compulsive disorder, whose side is developed in the Anxiety, OCD and trauma-related disorders notes. In the ICD-11 dimensional system the phenotype is captured by prominent **anankastia**, the fifth trait domain. Management is psychological-first: structured cognitive-behavioural therapy working on the rigidity and the need for control, with the schema-mode formulation informing the work where a full protocol is out of reach; medication is reserved for a comorbidity only, never for the personality, and had he also described genuine resisted obsessions and washing rituals, both diagnoses would stand together.

GOOD TO REMEMBER

- **Obsessive-compulsive (anankastic) personality disorder:** defining requirement is a pervasive, ego-syntonic preoccupation with orderliness, perfectionism and mental and interpersonal control at the expense of flexibility, openness and efficiency, four or more of the eight features, from early adulthood. First diagnostic step in the case: clear the general criteria, confirm the perfectionism and control are ego-syntonic trait, then search for true obsessions and compulsions; their absence, not the shared name, discriminates it from obsessive-compulsive disorder.
- **Cognitive-behavioural therapy (for cluster C):** mechanism is graded exposure to the avoided or feared situation plus cognitive restructuring of the core belief (the need for control in anankastic, inadequacy in avoidant, helplessness in dependent), with work on rigidity, assertiveness and autonomy; indication is all three cluster-C types, engaging fastest where the anxious behaviour distresses the person; signature caution is the alliance and pace (the anankastic patient resists surrendering control), and medication is for a comorbidity only, never disorder-specific.

18.9 The thread through the four cases, and the India lens

One method, four applications. Laid side by side, the four cases show the same reasoning applied to different pathology, and the grid below is the object to carry into the hall: in every row the diagnosis is reached by clearing the general criteria and then discriminating from a mimic, and in every row the first-line treatment is a structured psychotherapy, with medication either symptom-targeted and time-limited, reserved for a comorbidity, or absent altogether. The synthesis map alongside arranges the same four against the decision points of the chapter. The wider technique of the psychotherapies, beyond the disorder-specific models owned here, sits in the Psychotherapies, clinical assessment, MSE and nosology notes; the schizotypal-versus-schizophrenia boundary that a cluster-A case would raise is in the Schizophrenia: aetiology, phenomenology, classification and course notes.

WORKED CASE	THE DECISIVE FEATURE(S)	DIAGNOSIS	FIRST-LINE TREATMENT	MEDICATION
The self-harming young woman	Five or more of the nine; reactive hours-long affect on a floor of emptiness and unstable identity	**Borderline personality disorder**	Structured psychotherapy (DBT skills plus schema mode work)	Symptom-targeted, time-limited, adjunctive
"Query bipolar II, mood swings"	Hours-long, interpersonally cued lability; no autonomous days-long episode; identity disturbance and emptiness	**Borderline personality disorder (not bipolar II)**	Structured psychotherapy, not a mood stabiliser	Only for a genuine comorbidity
The remanded violent offender	Three or more of the seven; conduct disorder before fifteen; remorseless	**Antisocial personality disorder**	Group cognitive-behavioural therapy; structured risk assessment	None for the personality; adjunct for impulsive aggression only
The perfectionist "referred as OCD"	Ego-syntonic perfectionism and control; no true obsessions or compulsions	**Obsessive-compulsive (anankastic) personality disorder**	Structured CBT on rigidity and control	For a comorbidity only

The four worked cases mapped to their decisive feature, diagnosis, first-line treatment and medication role; the shared conclusion is that structured psychotherapy is primary across all four and the drug is never the treatment for the personality itself.

INDIA

Four genuine India angles converge on these cases. First, under-recognition: personality disorders are markedly under-recognised in Indian practice, so all four patients typically reach services through something else, the overdose, the "mood swings" referral, the legal remand, the burnout, and the underlying pattern is captured only when a corroborated developmental history is deliberately taken. Second, access: the structured psychotherapies the borderline and cluster-C cases need, dialectical behaviour therapy and schema therapy, are being delivered and culturally adapted at Indian tertiary and private centres rather than being widely available, so recognising that a patient needs psychotherapy rather than a mood stabiliser is only the first step; the mindfulness core of DBT sits naturally with Indian contemplative traditions, while its interpersonal-effectiveness scripts must be calibrated to family interdependence rather than imported wholesale. Third, the medico-legal frame of the antisocial case: the insanity defence rests on the unsoundness-of-mind test carried into the Indian Penal Code, which antisocial personality disorder rarely satisfies, and the dedicated forensic infrastructure to support the risk assessment is thin. Fourth, cultural expression: every criterion is judged against the person's own cultural, social and economic milieu, so that normative family interdependence is not scored as dependence, culturally sanctioned emotional or dissociative-trance display is not over-read as borderline reactivity, and survival-driven or subcultural offending is not read as trait antisociality; personality pathology must never be read through a single culture's norms.

HOLD THIS

Every personality-disorder case runs one spine: clear the general criteria, count and type, discriminate from the mimic (above all borderline from bipolar II), formulate on the biosocial and schema-mode models, and treat with structured psychotherapy first, the drug being only ever a symptom-targeted, time-limited adjunct for a comorbidity or a crisis, and never the first-line treatment for the personality itself.

19 · Consolidation

The last step is not new material, it is retrieval. This closing topic turns a chapter you have *read* into one you can *reproduce* under exam pressure. The single most examinable idea of the personality-disorders chapter, that the **general criteria** are a gate and that ICD-11 rates **severity first** before any trait, is learned not by rereading it but by forcing it out of memory. The evidence is one-directional: testing yourself on half-learned material, **retrieval practice**, fixes it far more durably than reading it again, and the efficient way to study is to **cover the answer** and pull each fact out 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 vaults 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, and the gaps you find are exactly where to go back. The wider technique of **active recall** and spaced retrieval sits alongside the clinical material, and

the general models and technique of the psychological therapies are developed in the Psychotherapies, clinical assessment, MSE and nosology notes.

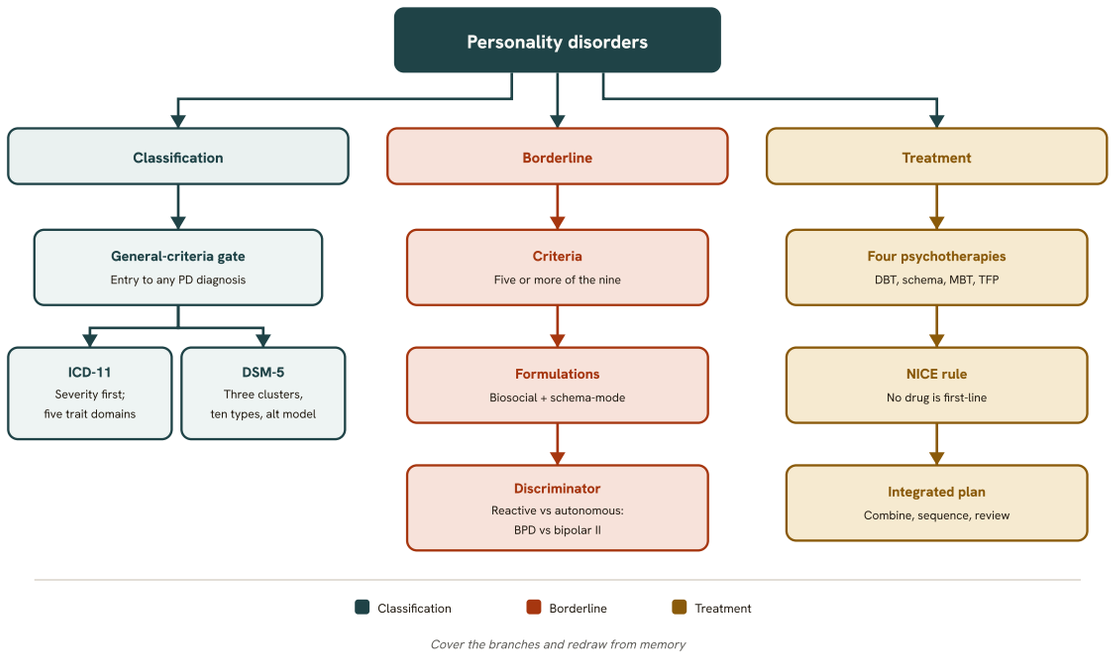


Fig 16.14 The chapter in one map: the general-criteria gate opening onto the ICD-11 dimensional and DSM-5 categorical classifications; borderline personality disorder with its criteria, its biosocial and schema-mode formulations and the bipolar-II discriminator; and the treatment branch of four structured psychotherapies with the rule that no drug is first-line. Cover the branches and redraw from memory.

19.1 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. Its four beats are the four countable skeletons of the day, the clusters and types, the ICD-11 trait domains, the borderline criterion count, and the core skills therapy, and saying the line should make the branches beneath each beat unfold. If the line is solid, the whole family, however you later classify it, is recoverable from it.

MNEMONIC

MAD BAD SAD, FIVE DOMAINS, NINE-FOR-FIVE, FOUR TO COPE

MAD BAD SAD are the three DSM-5-TR clusters holding the ten categorical types: *mad* is Cluster A, odd or eccentric (Paranoid, Schizoid, Schizotypal); *bad* is Cluster B, dramatic or erratic (Antisocial, Borderline, Histrionic, Narcissistic); *sad* is Cluster C, anxious or fearful (Avoidant, Dependent, Anankastic). **FIVE DOMAINS** are the ICD-11 trait qualifiers, hooked as *New Dogs Do Dig Antiques*: Negative affectivity, Detachment, Dissociality, Disinhibition, Anankastia, plus the optional borderline pattern qualifier that sits apart. **NINE-FOR-FIVE** is borderline personality disorder, defined by **five or more of the nine** criteria. **FOUR TO COPE** is dialectical behaviour therapy and its four skills modules (mindfulness at the core, plus distress tolerance, emotion regulation and interpersonal effectiveness). Four numbers, and the chapter rebuilds from one line.

3	10	5	4
DSM-5-TR CLUSTERS	CATEGORICAL PERSONALITY TYPES	ICD-11 TRAIT DOMAINS	DBT SKILLS MODULES

19.2 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 **Two anchors hold the chapter.** The general criteria are a gate that must clear before any type is named, and ICD-11 rates severity *first* (personality difficulty, then mild, moderate, severe) before adding the five trait domains; and in borderline personality disorder structured psychotherapy is primary while a drug is never a first-line treatment for the disorder. Recover those and the detail hangs off them.

19.3 Retrieval prompts: the general criteria, the clusters and the ICD-11 model

Cover the vaults below and answer from memory. Each prompt targets the single point the examiner tests, not a general description. Work them as **retrieval practice**, then check against the criterion vault.

1. State the six-part general personality-disorder gate (Criteria A to F), and why no specific type is named until every part is cleared.
2. Which two or more of the four channels (cognition, affectivity, interpersonal functioning, impulse control) must Criterion A involve, and what age of onset does Criterion D require?
3. What separates a personality *disorder* from a personality *style*, and how does ICD-11 code the style that falls short of the threshold?
4. Name the three DSM-5-TR clusters and the ten categorical types under them, and give each cluster's descriptor.
5. What did ICD-11 abolish, what did it put in its place, and in what order are the three moves made?
6. Name the five ICD-11 trait domains, and say which one is ICD-11-only and which AMPD domain has no ICD-11 counterpart.
7. Where does the borderline pattern qualifier sit in the ICD-11 model, and why was it the one pattern retained?
8. What did ICD-10 keep that ICD-11 removed, and why do the categorical names still get tested?

19.4 Retrieval prompts: the borderline criteria and the bipolar-II discriminator

Still covered. This is the highest-yield discrimination of the chapter, so retrieve it cleanly and check against the criterion vault.

1. Reproduce the nine DSM-5-TR borderline criteria and state the threshold; which four strands do they unpack into?
2. Which single criterion (its reactivity clause) is the pivot of the bipolar-II discrimination, and what does it specify about duration?
3. Read the borderline-versus-bipolar-II differential on tempo, trigger and baseline: which pattern points to each side?
4. Which features belong to borderline personality disorder and have no bipolar-II equivalent at all?
5. How long must a hypomanic episode and a major depressive episode last, and where does the bipolar mood return to between episodes?
6. When do borderline personality disorder and bipolar II both stand, and what does each then need?

GOOD TO REMEMBER

- **Personality disorder (general):** an enduring pattern of inner experience and behaviour, pervasive and inflexible, deviating markedly from cultural expectation in two or more of cognition, affectivity, interpersonal functioning and impulse control, with onset by adolescence, causing impairment or distress, and not better explained by another disorder, a substance or a medical condition. Discriminator from a personality *style*: the impairment or distress (Criterion C); ICD-11 codes the style short of this as personality difficulty. First step: clear all the general criteria before naming any type.
- **ICD-11 dimensional classification:** no types; rate severity *first* (personality difficulty, then mild, moderate, severe by self-and-interpersonal dysfunction and risk of harm), then add any of the five trait domains (negative affectivity, detachment, dissociality, disinhibition, anankastia), then the borderline pattern qualifier if present. Discriminator from DSM-5-TR: DSM keeps the categorical clusters in its main text (with the Section III AMPD hybrid alongside), so read fluently in both.
- **Borderline personality disorder:** a pervasive pattern of instability of relationships, self-image and affect with marked impulsivity, meeting **five or more of the nine** criteria. Discriminator: the affect is rapidly reactive, interpersonally triggered and lasts hours, versus the sustained autonomous episodes of bipolar II. First step: clear the general criteria, then count the nine. ICD-11 keeps the construct as the optional borderline pattern qualifier.
- **Bipolar II disorder (the discriminator):** at least one hypomanic episode (elevated or irritable mood, at least four consecutive days) plus at least one major depressive episode (at least two weeks), with a euthymic baseline between discrete, largely autonomous episodes. Discriminator: sustained, autonomous, days-to-weeks episodes, not minutes-to-hours interpersonal reactivity; the mood stabiliser helps here, not in personality-based instability.
- **The three classic boundary traps:** schizotypal is placed on the schizophrenia spectrum but is a personality disorder marked by enduring cognitive-perceptual oddness without sustained psychosis (the schizophrenia boundary sits in the aetiology-and-phenomenology notes); anankastic personality is an ego-syntonic *trait* of rigid perfectionism, not the ego-dystonic obsessions and compulsions of OCD; and antisocial personality disorder, never diagnosed before adulthood, carries the medico-legal weight of the chapter and is worked in the forensic notes.

19.5 Retrieval prompts: the psychotherapies and their mechanisms

Cover the therapy vault below. Each prompt asks for the *mechanism* and the signature technique, which is what the examiner rewards, not a bare name.

1. State the biosocial theory and the acceptance-change dialectic of dialectical behaviour therapy, and name its four skills modules and four treatment modes.
2. Which behaviours does the DBT target hierarchy address first, second and third within its first stage?
3. Define an early maladaptive schema and name the four borderline schema modes; what is limited reparenting?
4. How does mentalisation-based therapy read borderline pathology, and what is the mentalising stance it uses instead of interpretation?
5. How does transference-focused psychotherapy read the disorder, and by what sequence does it integrate the split object dyads?
6. Which of the four structured therapies is best-evidenced where recurrent self-harm is the priority, and does any head-to-head trial crown one overall?

19.6 Retrieval prompts: the NICE rule and the integrated plan

Still covered. These are the single most examinable management statements of the material; retrieve them and check against the therapy vault.

1. State the NICE (2009, CG78) rule on drug treatment in borderline personality disorder: is any drug licensed, and what are the only two windows for medication?
2. What does NICE say specifically about antipsychotics for the medium- and long-term treatment of the disorder?
3. Lay out the LOCUS, FOCUS, MODUS spine: where care happens, how the target shifts by phase, and by what primary means.
4. Where NICE, the Indian Psychiatric Society and WFSBP differ, what is the one line all three will sign, and which is most restrictive?
5. Which two agents do the guidelines steer away from, and why (dependence, and safety in overdose)?

GOOD TO REMEMBER

- **Dialectical behaviour therapy (Marsha Linehan):** mechanism is the biosocial theory (emotional vulnerability transacting with an invalidating environment to produce pervasive emotion dysregulation), treated by fusing behavioural change with mindfulness-based acceptance; four skills modules (mindfulness as the core, distress tolerance, emotion regulation, interpersonal effectiveness) through four modes (individual therapy, skills group, phone coaching, consultation team) on a target hierarchy of life-threatening, then therapy-interfering, then quality-of-life behaviours. Indication: borderline personality disorder with recurrent self-harm; the best-evidenced structured therapy for self-harm. Caution: must be comprehensive and delivered with fidelity, and trauma work waits for stage two.
- **Schema therapy (Jeffrey Young):** mechanism is healing early maladaptive schemas (self-defeating patterns from unmet childhood needs) through limited reparenting plus experiential mode work (imagery rescripting, chair work); the borderline patient is worked through four modes (vulnerable child, angry and impulsive child, detached protector, punitive parent) toward a strengthened Healthy Adult. Indication: chronic characterological pathology, strongest evidence in borderline personality disorder (superior to TFP in Giesen-Bloo 2006). Caution: strip out the reparenting and the experiential work and it collapses into ordinary CBT that leaves the schemas untouched.
- **Mentalisation-based therapy (Bateman and Fonagy):** mechanism is that borderline symptoms arise from a loss of mentalising under attachment-stress arousal, so the therapy restores it through a curious, not-knowing mentalising stance in the here and now, working close to conscious experience rather than interpreting the unconscious. Indication: borderline personality disorder (extended to antisocial and to adolescents). Caution: do *not* push interpretation while the patient is non-mentalising and highly aroused; lower arousal and re-open thinking first.
- **Transference-focused psychotherapy (Clarkin and Kernberg):** mechanism is an object-relations treatment reading the disorder as split, unintegrated self-and-other representations (object dyads) held apart by splitting, integrated by working the here-and-now transference through clarification, confrontation and interpretation, with identity diffusion the target and identity consolidation the goal. Indication: borderline and narcissistic organisation in a patient able to tolerate a long, interpretive treatment. Caution: interpretation needs a mind that can still mentalise, or it destabilises.

19.7 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 and three primary branches: a *classification* branch (the general-criteria gate splitting into the DSM-5-TR categorical clusters and the ICD-11 severity-first dimensional model with its five trait domains), a *borderline* branch (the five-of-nine criterion set and the reactive-versus-autonomous bipolar-II discriminator), and a *treatment* branch (the four structured psychotherapies with their mechanisms, and the NICE psychotherapy-primary, no-drug-first-line rule). Study it once, then cover every branch and **redraw from memory** on blank paper, writing the discriminator or the mechanism at each leaf. 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

Four Indian threads run the length of this chapter and belong on the synthesis map. First, *under-recognition*: personality disorders are diagnosed far less often in Indian practice than Western prevalence would predict, because the ego-syntonic pattern surfaces only when it is actively sought behind the self-harm, overdose or comorbidity that brings the patient in. Second, *availability and cultural adaptation*: dialectical behaviour therapy and schema therapy are being delivered and culturally adapted at Indian tertiary and metropolitan centres rather than being widely accessible, so a realistic plan blends what is available with skilled general management. Third, the *medico-legal frame*, sharpest around antisocial personality disorder, is where the diagnosis carries legal weight in offender assessment and court reports (the forensic notes). Fourth, the *cultural expression of pathology*: deviation is judged against the person's own cultural, social and economic milieu, so collectivist family-embeddedness, deference and religiously framed idioms are never read as dependent, avoidant or dissociative pathology; personality is not read through a single culture's norms.

TRAP **The chapter's recurring errors, in one line.** Diagnosing a type without first clearing the general criteria of an enduring, pervasive, inflexible pattern with onset by adolescence; calling reactive, hours-long affective lability bipolar II and starting a mood stabiliser; reaching for a drug first-line for borderline personality disorder against NICE; mislabelling schizotypal as schizophrenia; confusing anankastic personality with OCD; and reading personality pathology through one culture's norms.

HOLD THIS

If you can say *MAD BAD SAD, FIVE DOMAINS, NINE-FOR-FIVE, FOUR TO COPE* and, from it, regrow the general-criteria gate, the ICD-11 severity-first dimensional model with its five trait domains, the five-or-more-of-nine borderline count, the reactive-versus-autonomous bipolar-II split, and the psychotherapy-primary, no-drug-first-line NICE rule, the chapter is consolidated; wherever you cannot, that gap is the reread.

For the deep pharmacology of the mood stabilisers, low-dose antipsychotics and serotonin reuptake inhibitors that appear only as symptom-targeted adjuncts here see the Mood disorders: pharmacotherapy notes and the Schizophrenia: management, antipsychotics and adverse effects notes; for the bipolar-II side of the differential the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes; for the forensic implications of antisocial personality disorder the Mental health legislation and forensic psychiatry notes; for the complex-PTSD-versus-borderline boundary the Anxiety, OCD and trauma-related disorders notes; for the schizotypal-versus-schizophrenia boundary the Schizophrenia: aetiology, phenomenology, classification and course notes; and for the wider psychotherapy models and technique the Psychotherapies, clinical assessment, MSE and nosology notes.

Previously asked

This territory is examined at two levels at once: the disorder (its clinical features, its differential diagnosis, its aetiology and its course) and the treatment (which structured psychotherapy, the

integrated borderline plan, and the place of symptom-targeted medication). The reliable pattern is that borderline personality disorder carries far the most weight, asked at every length and from every angle, that the classification and the clinical assessment of personality recur as standalone notes and essays, and that a single therapy such as dialectical behaviour therapy or a single type such as avoidant personality disorder can be asked as a note in its own right. Every long essay ends in a management plan, so the rule that structured psychological therapy is primary and that a drug is not the first-line treatment for borderline personality disorder must be exam-ready. The genuine questions on record are grouped by band below. The deep pharmacology of the agents used in symptom-targeted treatment is developed in the Mood disorders: pharmacotherapy notes and the Schizophrenia: management, antipsychotics and adverse effects notes; the bipolar side of the differential in the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes; the forensic frame of antisocial personality disorder in the Mental health legislation and forensic psychiatry notes; and the wider psychotherapy models in the Psychotherapies, clinical assessment, MSE and nosology notes, while dialectical behaviour therapy, schema therapy, mentalisation-based therapy and transference-focused psychotherapy are owned here.

HOW THIS TERRITORY IS ACTUALLY TESTED

Q **Borderline personality disorder, the dominant essay and note.** This is by far the most heavily examined topic in the section. "Describe Kernberg's test of personality organisation. Discuss clinical features and management of Borderline Personality Disorder" and "Discuss aetiology and presentation of Borderline Personality Disorder" have both come as full 25-mark essays, while "Borderline personality disorder", "Borderline personality disorder, clinical features and differential diagnosis" and "Discuss clinical features and psychopharmacological management of Borderline Personality Disorder" recur as 10-mark notes across several sittings. Prepare borderline personality disorder end to end: the criteria and the threshold, the course and complications, the borderline-versus-bipolar-II differential, the Linehan biosocial and the schema-mode formulations, the integrated management plan, and the point that medication is symptom-targeted and adjunctive only. essay and short note, recurring

Q **The classification and the clinical assessment of personality.** "Classification of Personality Disorders" has come as a 10-mark note and "Discuss the clinical assessment of personality" as a 25-mark essay, with the assessment of personality also examined on the psychology side. Prepare the general criteria that gate every diagnosis, the ICD-11 dimensional model against the DSM-5 categorical clusters and the alternative model, the categorical-versus-dimensional debate, and the structured instruments and interviews with the state-versus-trait caution. essay and short note, recurring

Q **The approach to management and the named psychotherapies.** "Discuss approaches to personality disorders management" has come as a 25-mark essay and "Dialectical Behaviour Therapy (DBT)" as a standalone 10-mark note, and Kernberg's object-relations frame is drawn into the borderline essay. Prepare the guideline-anchored, structured-psychotherapy-first approach, dialectical behaviour therapy with its four skills modules and four modes, schema therapy on the modes framework, mentalisation-based and transference-focused psychotherapy, and the symptom-targeted pharmacotherapy with the not-first-line rule. essay and short note, recurring

Q **The individual types as standalone notes.** "Avoidant personality disorder" has come as a 10-mark note, and any single type can be asked this way. Prepare each cluster A, B and C type by its one defining feature

and its discriminator from its neighbours, so a type can be written up cleanly on its own. **short note, recurring**

Prepare this material to be diagnosed and treated, not just recited: the general criteria and the classification shift, the borderline criteria and the borderline-versus-bipolar-II discriminator, the criterion-anchored good-to-remember boxes, the psychotherapy frameworks and the integrated borderline plan in these notes are built to the way the block is examined. The deep pharmacology of the mood stabilisers, low-dose antipsychotics and serotonin reuptake inhibitors used in symptom-targeted treatment lives in the Mood disorders: pharmacotherapy notes and the Schizophrenia: management, antipsychotics and adverse effects notes; the forensic frame of antisocial personality disorder in the Mental health legislation and forensic psychiatry notes; and the wider psychotherapy models in the Psychotherapies, clinical assessment, MSE and nosology notes.

Quick review

THE FRAMEWORK: THE GENERAL CRITERIA AND THE CLASSIFICATION SHIFT	
THE GATE BEFORE ANY TYPE	No type is named until the **general criteria** hold: an **enduring**, **pervasive** and **inflexible** pattern across cognition, affectivity, interpersonal functioning and impulse control, **deviating from cultural expectation**, with **onset by adolescence or early adulthood**, causing distress or impairment, and not better explained by another disorder.
ICD-11 (THE HEADLINE SHIFT)	ICD-11 **abolished the categorical types**. Rate **severity first** (personality difficulty, then mild, moderate, severe), then the **five trait domains** (negative affectivity, detachment, dissociality, disinhibition, anankastia) and the **borderline pattern** qualifier. Severity leads because it predicts outcome and need better than type.
DSM-5-TR (CATEGORICAL) AND THE ALTERNATIVE MODEL	Ten types in three clusters: **A** odd or eccentric, **B** dramatic or erratic, **C** anxious or fearful. Section III **alternative model** = the level-of-personality-functioning scale plus five pathological trait domains (negative affectivity, detachment, antagonism, disinhibition, psychoticism) and six retained types. **ICD-10 kept** the categorical types, which is why exams still test them.
CATEGORICAL VERSUS DIMENSIONAL	Categorical is familiar and communicable but gives excessive comorbidity, arbitrary thresholds and a large not-otherwise-specified group; dimensional has better validity and covers the subthreshold majority. The field moved dimensional (ICD-11 and the DSM-5 alternative model).
THE DISORDERS BY CLUSTER	
CLUSTER A (ODD OR ECCENTRIC)	**Paranoid** (pervasive distrust and suspiciousness), **schizoid** (detachment and restricted affect), **schizotypal** (cognitive and perceptual distortions and eccentricity). Schizotypal sits on the **schizophrenia spectrum** (ICD-11 6A22, outside the personality disorders); the discriminator from schizophrenia is that the signs are attenuated and never become sustained frank psychosis.
BORDERLINE (THE HIGHEST-YIELD TYPE)	Abandonment fears, unstable intense relationships, identity disturbance, impulsivity, recurrent self-harm and suicidality, affective instability, chronic emptiness, inappropriate anger, transient stress-related paranoia or dissociation: **five or more of the nine criteria**. Course trends to **symptomatic remission** over years; sustained functional recovery is harder.

THE DISORDERS BY CLUSTER	
ANTISOCIAL	Pervasive disregard for the rights of others (deceit, impulsivity, aggression, recklessness, irresponsibility, lack of remorse); requires **conduct disorder before age fifteen** and is diagnosed **from age eighteen**. **Psychopathy** (the affective-interpersonal core, PCL-R) is a narrower construct nested within it. The forensic frame is cross-referenced.
HISTRIONIC AND NARCISSISTIC	Histrionic: excessive emotionality and **attention-seeking**. Narcissistic: grandiosity, need for admiration, lack of empathy, split into **grandiose** (overt, entitled) and **vulnerable** (covert, shame-prone) presentations over a shared fragile self-esteem.
CLUSTER C (ANXIOUS OR FEARFUL)	**Avoidant** (social inhibition, feels inadequate, wants closeness but withdraws), **dependent** (needs to be taken care of, submissive, fears separation), **anankastic / obsessive-compulsive** (order, perfectionism, control). Anankastic is **ego-syntonic** traits with **no true obsessions or compulsions**, which is the line from OCD.

THE HIGH-YIELD DISCRIMINATORS	
BORDERLINE VERSUS BIPOLAR II	Borderline mood shifts are **rapid (minutes to hours) and reactive** to interpersonal triggers, with identity disturbance and chronic emptiness; bipolar II episodes are **sustained (days to weeks) and autonomous**, with a bipolar family history and a response to a mood stabiliser. The trap is calling reactive personality-based lability bipolar and starting a mood stabiliser.
COMPLEX PTSD VERSUS BORDERLINE	Complex PTSD carries the PTSD triad plus disturbances in self-organisation with a **stable** negative self-view and relationships of avoidance, after prolonged trauma; borderline shows unstable identity and frantic abandonment fears. Missing complex PTSD behind a personality-disorder label is the classic error.
ANANKASTIC PERSONALITY VERSUS OCD	Anankastic personality is the ego-syntonic character of perfectionism and control with no discrete obsessions or compulsions; OCD is ego-dystonic intrusive obsessions and resisted compulsions.
DO NOT DIAGNOSE IN AN ACUTE STATE	The pattern must be enduring and pervasive. Do not diagnose a personality disorder during an acute episode of another disorder (the state-versus-trait caution); use a structured instrument (IPDE, SCID-5-PD) and corroborating history.

THE TREATMENT BLOCK, THE GUIDELINE SPINE AND THE HARD RULES	
THE GUIDELINE ORDER	Guidelines-first on the where-to-treat, target-by-phase, modes-of-treatment spine: **NICE leads** (the borderline and the antisocial guidelines), then the **Indian Psychiatric Society** and **WFSBP**. Structured psychological therapy is primary throughout.

THE TREATMENT BLOCK, THE GUIDELINE SPINE AND THE HARD RULES	
THE PSYCHOTHERAPIES	**Dialectical behaviour therapy**: four skills modules (mindfulness, distress tolerance, emotion regulation, interpersonal effectiveness) across four modes (individual, skills group, phone coaching, consultation team). **Schema therapy**: work the modes (vulnerable child, angry child, detached protector, punitive parent, healthy adult) by limited reparenting. **Mentalisation-based therapy**: restore the mentalising stance. **Transference-focused psychotherapy**: integrate split object relations in the here-and-now transference.
PHARMACOTHERAPY (THE KEY RULE)	**No drug is first-line for borderline personality disorder** and none is licensed (NICE); no drug reduces total borderline severity (Cochrane). Medication is **symptom-targeted and adjunctive**: a low-dose antipsychotic for cognitive-perceptual and impulsive-behavioural dyscontrol, a mood stabiliser for impulsive aggression, an SSRI for the affective and anxiety domain, only for a comorbidity or a crisis. **Avoid benzodiazepines** and **avoid polypharmacy**; review and deprescribe.
THE INTEGRATED BORDERLINE PLAN	LOCUS: outpatient and community by default, admission avoided and brief. FOCUS: short-term safety and crisis, mid-term the core features via structured therapy, long-term function and remission. MODUS: structured psychotherapy primary, symptom-targeted time-limited medication only, multidisciplinary care planning and a crisis plan.
THE FIVE TRAPS	Diagnosing a type without the general criteria; calling reactive affective lability bipolar and starting a mood stabiliser; giving a drug first-line for borderline personality disorder; reading schizotypal as schizophrenia; and confusing ego-syntonic anankastic personality with ego-dystonic OCD.

References

The personality-disorder content is grounded in the standard psychiatry sources (the source hierarchy below), with Kaplan and Sadock and the New Oxford Textbook as the depth bar for the phenomenology, the classification and the course, the ICD-11 Clinical Descriptions and Diagnostic Requirements and the DSM-5-TR for the criteria and the two classification systems, and the primary psychotherapy literature for dialectical behaviour therapy, schema therapy, mentalisation-based therapy and transference-focused psychotherapy. The management is guideline-first, led by NICE (the borderline and the antisocial guidelines), with the Indian Psychiatric Society, WFSBP and the American Psychiatric Association. Every criterion, trait domain, cluster mapping, severity anchor and guideline rule was checked against these sources and the adversarial content pass; anything that could not be confirmed was omitted and flagged rather than guessed, so several prevalence and effect-size figures are stated by direction rather than by a specific number. Each journal PMID below was verified against PubMed this build and

matched on title, first author and year. Books, manuals and guideline documents are cited by author, title and year without a PMID.

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PART X

X

Eating and Sleep-Wake Disorders

Disordered eating and disordered sleep: the eating disorders and the sleep-wake disorders in full.

01 Eating disorders

02 Sleep-wake disorders

03 Apply and consolidate

PAPER II

Eating and sleep-wake disorders

Two sections . one complete notes document

This material gathers the eating disorders and the sleep-wake disorders into one document, by syndrome and by treatment. The first band is **the eating disorders**: anorexia nervosa and its medical complications, the refeeding syndrome that endangers recovery, the guideline-anchored management, bulimia nervosa and binge eating disorder, avoidant/restrictive food intake disorder, the aetiology and neurobiology, the feeding disorders, and the atypical and non-fat-phobic presentations. The second band is **the sleep-wake disorders**: normal sleep physiology and architecture as the foundation, insomnia and the first-line place of cognitive behaviour therapy for insomnia, narcolepsy and its tetrad, obstructive sleep apnoea, the NREM and REM parasomnias including REM sleep behaviour disorder, the circadian rhythm disorders, and restless legs. The examinable skill is the safe, guideline-anchored diagnosis and treatment of each disorder: which eating disorder behind which weight and behaviour, how to refeed without killing the patient, which drug is licensed for which eating disorder, how a normal night is built, why CBT-I comes before a hypnotic, and how to recognise narcolepsy, sleep apnoea and REM sleep behaviour disorder behind their common disguises.

HOW TO USE THESE NOTES

Read the two bands as one continuous account of how the eating and the sleep-wake disorders are diagnosed and treated. The **eating disorders** band builds the frame: the individual disorders discriminated one from another, the medical complications of starvation, the refeeding syndrome and how to prevent it, and the guideline-anchored management (no drug is first-line for anorexia; cognitive behaviour therapy for eating disorders and family-based treatment carry the weight; fluoxetine is the licensed drug for bulimia). The **sleep-wake disorders** band opens with normal sleep physiology and the hypnogram as the foundation, then teaches insomnia and the first-line place of cognitive behaviour therapy for insomnia, narcolepsy and its orexin loss, obstructive sleep apnoea, the parasomnias including REM sleep behaviour disorder as a prodrome of a synucleinopathy, and the circadian disorders. Every management recommendation is guideline-anchored, led by NICE and MARSIPAN in the eating half and by the BAP guidelines with the Indian Psychiatric Society, NICE and the sleep-medicine consensus in the sleep half, on the where-to-treat, target-by-phase, modes-of-treatment spine; every named syndrome ends with a **Good to remember** box giving the criterion, the discriminator and the first step, and every named management step ends with one giving the principle and the one value or first-line to hold. Figures declare one axis and code it in colour: **petrol** marks structure and the neutral case, **coral** marks the trap or the danger, and **marigold** highlights the number to hold; the rating scales are reproduced as the actual instruments where the licence permits, credited to their sources, or typeset from their structure where it does not. Several boundaries are stated up front: the deep hypnotic pharmacology (melatonin, ramelteon, the dual orexin receptor antagonists, the z-drugs and the benzodiazepines) is taught in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes and cross-referenced here; the deep antidepressant pharmacology in the Mood disorders: pharmacotherapy notes; the sleep neurochemistry and the ascending-arousal anatomy in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes and the Functional Neuroanatomy and Neurophysiology notes; the anorexia neuroendocrine axes in the Neurochemistry notes; and the wider cognitive behaviour therapy technique in the Psychotherapies, clinical assessment, MSE and nosology notes. Cognitive behaviour therapy for insomnia, the eating-disorder cognitive behaviour therapy and family-based treatment, and the sleep architecture, the hypnogram and polysomnography 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 refeeding value, drug dose and scale cut-off is verified against a source or omitted and flagged.

Eating disorders

1 · Anorexia nervosa

The disorder to know cold. **Anorexia nervosa** is the eating disorder of self-imposed starvation: a **restriction of energy intake** that drives the body to a **significantly low weight**, held there by an **intense fear of weight gain** (or a persistent behaviour that blocks it) and a **disturbance in how body weight and shape are experienced**. It carries the highest mortality of any psychiatric illness, so the examiner treats it as a safety topic as much as a phenomenology one. This note owns the diagnosis: the three core features, the ICD-11 and DSM-5-TR criteria and the

amenorrhoea criterion that DSM-5 dropped, the **restricting subtype** and **binge-purge subtype**, the epidemiology and course, and the **medical complications** mapped body system by body system.

The chapter's master mnemonic for the starvation complications, **STARVED**, is seeded here and paid off at consolidation. The neuroendocrine axes that fail in anorexia (the hypothalamic-pituitary-gonadal, thyroid and adrenal changes) are derived in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes and are given here only in applied clinical form. The dangerous physiology of re-nourishing a starved patient sits in the refeeding-syndrome topic in these notes, and the guideline-anchored treatment in the anorexia-management account here; what follows is how to recognise and stage the disorder and read its body. First described clinically by Sir William Gull and Charles Lasègue in the 1870s, its most quoted teaching point is that *the fear need not be spoken to be present*.

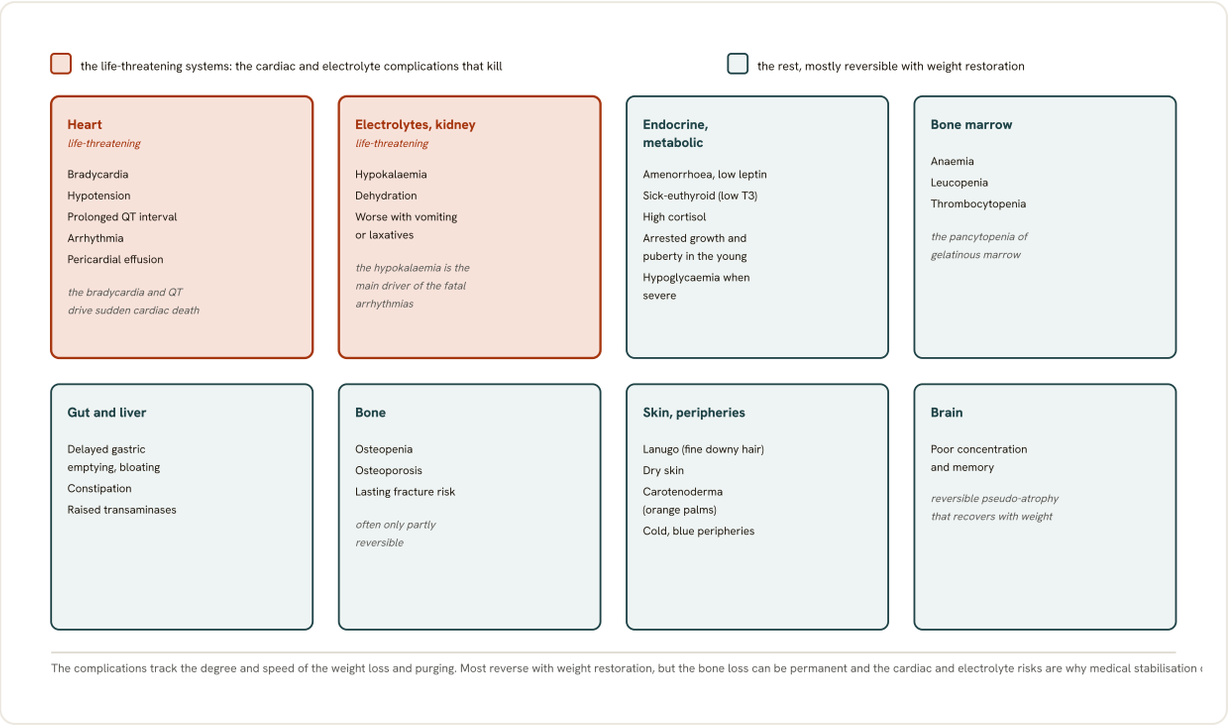


Fig 17.1 The medical complications of anorexia nervosa by body system. Starvation touches every organ. The cardiac and electrolyte complications (coral) are the ones that kill: bradycardia, hypotension, a prolonged QT interval and arrhythmia, and the hypokalaemia and dehydration made worse by vomiting or laxative misuse. The endocrine picture is the low-oestrogen amenorrhoea, a low-leptin and sick-euthyroid state with a raised cortisol, and, in a young patient, arrested growth and puberty; the marrow shows a pancytopenia; the gut is slow, with delayed emptying and constipation; the bone loses density with a fracture risk that may not fully recover; and the skin grows fine lanugo hair with carotenoderma and cold peripheries. Most reverse with weight restoration, but the bone loss can be permanent and the cardiac and electrolyte risks are why medical stabilisation, not psychotherapy, comes first.

SCOFF, THE 5-ITEM SCREEN (REPRODUCED)	SCORE ONE POINT FOR EACH "YES"
S ick	Do you make yourself **Sick** because you feel uncomfortably full?
C ontrol	Do you worry you have lost **Control** over how much you eat?
O ne stone	Have you recently lost more than **One** stone (about 6.35 kg) in a three-month period?
F at	Do you believe yourself to be **Fat** when others say you are too thin?
F ood	Would you say that **Food** dominates your life?
Cut-off	**Two or more "yes" answers is a positive screen for a probable eating disorder and prompts a full assessment**

SCOFF: reproduced from Morgan JF, Reid F, Lacey JH. The SCOFF questionnaire. BMJ. 1999;319:1467-1468. A freely used screening tool; for the free, non-commercial resident edition only.

EAT-26 (TYPESET STRUCTURE)	WHAT TO HOLD
Instrument	The Eating Attitudes Test, a 26-item self-report screen of eating attitudes and behaviours (Garner et al., 1982), across three subscales: dieting, bulimia and food preoccupation, and oral control
Cut-off	**A total score of 20 or more indicates a need for a full assessment; it is a screen, not a diagnosis**

EAT-26: structure and cut-off typeset (Garner DM, Olmsted MP, Bohr Y, Garfinkel PE. Psychol Med. 1982;12:871-878); free for non-commercial use with acknowledgement. For the free, non-commercial resident edition only.

Fig 17.2 The eating-disorder screening scales. SCOFF is a five-item screen whose name is its mnemonic (Sick, Control, One stone, Fat, Food); two or more positive answers is a positive screen and prompts a full assessment. The EAT-26 is a longer 26-item self-report across dieting, bulimia and food preoccupation, and oral control subscales, with a total of 20 or more flagging the need for assessment. Both are screens, not diagnoses: a positive screen leads to a clinical interview against the ICD-11 or DSM-5-TR criteria, and in India a non-fat-phobic presentation can screen negative on the shape-and-weight items yet still be anorexia, so a negative screen does not exclude an eating disorder.

1.1 The three core features

One engine, three faces. Every framework builds anorexia nervosa from the same triad. First, a persistent *restriction of energy intake* relative to requirements that produces a body weight below what is minimally normal for age, sex, developmental trajectory and physical health (in a growing child, this may be a failure to make expected weight gain rather than frank loss). Second, an *intense fear of becoming fat* or a persistent behaviour that interferes with weight gain, present even as the weight falls. Third, a *disturbance in the experience of weight or shape*: undue influence of shape on self-evaluation, or a persistent failure to recognise the medical seriousness of the low weight.

Restriction is the non-negotiable one. The fear and the body-image distortion can be inferred from behaviour; the significantly low weight driven by restriction cannot be worked around. This is why a patient who denies any wish to be thin but relentlessly starves still meets the diagnosis.

GOOD TO REMEMBER

- **Anorexia nervosa (the syndrome):** three essential features, (1) restriction of intake leading to a significantly low weight, (2) intense fear of weight gain *or* persistent behaviour that interferes with weight gain, and (3) disturbed experience of weight/shape or non-recognition of the danger; the defining criterion is the low body weight from restriction, and the fear does not have to be verbalised.

1.2 DSM-5-TR criteria and the severity bands

The three criteria to state. DSM-5-TR criterion A is restriction of energy intake leading to a significantly low body weight in the context of age, sex, developmental trajectory and physical health; B is intense fear of gaining weight or of becoming fat, or persistent behaviour that interferes with weight gain, even at a significantly low weight; and C is a disturbance in the way weight or shape is experienced, undue influence of weight/shape on self-evaluation, or persistent lack of recognition of the seriousness of the low weight (DSM-5-TR 2022). DSM-5 deliberately removed the old numeric anchor (the DSM-IV example of "85% of ideal body weight") so that no single figure is read as the definition.

Severity is scored on current BMI. For adults the severity is graded from the current body mass index, and this is the number set examiners like. The severity may be stepped up for clinical symptoms, functional disability or the need for supervision.

DSM-5-TR SEVERITY (ADULT)	BMI (KG PER M SQUARED)
Mild	≥ 17
Moderate	16 to 16.99
Severe	**15 to 15.99**
Extreme	**< 15**

DSM-5-TR severity grading for anorexia nervosa from current adult BMI (WHO thinness categories); for children and adolescents the corresponding BMI-for-age percentiles are used (DSM-5-TR 2022).

RULE **Significantly low weight from restriction is the anchor.** Diagnosis turns on a restriction-driven, significantly low body weight; the fear of fatness can be shown by persistent weight-defeating behaviour and need not be voiced, and there is no single BMI that defines "low" in either system.

1.3 ICD-11 criteria and the underweight specifiers

Where ICD-11 puts a number on it. ICD-11 requires a significantly low body weight for the individual's height, age and developmental stage, with a commonly used adult threshold of BMI below **18.5 kg per m squared** (or below the 5th percentile for age in children). A distinctive ICD-11 provision: *rapid weight loss of more than **20% of total body weight within 6 months*** can substitute for the low-weight requirement when the other features are met. The presentation

must be a persistent pattern of restrictive eating (or purging or over-exercise) aimed at abnormally low weight, not better explained by another condition or by lack of food (ICD-11 CDDR).

The two underweight grades. ICD-11 stages the danger directly: *anorexia nervosa with significantly low body weight* (BMI 18.5 down to 14.0) and *anorexia nervosa with dangerously low body weight* (BMI below 14.0), the latter flagging the high-complication, high-mortality patient. Fear of weight gain is explicitly *not* an absolute requirement in ICD-11.

FRAMEWORK	WEIGHT THRESHOLD TO DIAGNOSE (BMI IN KG PER M SQUARED)	GRADING OF SEVERITY
DSM-5-TR	No fixed cut-off; adult BMI under 17.0 usually "low", 17.0 to 18.5 possible on clinical grounds	Mild ≥ 17 / Moderate 16 to 16.99 / Severe 15 to 15.99 / Extreme < 15
ICD-11	**Commonly < 18.5, *or* rapid loss > 20% of body weight within 6 months**	Significantly low (18.5 to 14.0); dangerously low (< 14.0)
ICD-10 (historical)	BMI < 17.5 *plus* amenorrhoea in women	Self-induced weight loss with a "dread of fatness"

The weight thresholds compared; the number to quote is ICD-11's BMI under 18.5 (or a greater-than-20% loss in 6 months), against DSM-5-TR's deliberately non-numeric definition.

1.4 The amenorrhoea criterion, dropped in DSM-5

A favourite discriminator. DSM-IV required *amenorrhoea* (absence of at least three consecutive menstrual cycles) as a criterion for anorexia nervosa. DSM-5 removed it, because women who met every other criterion but continued to menstruate did not differ in course, prognosis or severity, and because the criterion could not apply to men, pre-menarcheal girls, post-menopausal women or those on oral contraception (Kaplan and Sadock 2024). ICD-10's F50.0 likewise leaned on a widespread endocrine disorder manifesting as amenorrhoea; ICD-11 has moved the same way, folding amenorrhoea in as a clinical *feature* and consequence of starvation rather than a required criterion.

The take-home. Amenorrhoea is no longer needed to diagnose anorexia nervosa in either modern system, but it remains one of the most reliable clinical *signs* of the hypothalamic-pituitary-gonadal suppression that starvation produces, so it stays in the complications, not the criteria.

GOOD TO REMEMBER

- **Amenorrhoea criterion:** a DSM-IV requirement that DSM-5 abolished (and ICD-11 does not require); it discriminated poorly, excluded men and non-menstruating women, and is now treated as a clinical sign of hypothalamic-pituitary-gonadal suppression rather than a diagnostic criterion.

1.5 The subtypes: restricting versus binge-purge

Defined by the last three months. The restricting subtype (DSM: restricting type) describes weight loss achieved through dieting, fasting or excessive exercise, with *no* recurrent binge-eating

or purging in the last three months. The binge-purge subtype (DSM: binge-eating/purging type) applies when, over the same recent window, the person has engaged in recurrent binge-eating or purging (self-induced vomiting, or misuse of laxatives, diuretics or enemas); some in this group do not binge but regularly purge after small amounts of food. ICD-11 uses the parallel "restricting pattern" and "binge-purge pattern" specifiers.

Crossover is common. Movement between the subtypes over the illness course is frequent, so the label describes *current* symptoms, not the lifetime picture. Clinically it matters because the binge-purge subtype carries more electrolyte derangement and cardiac risk, and it is the group whose bloods most often reveal **hypokalaemia** and metabolic alkalosis.

GOOD TO REMEMBER

- **Restricting subtype:** low weight by dieting, fasting or over-exercise with no binge-eating or purging in the last three months; the classic "pure restrictor".
- **Binge-purge subtype:** recurrent binge-eating or purging (vomiting, laxatives, diuretics, enemas) in the last three months, at a still significantly low weight; carries the higher electrolyte and cardiac risk, and crossover between subtypes is common.

1.6 Epidemiology, onset and course

The numbers. Lifetime prevalence of DSM-5 anorexia nervosa is roughly **1.4% in women** and **0.2% in men**, with a female-to-male ratio near **10 to 1**, though the male share is under-recognised (men present with a drive for muscularity and leanness and may be missed) (Kaplan and Sadock 2024). Point prevalence in girls and women is about 0.5% to 1%, and incidence is rising fastest in the high-risk **15 to 19**-year-old female band. Onset is typically adolescence or early adulthood (ages 10 to 24), often after a stressor and a diet that "works" and becomes self-reinforcing.

Course and lethality. On follow-up, roughly **30% to 50%** achieve full recovery, **10% to 20%** remain chronically ill, and the rest improve but retain some disordered behaviour. The mortality is the headline: individuals are up to **six times** more likely to die than the general population, most deaths from the medical consequences of starvation and about **one in five** from suicide. Shorter illness duration and full weight restoration predict better outcome, which is the whole argument for early detection.

PEARL

Anorexia nervosa has the highest mortality rate of any psychiatric disorder. Hold two numbers: a roughly six-fold excess mortality, and about one death in five by suicide rather than by starvation. This is why "she is just dieting" is never a safe formulation, and why weight, orthostatic vitals, potassium and the ECG QTc are checked at every review.

1.7 Medical complications, mapped by body system

Starvation writes on every organ. The **medical complications** follow from starvation, from the weight-control methods (vomiting, laxatives, over-exercise), and, dangerously, from re-nourishment. The figure alongside maps them onto the body; the table below is the system-by-

system checklist. Two of them are the ones that kill and must be sought at every visit: a prolonged QTc on the ECG and marked **hypokalaemia**.

SYSTEM	SIGNS AND ABNORMALITIES	MECHANISM / NOTE
Cardiovascular	**Bradycardia, hypotension, orthostasis, prolonged QTc, arrhythmia, peripheral oedema**	Commonest fatal pathway; QTc prolongation may occur even without electrolyte derangement
Endocrine / reproductive	Amenorrhoea, low LH/FSH and oestrogen or testosterone, sick-euthyroid pattern, hypercortisolaemia, low leptin	Energy-conserving; most reverse with refeeding
Skeletal / bone	Osteopenia and osteoporosis, fracture risk	Low oestrogen, high cortisol and malnutrition; bone may not fully recover
Haematological	Pancytopenia (leukopenia, anaemia, thrombocytopenia)	Gelatinous marrow transformation of starvation
Renal / electrolyte	**Hypokalaemia, hyponatraemia, hypochloraemia, metabolic alkalosis, dehydration**	Worse in the binge-purge subtype; hypokalaemia is the dangerous one
Gastrointestinal	Delayed gastric emptying, constipation, raised liver enzymes	Hypomotility of starvation
Dermatological	Lanugo, dry skin, hair thinning, carotenaemia (yellow skin)	Visible signs of the starved state
Refeeding (metabolic)	**Hypophosphataemia, hypomagnesaemia, thiamine deficiency, glucose intolerance**	The refeeding-syndrome cluster; detailed in the refeeding-syndrome topic in these notes

Medical complications of anorexia nervosa by body system; the two urgent findings are a prolonged QTc and marked hypokalaemia, and the refeeding cluster is led by hypophosphataemia (Kaplan and Sadock 2024).

MNEMONIC

STARVED

The chapter's master complication mnemonic, seeded here and paid off at consolidation. **S**kin (lanugo, dry skin, carotenaemia); **T**hyroid sick-euthyroid pattern and low Temperature (hypothermia); **A**menorrhoea and the hypothalamic-pituitary-gonadal or leptin axis; **R**ate and rhythm (bradycardia, hypotension, prolonged QTc, arrhythmia); **V**alues, the electrolytes (hypokalaemia, hyponatraemia, dehydration); **E**rythron and marrow (pancytopenia); **D**igestive and Density (delayed gastric emptying and constipation, plus low bone mineral density).

1.8 The neuroendocrine picture, in applied form

Every axis is set to conserve energy. The full endocrinology lives in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes; the clinical residues are these. The hypothalamic-pituitary-gonadal axis is virtually always suppressed, low LH, FSH and oestrogen or testosterone, mediated in part by low serum leptin that tracks the loss of fat

mass, giving the amenorrhoea and loss of libido. The thyroid shows a euthyroid sick syndrome (low total T3 and T4 with low or normal TSH and a raised reverse T3), an adaptive down-shift, not primary thyroid disease. The adrenal axis runs high, with hypercortisolaemia.

Two safety points. First, the sick-euthyroid pattern is *not* treated with thyroxine; it corrects with refeeding, and thyroxine would only accelerate weight loss and cardiac risk. Second, the low oestrogen plus high cortisol plus malnutrition drives the osteoporosis, which unlike the other endocrine changes may not fully reverse, and oral oestrogen or calcium alone does not restore bone density while the patient stays underweight.

COMMON TRAP

Do not read the low thyroid hormones as hypothyroidism and start thyroxine, and do not be reassured by "normal" bloods. Laboratory results are frequently normal even in a seriously ill, low-weight patient, so a normal panel never excludes danger. The sick-euthyroid pattern reverses with feeding; giving thyroxine only worsens weight loss and the cardiac risk.

1.9 The refeeding flag and the hypophosphataemia hallmark

The paradox of getting better. The most dangerous moment can be the start of re-nourishment. Refeeding syndrome is the shift of fluid, electrolytes and minerals that occurs when a severely malnourished patient is fed too quickly: insulin surges as carbohydrate returns, pushing phosphate, potassium and magnesium into cells. Its hallmark is hypophosphataemia, and it can precipitate cardiac failure, arrhythmia and death. The preventive discipline is a single phrase: correct the electrolytes, give thiamine, and start slow, monitoring closely through the opening days.

Why it is flagged here. The exact starting-calorie ranges, the phosphate, potassium and magnesium thresholds and the monitoring schedule are worked out in the refeeding-syndrome topic in these notes; the point for the anorexia diagnosis is simply to name refeeding syndrome as the risk that a low-BMI patient carries into treatment, with hypophosphataemia as the signature.

TRAP **Refeeding too fast, and missing the phosphate.** The classic lethal error is to correct starvation rapidly; the falling phosphate of refeeding syndrome is the thing to pre-empt, so electrolytes are corrected and thiamine given *before* calories are pushed, and phosphate is watched daily through the first several days of feeding.

1.10 Screening: the SCOFF and the EAT-26

The five-question bedside screen. The SCOFF is a five-item verbal screen for anorexia and bulimia that takes about two minutes: *S*ick (making yourself vomit when uncomfortably full), *C*ontrol (lost control over eating), *O*ne stone (lost more than one stone, about 6.35 kg, in three months), *F*at (believe yourself fat when others say too thin), and *F*ood (food dominates life). Each "yes" scores one, and a total of **two or more** is a positive screen warranting fuller assessment, with a reported sensitivity around 84.6% and specificity around 89.6% in general-practice women (Luck et al. 2002); the original specialist-clinic study (Morgan, Reid and Lacey 1999)

reported a 100% sensitivity for anorexia and bulimia. The instrument itself is reproduced on the accompanying scale plate.

The self-report questionnaire. The EAT-26 (Eating Attitudes Test) is a 26-item self-report of eating attitudes and behaviours; a total score of **20 or more** is the conventional threshold prompting referral for clinical evaluation (Garner 1982). The EDE and its questionnaire form (EDE-Q) are the fuller research-standard interview and questionnaire but give dimensional scores rather than a single screening cut-off. All are aids to case-finding, not diagnoses in themselves.

INSTRUMENT	FORM	POSITIVE THRESHOLD
SCOFF	5-item verbal screen (Sick, Control, One stone, Fat, Food)	**≥ 2 "yes" answers**
EAT-26 (Eating Attitudes Test)	26-item self-report questionnaire	**Total ≥ 20 warrants assessment**
EDE-Q / EDE	Questionnaire / semi-structured interview	Dimensional; no single cut-off (reference standard)

The eating-disorder screens and their verified thresholds; SCOFF two-or-more is the number to hold, and the actual forms sit on the accompanying scale plate.

GOOD TO REMEMBER

- **SCOFF:** five verbal questions, one point each, a score of two or more is a positive screen for a probable eating disorder (sensitivity around 84.6%, specificity around 89.6%); it screens, it does not diagnose.
- **EAT-26:** a 26-item self-report; a total of 20 or more warrants clinical evaluation; a screening/severity aid, not a diagnostic instrument.

2 SCOFF POSITIVE CUT-OFF (YES ANSWERS)	**15** SEVERE ANOREXIA THRESHOLD (ADULT BMI, KG PER M SQUARED)	**1.4%** LIFETIME PREVALENCE IN WOMEN (0.2% IN MEN)	**6×** EXCESS MORTALITY VS GENERAL POPULATION

1.11 The India lens: non-fat-phobic anorexia and under-recognition

The fat phobia may be absent. A genuinely Indian and pan-Asian point: the explicitly stated fear of becoming fat is *relatively more often absent* in Asian populations, where the person rationalises the restriction as gastrointestinal discomfort, loss of appetite, or a culturally sanctioned religious fast rather than a wish to be thin, the "non-fat-phobic" presentation (DSM-5-TR 2022; ICD-11 CDDR). Both modern systems accommodate this precisely because Criterion B accepts persistent weight-defeating *behaviour* in place of a voiced fear. The transcultural framing is developed in the Consultation-liaison, geriatric and transcultural psychiatry notes; the applied lesson is to not withhold the diagnosis just because the patient never says "I want to be thin".

Under-recognition is the other reality. Eating disorders are under-detected in India, dismissed as ordinary dieting or as a physical complaint, and are still misread as a disease only of affluent urban young women; they occur across socioeconomic strata and in men. The clinical antidote is a low threshold to screen (the SCOFF at the bedside) and to weigh, take orthostatic vitals, and check potassium and the ECG in any young person with unexplained weight loss.

INDIA

Two things travel differently here. First, **non-fat-phobic anorexia** is relatively more common in Indian and other Asian patients, who attribute the restriction to abdominal discomfort, poor appetite or a religious fast; the diagnosis still stands on behaviour and a significantly low weight, so do not dismiss it for want of a stated fear of fatness. Second, eating disorders are widely under-recognised and stereotyped as an affluent-urban-female problem, missing men and lower-income patients; screen early with the SCOFF and always attach a weight, orthostatic vitals, a potassium and an ECG QTc.

WORKED EXAMPLE

A 17-year-old girl is brought by her mother for amenorrhoea and fainting. She is emaciated with fine downy **lanugo** on the back and forearms, cold peripheries, a pulse of 44 and lying-to-standing blood-pressure drop; BMI is 15.4. She denies wanting to be thin but eats tiny measured portions and exercises for hours. Bloods show potassium 3.0 and the ECG QTc is prolonged. This is anorexia nervosa, restricting subtype (no binge-purge), DSM-5-TR severe by BMI, ICD-11 with significantly low body weight; the **bradycardia**, **hypokalaemia** and QTc make her a medical-admission and refeeding-risk patient, and the absent stated fear does not exclude the diagnosis.

HOLD THIS

Anorexia nervosa is restriction-driven, significantly low body weight plus a fear of fatness (or behaviour that blocks weight gain) plus disturbed body-image or non-recognition of the danger; DSM-5-TR grades severity by BMI (severe 15 to 15.99, extreme under 15) and dropped the amenorrhoea criterion, ICD-11 uses BMI under 18.5 with significantly-low versus dangerously-low (under 14) grades; it kills more than any other psychiatric illness (six-fold, one in five by suicide), the SCOFF is positive at two, and the urgent complications are the prolonged QTc, hypokalaemia and refeeding hypophosphataemia.

2 · Refeeding syndrome

Refeeding syndrome is the group of potentially fatal fluid and electrolyte shifts that follow the reintroduction of nutrition, especially carbohydrate, into a severely malnourished body. It is the sharpest paradox in eating-disorder medicine: the patient who is sickest from starvation is the one most endangered by the treatment for it, so the danger arrives not from the illness but from feeding it back too fast. Because the psychiatrist is often the physician who admits and refeeds the profoundly underweight patient with anorexia nervosa, this is examinable as a hands-on emergency, not a footnote.

Why it matters, and how this note is framed. Two facts carry the topic. First, the biochemical hallmark is **hypophosphataemia**, a falling serum **phosphate** in the opening days of feeding, accompanied by falls in potassium and magnesium and by fluid and sodium retention. Second, the whole of prevention reduces to three moves: correct the electrolytes first, give **thiamine** before or with the first feed, and start the calories low and climb slowly. This is a management topic, so it is built from the verified guideline rows for cautious refeeding and the medical

stabilisation of anorexia; every rate and schedule below is drawn from that verified base, and any exact numeric threshold the parsed library could not confirm is flagged rather than invented.

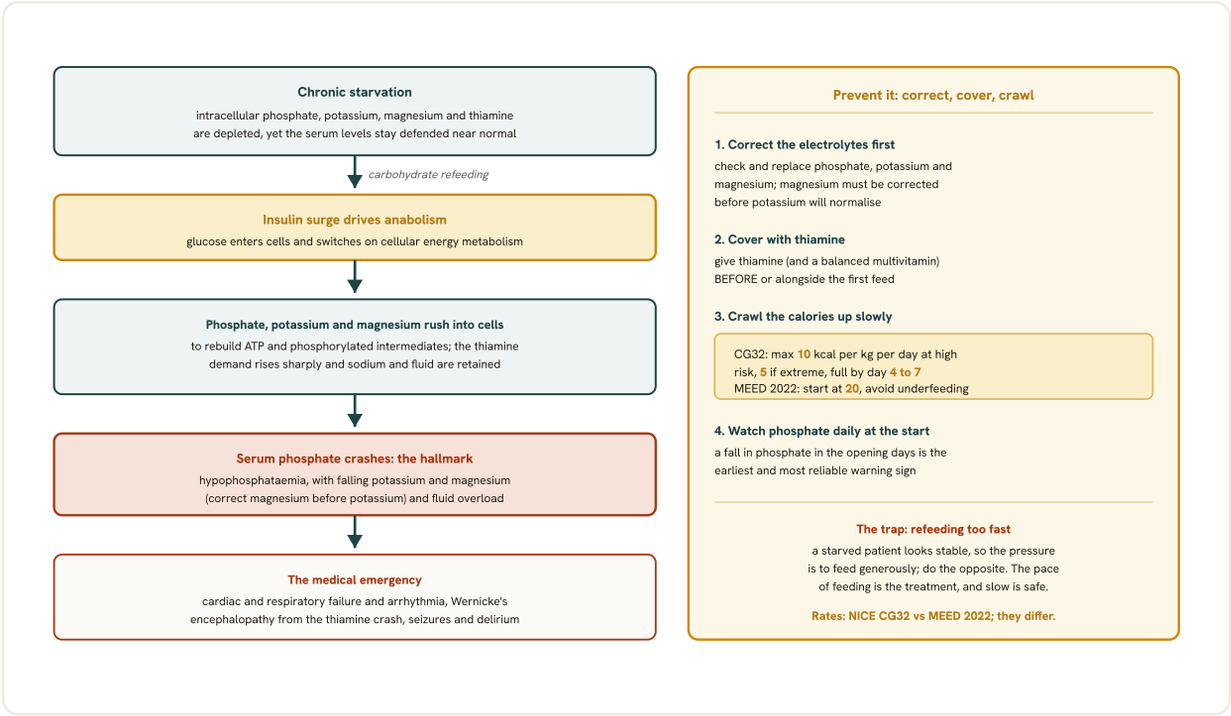


Fig 17.3 Refeeding syndrome. Starvation depletes the intracellular stores of phosphate, potassium, magnesium and thiamine while the serum levels stay defended near normal, so a routine blood test before feeding can look reassuring. When carbohydrate is reintroduced, the insulin surge switches anabolism back on and drives phosphate, potassium and magnesium into the cells to rebuild ATP, while the thiamine demand rises and sodium and fluid are retained. The serum pools then crash: the fall in phosphate is the hallmark and is what makes the syndrome lethal, and it is accompanied by hypokalaemia, hypomagnesaemia and fluid overload, producing cardiac and respiratory failure, arrhythmia, Wernicke's encephalopathy and seizures. Prevention is three moves: correct the electrolytes first (magnesium before potassium), cover with thiamine before the first feed, and crawl the calories up. The two numeric sources disagree and NICE NG69 sets no rate: NICE CG32 recommendation 1.4.8 caps the high-risk start at a maximum of 10 kcal per kg per day, uses only 5 at extreme risk with a body mass index under 14, and reaches full needs within 4 to 7 days, whereas RCPsych MEED CR233 (2022) starts at 20 kcal per kg per day, never below the immediate pre-admission intake, and warns against underfeeding. Quote whichever source the question names, and watch the phosphate daily. The pace of feeding is the treatment.

2.1 What refeeding syndrome is, and the deceptive normal serum level

The definition. Refeeding syndrome refers to the dangerous electrolyte and fluid shifts that occur when severely malnourished patients are refed too quickly; the clinical signature is fluid retention together with falling levels of phosphorus, magnesium and, less specifically, calcium (First Aid for Psychiatry). It is precipitated by the reintroduction of feeding, whether oral, enteral or parenteral, and the carbohydrate component is the main trigger.

The trap of the near-normal baseline. The catch that examiners love is that in the starved state the serum levels of phosphate, potassium and magnesium can sit near normal even though the intracellular and total-body stores are severely depleted, so a reassuring admission panel does not exclude risk. The fall only unmasks itself once feeding begins and the ions are driven out of the serum into the cells. The lesson is that risk is assessed from the degree of starvation and the feeding plan, not from a single normal set of electrolytes at presentation.

2.2 Pathophysiology: starvation depletion, then the insulin surge on carbohydrate

The starved state. Prolonged undernutrition switches the body to fat and protein catabolism, insulin secretion falls, and intracellular **phosphate**, potassium, magnesium and thiamine are steadily depleted while, as above, serum concentrations are defended and stay near-normal. The cell is running on empty even though the blood test looks acceptable.

The refeeding switch. When carbohydrate is reintroduced, blood glucose rises and insulin surges. Insulin drives glucose, and with it phosphate, potassium and magnesium, rapidly into the cells to rebuild ATP, phosphorylated intermediates and 2,3-diphosphoglycerate, so the already-depleted serum pools collapse. The same carbohydrate load sharply raises the demand for **thiamine**, an essential cofactor for glucose metabolism whose requirement rises with carbohydrate intake (KDT), and can outstrip depleted stores. Insulin also promotes renal sodium and water retention, expanding the extracellular volume. The mechanism is drawn together in the accompanying figure. This starvation-to-refeeding neuroendocrine backdrop, the wider hypothalamic-pituitary-gonadal, thyroid and adrenal changes of anorexia, is developed in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes.

MECHANISM IN ONE LINE

Starvation empties the intracellular stores while serum stays near-normal; carbohydrate refeeding triggers an insulin surge that pulls phosphate, potassium and magnesium into the cells and raises thiamine demand, so the serum pools crash and fluid is retained.

2.3 The hallmark: hypophosphataemia

Why phosphate is the marker. The defining biochemical abnormality of **refeeding syndrome** is hypophosphataemia. Phosphate is consumed on a large scale the moment anabolism restarts, because it is needed for ATP, for the phosphorylation of glucose entering the cell, and for 2,3-diphosphoglycerate in red cells; the newly insulin-stimulated cells strip it out of the serum faster than a depleted body can replace it. A falling serum phosphate in the first days of feeding is therefore the earliest and most reliable warning that refeeding syndrome is developing.

What low phosphate does. Because phosphate underpins cellular energy, severe hypophosphataemia is what makes the syndrome lethal: it impairs cardiac and respiratory muscle, red and white cell function and neuronal membranes, and is the proximate driver of the cardiac failure, arrhythmia and respiratory failure below. Serum phosphate is the single number watched most closely once feeding starts.

GOOD TO REMEMBER

- **Refeeding syndrome (defining criterion):** a fatal cluster of fluid and electrolyte shifts triggered by reintroducing nutrition (chiefly carbohydrate) into a severely malnourished patient; the defining biochemical criterion is *hypophosphataemia*, a falling serum phosphate in the opening days of feeding, alongside hypokalaemia, hypomagnesaemia and fluid/sodium retention; the discriminator from the starved baseline is that serum ions are near-normal until feeding drives them intracellularly.

2.4 The other shifts: hypokalaemia, hypomagnesaemia, and fluid and sodium retention

The two accompanying electrolyte falls. Insulin drives potassium into cells through the sodium-potassium pump, producing hypokalaemia, and shifts magnesium intracellularly, producing hypomagnesaemia. Both develop transiently during the early phases of refeeding even when the presenting electrolytes were normal (Tasman Psychiatry). Hypomagnesaemia matters doubly, because a low magnesium blocks the correction of potassium and prolongs the hypokalaemia, so the magnesium must be replaced for the potassium to normalise.

Fluid and sodium retention. The carbohydrate-driven insulin surge causes the kidney to retain sodium and water, expanding extracellular volume. In a heart already weakened by starvation, with reduced cardiac mass, this fluid load can precipitate peripheral oedema and, when refeeding is too rapid, congestive cardiac failure (Tasman Psychiatry). The salt-and-water retention is therefore not a benign cosmetic swelling but part of the cardiac danger.

ION OR COMPARTMENT	DIRECTION IN REFEEDING	MECHANISM AND CONSEQUENCE
Phosphate	**Falls, hypophosphataemia (the hallmark)**	Insulin drives it into cells for ATP, glucose phosphorylation, 2,3-DPG; drives cardiac, respiratory and neuronal failure
Potassium	Falls, hypokalaemia	Insulin-driven intracellular shift via the sodium-potassium pump; arrhythmia risk
Magnesium	Falls, hypomagnesaemia	**Intracellular shift; a low magnesium blocks correction of potassium**
Sodium and water	Retained	Carbohydrate/insulin-driven renal retention; oedema and volume overload, cardiac failure
Thiamine	Demand rises, stores deplete	**Cofactor for carbohydrate metabolism; deficiency risks Wernicke's encephalopathy**

The refeeding electrolyte and fluid shifts; the load-bearing points are hypophosphataemia as the hallmark and the rule that magnesium must be corrected before potassium will normalise.

2.5 The clinical consequences: cardiac, neurological, respiratory, muscular

The organ failures. The consequences follow directly from an energy-starved, electrolyte-depleted body being loaded too fast. The cardiac complications are the ones that kill: arrhythmia, sinus bradycardia, nonspecific electrocardiographic changes, QT prolongation and, with overly rapid refeeding, congestive cardiac failure and sudden death (Tasman Psychiatry). Neurologically, the surge in thiamine demand can precipitate Wernicke's encephalopathy (confusion, ophthalmoplegia, ataxia), and the electrolyte shifts can cause seizures and delirium.

Respiratory and muscular. Hypophosphataemia weakens the diaphragm and respiratory muscles and can cause respiratory failure, and the same energy failure of muscle produces rhabdomyolysis and profound weakness. The picture is thus a multi-organ collapse driven by

phosphate and thiamine depletion, which is why it is treated as a medical emergency and why the pace of feeding is the thing under a clinician's control.

SYSTEM	CONSEQUENCE OF REFEEDING SYNDROME
Cardiac	**Arrhythmia, bradycardia, QT prolongation, congestive cardiac failure, sudden death**
Neurological	Wernicke's encephalopathy (thiamine), seizures, delirium
Respiratory	Respiratory muscle weakness and failure (hypophosphataemia)
Musculoskeletal	Rhabdomyolysis, weakness
Haematological/immune	Haemolysis, leucocyte dysfunction, impaired immunity

Clinical consequences; the cardiac complications carry the mortality, and Wernicke's encephalopathy is the neurological reason thiamine is given before feeding.

GOOD TO REMEMBER

- **Wernicke's encephalopathy (in refeeding, criterion):** the triad of confusion, ophthalmoplegia and ataxia from thiamine deficiency, precipitated when carbohydrate refeeding raises thiamine demand beyond depleted stores; the defining prevention step is to give thiamine *before or with* the first carbohydrate, never after.

2.6 Identifying the high-risk patient

Who is at risk. Risk tracks the depth and duration of undernutrition, not the presenting electrolytes. The recognised high-risk features, drawn from the nutrition-support criteria that MARSIPAN and NICE adopt, are a very low body mass index, large recent unintentional weight loss, a period of little or no nutritional intake before feeding, and low pre-feeding levels of potassium, phosphate or magnesium; extreme risk is flagged by a very low BMI (in the verified eating-disorder guidance, a BMI under 14) or negligible intake over a prolonged period. The exact numeric cut-offs for each of these criteria are guideline-specific and are flagged below rather than stated from memory.

Beyond anorexia. The psychiatrist meets refeeding risk outside the eating-disorder clinic too. In severe alcohol dependence with little food intake before withdrawal, refeeding risk must be actively considered (Saunders, Addiction medicine), and it arises in chronic neglect, prolonged fasting and the post-critical-illness patient. The common thread is a sustained period of near-starvation followed by a feed, whatever the psychiatric context.

GOOD TO REMEMBER

- **High-risk identification (first step):** stratify by *depth and duration of starvation*, very low BMI, large recent weight loss, prolonged negligible intake, and low pre-feeding phosphate/potassium/magnesium, with a BMI under 14 marking extreme risk (verified NICE NG69 eating-disorder guidance); a near-normal admission electrolyte panel does *not* exclude risk, and the same risk applies in severe alcohol dependence and chronic neglect, not only anorexia.

2.7 Prevention: correct the electrolytes, cover with thiamine, crawl the calories up

The three moves. **Prevention** is the whole game, and it is built into every guideline: identify the at-risk patient, correct pre-existing electrolyte abnormalities, supplement phosphate, magnesium, potassium and thiamine from the outset, and begin feeding at a deliberately low energy level with a slow climb. The numeric supplementation schedule belongs to NICE CG32 (recommendation 1.4.8), not to NG69: oral thiamine 200 to 300 mg daily, vitamin B co strong and a balanced multivitamin immediately before and during the first 10 days of feeding, with *daily* potassium, phosphate and magnesium supplements unless pre-feeding plasma levels are high. Thiamine is given before or alongside the first carbohydrate, because loading glucose into a thiamine-deplete brain is what precipitates Wernicke's encephalopathy.

Correct first, do not chase. Electrolytes are replaced proactively rather than only when they fall, because waiting for hypophosphataemia to appear means waiting for the syndrome to start. Feeding is not withheld for a low phosphate; instead the phosphate is replaced while feeding continues cautiously. Thiamine and a balanced multivitamin and multimineral supplement are standard from the beginning.

MNEMONIC

CORRECT, COVER, CRAWL

Correct the electrolytes (replace phosphate, potassium, magnesium proactively); **Cover** with thiamine before or with the first feed; **Crawl** the calories up from a low starting rate. The three prevention moves for refeeding syndrome, in order.

RULE **Correct the electrolytes, give thiamine, start slow.** Refeeding syndrome is prevented, not treated: replace phosphate, potassium and magnesium proactively, give thiamine before or with the first carbohydrate, and begin feeding at a low energy level with a gradual climb.

GOOD TO REMEMBER

- **Prevention (the principle):** correct electrolytes first, cover with thiamine before or with feeding, and crawl the calories up from a low start, with daily phosphate, magnesium, potassium and thiamine supplementation (NICE CG32 recommendation 1.4.8; NICE NG69 2017 carries no numbers of its own and refers to MEED); a low phosphate is replaced *while* feeding continues cautiously, not a reason to starve the patient further.

2.8 The refeeding rate: start low, climb slow

The starting energy. The controllable variable is the **refeeding rate**, and this is the one place in the topic where the guidelines openly disagree. NICE NG69 (2017) carries *no* kcal figure at all: recommendation 1.11.10 asks only for a standard operating procedure that avoids both under-nutrition and refeeding syndrome, and refers the clinician to the Royal College of Psychiatrists' Medical Emergencies in Eating Disorders guidance. The numbers therefore come from two other places, and they do not agree. The general nutrition-support guidance, NICE CG32 (recommendation 1.4.8), starts the patient at high risk of refeeding problems at a *maximum* of **10 kcal per kg per day**, increasing slowly to meet or exceed full needs within **4 to 7 days**, and uses

only **5 kcal per kg per day** in extreme cases (for example a BMI under 14, or negligible intake for more than 15 days) with continuous cardiac-rhythm monitoring. The eating-disorder guidance, RCPsych MEED CR233 (2022), starts higher, at **20 kcal per kg per day** and never below the immediate pre-admission intake, builds the calories up swiftly, and warns explicitly against underfeeding. Quote whichever source the question names.

Slow enough to be safe. The rate is set low precisely because the insulin response scales with the carbohydrate delivered: feed cautiously and the intracellular shift is gentle enough for replacement to keep pace; feed fast and the serum phosphate, potassium and magnesium crash faster than they can be corrected. Historically some clinicians started even lower than these figures; the exact lowest-risk starting rate and the tightest escalation schedule differ between guidelines and are flagged below.

SOURCE	STARTING RATE (KCAL PER KG PER DAY)	NOTE
NICE NG69 2017 (eating disorders)	no kcal figure given	Recommendation 1.11.10: use a standard operating procedure that avoids under-nutrition *and* refeeding syndrome; refers to MEED
NICE CG32 1.4.8, high risk of refeeding problems	**maximum 10**	Increase slowly to meet or exceed full needs within 4 to 7 days
NICE CG32 1.4.8, extreme cases (BMI under 14, or negligible intake over 15 days)	**only 5**	Slowest start; continuous cardiac-rhythm monitoring
RCPsych MEED CR233 2022 (eating disorders)	**start at 20**	**Never below the immediate pre-admission intake; build up swiftly; avoid underfeeding**

The two numeric refeeding positions, and they disagree: NICE CG32 caps the high-risk start at 10 kcal per kg per day, RCPsych MEED CR233 2022 opens at 20 and warns against underfeeding. NICE NG69 itself sets no rate. Quote whichever source the question names.

10	5	20	4 to 7
NICE CG32 MAXIMUM START AT HIGH RISK (KCAL PER KG PER DAY)	CG32 EXTREME CASES, BMI UNDER 14 (KCAL PER KG PER DAY)	RCPSYCH MEED CR233 2022 START (KCAL PER KG PER DAY)	CG32 DAYS TO MEET OR EXCEED FULL NEEDS

GOOD TO REMEMBER

- **Refeeding rate (the value):** NICE NG69 gives no kcal figure at all, and the two sources that do disagree, so quote the one the question names. NICE CG32 1.4.8: a *maximum* of 10 kcal per kg per day at high risk, only 5 in extreme cases (BMI under 14), increasing slowly to meet or exceed full needs within 4 to 7 days. RCPsych MEED CR233 2022: start at 20 kcal per kg per day, no lower than the immediate pre-admission intake, build up swiftly and avoid underfeeding. Either way the rate is the one variable fully under the clinician's control and the single strongest lever against the syndrome.

2.9 Monitoring: what to watch, and when

What is monitored. **Monitoring** centres on the electrolytes that move: serum phosphate above all, plus potassium and magnesium, together with glucose, sodium and fluid balance, and clinical signs of cardiac overload. Physical monitoring in the underweight patient also covers weight, orthostatic blood pressure, pulse, temperature and the electrocardiogram (QTc), with the eating-disorder guidance emphasising potassium, magnesium and phosphate specifically (NICE NG69 2017).

The schedule. Electrolytes are checked frequently once feeding starts, monitored daily for the first week in the verified guidance, with the tightest surveillance over the opening days when the intracellular shift is steepest; the extreme-risk patient warrants continuous cardiac monitoring from the outset. As feeding stabilises and the electrolytes hold, the frequency is relaxed. The phosphate trend across the first three days is the earliest signal that the syndrome is developing.

TRAP **Refeeding too fast and trusting a normal admission panel.** The classic errors are pushing calories up quickly to correct weight and being reassured by near-normal presenting electrolytes; both miss the falling phosphate of the opening days. Feed slowly and re-check the electrolytes, do not chase the weight.

GOOD TO REMEMBER

- **Monitoring (the value):** watch phosphate, potassium and magnesium (plus glucose, sodium, fluid balance and the electrocardiogram), checked daily for the first week and most intensively over the opening days (NICE NG69 2017); a falling serum phosphate in the first three days is the earliest warning, and the extreme-risk patient needs continuous cardiac monitoring from the outset.

2.10 The guideline positions, and the Indian frame

MARSIPAN leads. For the medical stabilisation of the severely ill patient with anorexia, the lead guidance is MARSIPAN (Management of Really Sick Patients with Anorexia Nervosa; RCPsych, MARSIPAN 2014, updated as the Medical Emergencies in Eating Disorders guidance 2022). Its position is a coordinated medical-and-psychiatric approach: assess refeeding risk early, refeed cautiously, replace electrolytes and give thiamine, and monitor cardiac status closely, with escalation of care for the highest-risk patient. Alongside it, NICE NG69 (2017) sets the eating-disorder care pathway but carries no kcal figure of its own, asking at recommendation 1.11.10 only for a standard operating procedure that avoids both under-nutrition and refeeding syndrome and referring to MEED; the general nutrition-support guidance NICE CG32 (recommendation 1.4.8) supplies the numeric refeeding-risk criteria, the cautious maximum rate and the replacement doses; MEED CR233 (2022) supplies the higher eating-disorder-specific starting rate that CG32 disagrees with; and the Indian Psychiatric Society CPG (IPS 2017) frames eating-disorder care for local practice. Where an exact MARSIPAN threshold, dose or criterion could not be confirmed against the parsed library, it is flagged below rather than stated.

The Indian reality. Refeeding is a recognition problem before it is a resource problem, and phosphate, potassium, magnesium and thiamine replacement are all inexpensive and widely stocked in India.

INDIA

Eating disorders are under-recognised in India, and the atypical, non-fat-phobic presentation, weight loss framed as appetite loss or a somatic complaint rather than a fear of fatness, means the severely underweight patient can reach care late and profoundly starved, exactly the patient most at risk of refeeding syndrome. The psychiatrist also meets the risk in severe alcohol dependence with poor intake and in chronic self-neglect. The corrective is cheap and available: thiamine, phosphate, potassium and magnesium replacement cost little, so the failure that kills is not funding but not thinking of the syndrome and refeeding too fast. Correct the electrolytes, give thiamine first, and start the calories low.

HOLD THIS

Refeeding syndrome is carbohydrate-triggered, insulin-driven, and defined by *hypophosphataemia* with falling potassium and magnesium and fluid retention in a severely starved patient whose admission electrolytes looked normal; you prevent it, not treat it, by correcting electrolytes, giving thiamine before or with the first feed, and starting low, on whichever source the question names (NICE CG32 1.4.8: a maximum of 10 kcal per kg per day, only 5 if BMI is under 14, full needs within 4 to 7 days; RCPsych MEED CR233 2022: start at 20 and avoid underfeeding), with daily electrolyte monitoring for the first week.

3 · The management of anorexia nervosa

The **management of anorexia** nervosa is the topic where examiners test whether you can hold a hierarchy in your head: this is a disorder with the highest mortality in psychiatry, yet the treatment that saves lives is not a drug at all. The answer is a ladder. You decide *where* to treat (least restrictive, but escalate for medical danger), you stabilise the failing body (the medical-risk assessment and, if needed, admission), you restore weight through **nutritional rehabilitation** done slowly enough to avoid killing the patient by feeding them, and you deliver a manualised psychological therapy as the primary treatment. Medication sits off to one side, treating comorbidity, never inducing the weight gain. The single fact that anchors every answer is that recovery is driven by weight restoration plus a specialist talking therapy, and that **no drug is first-line.**

Why it matters clinically. This is a management topic, so the marks sit in the guideline-level plan, the setting, the medical stabilisation, the refeeding rate, the named therapies and the medication caveat, more than in the phenomenology, which belongs to the anorexia diagnosis topic and the eating-disorder differentiation grid. The lead position is NICE NG69 (2017), with the MARSIPAN framework for the medically compromised patient, then RANZCP and WFSBP where they add or qualify. The neuroendocrine axes wrecked by starvation (the hypothalamic-pituitary-gonadal, thyroid and adrenal changes) are developed in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes; the general cognitive-behavioural technique that CBT-ED specialises belongs to the Psychotherapies, clinical assessment, MSE and nosology notes; the fine detail of the refeeding electrolyte thresholds and the monitoring schedule is owned by the refeeding-syndrome topic and its diagram. This topic

leans on the differentiation grid and the accompanying refeeding figure rather than adding a new figure of its own.

3.1 The setting: least-restrictive care, an outpatient or a day-patient by default

Treat in the least restrictive place that is safe. Most patients with anorexia nervosa are managed as outpatients or on a day-patient basis; hospital admission is reserved for those at risk of medical or psychological compromise (RANZCP 2014). The principle is explicit: use the least restrictive environment, escalating only when the body or the risk demands it. NICE frames specialist eating-disorder services, whether the patient is having a specific intervention or not, as the setting where psychoeducation and coordinated multidisciplinary monitoring of weight, mental and physical health, and risk are provided (NICE NG69 2017).

What tips the decision to admit. Emergency admission is indicated for the patient with severe electrolyte imbalance, severe malnutrition, severe dehydration, or signs of incipient organ failure (NICE NG69 2017). The choice of setting is therefore a medical-risk decision as much as a psychiatric one, which is why the medical-risk assessment comes next.

GOOD TO REMEMBER

- **Setting (the where-to-treat step):** the governing value is *least restrictive that is safe*; anorexia nervosa is treated as an outpatient or a day patient in most instances, with admission reserved for medical or psychological compromise (severe electrolyte imbalance, severe malnutrition or dehydration, incipient organ failure).

3.2 Medical stabilisation: the medical-risk assessment and when to admit

Assess the risk before you feed. **Medical stabilisation** begins with a structured medical-risk assessment. Monitor weight, orthostatic (lying and standing) blood pressure, pulse, temperature and peripheral circulation, and check full blood count, urea and electrolytes with particular attention to potassium, magnesium and phosphate, liver function, glucose, an ECG for the QTc interval, and bone density by DEXA (consider a scan after one year of underweight in children and young people, after two years of underweight in adults, or earlier if there is bone pain or recurrent fractures; NG69 explicitly says not to use single measures such as BMI or duration of illness to drive decisions) (NICE NG69 2017). Frequency rises for the severely underweight or the rapidly losing patient.

MARSIPAN, the framework for the really sick patient. The MARSIPAN guidance (Management of Really Sick Patients with Anorexia Nervosa) stratifies medical risk from parameters such as BMI and rate of weight loss, bradycardia and postural blood-pressure drop, hypothermia, the core biochemistry (potassium, phosphate, magnesium, glucose), ECG changes, and muscle power gauged clinically by the sit-up-squat-stand manoeuvre, sorting patients into risk zones that dictate the urgency and the site of care. It is the medically-led companion to NICE NG69 and the reference point when a patient is too unwell for routine outpatient refeeding. Its exact red-zone numeric cut-offs are not reproduced here (see gaps) and are cross-referenced to the refeeding-syndrome topic.

GOOD TO REMEMBER

- **Medical stabilisation (the safety step):** the principle is *assess medical risk first, correct it before you push calories*; monitor weight, orthostatic BP, pulse, temperature, U&E (potassium, magnesium, phosphate), LFT, glucose, ECG (QTc) and DEXA, and use the MARSIPAN risk stratification to decide who is too unwell for outpatient care.

3.3 Refeeding safely: the low-and-slow principle and the daily electrolyte watch

Feeding can kill; feed slowly. **Nutritional rehabilitation** must be started cautiously to avoid refeeding syndrome, the fluid, electrolyte and mineral catastrophe that follows when a starved body meets a carbohydrate load: insulin surges, intracellular uptake of phosphate, potassium and magnesium empties the serum, and the hallmark is hypophosphataemia (NICE NG69 2017; Kaplan and Sadock CTP 2024). The instruction is therefore to start low and go slow, and the two sources that carry numbers disagree about how low. NICE NG69 gives no kcal figure: recommendation 1.11.10 asks only for a standard operating procedure that avoids both under-nutrition and refeeding syndrome, and refers to the RCPsych Medical Emergencies in Eating Disorders guidance. The general nutrition-support guidance NICE CG32 (recommendation 1.4.8) starts the high-risk patient at a maximum of 10 kcal per kg per day, and only 5 kcal per kg per day in extreme cases such as a BMI under 14, with oral thiamine and a balanced multivitamin before and during the first 10 days of feeding plus potassium, phosphate and magnesium supplements, increasing slowly to meet or exceed full needs within 4 to 7 days. RCPsych MEED CR233 (2022) starts higher, at 20 kcal per kg per day and no lower than the immediate pre-admission intake, builds up swiftly and warns against underfeeding. Quote whichever source the question names.

Watch the electrolytes early and often. Monitor electrolytes daily through the first week, when the risk of the intracellular shift is highest, checking phosphate over the first three days especially, and give thiamine from the outset to pre-empt Wernicke encephalopathy. The precise phosphate, potassium and magnesium thresholds and the full monitoring schedule are owned by the refeeding-syndrome topic and its diagram; the message to carry into a management answer is the sequence *correct the electrolytes, give thiamine, start slow*.

SOURCE AND CLINICAL STATE	STARTING RATE (KCAL PER KG PER DAY)	SUPPLEMENT DAILY	ESCALATION
NICE CG32 1.4.8, high risk of refeeding problems	**maximum 10**	Phosphate, magnesium, potassium, thiamine	Increase slowly to meet or exceed full needs within 4 to 7 days
NICE CG32 1.4.8, extreme (e.g. BMI under 14)	**only 5**	Phosphate, magnesium, potassium, thiamine	Continuous cardiac-rhythm monitoring, electrolytes daily
RCPsych MEED CR233 2022, eating disorders	**start at 20**	Phosphate, magnesium, potassium, thiamine	Build up swiftly, never below the pre-admission intake
NICE NG69 2017, eating disorders	no kcal figure given	Recheck electrolytes daily	**Standard operating procedure only; refers to MEED**

The two numeric refeeding positions in anorexia nervosa, and they disagree: NICE CG32 caps the high-risk start at 10 kcal per kg per day, RCPsych MEED CR233 2022 opens at 20 and warns against underfeeding, and NG69 sets no rate at all. Quote whichever source the question names; exact electrolyte thresholds are held in the refeeding-syndrome topic and the accompanying refeeding figure.

TRAP **Refeeding too fast.** Piling on calories to correct a dangerously low weight is the classic lethal error: the carbohydrate load drives phosphate, potassium and magnesium into cells, hypophosphataemia precipitates cardiac failure, arrhythmia and death. Start low (NICE CG32: a maximum of 10 kcal per kg per day, only 5 if extreme; RCPsych MEED CR233 2022 opens at 20 and warns against underfeeding, so quote the source the question names), supplement phosphate and thiamine, and check electrolytes daily through the first week.

GOOD TO REMEMBER

- **Refeeding (the nutritional-rehabilitation safety step):** the hallmark of refeeding syndrome is *hypophosphataemia* and the prevention is *correct the electrolytes, give thiamine, start slow*; open low on NICE CG32 (a maximum of 10 kcal per kg per day, only 5 if extreme with a BMI under 14, full needs within 4 to 7 days) or at 20 kcal per kg per day on RCPsych MEED CR233 2022, which warns against underfeeding, quoting whichever the question names; supplement phosphate, magnesium, potassium and thiamine, and monitor electrolytes daily through the first week.

3.4 Nutritional rehabilitation: weight restoration is the treatment goal

Healthy weight is the target, not an afterthought. NICE is explicit that helping the person reach a healthy body weight or BMI for their age is a key treatment goal, because weight gain is what unlocks the psychological, physical and quality-of-life recovery (NICE NG69 2017). Once refeeding is safely under way, the calorie prescription is escalated toward weight restoration within a multidisciplinary plan; dietary counselling is offered only as part of that multidisciplinary approach, alongside an age-appropriate oral multivitamin and multimineral supplement until the diet meets reference values (NICE NG69 2017). WFSBP adds that zinc supplementation may be considered as an adjunct (WFSBP grade B evidence, 2011).

Bone and endocrine sequelae. Consider a bone-mineral-density scan after one year of underweight in the young and after two years in adults (not repeated more than once a year), and for young women aged 13 to 17 with long-standing low weight, low bone density and a bone age

over 15, transdermal 17-beta-estradiol with cyclic progesterone may be considered (NICE NG69 2017). The starvation endocrinopathy that drives the bone loss (the suppressed hypothalamic-pituitary-gonadal axis, the low-T3 state) is developed in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes.

PEARL

In anorexia nervosa, food is the medicine and weight is the vital sign. Almost every physical and cognitive abnormality, the bradycardia, the amenorrhoea, the concrete rigid thinking, the low bone density, is downstream of the starved state and reverses, at least partly, with weight restoration. This is why the plan refuses to treat the low weight as a side issue: restore the weight and most of the pathology follows it back toward normal.

GOOD TO REMEMBER

- **Nutritional rehabilitation (the weight-restoration step):** the governing value is that *reaching a healthy weight/BMI is the key treatment goal* because weight gain unlocks recovery; deliver it in a multidisciplinary plan with an oral multivitamin-multimineral supplement (zinc as a possible adjunct), and screen bone density by DEXA after one year (young) or two years (adults) of underweight.

3.5 Psychological therapy is the primary treatment in adults: CBT-ED, MANTRA, SSCM

The first-line psychological choice. For adults with anorexia nervosa, NICE offers a choice of three manualised psychological therapies as first-line: individual eating-disorder-focused cognitive behavioural therapy (CBT-ED), the Maudsley Anorexia Nervosa Treatment for Adults (MANTRA), or Specialist Supportive Clinical Management (SSCM) (NICE NG69 2017). CBT-ED runs typically to 40 sessions over about 40 weeks. If all three are unacceptable, contraindicated or ineffective, eating-disorder-focused focal psychodynamic therapy (FPT) is the next option (NICE NG69 2017). RANZCP concurs at the guideline level: use specialist, therapist-led, manualised psychological therapies across all age groups, within a multi-axial approach that addresses nutritional, medical and psychological aspects together (RANZCP 2014).

Therapy is primary, not adjunctive. The talking therapy is not an add-on to feeding; it is the treatment that changes the disorder, delivered in parallel with weight restoration. The general CBT technique on which CBT-ED is built belongs to the Psychotherapies, clinical assessment, MSE and nosology notes; this topic owns the disorder-specific therapies.

RULE **Weight plus a specialist therapy, and no drug first-line.** The primary treatment of anorexia nervosa is nutritional rehabilitation to a healthy weight *plus* a manualised psychological therapy (in adults CBT-ED, MANTRA or SSCM; in the young, family-based treatment). Medication has no established role in inducing weight gain: *no drug first-line*.

WORKED EXAMPLE

A 24-year-old woman with a BMI of 15.5, amenorrhoeic, restricting and over-exercising but medically stable, is seen in a specialist eating-disorder clinic. The correct plan is outpatient care (least restrictive, she is not medically compromised), a multidisciplinary weight-restoration programme with dietetic input and a multivitamin-multimineral supplement, and a choice of first-line psychological therapy, CBT-ED, MANTRA or SSCM, agreed with her. She does not need, and should not be started on, an antidepressant or an antipsychotic to make her gain weight; if she had comorbid depression that persisted after some weight gain, that would be treated on its own merits, with ECG monitoring given the cardiac risk at low weight.

3.6 The three adult therapies up close: CBT-ED, MANTRA and SSCM

What each therapy is. **CBT-ED** is the eating-disorder-specific adaptation of CBT: it targets the over-evaluation of shape, weight and control, the dietary rules and the safety behaviours, and it builds regular eating and behavioural experiments that disconfirm the feared consequences of weight gain, all while working toward weight restoration. **MANTRA**, the Maudsley model, is a manualised individual therapy built around the maintaining factors of the illness (a rigid, detail-focused, harm-avoidant cognitive style, unhelpful beliefs about the value of anorexia to the self, and interpersonal and emotional-avoidance patterns), working collaboratively to shift them. **SSCM** combines supportive psychotherapy with clinical management: education, careful physical and weight monitoring, and a focus on the core anorexic symptoms that keep the patient underweight, without a specific psychological technique. All three are first-line and chosen by patient preference and availability.

THERAPY	WHAT IT IS	POPULATION AND COURSE
CBT-ED	Eating-disorder-focused CBT: over-evaluation of shape/weight/control, dietary rules, regular eating, behavioural experiments	Adults, first-line; typically 40 sessions over about 40 weeks
MANTRA	Maudsley individual model targeting the cognitive style and beliefs that maintain the illness	Adults, first-line
SSCM	Supportive psychotherapy plus clinical management; education, monitoring, focus on core anorexic symptoms	Adults, first-line
FPT	Eating-disorder-focused focal psychodynamic therapy	Adults, second-line if the three above fail or are unacceptable

The adult first-line psychological therapies for anorexia nervosa and the second-line psychodynamic option (NICE NG69 2017); chosen by patient preference and local availability.

GOOD TO REMEMBER

- **CBT-ED, MANTRA and SSCM (the adult primary therapies):** the one value is that *all three are first-line and equivalent*, chosen by patient preference; CBT-ED (typically 40 sessions over about 40 weeks) targets the over-evaluation of shape and weight, MANTRA targets the maintaining cognitive style, SSCM pairs supportive therapy with clinical monitoring, and FPT is the second-line fallback.

3.7 Children and young people: family-based treatment first

Treat the family, not just the child. For children and young people with anorexia nervosa, the first-line treatment is anorexia-nervosa-focused family therapy (FT-AN), the manualised **family-based treatment** in which parents are coached to take charge of refeeding and weight restoration before control is gradually handed back to the young person as they recover (NICE NG69 2017; RANZCP 2014). It is delivered as single-family therapy or as combined single- and multi-family work, typically over 18 to 20 sessions across about one year. If FT-AN is unacceptable, contraindicated or ineffective, the alternatives are individual CBT-ED or adolescent-focused psychotherapy for anorexia nervosa (AFP-AN) (NICE NG69 2017).

Why the family is the lever. In the young, still-dependent patient the parents are the practical agents of weight restoration, which is why the evidence base (originally the Maudsley model) puts family work, not individual therapy, at the front of the line for this age group.

GOOD TO REMEMBER

- **Family-based treatment (the first-line therapy in the young):** the anchor is that *FT-AN is first-line for children and young people*, parents lead refeeding then hand control back; typically 18 to 20 sessions over about one year, single- or multi-family, with individual CBT-ED or AFP-AN as the fallback.

3.8 No drug is first-line: medication, comorbidity and the cardiac caveat

The rule stated plainly. Do not offer medication as the sole treatment for anorexia nervosa (NICE NG69 2017). There is **no drug first-line**: no agent has an established role in inducing weight gain, and the pharmacology that works is aimed at comorbidity, treated on its own merits and preferably once some weight has been regained. If an SSRI is considered (for comorbid depression or OCD), remember that the evidence for efficacy in the low-weight patient is poor and the cardiac risk (QTc prolongation) is increased at low body weight, so an ECG and electrolyte check accompany any such prescription (NICE NG69 2017). The deep SSRI pharmacology, including fluoxetine (Indian brands Prodep, Fludac, Flunil), which is the licensed drug for *bulimia* and not for anorexia, is developed in the Mood disorders: pharmacotherapy notes.

Olanzapine, the only agent with a signal. Low-dose olanzapine (of the order of 2.5 to 5 mg per day) may have a small weight-gain effect and can help the severe, unremitting, ruminative patient, but it is explicitly not first-line and not for routine use (NICE NG69 2017); WFSBP grades the olanzapine weight-gain signal as grade B and other atypicals as weaker (WFSBP 2011). The

through-line for an answer: medication is adjunctive and comorbidity-directed, never the treatment that restores weight.

TRAP **Prescribing a drug to make the patient gain weight.** Reaching for an antidepressant, or an antipsychotic, as the way to treat anorexic low weight is the classic management error: no drug is first-line and none reliably induces weight gain. Weight is restored by nutrition and a specialist therapy; medication only treats comorbidity, with an ECG because the QTc is vulnerable at low weight.

GOOD TO REMEMBER

- **Medication in anorexia (no drug first-line):** the one value is that *no drug is first-line and none induces weight gain reliably*; do not offer medication as the sole treatment, treat comorbidity on its merits (SSRI cautiously, poor low-weight efficacy and QTc risk, ECG advised), and reserve low-dose olanzapine (2.5 to 5 mg per day) as a non-routine adjunct, not a first-line agent.

3.9 Guideline convergence, compulsory treatment and the enduring illness

Where the guidelines land. Read together, the guidelines converge: least-restrictive setting, medical stabilisation with safe refeeding, weight restoration as the goal, a manualised psychological therapy as the primary treatment, and medication kept for comorbidity (NICE NG69 2017; RANZCP 2014; WFSBP 2011). For the small group who lack capacity because of the eating disorder, refuse treatment and face a significant risk to life or long-term health, compulsory treatment under mental-health legislation may be considered, coordinated with a hospital ethics committee; in India the Mental Healthcare Act 2017 supported-admission provision (Section 89) may apply, and the co-occurring physical illness is treated under the ordinary treatment and consent provisions of that admission, not under Section 105, which deals with the question of mental illness in a judicial process (MHCA 2017). In chronic, severe and enduring anorexia nervosa, a harm-minimisation approach is adopted rather than repeated coercive weight restoration (RANZCP 2014).

GUIDELINE	SETTING	PRIMARY TREATMENT	MEDICATION STANCE
NICE NG69 2017	Least restrictive; admit for medical/psychological compromise	Adults: CBT-ED, MANTRA or SSCM. Young: FT-AN first-line	**Not the sole treatment; no drug first-line; olanzapine non-routine**
MARSIPAN 2014	Medical risk stratification decides site and urgency for the sick patient	Safe, low-and-slow refeeding under medical oversight	Medical management, not drug-led weight gain
RANZCP 2014	Outpatient or a day patient in most instances; least restrictive	Specialist therapist-led manualised therapy; family-based in the young	Harm minimisation in chronic (SEED) illness

GUIDELINE	SETTING	PRIMARY TREATMENT	MEDICATION STANCE
WFSBP 2011	Multidisciplinary	Nutritional and psychological rehabilitation	Olanzapine grade B (weight gain), zinc adjunct grade B; not first-line

Guideline-level management of anorexia nervosa, NICE NG69-led with MARSIPAN for the medically compromised, converging on least-restrictive care, safe refeeding, psychological therapy as primary and no drug first-line (verified from the drug-guideline supplement).

INDIA

Two India-specific points earn marks. First, the atypical, non-fat-phobic presentation: the absence of an expressed intense fear of weight gain ("fat phobia") is relatively more common in Asian populations, where dietary restriction is attributed to a culturally sanctioned complaint such as gastrointestinal discomfort, or to cultural or religious motives such as fasting, so an Indian patient can meet criteria for anorexia nervosa with prominent low weight but little overt shape-and-weight talk (DSM-5-TR 2022; ICD-11 CDDR). Missing this, and calling a non-fat-phobic starving patient "not anorexia", is the classic error. Second, eating disorders remain under-recognised and under-served in India, which delays the medical-risk assessment and safe refeeding that this ladder depends on. Where capacity is lost and life is at risk, compulsory care runs through the Mental Healthcare Act 2017 (supported admission under Section 89; Section 105 is *not* the physical-illness provision, it covers the question of mental illness in a judicial process), coordinated with the hospital ethics committee.

10 or 20
REFEEDING START (KCAL PER KG PER DAY): CG32 CAPS AT 10 (5 IF EXTREME), MEED 2022 OPENS AT 20

40
CBT-ED SESSIONS OVER ABOUT 40 WEEKS IN ADULTS (NICE NG69)

18 to 20
FT-AN FAMILY-THERAPY SESSIONS OVER ABOUT ONE YEAR IN THE YOUNG

None
DRUG IS FIRST-LINE FOR ANOREXIA (MEDICATION HAS NO WEIGHT-GAIN ROLE)

HOLD THIS

The management of anorexia nervosa is a ladder: treat in the least restrictive safe setting, stabilise the body (medical-risk assessment, MARSIPAN, safe low-and-slow refeeding to pre-empt hypophosphataemia), restore weight as the treatment goal, and deliver the primary treatment, a manualised psychological therapy (adults: CBT-ED, MANTRA or SSCM; young: family-based treatment), while no drug is first-line and medication only treats comorbidity.

4 · Bulimia nervosa

The normal-weight eating disorder. **Bulimia nervosa** is the disorder of the **binge-purge cycle**: recurrent episodes of binge eating with a sense of loss of control, followed by recurrent inappropriate compensatory behaviour to undo the calories, in a person whose self-worth is hostage to weight and shape and whose body weight is typically normal or above normal. That last fact is why it hides: unlike anorexia nervosa, there is no emaciation to give it away, so the examiner tests whether you can recognise it from the behaviour and read the physical footprint of purging on the body. This note owns both halves of the exam question: it *teaches* the features, the ICD-11 and DSM-5-TR criteria with their frequency and duration, and the **medical**

complications of purging (the electrolyte derangement and **hypokalaemia**, the metabolic alkalosis, **Russell's sign**, dental erosion, parotid swelling, and the oesophageal and cardiac risks), and it delivers the guideline-anchored *management*.

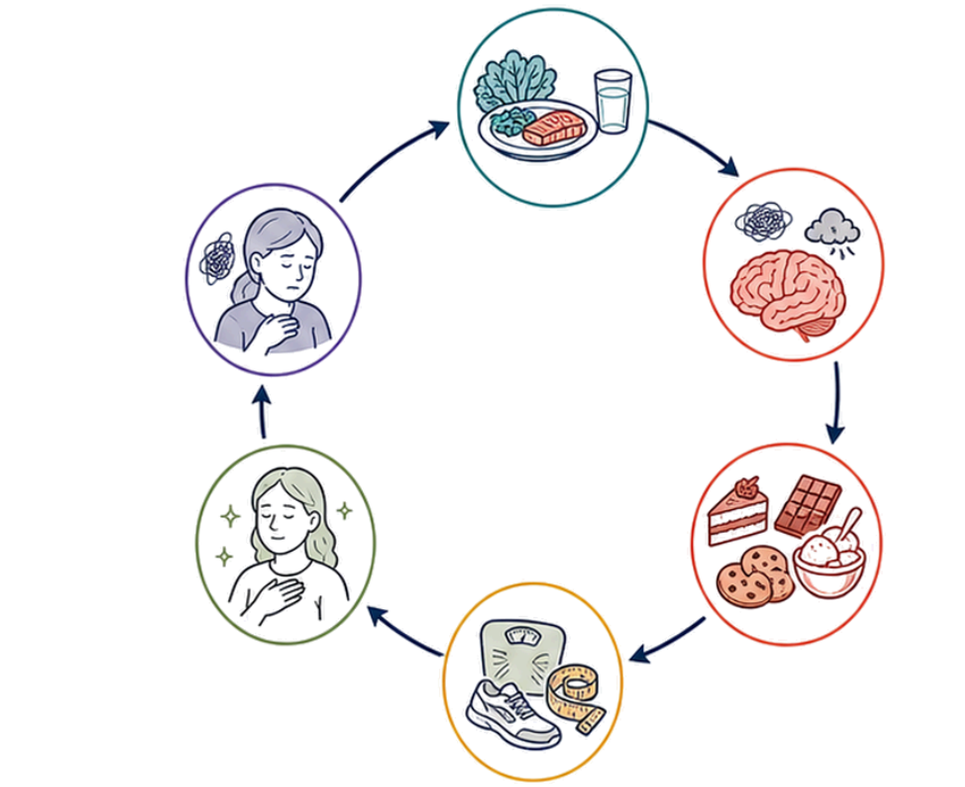


Fig 17.4 The bulimia nervosa cycle links rigid dietary restraint with hunger or negative affect, binge eating, compensatory behaviour, brief relief and shame that renews further restraint.

Why the management matters. This is a management topic, so the marks sit in the stepped-care plan and the one licensed drug. The lead position is NICE NG69 (2017): a psychological therapy first (guided self-help, then eating-disorder-focused CBT), with medication never the sole treatment. There is one drug to know cold, fluoxetine at a dose *higher* than its antidepressant dose, and one drug that is dangerous here. The deep SSRI pharmacology (fluoxetine's kinetics, adverse effects and interactions) is developed in the Mood disorders: pharmacotherapy notes; the general cognitive-behavioural technique that CBT-ED specialises belongs to the Psychotherapies, clinical assessment, MSE and nosology notes. The accompanying figure maps the binge-purge cycle and the purging complications onto the body; the SCOFF form sits on the accompanying scale plate.

4.1 The clinical picture: the binge-purge cycle

One cycle, four moving parts. Bulimia nervosa is built from a self-perpetuating loop. First, a *binge*: eating, in a discrete period usually under two hours, an amount of food definitely larger than most people would eat in similar circumstances, accompanied by a *sense of loss of control*, the inability to stop or limit what is eaten (DSM-5-TR 2022). The binge is usually secret, driven most often by negative affect, and continues until the person is uncomfortably or painfully full. Second, the guilt and fear of weight gain that follow. Third, a *compensatory behaviour* to undo it: most commonly self-induced vomiting, or misuse of laxatives, diuretics or other medications, fasting, or excessive exercise. Fourth, transient relief, then renewed dietary restraint that primes the next binge. The over-evaluation of shape and weight sits at the centre, keeping the whole cycle turning.

The weight is the giveaway that there is no giveaway. Because the binges and the purging roughly cancel, the person with bulimia nervosa is characteristically of *normal or above-normal weight* (Kaplan and Sadock CTP 2024). This is the single most useful discriminator from the binge-purge subtype of anorexia nervosa, where the weight is significantly low, and it is why bulimia nervosa is so often missed on inspection alone.

> **GOOD TO REMEMBER**
>
> - **Bulimia nervosa (the syndrome):** the defining criterion is the recurrent *binge-purge cycle*, binge eating with a sense of loss of control plus recurrent inappropriate compensatory behaviour (most often self-induced vomiting), with self-evaluation unduly influenced by weight and shape; the discriminating clinical fact is that the weight is *typically normal or above normal*, not low.

4.2 DSM-5-TR criteria and the severity bands

The five criteria to state. DSM-5-TR sets five criteria (DSM-5-TR 2022). *A*, recurrent episodes of binge eating (both the large amount, A1, and the sense of lack of control, A2). *B*, recurrent inappropriate compensatory behaviours to prevent weight gain (self-induced vomiting; misuse of laxatives, diuretics or other medications; fasting; or excessive exercise). *C*, the binge eating *and* the compensatory behaviours both occur, on average, **at least once a week for 3 months**. *D*, self-evaluation is unduly influenced by body shape and weight. *E*, the disturbance does not occur exclusively during episodes of anorexia nervosa (so a low-weight binge-purge patient is coded as anorexia nervosa, binge-purge type, not as bulimia nervosa).

Severity is scored on the compensatory behaviour. DSM-5-TR grades current severity by the average number of episodes of inappropriate compensatory behaviour per week, and this is the band examiners like to test.

DSM-5-TR SEVERITY	COMPENSATORY BEHAVIOUR (EPISODES PER WEEK)
Mild	1 to 3
Moderate	4 to 7
Severe	**8 to 13**
Extreme	**14 or more**

DSM-5-TR severity grading for bulimia nervosa by the average weekly frequency of inappropriate compensatory behaviour (DSM-5-TR 2022); severity may be raised for other symptoms or functional disability.

GOOD TO REMEMBER

- **DSM-5-TR criteria (the diagnostic anchor):** binge eating with loss of control *plus* recurrent compensatory behaviour, both on average *at least once a week for 3 months*, with self-evaluation unduly influenced by weight/shape, and *not* exclusively during anorexia nervosa; severity is banded by compensatory episodes per week (mild 1 to 3, moderate 4 to 7, severe 8 to 13, extreme 14 or more).

4.3 ICD-11 criteria and the frequency-duration discriminator

Where the two systems diverge. ICD-11 (6B81) requires frequent, recurrent binge eating (loss of control, eating notably more or differently than usual) *and* repeated inappropriate compensatory behaviour to prevent weight gain, each occurring **once a week or more over a period of at least 1 month**, with an excessive preoccupation with body weight or shape and marked distress or functional impairment (ICD-11 CDDR). The high-yield discriminator is the timeframe: *ICD-11 asks for at least one month, DSM-5-TR asks for three months*. ICD-11 also notes that self-induced vomiting, the commonest compensatory behaviour, typically occurs within an hour of the binge.

The historical anchor. Bulimia nervosa was first delineated by Gerald Russell in 1979 as an "ominous variant" of anorexia nervosa, and his name is attached to the hand sign of chronic self-induced vomiting (below). Where the person meets full criteria but at lower frequency or shorter duration, the residual category is *bulimia nervosa of low frequency and/or limited duration*, coded under OSFED (DSM-5-TR) or "other specified" in ICD-11.

FRAMEWORK	BINGE + COMPENSATORY FREQUENCY AND DURATION	WEIGHT AND BOUNDARY
DSM-5-TR	**On average at least once a week for 3 months**	Normal or above; excluded if only during anorexia nervosa
ICD-11 (6B81)	**Once a week or more over at least 1 month**	Not significantly low weight (else anorexia nervosa, binge-purge pattern)
ICD-10 (historical)	Recurrent overeating with pre-occupation and craving	Morbid dread of fatness; often past anorexic episode

The frequency-and-duration thresholds compared; the number to hold is the split, DSM-5-TR three months against ICD-11 one month, both at once weekly (DSM-5-TR 2022; ICD-11 CDDR).

RULE **Once weekly, but three months versus one month.** Both systems require binge eating and compensatory behaviour at least once a week; the duration is the discriminator, *three months in DSM-5-TR, one month in ICD-11*, and the patient is not significantly underweight (or the diagnosis becomes anorexia nervosa, binge-purge type).

4.4 Distinguishing bulimia from its neighbours

Three look-alikes. The differential turns on weight and on whether purging is present. *Anorexia nervosa, binge-purge subtype* shares the binge-purge behaviour but the weight is *significantly low*,

and by the exclusion rule that low-weight patient is diagnosed as anorexia nervosa, not bulimia nervosa. *Binge-eating disorder* shares the binges and the loss of control but has *no* regular compensatory behaviour, so the person is commonly overweight or obese and does not purge. *Purging disorder* (an OSFED presentation) is recurrent purging to control weight *without* objective binges. The presence of purging plus objective binges plus a non-low weight is what pins the label to bulimia nervosa.

DISORDER	OBJECTIVE BINGES	COMPENSATORY PURGING	TYPICAL WEIGHT
Bulimia nervosa	Yes	**Yes, recurrent**	Normal or above
Anorexia nervosa, binge-purge type	Yes	Yes	**Significantly low**
Binge-eating disorder	Yes	No	Overweight or obese
Purging disorder (OSFED)	No	Yes	Normal

Bulimia nervosa against its differentials; the two questions are "are there objective binges?" and "is there recurrent purging at a non-low weight?" (DSM-5-TR 2022).

GOOD TO REMEMBER

- **Boundary rule (the discriminator):** objective binges plus recurrent purging at a *normal or above-normal* weight is bulimia nervosa; the same behaviour at a significantly *low* weight is anorexia nervosa binge-purge type; binges *without* compensation is binge-eating disorder; purging *without* binges is purging disorder.

4.5 Medical complications: the footprint of purging

The body records the vomiting. The **medical complications** of bulimia nervosa come almost entirely from the compensatory behaviours, above all self-induced vomiting (Kaplan and Sadock CTP 2024). Repeated acid vomit dissolves the lingual dental enamel (erosion and multiple caries), swells the parotid and salivary glands (sialadenosis, with a raised serum amylase), and, over time, lays down Russell's sign, the callus or abrasion over the knuckles of the dominant hand where the teeth scrape the skin during gag-induced vomiting. The dangerous consequences are biochemical and cardiac. The two findings that demand urgent evaluation are a prolonged QTc on the ECG and marked hypokalaemia.

The electrolyte and acid-base signature. Recurrent vomiting loses hydrogen and chloride and drives a hypochloraemic, hypokalaemic metabolic alkalosis; laxative misuse instead loses bicarbonate in the stool and tends toward a metabolic acidosis. *Hypokalaemia* is the killer: a low serum potassium is a particularly dangerous imbalance that precipitates cardiac arrhythmia, so potassium is monitored in anyone who purges (Kaplan and Sadock CTP 2024). Forceful vomiting also risks the oesophagus, oesophagitis, Mallory-Weiss mucosal tears with haematemesis, and, rarely, catastrophic oesophageal or gastric rupture, while ipecac (emetine) abuse can cause a skeletal and cardiac myopathy.

SYSTEM	SIGN OR ABNORMALITY	MECHANISM / NOTE
Oral / dental	**Erosion of dental enamel (perimylolysis), multiple caries**	Acid gastric contents dissolve the lingual enamel with chronic vomiting
Salivary	Swollen parotid and salivary glands (sialadenosis), raised serum amylase	Recurrent self-induced vomiting; the "chipmunk" facies
Hands	**Russell's sign: knuckle calluses / abrasions**	Teeth scraping the dorsum of the hand during gag-induced vomiting
Electrolyte	**Hypokalaemia, hypochloraemia, hyponatraemia**	Loss through vomiting and purging; hypokalaemia is the dangerous one
Acid-base	Metabolic alkalosis (vomiting); metabolic acidosis (laxatives)	Vomiting loses hydrogen and chloride; laxatives lose bicarbonate
Cardiac	**Prolonged QTc, arrhythmia; ipecac cardiomyopathy**	Driven by hypokalaemia and QTc; urgent findings on the ECG
Oesophageal / gastric	Oesophagitis, Mallory-Weiss tears, rare oesophageal or gastric rupture	Forceful vomiting; gastric distension from bingeing
Gastrointestinal (lower)	Constipation, cathartic (atonic) colon, rectal prolapse	Chronic laxative misuse and dependence

Medical complications of bulimia nervosa by system; the eye-catchers are Russell's sign, parotid swelling and dental erosion, and the two that kill are hypokalaemia and a prolonged QTc (Kaplan and Sadock CTP 2024).

TRAP **A normal weight is falsely reassuring.** The classic error is to discount an eating disorder because the patient looks well and weighs normally, and to miss the silent *hypokalaemia* and prolonged QTc of covert purging. Weigh, ask directly about bingeing and vomiting, look for Russell's sign, parotid swelling and dental erosion, and always check potassium and an ECG.

GOOD TO REMEMBER

- **Complications of purging (the criterion sign to seek):** the physical footprint is dental erosion, parotid swelling and Russell's sign; the biochemistry is a *hypochloraemic, hypokalaemic metabolic alkalosis* from vomiting; and the two urgent findings that must be actively sought are marked *hypokalaemia* and a *prolonged QTc*, because hypokalaemia drives fatal arrhythmia.

4.6 Management, the NICE NG69 stepped plan: guided self-help first

Psychological therapy leads, in a defined order. The lead guideline is NICE NG69 (2017), and its first step for adults with bulimia nervosa is bulimia-nervosa-focused guided self-help: structured self-help materials supported by four to nine brief supportive sessions over about 16 weeks (NICE NG69 2017). If guided self-help is unacceptable, contraindicated or ineffective after four weeks, the next step is individual eating-disorder-focused cognitive behavioural therapy (CBT-ED), typically 20 sessions over about 20 weeks. For children and young people the first-line psychological treatment is *bulimia-nervosa-focused family therapy* (FT-BN) (NICE NG69 2017).

NICE is explicit that medication is *not* offered as the sole treatment for bulimia nervosa, and that ineffective physical therapies (transcranial magnetic stimulation, acupuncture, weight training, yoga, warming therapy) should not be used.

Where the other guidelines land. RANZCP (2014) puts therapist-led CBT as the first-line psychological treatment and allows an antidepressant, topiramate or orlistat (the last in comorbid obesity) as adjunctive or alternative medication. This is the point where NICE (self-help first, then CBT-ED) and RANZCP (CBT first) differ in emphasis while agreeing that a talking therapy, not a drug, is primary.

GOOD TO REMEMBER

- **NICE NG69 stepped care (the management order):** the value is *a psychological therapy first, medication never alone*; adults start with bulimia-nervosa-focused *guided self-help* (4 to 9 sessions over about 16 weeks), stepping up to individual *CBT-ED* (about 20 sessions over 20 weeks) if that fails after four weeks; young people get FT-BN; RANZCP places therapist-led CBT first-line.

4.7 CBT-ED, the definitive psychological treatment

The best-evidenced therapy. **CBT-ED** is the eating-disorder-specific form of cognitive behavioural therapy and has the strongest evidence base in bulimia nervosa, established across dozens of controlled trials as the most effective single treatment (Lewis Child and Adolescent Psychiatry; NICE NG69 2017). It targets the machinery of the binge-purge cycle directly: it establishes a pattern of regular eating to break the restrict-binge see-saw, dismantles dietary rules and the compensatory behaviours, and works on the over-evaluation of shape and weight that drives the whole loop. A typical course runs to about 20 sessions over 20 weeks. The general CBT technique on which it is built is developed in the Psychotherapies, clinical assessment, MSE and nosology notes; this topic owns the disorder-specific adaptation.

Therapy is primary, drug is adjunctive. The through-line for an answer is that CBT-ED (or, as a first step in NICE, guided self-help built on the same model) is the treatment that changes the disorder, and any medication rides alongside it rather than replacing it.

GOOD TO REMEMBER

- **CBT-ED (the primary therapy):** the anchor is that *eating-disorder-focused CBT is the best-evidenced treatment for bulimia nervosa*; it installs regular eating, targets dietary rules, purging and the over-evaluation of shape and weight, typically 20 sessions over about 20 weeks, and medication is adjunctive to it, never a substitute.

4.8 Fluoxetine, the one licensed drug, at 60 mg per day

The drug to know cold. If one pharmacological fact is tested on this topic, it is this: fluoxetine is the medication of choice and the licensed drug for bulimia nervosa, and it works at a dose *higher* than its antidepressant dose. Fluoxetine is traditionally prescribed at 20 to 40 mg per day for depressive disorders, but studies show that a higher dose, **60 mg per day**, is more effective at disrupting binge eating and purging, and even at that higher dose it is better tolerated than the older alternatives (Kaplan and Sadock CTP 2024). WFSBP (2023) names fluoxetine as the first-line pharmacological treatment for bulimia nervosa in adults (its strongest recommendation

grade). The effect is largely independent of whether the patient is depressed, so it is not merely treating a comorbid mood disorder. In India, fluoxetine is widely available and inexpensive, led by the brands Prodep, Fludac and Flunil; the deep pharmacology (kinetics, adverse effects, the long half-life, CYP interactions) is cross-referenced to the Mood disorders: pharmacotherapy notes.

RULE **Fluoxetine 60 mg per day is THE licensed drug for bulimia.** The one antibulimic dose to memorise is fluoxetine *60 mg per day*, the antibinge dose that sits above the 20 to 40 mg antidepressant range; it is the medication of choice and licensed for bulimia nervosa, given alongside, not instead of, a psychological therapy.

WORKED EXAMPLE

A 22-year-old woman of normal weight (BMI 22) discloses that she binges several evenings a week and then makes herself vomit; she has knuckle calluses, bilateral parotid fullness, and a serum potassium of 3.1 with a borderline-prolonged QTc. This is bulimia nervosa. The correct plan is to correct and monitor the potassium, arrange an ECG, and start a psychological therapy, bulimia-nervosa-focused guided self-help stepping up to CBT-ED per NICE NG69. If a drug is added for the bingeing and purging, the answer is **fluoxetine** titrated to 60 mg per day, alongside the therapy. She should not be started on bupropion (see below), and normal weight does not make the hypokalaemia any less dangerous.

GOOD TO REMEMBER

- **Fluoxetine (the licensed drug):** the one value is *60 mg per day*, above the 20 to 40 mg antidepressant dose, as the antibinge dose; fluoxetine is the medication of choice and the licensed agent for bulimia nervosa (WFSBP 2023 first-line), works whether or not the patient is depressed, and is given *with* a psychological therapy, not alone.

4.9 The rest of the drug shelf, and the one to avoid

Adjuncts and alternatives. Beyond fluoxetine, topiramate reduces binge eating and purging and is recommended by WFSBP (2023) and offered by RANZCP (2014) as an adjunct or alternative, though its cognitive and paraesthetic side effects limit tolerability. Older tricyclic antidepressants can reduce bulimic symptoms but carry a moderate risk-benefit ratio and are dangerous in overdose (WFSBP 2011). Because eating-disorder patients on medication may develop electrolyte disturbance, bradycardia, hypokalaemia or QT prolongation, NICE NG69 (2017) advises ECG monitoring for anyone whose drug could compromise cardiac function.

The contraindication that is a favourite question. Bupropion is *contraindicated* in bulimia nervosa: it lowers the seizure threshold, and actively purging patients had an unacceptably high rate of seizures, so it is not used in current or past bulimia or anorexia nervosa (Kaplan and Sadock CTP 2024). Naming bupropion as an antidepressant for a bulimic patient is a classic exam trap.

TRAP **Reaching for bupropion.** Bupropion is contraindicated in bulimia nervosa because it lowers the seizure threshold and precipitated seizures in actively purging patients; the antidepressant that is *indicated and licensed* here is fluoxetine at 60 mg per day, not bupropion.

GOOD TO REMEMBER

- **Other drugs and the caution (the value):** topiramate is the evidenced adjunct or alternative (WFSBP 2023; RANZCP 2014) and tricyclics are second-line with a poor overdose margin; monitor the ECG on any cardiac-risk drug (NICE NG69 2017); and *bupropion is contraindicated* because it lowers the seizure threshold in purging patients.

4.10 Screening, guideline convergence and the India lens

Case-finding and where the guidelines meet. The bedside screen is the SCOFF, the same five-item verbal tool used for anorexia nervosa (Sick, Control, One stone, Fat, Food), positive at **two or more** "yes" answers and reproduced on the accompanying scale plate. Read together, the guidelines converge: a psychological therapy is primary (NICE NG69 guided self-help then CBT-ED; RANZCP therapist-led CBT), medication is never the sole treatment, and where a drug is used the licensed choice is fluoxetine at 60 mg per day.

GUIDELINE	FIRST-LINE PSYCHOLOGICAL	MEDICATION STANCE
NICE NG69 2017	**Guided self-help, then CBT-ED; FT-BN in the young**	Not the sole treatment; ECG-monitor cardiac-risk drugs
RANZCP 2014	Therapist-led CBT first-line	Antidepressant, topiramate or orlistat as adjunct/alternative
WFSBP 2023	Psychotherapy embedded	**Fluoxetine first-line; topiramate to reduce binge/purge**
WFSBP 2011	Psychotherapy embedded	Tricyclics usable, moderate risk-benefit

Guideline-level management of bulimia nervosa, NICE NG69-led, converging on a psychological therapy first and fluoxetine as the licensed drug (verified from the drug-guideline supplement; WFSBP fluoxetine and dose from Kaplan and Sadock CTP 2024).

INDIA

Bulimia nervosa is under-recognised in India, and doubly hidden: unlike anorexia nervosa it leaves no visible emaciation, the binges and vomiting are intensely secret and shaming, and the presentation is often to a physician or dentist with dental erosion, parotid swelling or unexplained **hypokalaemia** rather than to a psychiatrist. The stereotype of the affluent urban young woman misses men and lower-income patients. The clinical antidotes are a low threshold to screen (the SCOFF), a direct question about bingeing and self-induced vomiting in any young person with normal weight and a low potassium or eroded teeth, and reassurance that the licensed, cheap and available drug, **fluoxetine** (Prodep, Fludac, Flunil), sits behind an accessible first-line therapy.

PEARL

Bulimia nervosa is the eating disorder you diagnose by *asking*, not by looking. The weight is normal, so the tells are behavioural and physical residue: the secret binge-purge cycle on history, and Russell's sign, parotid fullness, eroded teeth and a quietly low potassium on examination. Treat the cycle with CBT-ED and, if a drug is needed, fluoxetine at 60 mg per day.

60	2	3	8 to 13
FLUOXETINE DOSE FOR BULIMIA (MG PER DAY); THE LICENSED DRUG	SCOFF POSITIVE SCREEN (YES ANSWERS)	DSM-5-TR MINIMUM DURATION (MONTHS AT ONCE WEEKLY)	SEVERE BULIMIA (COMPENSATORY EPISODES PER WEEK)

HOLD THIS

Bulimia nervosa is the recurrent binge-purge cycle, binge eating with loss of control plus recurrent compensatory behaviour (most often self-induced vomiting), at least once weekly for three months in DSM-5-TR (one month in ICD-11), in a person of normal or above-normal weight; the footprint of purging is Russell's sign, parotid swelling, dental erosion and a hypokalaemic metabolic alkalosis with the deadly duo of hypokalaemia and a prolonged QTc; management is a psychological therapy first (NICE NG69 guided self-help then CBT-ED), medication never alone, and the one licensed drug is fluoxetine at 60 mg per day, with bupropion contraindicated.

5 · Binge eating disorder

Binge eating disorder (BED) is the eating disorder examiners use to test one clean discrimination: it is **recurrent binge eating with loss of control, but without the regular compensatory behaviour that defines bulimia.** Take away the self-induced vomiting, the laxatives, the fasting and the driven exercise, and a bulimia-shaped binge picture becomes binge eating disorder. It is the most common eating disorder, it sits alongside overweight and obesity rather than emaciation, and its management is a guideline-led ladder in which a psychological therapy, not a drug, is the primary treatment.

Why it matters clinically. This is a hybrid topic. The diagnosis half turns on three things you must be able to state cold: the binge (a discrete period of overeating with loss of control eating), the associated features and marked distress, and the absence of regular compensation. The management half is a guideline plan led by NICE NG69 (2017): binge-eating-disorder-focused guided self-help first, then group CBT-ED, with medication kept adjunctive and weight loss explicitly not a therapy target. The general cognitive-behavioural technique that CBT-ED specialises belongs to the Psychotherapies, clinical assessment, MSE and nosology notes, and the high-dose bulimia pharmacology of fluoxetine belongs to the Mood disorders: pharmacotherapy notes. This topic leans on the differentiation grid and the accompanying scale plate rather than adding a figure of its own.

5.1 What binge eating disorder is: the recurrent binge with loss of control

The binge, defined. A binge is eating, in a discrete period of time (usually under two hours), an amount of food that is definitely larger than most people would eat in a similar period under similar circumstances, accompanied by a **sense of lack of control** (DSM-5-TR 2022, Criterion A). The loss of control is the core: the inability to stop once started, or, in longer-standing illness, a generalised feeling of uncontrolled eating even where the acute out-of-control quality has faded. ICD-11 broadens this usefully, allowing the binge to be "objective" (a genuinely large amount) or "subjective" (an amount that is normal in quantity but experienced as a binge), because the defining experience is the loss of control, not the calorie count (ICD-11 CDDR).

Recurrent, and distressing. The pattern must be a **recurrent binge**, not an isolated lapse: on average at least once per week for three months (DSM-5-TR 2022, Criterion D; ICD-11 uses "once a week or more over three months"). The binge eating is marked by significant distress (DSM-5-TR 2022, Criterion C), the shame and secrecy that make patients conceal it, so most binge eating happens alone and hidden.

MNEMONIC

RAG (Rapid, Alone, Guilt) around a Loss-of-control binge

The core of a binge eating disorder episode is a *Loss-of-control* binge; the three most memorable of the five associated features are eating *Rapidly*, eating *Alone* from embarrassment, and *Guilt* afterwards. Loss of control is required; three or more of the five features are required.

5.2 The five associated features and the "three of five" rule

Marked distress plus three of five. Beyond the binge itself, the diagnosis requires marked distress and at least three of five associated behavioural features (DSM-5-TR 2022, Criterion B): eating much more rapidly than normal; eating until feeling uncomfortably full; eating large amounts of food when not feeling physically hungry; eating alone because of feeling embarrassed by how much one is eating; and feeling disgusted with oneself, depressed, or very guilty afterward. These are the phenomenology of a compulsive, secretive, affect-driven episode; negative affect is the most common trigger.

CRITERION	REQUIREMENT
A. The binge	**Discrete period (usually under two hours), amount definitely larger than most would eat, with a sense of lack of control**
B. Associated features (three or more)	Eating much more rapidly than normal
B (feature)	Eating until uncomfortably full
B (feature)	Eating large amounts when not physically hungry
B (feature)	Eating alone from embarrassment at the amount
B (feature)	Feeling disgusted, depressed or very guilty afterward
C. Distress	**Marked distress about the binge eating**
D. Frequency	**On average at least once per week for three months**
E. No compensation	**Not regularly accompanied by inappropriate compensatory behaviour; not occurring exclusively during anorexia nervosa or bulimia nervosa**

The binge eating disorder criteria (DSM-5-TR 2022; ICD-11 6B82 aligns, adding the objective/subjective binge distinction). The instrument forms are held on the accompanying scale plate.

GOOD TO REMEMBER

- **Binge eating disorder (the defining criteria):** the anchor is a *recurrent binge with loss of control, marked distress, three or more of five associated features, at least once per week for three months, and no regular compensation*; loss of control is required, the "three of five" is the behavioural gate, and the absence of compensation is what separates it from bulimia.

5.3 The discriminator: binge without compensation

Take away the compensation and it is BED. The single fact that fixes the diagnosis is **binge without compensation**: binge eating disorder shares recurrent binge eating with bulimia nervosa but lacks the regular inappropriate compensatory behaviour (vomiting, laxatives, fasting, driven exercise) that bulimia requires (DSM-5-TR 2022). A second, softer discriminator: unlike bulimia, patients with binge eating disorder do not show marked sustained dietary restriction between binges, and the sequence is reversed, dieting typically *follows* the binge eating in BED, whereas dysfunctional dieting usually *precedes* binge eating in bulimia (DSM-5-TR 2022). The three eating-disorder diagnoses are mutually exclusive for a single episode, so a patient who binges and purges is bulimia, not BED.

FEATURE	BINGE EATING DISORDER	BULIMIA NERVOSA
Recurrent binge with loss of control	**Yes**	Yes
Regular inappropriate compensation	**Absent (the discriminator)**	Present (defining)
Dietary restriction between binges	Not marked; dieting follows binges	Marked; dieting precedes binges
Typical body weight	Overweight or obese (but distinct from obesity)	Often normal weight
Treatment response	**Higher remission rates**	Lower remission rates

Binge eating disorder versus bulimia nervosa; the presence or absence of regular compensatory behaviour is the pivotal discriminator (DSM-5-TR 2022).

RULE **Recurrent binge, loss of control, no regular compensation.** If the binges are recurrent and out of control but there is no regular purging, fasting or driven exercise, the diagnosis is binge eating disorder, not bulimia nervosa. The compensation is the fork in the road.

5.4 Frequency, severity bands and the ICD-11 framing

Severity is graded by binge frequency. Once the diagnosis is made, DSM-5-TR grades severity purely by the average number of binge-eating episodes per week: mild is 1 to 3, moderate 4 to 7, severe 8 to 13, and extreme 14 or more (DSM-5-TR 2022). ICD-11 does not use the same numeric grid but allows the diagnosis to be assigned after a shorter period (for example one month) when multiple binges per week cause significant distress (ICD-11 CDDR). Remission is more common than in bulimia or anorexia, sometimes spontaneously, and crossover to another eating disorder is uncommon.

DSM-5-TR SEVERITY	FREQUENCY (BINGE EPISODES PER WEEK)
Mild	**1 to 3**
Moderate	**4 to 7**
Severe	8 to 13
Extreme	14 or more

DSM-5-TR severity bands for binge eating disorder, graded by weekly binge frequency (severity may be raised for greater distress or disability).

GOOD TO REMEMBER

- **Severity grading (the frequency step):** the anchor value is that *DSM-5-TR grades binge eating disorder by binge episodes per week*, mild 1 to 3, moderate 4 to 7, severe 8 to 13, extreme 14 or more; ICD-11 keeps the "once a week over three months" threshold and may diagnose after one month if binges are frequent and distressing.

5.5 Association with obesity and the metabolic risk

Sits with obesity, but is not obesity. Binge eating disorder occurs in normal-weight, overweight and obese individuals and is reliably associated with overweight and obesity in treatment-seeking samples, carrying the attendant metabolic and cardiovascular morbidity, the weight-related quality-of-life loss, and increased health-care use (DSM-5-TR 2022). But it is distinct from simple obesity: most obese people do not binge; compared with weight-matched obese controls without the disorder, those with BED eat more in laboratory studies, place higher overvaluation on shape and weight, and carry far more psychiatric comorbidity, most commonly major depressive disorder and alcohol use disorder. Crucially, that comorbidity tracks the *severity of the binge eating*, not the degree of obesity (DSM-5-TR 2022).

The clinical implication. This is why weight loss is the wrong therapeutic target (see the management half): the disorder is a psychiatric one that happens to co-travel with obesity, and treating the obesity does not treat the binge eating. Screen the overweight or obese patient who describes secret, out-of-control eating with the eating-disorder screen on the accompanying scale plate rather than reaching only for a weight-loss plan.

PEARL

Binge eating disorder is the eating disorder of the medical and bariatric clinic, not the emaciation ward. It is over-represented among people seeking weight-loss treatment, and the binge eating almost always predates the weight-loss attempt rather than being caused by it. Ask any patient presenting for obesity management about secret, rapid, out-of-control eating; you will find binge eating disorder hiding behind a body mass index far more often than behind a low weight.

5.6 Management, the NICE NG69 lead: guided self-help then CBT-ED

Psychological therapy is the primary treatment. Leading with NICE NG69 (2017): for adults with binge eating disorder, offer a binge-eating-disorder-focused guided self-help programme as the first step. If guided self-help is unacceptable, contraindicated, or ineffective after four weeks, offer group eating-disorder-focused cognitive behavioural therapy (CBT-ED); if group CBT-ED is

unavailable or declined, offer individual CBT-ED (NICE NG69 2017). Children and young people are offered the same treatments as adults (NICE NG69 2017). RANZCP concurs at guideline level that individual therapist-led CBT is the best-evidenced psychological treatment for the disorder (RANZCP 2014).

Weight loss is not the target. NICE is explicit: explain to patients that psychological treatment has a limited effect on body weight, and weight loss is not itself a therapy target (NICE NG69 2017). The goal is to stop the binge eating and the distress it causes; any weight change is a separate, secondary matter managed on its own merits.

TRAP **Chasing weight loss as the treatment.** Treating binge eating disorder as if it were obesity, prescribing a diet and a weight-loss target, is the classic error: NICE says weight loss is not a therapy target and psychological treatment has limited effect on weight. Treat the binge eating with guided self-help and CBT-ED; manage weight, if at all, separately.

GOOD TO REMEMBER

- **Psychological therapy (the first-line step):** the one value is that *binge-eating-disorder-focused guided self-help is first-line, then group CBT-ED, then individual CBT-ED* (NICE NG69 2017), and that *weight loss is not a therapy target*; the goal is to stop the binge eating and its distress, not to slim the patient.

5.7 Pharmacotherapy: adjunctive, never the sole treatment

Medication is an add-on, not a substitute. Do not offer medication as the sole treatment for binge eating disorder (NICE NG69 2017, strong); NICE keeps the psychological therapies primary and does not license a specific drug for the disorder in the UK. Where a drug is used, it sits within a plan that includes psychotherapy and, where relevant, nutritional counselling and treatment of medical complications (WFSBP 2011). The named agents, drawn from the guidelines and the licensing position:

- **Lisdexamfetamine** is the one drug with a specific regulatory licence for binge eating disorder (US FDA), a prodrug stimulant given at 50 to 70 mg per day (initial 30 mg per day, titrated), and is the first medication ever approved for the disorder (Stahl, Prescriber's Guide). It reduces binge frequency; it is a Schedule-controlled stimulant and is not a NICE-recommended UK option.
- An **SSRI, sertraline**, is supported for binge eating disorder (WFSBP 2011, grade A), and RANZCP lists an antidepressant as an adjunctive or alternative option (RANZCP 2014).
- **Topiramate**, the antiepileptic, reduces binge eating (WFSBP 2011, grade A; RANZCP 2014), useful where weight is a concern given its anorectic effect.
- **Orlistat**, the pancreatic lipase inhibitor, is considered for the patient with comorbid obesity, aiding weight but with little direct effect on the binge eating itself (RANZCP 2014; New Oxford Textbook).

GOOD TO REMEMBER

- **Lisdexamfetamine and the adjuncts (the medication step):** the anchor value is that *medication is adjunctive, never the sole treatment* (NICE NG69 2017); lisdexamfetamine (50 to 70 mg per day) is the only agent specifically licensed for binge eating disorder (US FDA, Stahl), with the SSRI sertraline and topiramate (both WFSBP grade A) and orlistat (for comorbid obesity) as the other named options.

5.8 Guideline convergence and the Indian picture

Where the guidelines land. Read together they converge: a psychological therapy is primary (guided self-help then CBT-ED per NICE NG69; individual CBT per RANZCP), weight loss is not the treatment goal, and medication is adjunctive within a psychotherapy-anchored plan (NICE NG69 2017; RANZCP 2014; WFSBP 2011). The one point of divergence is regulatory rather than clinical: only lisdexamfetamine holds a specific licence, and only in the US.

GUIDELINE / SOURCE	PRIMARY TREATMENT	MEDICATION STANCE
NICE NG69 2017	Guided self-help first, then group CBT-ED, then individual CBT-ED; weight loss not a target	**Not the sole treatment; no UK-licensed drug**
RANZCP 2014	Individual therapist-led CBT (best-evidenced)	Antidepressant or topiramate as adjunct; orlistat if comorbid obesity
WFSBP 2011	Psychotherapy plus nutritional counselling	Sertraline (grade A), topiramate (grade A)
US FDA (Stahl)	Adjunct to psychological therapy	**Lisdexamfetamine 50 to 70 mg per day, the only licensed agent**

Guideline-level management of binge eating disorder, NICE NG69-led, converging on a psychological therapy as primary and medication as adjunctive (verified from the drug-guideline supplement; the FDA licence and dose from Stahl).

INDIA

Two India-specific points earn marks. First, under-recognition: eating disorders, and binge eating disorder in particular, are substantially under-recognised in Indian practice, and because binge eating disorder hides behind overweight rather than emaciation it is especially easy to miss in the diabetes, cardiac and bariatric clinics where these patients actually present. Ask the overweight patient with secret, out-of-control eating the eating-disorder screen rather than assuming simple obesity. Second, drug availability: lisdexamfetamine, the only agent with a specific licence for binge eating disorder, is a US FDA licence and the drug is not marketed in India, so Indian practice necessarily leans on the psychological therapies with off-label SSRI (sertraline) or topiramate as the adjunct where one is used. (This availability point is a clinical-practice observation and is flagged for local verification.)

1	3 of 5	50 to 70	4 to 7
BINGE EPISODES FOR DIAGNOSIS, MINIMUM (PER WEEK, OVER THREE MONTHS)	ASSOCIATED FEATURES REQUIRED (WITH MARKED DISTRESS AND LOSS OF CONTROL)	LISDEXAMFETAMINE, THE ONLY LICENSED AGENT (MG PER DAY)	DSM-5-TR MODERATE SEVERITY (BINGE EPISODES PER WEEK)

WORKED EXAMPLE

A 38-year-old man attending a diabetes clinic, BMI 34, discloses that two or three evenings a week he eats a very large amount of food rapidly, alone, after the household is asleep, cannot stop once he starts, and feels disgusted and guilty afterward. He never vomits, fasts or over-exercises to compensate, and does not diet restrictively between episodes. This is binge eating disorder (recurrent binge, loss of control, three or more associated features, marked distress, no compensation), moderate severity by DSM-5-TR frequency. The correct plan is binge-eating-disorder-focused guided self-help first, escalating to group then individual CBT-ED if it fails, with the explicit message that weight loss is not the treatment target; if a drug is added it is adjunctive (an SSRI such as sertraline, or topiramate), not a substitute for the therapy, and lisdexamfetamine is unavailable to him in India.

HOLD THIS

Binge eating disorder is recurrent binge eating with loss of control and marked distress but no regular compensatory behaviour (the discriminator from bulimia); it co-travels with obesity yet is distinct from it; and management is led by NICE NG69, binge-eating-disorder-focused guided self-help then CBT-ED, with weight loss explicitly not a target and medication only ever adjunctive (lisdexamfetamine 50 to 70 mg per day the sole licensed agent, US FDA; sertraline or topiramate the other named options).

6 · Avoidant/restrictive food intake disorder (ARFID)

The eating disorder without the body image. **Avoidant/restrictive food intake disorder** (ARFID) is the disorder of restricted eating that is *not* driven by any wish to be thin. The person avoids or restricts food because of the sensory qualities of food, an apparent lack of interest in eating, or a fear of an aversive consequence such as choking or vomiting, and this **food avoidance** produces weight loss, nutritional deficiency, dependence on supplements or feeds, or psychosocial impairment. What it lacks is the whole engine of anorexia: there is **no body-image disturbance**, no overvaluation of weight and shape, and no fear of gaining weight. That single absence is the examiner's discriminator and the reason the topic exists.

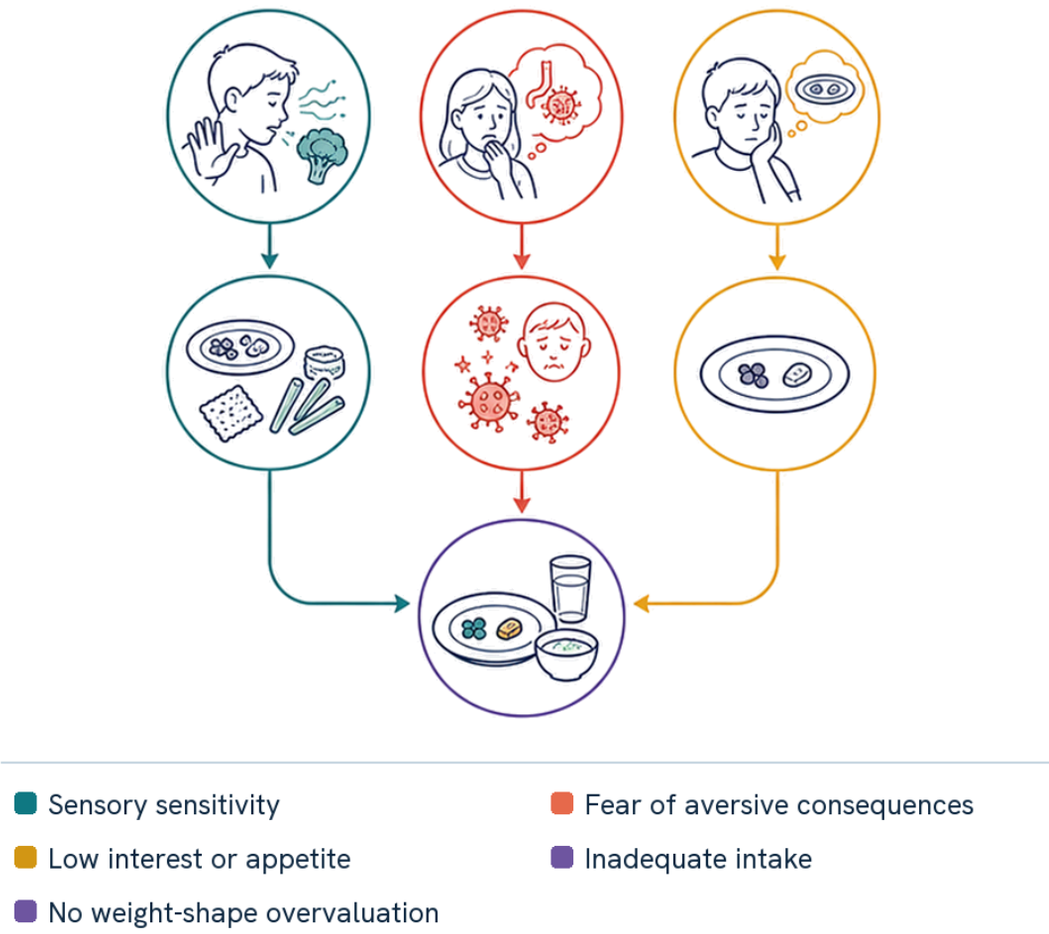


Fig 17.5 Avoidant/restrictive food intake disorder may arise from sensory sensitivity, fear of aversive consequences or low interest in food, leading to inadequate intake without weight-shape overvaluation.

A DSM-5 creation. ARFID was introduced in DSM-5 (carried into DSM-5-TR) to replace and widen the old DSM-IV "feeding disorder of infancy or early childhood", extending it to older children, adolescents and adults; ICD-11 then adopted the same category (6B83). The full name, *avoidant restrictive food intake disorder*, is worth stating in the viva because the components name the syndrome: avoidance or restriction of intake, with functional and nutritional consequences, and without the weight-and-shape psychopathology of anorexia. This note owns the diagnosis and the discrimination from anorexia; the SSRIs sometimes used for comorbid anxiety are detailed in the Mood disorders: pharmacotherapy notes, and the family and cognitive-behavioural techniques in the Psychotherapies, clinical assessment, MSE and nosology notes.

6.1 The DSM-5-TR criteria: an eating disturbance plus one consequence

Criterion A is a disturbance with a consequence. DSM-5-TR criterion A is an eating or feeding disturbance (for example, an apparent lack of interest in eating or food, avoidance based on the sensory characteristics of food, or concern about the aversive consequences of eating) that is associated with *one or more* of four consequences: significant weight loss (or, in a growing child, a

failure to make expected weight gain or faltering growth); significant nutritional deficiency; dependence on **enteral feeding or oral nutritional supplements**; or marked interference with psychosocial functioning (DSM-5-TR 2022).

The exclusions do the diagnostic work. Criterion B excludes restriction better explained by unavailability of food or by a culturally sanctioned practice (a religious fast is not ARFID). Criterion C is the pivotal one: the disturbance does not occur only during anorexia or bulimia, *and there is no disturbance in the way body weight or shape is experienced*. Criterion D excludes restriction fully attributable to a concurrent medical condition or better explained by another mental disorder, unless it exceeds what that condition usually causes.

GOOD TO REMEMBER

- **ARFID (the syndrome):** an eating/feeding disturbance from sensory-based avoidance, low interest in food, or fear of aversive consequences, plus at least one of four consequences (weight loss or faltering growth, nutritional deficiency, dependence on enteral feeding or oral nutritional supplements, or marked psychosocial impairment); the defining exclusion is Criterion C, no body-image disturbance and no overvaluation of weight or shape, which is what separates it from anorexia.

6.2 The three drivers of restriction

Same restriction, three routes in. DSM-5-TR frames the food avoidance through three prototypical, non-exclusive drivers, and naming them is the fastest way to show you understand the disorder.

Sensory-based avoidance

Restriction driven by the appearance, colour, smell, texture, temperature or taste of food; the "selective", "choosy" or "picky" eater and food neophobia, often refusing whole food groups or particular brands, and overlapping with autism-spectrum sensory sensitivity.

Apparent lack of interest in eating or food

A longstanding low appetite, poor recognition of hunger, or simple lack of drive to eat, so intake falls without any conscious avoidance of a feared outcome.

Fear of aversive consequences

A conditioned avoidance following, or in anticipation of, a frightening experience such as choking, vomiting or a traumatic gastrointestinal procedure; the person restricts to prevent the feared event, not to lose weight.

6.3 The line from anorexia nervosa

One question settles it. Is the restriction motivated by weight or shape concern? If the person restricts to be thin, fears fatness, or overvalues weight and shape, it is anorexia nervosa; if the restriction is sensory, appetite-driven or fear-of-consequence driven and body image is intact, it is ARFID. Both can present with a significantly low weight and the same starvation physiology (bradycardia, hypothermia, anaemia), so the differentiation is psychological, not physical. The figure alongside lays the two side by side.

FEATURE	ARFID	ANOREXIA NERVOSA
Body-image / weight-shape overvaluation	**Absent (no body-image disturbance)**	Present and central
Fear of weight gain / drive for thinness	Absent	Present, or weight-defeating behaviour
Reason for restriction	**Sensory aversion, low interest/appetite, or fear of choking or vomiting**	To lose weight or avoid becoming fat
Typical age at onset	Infancy or early childhood	Adolescence or early adulthood
Sex distribution	More often male than in anorexia	Strongly female
Weight	Low or faltering growth (may be normal)	Significantly low (required)

ARFID versus anorexia nervosa; the discriminator is the presence or absence of body-image disturbance and weight/shape overvaluation, not the weight itself (DSM-5-TR 2022; New Oxford Textbook of Psychiatry).

RULE **No body-image disturbance is the line.** Restricted eating with weight loss but no overvaluation of weight or shape and no fear of fatness is ARFID, not anorexia; the absence of the body-image psychopathology is the diagnosis, even when the weight is dangerously low.

GOOD TO REMEMBER

- **ARFID versus anorexia (the discriminator):** both can reach a significantly low weight, but ARFID has intact body image and no drive for thinness, restricting for sensory, appetite or aversive-consequence reasons; anorexia is defined by weight/shape overvaluation and fear of weight gain. Ask why the person restricts.

6.4 The typical presentation, comorbidity and epidemiology

A disorder of childhood, more often male. ARFID most commonly begins in infancy or early childhood and can persist into adulthood. Compared with anorexia, patients are younger, more likely to be male, more likely to carry a comorbid medical condition or an anxiety disorder, and less likely to have a mood disorder (New Oxford Textbook of Psychiatry). The strongest associations are with autism spectrum disorder and neurodevelopmental conditions (the sensory subtype), with anxiety disorders, and with low bone mineral density from chronic undernutrition. Very young infants may present with food refusal, gagging or vomiting and failure to engage with the caregiver at feeds.

Prevalence is uncertain but not trivial. The community prevalence is not established, but among paediatric eating-disorder inpatients ARFID accounts for roughly **5% to 14%** of admissions, so it is a real and separable slice of eating-disorder practice rather than a rarity (New Oxford Textbook of Psychiatry).

3	4	14%
RECOGNISED DRIVERS OF RESTRICTION	CRITERION A CONSEQUENCES (AT LEAST ONE REQUIRED)	UPPER ARFID SHARE OF PAEDIATRIC ED INPATIENTS (FROM 5%)

TRAP **Dismissing it as ordinary fussy eating.** The classic error is to write off ARFID as a phase of picky eating and miss the weight loss, nutritional deficiency or supplement dependence that defines it; conversely, do not label a restrictor as ARFID if body-image concern is actually present, because that is anorexia.

6.5 Management in brief, and the Indian frame

Multidisciplinary, and no first-line drug. There are no randomised trials and no established medication for ARFID; management rests on a multidisciplinary team providing medical stabilisation, nutritional rehabilitation and psychological treatment, with family therapy central for a child so that parents can manage intake. Feared-consequence presentations respond to graded exposure and cognitive-behavioural work; sensory presentations to structured food-chaining and desensitisation. Where drugs are used they are adjuncts of unproven efficacy: SSRIs (for example fluoxetine or sertraline) for a comorbid anxiety disorder, mirtazapine or olanzapine to promote weight gain, and the antihistaminic appetite stimulant cyproheptadine, all off-label and evidence-thin (Maudsley Guidelines). The dosing of the antidepressants sits in the Mood disorders: pharmacotherapy notes, and the exposure and family techniques in the Psychotherapies, clinical assessment, MSE and nosology notes.

GOOD TO REMEMBER

- **ARFID management (the value):** no drug is first-line and none is licensed; the treatment is multidisciplinary nutritional rehabilitation plus psychological therapy (family-based for children, graded exposure for the choking/vomiting-fear type, sensory desensitisation for the selective type), with SSRIs, mirtazapine, olanzapine or cyproheptadine used only as unproven off-label adjuncts (New Oxford Textbook of Psychiatry; Maudsley Guidelines).

INDIA

Two Indian realities meet in this diagnosis. First, eating disorders are widely under-recognised here, and a child's restrictive or "choosy" eating with faltering growth is easily normalised as fussiness rather than assessed, so ARFID reaches care late and undernourished. Second, ARFID is easy to confuse with the **non-fat-phobic** anorexia presentation that is relatively common in Indian and other Asian patients, where a person rationalises restriction as poor appetite or abdominal discomfort. The discriminator is the same one question: is there any overvaluation of weight or shape? If yes, it is anorexia despite the somatic framing; if the body image is genuinely intact, it is ARFID. Weigh the child, plot growth, and screen for the anxiety and neurodevelopmental comorbidity that so often sits underneath.

HOLD THIS

ARFID is restricted eating from sensory-based avoidance, an apparent lack of interest in food, or fear of an aversive consequence (choking, vomiting), causing weight loss or faltering growth, nutritional deficiency, dependence on supplements or feeds, or psychosocial impairment, with no fear of fatness and no body-image disturbance; it is a DSM-5 creation (also ICD-11 6B83), usually starts in early childhood and more often in boys, and is managed by a multidisciplinary team with no first-line drug.

7 · The aetiology and neurobiology of eating disorders

Why one disorder becomes an eating disorder. No single gene, family or magazine causes an eating disorder; each arises where a heritable temperament meets a developmental window and a culture that rewards thinness. The examiner tests this as a **biopsychosocial** synthesis, so the safe answer names risk from three tiers at once and refuses a single cause. This note owns the causal model: the **aetiology of eating disorders** (the genetic and heritability data, the temperament and personality risk, the sociocultural and family factors, the life-event triggers) and the **neurobiology of eating disorders** (the appetite-regulation circuitry, the hypothalamic homeostatic control, leptin and ghrelin, the serotonin and dopamine systems, the reward and cognitive-control circuits, and the interoception literature).

The diagnoses themselves are set out in the anorexia-nervosa, bulimia-nervosa and binge-eating-disorder topics in these notes; the neuroendocrine axes that fail in the starved state (the hypothalamic-pituitary-gonadal, thyroid and adrenal changes) are derived in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes and used here only in causal form. What follows is the machinery: which risks load the dice before the illness, and which brain systems keep it running once weight is lost. The single most useful teaching point sits underneath the whole account, that many of the "biological findings" in a low-weight patient are *consequences* of starvation rather than its cause, which is why the state-versus-trait distinction runs through every subtopic.

7.1 The biopsychosocial model as the organising frame

Three tiers, one threshold. The **biopsychosocial** model holds that eating disorders emerge from an interaction of predisposing, precipitating and perpetuating factors across biological, psychological and social tiers, none sufficient alone. A heritable vulnerability (a particular temperament, a susceptibility to anxiety or affective instability, or a neurocircuitry difference) is expressed only under adverse conditions such as ill-judged dieting or emotional stress, and is then locked in by the effects of starvation itself. This gene-environment framing is why the aetiology of eating disorders is written as a convergence, not a chain.

Predispose, precipitate, perpetuate. A clean way to hold it is by timing: *predisposing* factors (genes, temperament, family and cultural context) set the vulnerability; a *precipitant* (a diet that "works", a life event, puberty) tips a susceptible person over; and *perpetuating* factors (the neuroendocrine and cognitive effects of the starved or binge-purge state) hold the disorder in place and make it self-reinforcing.

RULE **No single cause; name three tiers.** Eating disorders are multifactorial and biopsychosocial. A heritable temperament, a precipitating stressor or diet, and a perpetuating starved or binge-purge state converge, so the correct answer refuses a monocausal ("it is just the media") account.

7.2 Genetics and heritability

The disorders run in families and twins. Family studies show an increased risk of anorexia nervosa and bulimia nervosa in the relatives of affected probands, and more binge-eating disorder among the first-degree relatives of people with binge-eating disorder. Twin studies

comparing identical with fraternal pairs give heritability estimates of roughly **33% to 84%** for anorexia nervosa, **28% to 83%** for bulimia nervosa, and **41% to 57%** for binge-eating disorder, with the remaining variance attributed largely to unique (non-shared) environment; adoption data for disordered-eating symptoms fall in the same high range (Kaplan and Sadock 2024). The wide bands reflect small early samples, but the message is consistent, that these are substantially heritable conditions.

Candidate genes and the modern reframe. Linkage work put signals on chromosome 1 near the serotonin 1D receptor gene (HTR1D) and the delta-opioid receptor gene (OPRD1), and the BDNF Met66 variant has been associated with the *restricting* type of anorexia nervosa, consistent with the disorder's serotonergic, reward and neurotrophic biology. Early candidate-gene and small genome-wide studies were largely non-replicating; the field has since moved to large collaborative genome-wide association studies, which point to overlap of anorexia nervosa with *metabolic and anthropometric* traits as well as psychiatric ones, an emerging reframe of anorexia as partly a "metabo-psychiatric" condition rather than a purely psychological one.

GOOD TO REMEMBER

- **Heritability of the eating disorders:** twin-study estimates are roughly 33% to 84% for anorexia nervosa, 28% to 83% for bulimia nervosa and 41% to 57% for binge-eating disorder; substantial genetic loading with the rest largely non-shared environment, and the candidate signals cluster on serotonergic (HTR1D), opioid (OPRD1) and neurotrophic (BDNF) genes.

7.3 Temperament and personality risk

The anorexic and bulimic temperaments differ. A meta-analysis of the Temperament and Character Inventory found significantly higher *harm avoidance* and *persistence* in anorexia nervosa than in bulimia nervosa, binge-eating disorder or controls, whereas *novelty seeking* was elevated specifically in bulimia nervosa, which also shows greater impulsivity and low self-directedness (Kaplan and Sadock 2024). The restricting anorexic tends toward an anxious, rigid, over-controlled style; the bulimic toward an impulsive, dysregulated one, mirroring the behaviours of restriction versus binge-purge.

Perfectionism is the signature trait. Perfectionism was the psychological feature first tied to anorexia nervosa; higher scores track lower body weight, more eating rituals and less motivation to change, and are most severe where obsessive-compulsive personality disorder or obsessive-compulsive disorder co-occur. Trait anxiety, a common accompaniment, predicts a lower likelihood of recovery. Crucially, harm avoidance falls and reward dependence rises after weight restoration, a reminder that some "traits" are partly state effects of starvation.

PEARL

Read the temperament off the behaviour: **harm avoidance, persistence and perfectionism** load the over-controlled, anxious restricting anorexic; **novelty seeking, impulsivity and low self-directedness** load the dysregulated bulimic. This is why obsessive-compulsive traits cluster with anorexia and impulse-control and affective-dysregulation problems cluster with bulimia.

7.4 Sociocultural and family factors

The thin ideal, internalised. The social tier is the internalisation of a culturally transmitted *thin ideal*: exposure to media and peer messages that equate slimness with worth, body dissatisfaction, and dieting as the normative response, amplified in weight-sensitive settings (ballet, modelling, gymnastics, wrestling). Sociocultural pressure is neither necessary nor sufficient, but it shapes which vulnerable individuals cross into disordered eating and gives the illness its content (the fear of fatness, the over-valuation of shape).

Family influence, without blame. Family factors include parental psychiatric illness (eating disorders, anxiety, affective disorders and substance use aggregate in families), parental attitudes to weight and eating, and interaction patterns. The modern reading is that the family is a legitimate arena for *treatment* (family-based therapy is first-line for young people, set out in the anorexia-management topic in these notes) rather than a place to assign cause; the old "psychosomatic family" typology is not an evidence-based aetiology.

7.5 Life-event triggers and the developmental window

Adolescence is the vulnerable window. Onset clusters in adolescence and early adulthood because puberty, identity formation and the surge in body concern coincide with rising autonomy over eating; puberty and age themselves moderate the genetic influence on eating-disorder traits. The commonest precipitant is a diet that "works", producing early praise or a sense of control that becomes self-reinforcing as weight falls.

Stressors and adversity as precipitants. Life events (loss, transition, bullying about weight, interpersonal or family stress) and, in a subset, childhood adversity or trauma act as non-specific precipitants that tip a predisposed person over the threshold. They are risk-raising rather than disorder-specific, which is why the same adversity elsewhere produces mood or anxiety pathology instead.

7.6 The homeostatic appetite-regulation circuit

Where appetite is computed. Normal **appetite regulation** is a distributed network, but its homeostatic core is the hypothalamic arcuate nucleus, which holds two opposing neuron populations. One expresses pro-opiomelanocortin and releases alpha-melanocyte-stimulating hormone (an *anorexigenic*, satiety signal); the other co-expresses neuropeptide Y, agouti-related peptide and GABA (an *orexigenic*, hunger signal). The two are mutually inhibitory and project to the paraventricular nucleus (satiety) and the lateral hypothalamic area (feeding, via orexin and melanin-concentrating hormone) (Kaplan and Sadock 2024).

The melanocortin switch. The opposing arcuate signals converge on a single receptor, the melanocortin-4 receptor: alpha-MSH is its agonist (dampening intake) and agouti-related peptide its inverse agonist (driving intake). Loss-of-function mutations in the melanocortin-4 receptor or in pro-opiomelanocortin are the commonest monogenic human obesity, seen in about **1% to 2%** of people with obesity, which shows how much of intake this one node sets. Downstream, the mesolimbic dopamine projection from the ventral tegmental area to the nucleus accumbens adds the *hedonic* layer that homeostasis alone does not.

GOOD TO REMEMBER

- **Arcuate two-population model:** pro-opiomelanocortin/alpha-MSH neurons are anorexigenic (satiety) and the neuropeptide Y/agouti-related peptide/GABA neurons are orexigenic (hunger); they act oppositely on the melanocortin-4 receptor, with alpha-MSH the agonist and agouti-related peptide the inverse agonist. Leptin excites the anorexigenic set and inhibits the orexigenic set; ghrelin does the reverse.

7.7 Leptin and ghrelin: the two hormones to know

Leptin, the adiposity signal. Leptin is a hormone secreted by adipocytes in proportion to fat mass; its physiological job is to *defend against underweight* (a fall in leptin powerfully increases eating and lowers energy expenditure, while a rise above the usual level does little). In anorexia nervosa serum leptin is markedly low and tracks the loss of adipose tissue, rising as weight is regained, and the same low leptin drives the suppression of the reproductive axis and the amenorrhoea. It is almost certainly a *consequence* of the low weight, not a cause. The leptin (ob) gene was cloned in 1994, and the name comes from the Greek *leptos*, "thin".

Ghrelin, the hunger signal. Ghrelin is a peptide released mainly by enteroendocrine cells of the stomach; plasma levels rise before meals and during fasting and fall after eating, making it the principal circulating *orexigenic* signal (it acts on the arcuate nucleus and on reward nodes such as the ventral tegmental area and nucleus accumbens). In anorexia nervosa ghrelin is *elevated* and falls toward normal with weight gain, read as an unheeded hunger drive; in bulimia nervosa the post-meal suppression of ghrelin is blunted, consistent with impaired post-ingestive satiety that may permit binges.

HORMONE	SOURCE AND DIRECTION	ANOREXIA NERVOSA	BULIMIA NERVOSA
Leptin	Adipocytes; anorexigenic (satiety), defends against underweight, rises with fat mass	**Markedly low; rises as weight is regained (drives amenorrhoea)**	Variable / near-normal
Ghrelin	Stomach; orexigenic (hunger), rises before meals, falls after eating	**Elevated; falls toward normal with weight gain**	Blunted post-meal suppression (impaired satiety)

The two examinable appetite hormones and their direction of change; leptin falls and ghrelin rises in the starved anorexic state, both largely as consequences of low weight (Kaplan and Sadock 2024).

MNEMONIC

LEPTIN LEAN, GHRELIN GROWLS

Leptin from fat says "we are fat enough" (Greek *leptos*, thin), an anorexigenic satiety signal that is *low* when the anorexic is lean; **Ghrelin** is the growling-stomach hunger hormone that rises before meals and is *high* in anorexia nervosa, an ignored drive to eat.

TRAP **Calling starvation biology the cause.** The low leptin, high ghrelin, sick-euthyroid pattern and high cortisol of a low-weight patient are largely *state* effects of starvation that reverse with refeeding, not proof of a primary hormonal cause; reading them as causal (or as a hormone disorder to correct) is the classic reverse-causation error.

7.8 The serotonin and dopamine systems

Serotonin, anxiety and restraint. Of all the transmitter systems the serotonin eating disorder link is the most studied, because serotonin is both anxiogenic and inhibitory to feeding, exploration and sexual behaviour, all of which are suppressed in anorexia nervosa. PET studies of *recovered* patients (studied off the confounding starved state) show reduced 5-HT2A receptor binding in the insula, cingulate and temporal cortex, and the degree of binding change correlates positively with harm avoidance, tying the serotonin abnormality to the anxious temperament; in bulimia nervosa altered orbitofrontal serotonin binding maps instead onto impulsivity. This is the neurochemical rationale behind fluoxetine's role in bulimia nervosa (developed in the bulimia-nervosa and Mood disorders: pharmacotherapy notes).

Dopamine and altered reward. The dopamine reward system is also disturbed: recovered anorexia-nervosa women show increased dopamine D2/3 receptor availability in the ventral striatum and an altered striatal response to food and monetary reward, so the normal link between a rewarding stimulus and the striatal "wanting" signal is uncoupled. One reading is that in anorexia nervosa restraint is experienced as rewarding and eating as anxiety-provoking, an inversion of the usual valence of food.

GOOD TO REMEMBER

- **Serotonin and dopamine in the eating disorders:** recovered anorexia nervosa shows reduced 5-HT2A binding (insula and cingulate) that correlates with harm avoidance, and increased striatal dopamine D2/3 availability with altered reward processing; bulimia nervosa maps onto orbitofrontal serotonin changes and impulsivity. Studying recovered patients separates trait from the state effects of starvation.

RULE **Study the recovered brain to find the trait.** Because starvation itself distorts every measure, the reliable neurobiological signatures (reduced 5-HT2A binding tied to harm avoidance, altered striatal reward) come from *recovered* patients; a finding present only in the ill state is a state marker, not a cause.

7.9 Reward, cognitive control and interoception

Three circuits carry the pathology. The neurobiology of eating disorders converges on three overlapping systems: *taste and reward* processing (ventral striatum, orbitofrontal cortex and the mesolimbic dopamine projection), *cognitive and inhibitory control* (dorsolateral prefrontal and frontostriatal circuits, over-active in the rigid restraint of anorexia, under-active in the disinhibited binge of bulimia and binge-eating disorder), and *interoception*. The balance between an over-controlling executive system and a dysregulated reward system helps explain why the same broad biology yields restriction in one patient and bingeing in another.

The interoceptive insula. Interoception, the sensing of internal bodily states such as hunger, fullness and affect, is served by the anterior insula, and it is disturbed across the eating disorders: anorexic patients show altered insular responses to taste and an impaired reading of hunger and satiety, so the normal interoceptive signals that should start and stop a meal are mis-weighted. This interoceptive account links the appetite abnormality to the body-image disturbance and is one of the more active research frontiers.

33–84% AN TWIN-STUDY HERITABILITY RANGE	**28–83%** BN TWIN-STUDY HERITABILITY RANGE	**57%** BED HERITABILITY (UPPER TWIN ESTIMATE)	**1994** YEAR THE LEPTIN (OB) GENE WAS CLONED

7.10 The India lens: culture, the thin ideal and under-recognition

The sociocultural story is not only Western. The thin-ideal account was built on affluent Western samples, yet the classic Indian and pan-Asian presentation is the *non-fat-phobic* anorexia, in which the person attributes restriction to abdominal discomfort, poor appetite or a religious fast rather than a stated wish to be thin. This both complicates a purely sociocultural aetiology (the fear of fatness is not universal) and matters practically, because the diagnosis still stands on behaviour and a significantly low weight. As the globalised thin ideal reaches urban India, presentations are rising and shifting toward the Western fat-phobic pattern, so the cultural tier is a moving target here.

Under-recognition is the clinical reality. Eating disorders are under-detected in India, dismissed as ordinary dieting or a physical complaint and stereotyped as an affluent-urban-female problem, so men and lower-income patients are missed; the genetic, temperamental and interoceptive vulnerabilities described above are not culture-bound even where the presentation is atypical. The applied lesson from the aetiology is a low threshold to screen (the SCOFF, in the anorexia-nervosa topic in these notes) rather than to wait for a textbook fear of fatness.

INDIA

Two aetiological points travel differently here. First, the **non-fat-phobic** presentation is relatively common in Indian and Asian patients, undercutting a purely thin-ideal causal story and reminding you the biology (heritable temperament, altered serotonin, dopamine and interoception) is not culture-bound. Second, as the globalised thin ideal spreads to urban India, incidence is rising even as eating disorders stay widely under-recognised, so weigh, screen and take the risk seriously in men and lower-income patients too, not only affluent young women.

WORKED EXAMPLE

A 16-year-old girl develops anorexia nervosa after a "successful" diet before an exam. The biopsychosocial formulation writes the causes in tiers: *predisposing*, a family history of anxiety and her own perfectionism and high harm avoidance (heritable temperament, with candidate serotonergic and neurotrophic loading); *precipitating*, the pubertal window, exam stress and the diet that drew praise; *perpetuating*, the starved state itself, low leptin and high ghrelin, a 5-HT2A and striatal-reward picture that makes restraint feel rewarding and eating anxiety-provoking, and mis-read interoceptive hunger and satiety signals. No tier alone explains her; together they do.

HOLD THIS

Eating disorders are biopsychosocial and substantially heritable (twin estimates roughly 33% to 84% for anorexia nervosa); a perfectionistic, harm-avoidant temperament plus a precipitant (dieting, puberty, stress) meets an appetite-regulation circuit (the arcuate pro-opiomelanocortin/alpha-MSH satiety versus neuropeptide Y/agouti-related peptide hunger neurons acting on the melanocortin-4 receptor) tuned by leptin (low in anorexia, defends against underweight) and ghrelin (high in anorexia, the hunger signal), with reduced 5-HT2A binding tied to harm avoidance, altered striatal dopamine reward, and disturbed insular interoception; most hormonal findings in a low-weight patient are consequences of starvation, not its cause.

8 · Pica, rumination and other feeding disorders

The feeding disorders below the famous eating disorders. Beneath anorexia, bulimia and binge-eating sit three quieter diagnoses the syllabus still expects you to define crisply: **pica**, **rumination disorder**, and the residual "**other feeding disorders**" that capture presentations falling short of a full syndrome. They are grouped with the eating disorders because each is a persistent disturbance of eating or eating-related behaviour that harms physical health or functioning, but they carry no drive for thinness and no body-image psychopathology. The reward in a viva is precise criterion recall, the developmental and comorbidity context (they cluster with intellectual disability and autism), and two or three sharp discriminators.

Small topic, clean marks. DSM-5-TR and ICD-11 both give each entity its own code (ICD-11: **6B84 Pica**, **6B85 Rumination-regurgitation disorder**, **6B8Y** other specified and **6B8Z** unspecified). This note owns the definitions, the developmental frame and the brief management; the full anorexia and bulimia syndromes have their own notes, the SSRI dosing used for comorbidity sits in the Mood disorders: pharmacotherapy notes, and the behavioural and family techniques in the Psychotherapies, clinical assessment, MSE and nosology notes.

8.1 Pica: the persistent eating of non-nutritive substances

Four criteria, and a suggested floor age. Pica is the persistent eating of **non-nutritive substances** (paper, soap, cloth, hair, soil, chalk, paint, metal, pebbles, charcoal, clay, starch or ice) over a period of at least one month (Criterion A), that is inappropriate to the person's developmental level (Criterion B) and is not part of a culturally supported or socially normative practice (Criterion C); if it occurs within another disorder or in pregnancy, it counts only when severe enough to warrant independent attention (Criterion D). A minimum age of **2 years** is suggested, to exclude the ordinary mouthing and swallowing of objects by infants (DSM-5-TR 2022).

Two exclusions carry the diagnosis. Criterion B and Criterion C are what the examiner is testing. The substance must be developmentally out of place (a toddler mouthing a toy is not pica; a school-age child eating chalk daily may be), and it must not be a sanctioned cultural practice, because in several communities the eating of earth or clay is a valued or normative behaviour rather than a disorder.

RULE **Non-nutritive, non-food, out of place, not cultural.** Pica is diagnosed only when the eaten substance is nutritionally empty, developmentally inappropriate (suggested floor of two years) and outside any culturally sanctioned practice; take away any one of those and it is not pica.

8.2 Pica: the classic subtypes and the real danger

Name the substance, name the risk. Pica is traditionally sub-labelled by what is eaten, and the medical danger tracks the specific substance rather than the behaviour itself.

Geophagia

Eating of earth, soil, mud or clay; the commonest form worldwide, frequent in pregnancy and where soil is culturally used, and a route to parasitic infection and lead exposure.

Pagophagia

Compulsive eating of ice; the form most tightly linked to iron-deficiency anaemia and often the first clue to it.

Amylophagia

Eating of raw starch (uncooked rice, flour, laundry starch), again associated with pregnancy and iron deficiency.

Trichophagia and coprophagia

Eating of hair (risking a trichobezoar and bowel obstruction) and of faeces; the hair form overlaps with trichotillomania.

The complications are what kill. Pica reaches medical attention through its consequences: broken teeth, choking, intestinal obstruction or perforation, a **bezoar**, infection (toxoplasmosis, toxocariasis from soil or faeces) and **lead or other poisoning**. It is significantly associated with anaemia, low haemoglobin and low plasma zinc, though the direction of causation is uncertain (New Oxford Textbook of Psychiatry). Prevalence is roughly **5%** among school-age children, and a worldwide meta-analysis put it near **28%** in pregnancy or the postpartum period.

GOOD TO REMEMBER

- **Pica (the syndrome):** persistent eating of non-nutritive, non-food substances for at least one month, developmentally inappropriate (suggested minimum age two years) and not culturally sanctioned; the defining discriminator from anorexia is that the substance is nutritionally empty and not eaten to control weight, and the danger (obstruction, bezoar, lead poisoning, infection) tracks the specific item ingested (DSM-5-TR 2022; New Oxford Textbook of Psychiatry).

WORKED EXAMPLE

A pregnant woman is brought with weeks of compulsively crunching ice and, on questioning, eating small amounts of raw rice. Her haemoglobin is low. This is pagophagia with amylophagia, developmentally inappropriate and not a sanctioned practice, so it meets pica; the pica is best read as a flag for iron-deficiency anaemia, which is corrected while the eating is monitored. Contrast this with a woman from a community where a little clean earth is eaten in pregnancy as an accepted custom: Criterion C removes that from the diagnosis.

8.3 Rumination disorder: repeated, effortless regurgitation

Bring it up, re-chew, re-swallow or spit. Rumination disorder is the repeated regurgitation of food over at least one month, where the regurgitated food may be re-chewed, re-swallowed or spat out (Criterion A); the behaviour is *not* attributable to a gastrointestinal or other medical condition such as gastro-oesophageal reflux or pyloric stenosis (Criterion B), does not occur exclusively during anorexia, bulimia, binge-eating disorder or ARFID (Criterion C), and when it sits inside another disorder must be severe enough to warrant separate attention (Criterion D). The regurgitation is frequent, at least several times a week and typically each day (DSM-5-TR 2022).

The signature is that it is effortless and calm. Previously swallowed food is brought up without apparent nausea, retching or disgust, often preceded only by an urge or a belching sensation, and the regurgitant is recognisable and may taste pleasant, tending to stop once it turns acidic. In infants and in people with intellectual disability the act appears to serve a self-soothing or self-stimulating function, akin to other repetitive behaviours; infants show a characteristic straining, back-arching posture with tongue-sucking movements.

TRAP **Reading rumination as vomiting or reflux.** The classic error is to chase regurgitation as gastro-oesophageal reflux or vomiting and miss the psychiatric diagnosis; rumination is effortless, painless, without nausea or retching, brings up recognisable pleasant-tasting food, and is a diagnosis made only once genuine gastrointestinal disease is excluded.

PEARL

A quiet nosological difference is worth a mark: DSM-5-TR allows rumination disorder to be diagnosed across the lifespan, *including in infancy*, whereas ICD-11 states it should *not* be diagnosed in infants and frames the regurgitation as intentional and relatively easy to induce. Same phenomenon, two thresholds.

GOOD TO REMEMBER

- **Rumination disorder (the syndrome):** repeated, effortless regurgitation of food for at least one month, re-chewed, re-swallowed or spat out, with no nausea or retching, once a gastrointestinal cause (reflux, pyloric stenosis, achalasia, Sandifer syndrome) is excluded and it is not confined to another eating disorder; the discriminator from vomiting is the calm, painless, non-nauseated bringing-up of pleasant recognisable food (DSM-5-TR 2022).

8.4 The other feeding disorders: the residual categories

Clinically real, but sub-threshold or unnamed. The "**other feeding disorders**" are the two residual boxes: other specified feeding or eating disorder (OSFED; ICD-11 6B8Y), where the clinician states why the full criteria are not met, and unspecified feeding or eating disorder (UFED; ICD-11 6B8Z), used when the reason is not specified or information is incomplete, as in an emergency setting. OSFED is not a soft or trivial category, it is often the commonest eating-disorder diagnosis in the community.

Five OSFED presentations are worth naming. DSM-5-TR lists them explicitly.

Atypical anorexia nervosa

All anorexia criteria are met except that, despite significant weight loss, weight is within or above the normal range; the physiological complications can still be severe.

Bulimia / binge-eating disorder of low frequency or limited duration

The full behavioural pattern, but below the frequency or duration threshold of the parent diagnosis.

Purging disorder

Recurrent purging (self-induced vomiting, laxative or diuretic misuse) to influence weight or shape, in the absence of binge eating.

Night eating syndrome

Recurrent eating after waking from sleep, or excessive intake after the evening meal, with awareness and recall, causing distress and not better explained by binge-eating disorder or a circadian shift.

GOOD TO REMEMBER

- **Other feeding disorders (the residual categories):** OSFED (6B8Y) names the reason full criteria are unmet, UFED (6B8Z) does not; the five OSFED exemplars are atypical anorexia nervosa (weight normal or above despite the illness), low-frequency or limited-duration bulimia and binge-eating disorder, purging disorder (purging without binges) and night eating syndrome; the defining move is that a clinically significant eating disturbance is present but sits below a named threshold (DSM-5-TR 2022).

8.5 The shared developmental and comorbidity thread

One question opens all three. Pica and rumination in particular cluster with intellectual developmental disorder and autism spectrum disorder, begin most often in childhood, and can serve a self-soothing or self-stimulating function; both can also arise in otherwise typically developing children and persist or first appear in adults. Screening for neurodevelopmental disorder, neglect and understimulation, and for the medical consequences, belongs in every assessment. The figure alongside sets the three residual feeding disorders next to their discriminators.

ENTITY	DEFINING FEATURE	KEY DISCRIMINATOR
Pica	Eating of non-nutritive, non-food substances for at least one month	**Developmentally inappropriate and not culturally sanctioned; substance not eaten to control weight**
Rumination disorder	Effortless repeated regurgitation, re-chewed or spat out, for at least one month	**No nausea or retching; not due to a gastrointestinal condition; not confined to another eating disorder**
Atypical anorexia (OSFED)	Full anorexia picture despite weight in or above the normal range	Weight is not significantly low, so it falls outside anorexia proper
Shared context	Onset usually in childhood; self-soothing function common	Strong link to intellectual disability and autism; screen for both

The three residual feeding disorders and the single discriminating question that separates each from its neighbours (DSM-5-TR 2022; ICD-11).

2 MINIMUM SUGGESTED AGE FOR PICA (YEARS)	**5%** PICA PREVALENCE, SCHOOL-AGE CHILDREN	**28%** PICA IN PREGNANCY OR POSTPARTUM (META-ANALYSIS)	**1 to 2%** RUMINATION DISORDER, GRADE-SCHOOL CHILDREN

8.6 Management in brief: assess, correct, behavioural first

No first-line drug; behaviour and the underlying deficit lead. There are no adequately powered randomised trials for either pica or rumination, so treatment is pragmatic. For pica, begin with a thorough medical and psychological assessment, treat any complication (obstruction, poisoning, infection) and correct an identified deficiency such as iron-deficiency anaemia, which may reduce the behaviour, although supplementation trials have been negative and resolution is not guaranteed; the best-evidenced psychological approach is behavioural, with applied behaviour analysis and reinforcement-plus-response-reduction procedures, delivered by a multidisciplinary team. Medication is reserved for comorbidity: an SSRI such as fluoxetine (Indian brands Prodep, Fludac, Flunil) where depression or obsessive-compulsive features co-occur, and methylphenidate where ADHD co-occurs, both on case-report evidence only.

Rumination is treated behaviourally across all ages. After excluding gastrointestinal disease (reflux, achalasia, gastroparesis, Sandifer syndrome; manometry with impedance can help), the mainstay is behavioural. Diaphragmatic breathing, which competes with the urge to regurgitate, is the first-line technique for children, adolescents and adults; in young children, changing feeding posture, distraction at onset and parent-directed work are added. Antidepressant dosing is detailed in the Mood disorders: pharmacotherapy notes and the wider behavioural frame in the Psychotherapies, clinical assessment, MSE and nosology notes.

GOOD TO REMEMBER

- **Pica and rumination management (the value):** no drug is first-line and none is licensed for either; treat the medical complications, correct any deficiency (iron), and lead with behavioural therapy (applied behaviour analysis for pica, diaphragmatic breathing for rumination across all ages), reserving an SSRI or methylphenidate only for comorbid depression, OCD or ADHD on weak evidence (New Oxford Textbook of Psychiatry).

INDIA

Three Indian realities meet here. First, *geophagia* and the eating of raw starch are genuinely common in pregnancy and in some communities, so Criterion C does heavy lifting: earth-eating that is a sanctioned local custom is not pica, whereas the same behaviour causing anaemia, obstruction or lead exposure is. Second, the high background rate of iron-deficiency anaemia in Indian women and children makes pagophagia and amylophagia frequent and clinically useful red flags for anaemia that are easily dismissed as habit. Third, feeding and eating disorders are broadly under-recognised in India, and the **non-fat-phobic** presentations that dominate here mean a clinician must actively ask why a person is eating, or not eating, rather than assume the classic picture. Weigh, check haemoglobin, and consider lead where pica is chronic.

HOLD THIS

Pica is the persistent eating of non-nutritive, non-food substances for at least one month, developmentally inappropriate and not culturally sanctioned (suggested floor of two years); rumination disorder is repeated, effortless, non-nauseated regurgitation for at least one month once gastrointestinal disease is excluded; the "other feeding disorders" (OSFED and UFED) catch sub-threshold or unspecified presentations such as atypical anorexia; all three cluster with intellectual disability and autism, carry no drive for thinness, and are led by behavioural treatment with no first-line drug.

9 · Atypical eating disorders, orthorexia and OSFED

The residual bin that is not a dustbin. Not every disordered eater fits the tidy boxes of anorexia, bulimia, binge-eating disorder, ARFID, pica or rumination. A large share of real, impairing eating pathology sits just outside those thresholds, and the classifications capture it in a residual category: **other specified feeding or eating disorder** (OSFED) in DSM-5-TR, the successor to the old DSM-IV EDNOS. This note owns that residual space, plus two entities the exam loves to test at its edges: orthorexia nervosa, a pathological preoccupation with "healthy" or "pure" eating that is not a formal diagnosis, and the **non-fat-phobic** anorexia presentation that is comparatively common in Indian and other Asian patients.

Why it earns exam time. The whole point of these categories is to stop the clinician dismissing a genuine, dangerous disorder because it misses a threshold by a hair. An **atypical anorexia** patient can be normal-weight and still be in starvation physiology; a non-fat-phobic patient can deny any fear of fatness and still be dying of anorexia. The examiner's trap, and the clinical error, is the same sentence: "that is not a real eating disorder." The diagnosis lives in this note; the nutritional rehabilitation and psychological therapies that follow are detailed in the anorexia-management material, and the general cognitive-behavioural and family techniques in the Psychotherapies, clinical assessment, MSE and nosology notes.

9.1 OSFED and USFED: the two residual categories

OSFED names the reason; USFED does not. DSM-5-TR provides two residual codes for presentations that cause clinically significant distress or impairment but do not meet the full criteria for any specific feeding or eating disorder. **Other specified feeding or eating disorder** (OSFED, F50.89) is used when the clinician wants to state the specific reason the presentation falls short, recorded as "other specified feeding or eating disorder" followed by the reason, for example "bulimia nervosa of low frequency". Unspecified feeding or eating disorder (USFED, F50.9) is used when the clinician chooses not to specify a reason, or when information is insufficient to be more precise, as in an emergency setting (DSM-5-TR 2022).

A residual code is not a mild code. OSFED is emphatically not a diagnosis of triviality. Many OSFED presentations, atypical anorexia in particular, carry the same medical danger as their full-threshold counterparts; the label describes which criterion is not met, not how sick the patient is. ICD-11 mirrors the structure with its own residual categories.

GOOD TO REMEMBER

- **OSFED versus USFED (the discriminator):** both are residual codes for a clinically significant feeding or eating disturbance that misses full criteria; OSFED (F50.89) is chosen when the clinician *states* the specific reason it falls short (e.g. "bulimia nervosa of low frequency"), USFED (F50.9) when the reason is not specified or information is insufficient (DSM-5-TR 2022).

9.2 The five named OSFED presentations

Learn the list; the reason for each is the answer. DSM-5-TR names five prototypical OSFED presentations. For every one, the whole disorder is present except a single missing element, and that missing element is exactly what the examiner wants you to name.

OSFED PRESENTATION	WHAT IS MET	THE SINGLE MISSING ELEMENT
Atypical anorexia nervosa	All anorexia criteria, with significant weight loss	**Weight is within or above the normal range**
Bulimia nervosa (low frequency and/or limited duration)	All bulimia criteria	**Binge-purge cycles on average less than once a week and/or for less than three months**
Binge-eating disorder (low frequency and/or limited duration)	All binge-eating-disorder criteria	Binges on average less than once a week and/or for less than three months
Purging disorder	Recurrent purging to influence weight or shape	**No binge eating (purging occurs without binges)**
Night eating syndrome	Recurrent night eating with awareness and recall, causing distress or impairment	Not better explained by binge-eating disorder, sleep-wake change or local norms

The five DSM-5-TR OSFED exemplars; in each, name the one criterion that is not met, because that is the diagnosis (DSM-5-TR 2022).

5	<3	2
NAMED OSFED PRESENTATIONS IN DSM-5-TR	LIMITED-DURATION THRESHOLD (MONTHS)	SCOFF SCREEN POSITIVE CUT-OFF (OF FIVE)

TRAP **Reading "subthreshold" as "safe".** Do not treat an OSFED label as a reassurance; atypical anorexia and purging disorder carry the same electrolyte and cardiac risk as the full syndromes, so the medical work-up is unchanged by the qualifier.

9.3 Atypical anorexia nervosa: the weight is not the diagnosis

All the anorexia, minus the low weight. **Atypical anorexia nervosa** is the OSFED presentation in which every criterion for anorexia is met, including significant weight loss and the whole cognitive engine of weight and shape overvaluation, except that despite that weight loss the person's weight remains within or above the normal range. DSM-5-TR is explicit that adults who are not underweight by population standards, for example a body mass index of 19.0 kg/m^2 or above, should not be diagnosed with anorexia nervosa but may warrant atypical anorexia nervosa instead. Crucially, these patients "may experience many of the physiological complications associated with anorexia nervosa": bradycardia, hypotension, electrolyte disturbance and

amenorrhoea can all appear at a normal weight if the *rate* and *magnitude* of loss are large (DSM-5-TR 2022).

RULE **Rate of loss, not the number on the scale.** A normal or high absolute weight does not exclude anorexic physiology; a large, rapid loss with intact weight-shape overvaluation is atypical anorexia and is medically managed as anorexia.

GOOD TO REMEMBER

- **Atypical anorexia nervosa (the criterion):** all anorexia criteria are met, including the weight-and-shape overvaluation and significant weight loss, but the weight is within or above the normal range (DSM-5-TR anchors this at an adult body mass index of about 19.0 kg/m^2 or above); the physiological complications of starvation can still be present, so it is coded and treated as a serious eating disorder, not a mild one.

9.4 Non-fat-phobic anorexia: food refusal without the stated fear of fatness

The engine can be silent. A recognised variant, comparatively common in Asian and Indian populations, is **non-fat-phobic** anorexia, in which the patient shows the weight loss and the food refusal of anorexia but does *not* voice the classic intense fear of gaining weight or of becoming fat. Instead the dietary restriction is rationalised through a more culturally sanctioned complaint, most often gastrointestinal discomfort ("bloating", "no appetite", "food does not suit me"), or through cultural and religious motives such as fasting. Both DSM-5-TR and ICD-11 note this presentation and instruct that the absence of an *expressed* fat phobia does not exclude anorexia: if collateral history, observation or longitudinal course supports an underlying preoccupation with, or behaviour aimed at, weight and shape, the diagnosis stands (DSM-5-TR 2022; ICD-11 CDDR).

Screen actively, because it hides. Under-recognition is the rule when the presenting language is somatic, so a low-friction case-finding tool earns its place. The five-item SCOFF questionnaire is the standard screen; a score of two or more affirmative answers (out of five) is a positive screen that warrants full eating-disorder assessment. The instrument itself is reproduced in the accompanying scale plate. The somatic framing and its overlap with medical presentations connect to the Somatic-symptom, sexual, impulse-control disorders and suicide notes and the transcultural material in the Consultation-liaison, geriatric and transcultural psychiatry notes.

MNEMONIC

S·C·O·F·F

Sick (do you make yourself sick when uncomfortably full), **C**ontrol (have you lost control over eating), **O**ne stone (lost more than about 6.35 kg in a three-month span), **F**at (do you believe yourself fat when others say thin), **F**ood (does food dominate your life). Two or more "yes" answers is a positive screen.

RULE **Absence of stated fat-phobia does not exclude anorexia.** If the restriction is driven by inferable weight-shape concern behind a somatic or cultural rationale, it is anorexia; treat the food refusal, not the offered excuse.

TRAP **"She never said she wanted to be thin, so it is not anorexia."** The classic error is to reject a non-fat-phobic or atypical presentation as "not a real eating disorder"; the fear of fatness may be unexpressed, denied, or culturally recoded as GI complaint, while the starvation is entirely real.

GOOD TO REMEMBER

- **Non-fat-phobic anorexia (the criterion):** weight loss and food refusal with the underlying anorexic drive present but the fear of weight gain not verbally expressed, the restriction attributed instead to gastrointestinal discomfort or cultural or religious reasons; both DSM-5-TR and ICD-11 retain the anorexia diagnosis when collateral or longitudinal evidence supports weight-shape motivation, so screen (SCOFF two or more of five) rather than take the somatic story at face value.

INDIA

This is the most India-relevant slice of eating-disorder practice. The **non-fat-phobic** pattern is comparatively common here, so an adolescent who has lost dangerous weight but frames it as "gas", "acidity" or "no hunger" is easily routed through gastroenterology rather than psychiatry, and eating disorders are under-recognised across Indian settings for exactly this reason. The remedy is a low threshold to weigh, plot the trajectory, take collateral from family, and screen actively; do not require the textbook sentence about fearing fatness before you take the illness seriously. The same somatising instinct that hides anorexia here also drives the over-medicalisation of distress, so the transcultural lens in the Consultation-liaison, geriatric and transcultural psychiatry notes applies directly.

9.5 Purging disorder and night eating syndrome

Two well-characterised residual entities. Beyond the subthreshold variants, two OSFED presentations are distinct enough to have their own literature, though neither is a standalone formal category in DSM-5 or ICD-11.

Purging disorder

Recurrent purging behaviour to influence weight or shape (self-induced vomiting; misuse of laxatives, diuretics or other medications) in the *absence* of objective binge eating; the missing bulimic binge is the whole distinction, and body weight is typically normal.

Night eating syndrome

Recurrent episodes of night eating, evening hyperphagia after the evening meal and/or nocturnal awakenings with food ingestion, with full awareness and recall of the eating, causing distress or impairment, and not better explained by an altered sleep-wake cycle, local norms, binge-eating disorder or another disorder. The awareness and recall separate it from the parasomnia of sleep-related eating.

GOOD TO REMEMBER

- **Purging disorder (the criterion):** recurrent purging to control weight or shape *without* binge eating; the absent binge is what separates it from bulimia nervosa.
- **Night eating syndrome (the criterion):** recurrent evening or nocturnal eating with *awareness and recall*; the retained awareness distinguishes it from the sleep-related eating parasomnia, which is amnesic.

9.6 Orthorexia nervosa: a preoccupation, not a diagnosis

An obsession with purity, not thinness. Orthorexia nervosa, a term coined by the physician Steven Bratman in 1997, describes a pathological preoccupation with eating food perceived as "healthy", "clean" or "pure". The drive is not toward a thin body but toward dietary righteousness: escalating self-imposed rules about food quality and preparation, mounting time and anxiety spent policing the diet, guilt or distress when rules are broken, and social withdrawal to protect the regimen. In the extreme it produces genuine malnutrition and impairment. Critically, orthorexia is **not** a formal diagnosis in DSM-5-TR or ICD-11; proposed criteria exist but are not validated, and in exam terms it is best framed as a described phenomenon on the boundary of an eating disorder and obsessive-compulsive spectrum pathology, captured, where impairing, under OSFED rather than as a category of its own.

GOOD TO REMEMBER

- **Orthorexia nervosa (the defining feature):** a pathological preoccupation with the perceived healthiness or purity of food, with rigid escalating rules, distress when they are broken, and functional or nutritional harm; the fixation is on food *quality*, not on weight or shape, and it is *not* a formal DSM-5-TR or ICD-11 diagnosis (Bratman, 1997).

HOLD THIS

OSFED (other specified feeding or eating disorder) is the DSM-5-TR residual category, successor to EDNOS, for clinically significant eating pathology that misses full criteria by one element; its five exemplars are atypical anorexia (all anorexia features but weight within or above the normal range), low-frequency/limited-duration bulimia and binge-eating disorder (less than once a week and/or under three months), purging disorder (purging without binges) and night eating syndrome; the non-fat-phobic anorexia common in India is still anorexia when weight-shape drive is inferable behind a somatic rationale; and orthorexia, a preoccupation with "pure" eating, is a described phenomenon, not a formal diagnosis. The one trap is calling any of these "not a real eating disorder".

10 · Discriminating the eating disorders

The step that turns six syndromes into one decision. Every eating-disorder topic in these notes teaches a single entity in full; this note is the **differentiation** step that lays them side by side and asks the one question that separates each from its neighbour. The examiner rarely gives you a textbook case; the marks are in telling anorexia nervosa restricting from anorexia nervosa binge-purge, telling anorexia from bulimia nervosa, telling bulimia from binge eating disorder, and telling all four from ARFID and from the residual OSFED presentations. The whole map collapses onto a small set of axes: the *weight* (significantly low versus normal-or-above), the presence of an objective *binge*, the presence of a **compensatory behaviour**, the presence or absence of **body image** and weight-shape overvaluation, and the *medical picture* that follows from each.

Why this note exists. The individual diagnoses are owned by their own topics in these notes; this one owns the *discrimination*, the decision tree and the master grid that the exam draws its viva questions from. Two of the axes do most of the work: *weight* forks anorexia (and its atypical

variant) from the rest, and *compensatory behaviour* forks bulimia from binge eating disorder, while *body image* is the axis that pulls ARFID out of the whole family. The accompanying figure typesets these axes as a decision grid, and the eating-disorder screens sit on the accompanying scale plate; the guideline-anchored treatment that each label buys is developed in the individual management topics in these notes.

Read each row across	Anorexia restricting	Anorexia binge-purge	Bulimia nervosa	Binge eating disorder	ARFID no body image	OSFED / atypical
Body weight	Low	Low	Normal or above	Often raised	Low or faltering	Variable
Binge episodes	Absent	Present	Present	Present	Absent	Variable
Compensatory behaviour	None (restrict)	Purge / exercise	Vomiting, laxatives, exercise	None	None	Variable
Weight-shape overvaluation	Yes	Yes	Yes	Often	No	Sub-threshold
The one discriminator	Low weight, pure restriction	Low weight + binge-purge	Normal weight + binge-purge	Binge, no compensation	Restriction, no weight concern	Some but not all criteria

The shaded bottom row is the deciding line. ARFID is the one to hold apart (coral): food restriction with weight loss but no weight-shape overvaluation, so it is not anorexia.

Fig 17.6 The eating disorders discriminated. Read each row across the six presentations. Body weight separates the low-weight anorexias from the normal-or-above bulimia and the often-raised binge eating disorder; the presence of a binge separates restricting anorexia from the rest; and the compensatory behaviour separates bulimia and binge-purge anorexia (which purge) from binge eating disorder (which does not). The deciding line is the bottom row: anorexia is low weight with restriction, bulimia is normal weight with binge-purge, binge eating disorder is bingeing without compensation, and ARFID, the one most often mislabelled, is food restriction with weight loss but no overvaluation of weight or shape, so the body-image disturbance that defines anorexia is simply absent. OSFED and atypical anorexia meet some but not all of the full criteria.

10.1 The five axes that do all the work

Learn the axes, not the cases. Every discrimination in the family is settled by asking, in order, about five variables. Naming them in this sequence is the fastest route through any differentiation question, because each answer prunes the tree.

Weight

Significantly low (from restriction) versus normal-or-above. The first fork: a significantly low, restriction-driven weight points to anorexia nervosa; a normal or raised weight points toward bulimia nervosa, binge eating disorder or a residual presentation.

The binge

Present or absent. An objective binge (a discrete overeating episode with loss of control) is required for bulimia and for binge eating disorder, and defines the binge-purge subtype of anorexia; it is absent in restricting anorexia, in ARFID and in purging disorder.

Compensatory behaviour

Present or absent. Recurrent inappropriate compensation (self-induced vomiting, laxatives, diuretics, fasting, driven exercise) defines bulimia and is what its absence removes to leave binge eating disorder.

Body image and weight-shape overvaluation

Present or absent. Overvaluation of weight and shape is central to anorexia and bulimia and partly present in binge eating disorder, but it is *absent* in ARFID, which is the disorder without a body image disturbance.

The medical picture

The physiological footprint each pattern leaves: starvation physiology and refeeding risk at low weight, a hypokalaemic alkalosis and Russell's sign with purging, and metabolic morbidity with binge-driven obesity.

MNEMONIC

Weight? · Binge? · Compensation? · Body-image?

Four questions in order settle almost every eating-disorder differentiation. **Weight** low or not (anorexia versus the rest); **Binge** present or not (bulimia and binge eating disorder versus restricting anorexia and ARFID); **Compensation** present or not (bulimia versus binge eating disorder); **Body-image** overvaluation present or not (the eating disorders proper versus ARFID). The fifth axis, the medical picture, then tells you how urgent the patient is.

10.2 The master differentiation grid

The whole family on one page. The grid below reads each entity against the five axes; the highlighted cell in each row is the discriminator that pins that label, the single answer that no neighbour shares. The figure alongside renders the same information as a branching decision tree. Read any real patient down the columns and the diagnosis falls out of the pattern rather than out of a remembered vignette.

DISORDER	TYPICAL WEIGHT	OBJECTIVE BINGE	COMPENSATORY BEHAVIOUR	BODY IMAGE / WEIGHT-SHAPE OVERVALUATION	MEDICAL SIGNATURE
Anorexia nervosa, restricting	Significantly low	**Absent (no binge or purge in the last three months)**	None (dieting, fasting, over-exercise)	Present and central	Starvation physiology; refeeding risk
Anorexia nervosa, binge-purge	**Significantly low (the fork from bulimia)**	Present	Present (vomiting, laxatives, diuretics)	Present and central	Hypokalaemia, alkalosis, prolonged QTc; refeeding risk
Bulimia nervosa	Normal or above	Present	**Present, recurrent (the defining feature)**	Present and central	Hypokalaemic hypochloraemic alkalosis, Russell's sign, dental erosion, prolonged QTc

DISORDER	TYPICAL WEIGHT	OBJECTIVE BINGE	COMPENSATORY BEHAVIOUR	BODY IMAGE / WEIGHT-SHAPE OVERVALUATION	MEDICAL SIGNATURE
Binge eating disorder	Overweight or obese	Present	**Absent (no regular compensation)**	Partly present (overvaluation common, not required)	Metabolic and cardiovascular morbidity of obesity
ARFID	Low or faltering growth (may be normal)	Absent	None	**Absent (no body image disturbance)**	Nutritional deficiency, faltering growth, supplement or feed dependence
Atypical anorexia (OSFED)	**Within or above the normal range despite marked loss**	Absent (restricting pattern)	None or purging	Present and central	Starvation physiology can occur at a normal weight
Purging disorder (OSFED)	Normal	**Absent (purging without a binge)**	Present (purging to control weight)	Present	Electrolyte disturbance from purging

The eating-disorder differentiation grid; in each row the highlighted cell is the one axis that fixes the label, and the two axes that decide most cases are weight (low versus normal-or-above) and compensatory behaviour (present versus absent) (DSM-5-TR 2022; ICD-11 CDDR; Kaplan and Sadock CTP 2024).

RULE **Three questions before you name it.** Ask "is the weight significantly low?", "is there an objective binge?" and "is there a compensatory behaviour?"; those three answers separate restricting anorexia, binge-purge anorexia, bulimia and binge eating disorder, and a fourth question, "is body image intact?", peels ARFID away from all of them.

10.3 Restricting versus binge-purge anorexia

Same low weight, split by the last three months. Within anorexia nervosa the subtype is decided by behaviour over the recent three-month window at a still significantly low weight. The *restricting* subtype loses weight through dieting, fasting or excessive exercise with *no* recurrent binge eating or purging in that window, the classic pure restrictor. The *binge-purge* subtype has engaged, over the same window, in recurrent binge eating or purging (self-induced vomiting, or misuse of laxatives, diuretics or enemas), while the weight remains significantly low. Crossover between the two over the illness course is common, so the label describes the current picture, not the lifetime one, and clinically the binge-purge subtype carries the higher electrolyte and cardiac risk.

GOOD TO REMEMBER

- **Anorexia subtypes (the discriminator):** both share a significantly low, restriction-driven weight and the same body-image psychopathology; the split is the presence of recurrent binge eating or purging in the last three months, absent in the restricting subtype and present in the binge-purge subtype, which carries the greater hypokalaemia and cardiac risk.

10.4 Binge-purge anorexia versus bulimia nervosa: the weight fork

Identical behaviour, decided by the scale. The binge-purge subtype of anorexia and bulimia nervosa can look behaviourally identical, the same objective binge followed by the same compensatory behaviour. The discriminator is weight, and it is enforced by an exclusion rule: if the patient is at a *significantly low* weight, the diagnosis is anorexia nervosa, binge-purge subtype; if the weight is *normal or above*, it is bulimia nervosa (DSM-5-TR Criterion E: bulimia is not diagnosed if the disturbance occurs exclusively during anorexia nervosa). Bulimia is therefore the normal-weight eating disorder, and because there is no emaciation to give it away it is diagnosed by asking, not by looking. Its timeframe threshold is once weekly for three months in DSM-5-TR and once weekly for one month in ICD-11.

FEATURE	ANOREXIA NERVOSA, BINGE-PURGE	BULIMIA NERVOSA
Objective binge	Yes	Yes
Compensatory behaviour	Yes	Yes, recurrent
Typical weight	**Significantly low**	**Normal or above**
Coding rule	Low weight wins: coded as anorexia	Only if not exclusively during anorexia

The weight fork between binge-purge anorexia and bulimia nervosa; when binge-purge behaviour occurs, the weight decides the label and the low-weight patient is always coded as anorexia (DSM-5-TR 2022).

TRAP **A normal weight is falsely reassuring.** The classic error is to discount an eating disorder because the patient looks well and weighs normally, missing the silent hypokalaemia and prolonged QTc of covert purging; a normal weight moves the label from anorexia to bulimia, it does not lower the medical danger.

GOOD TO REMEMBER

- **Bulimia nervosa (the criterion boundary):** objective binges plus recurrent compensatory behaviour at a *normal or above-normal* weight is bulimia nervosa; the identical behaviour at a significantly *low* weight is anorexia nervosa, binge-purge subtype, because the low-weight exclusion rule always codes to anorexia.

10.5 Bulimia versus binge eating disorder: the compensation fork

Take away the compensation and it is binge eating disorder. Binge eating disorder shares the recurrent binge and the loss of control with bulimia nervosa but lacks the regular inappropriate **compensatory behaviour** that bulimia requires. Remove the vomiting, the laxatives, the fasting and the driven exercise from a bulimia-shaped picture and what remains is binge eating disorder. A softer second discriminator: in bulimia dysfunctional dieting usually *precedes* the binge,

whereas in binge eating disorder dieting typically *follows* it, and the patient is commonly overweight or obese rather than of normal weight. The three eating-disorder diagnoses are mutually exclusive for a single episode, so a patient who both binges and purges is bulimia, not binge eating disorder.

RULE **The compensation is the fork in the road.** Recurrent binge with loss of control *plus* regular compensatory behaviour is bulimia nervosa; the same recurrent binge *without* regular compensation is binge eating disorder. Ask whether the binge is undone, not just whether it happens.

GOOD TO REMEMBER

- **Binge eating disorder (the discriminator):** recurrent binge eating with loss of control and marked distress but *no* regular compensatory behaviour; it co-travels with overweight and obesity, dieting follows the binge rather than preceding it, and it is the single most common eating disorder.

10.6 ARFID: the disorder without a body image

One axis pulls it out of the family. ARFID (avoidant/restrictive food intake disorder) is restricted eating that is *not* driven by any wish to be thin. The person avoids or restricts food because of its sensory qualities, an apparent lack of interest in eating, or a fear of an aversive consequence such as choking or vomiting, and this produces weight loss or faltering growth, nutritional deficiency, dependence on supplements or feeds, or psychosocial impairment. What it lacks is the whole engine of anorexia: no fear of weight gain and no disturbance of **body image** or overvaluation of weight and shape. Because both ARFID and anorexia can reach a dangerously low weight with the same starvation physiology, the differentiation is psychological, not physical, and it is settled by a single question, "why does the person restrict?". If the answer is to be thin or to avoid becoming fat, it is anorexia; if it is sensory aversion, low appetite or fear of a feared event, and the body image is intact, it is ARFID.

RULE **No body image disturbance is the line.** Restricted eating with weight loss but no overvaluation of weight or shape and no fear of fatness is ARFID, not anorexia; the absence of the body image psychopathology is the diagnosis, even when the weight is dangerously low.

GOOD TO REMEMBER

- **ARFID (the discriminator):** restriction from sensory aversion, apparent lack of interest in food, or fear of an aversive consequence, with the same possible low weight as anorexia but *no* body image disturbance and no drive for thinness; ask why the person restricts, because the motive, not the weight, separates it from anorexia.

10.7 OSFED, atypical and non-fat-phobic anorexia: subthreshold is not safe

The residual bin that is not a dustbin. A large share of real, impairing eating pathology misses full criteria by a single element and lands in OSFED (other specified feeding or eating disorder), the DSM-5-TR successor to the old EDNOS. Its named exemplars each keep the whole syndrome minus one item: atypical anorexia nervosa is every anorexia feature, including significant weight loss and the weight-shape overvaluation, except that the weight stays within or above the normal

range (DSM-5-TR anchors this at an adult BMI of about **19 kg per m squared** or above); purging disorder is recurrent purging without objective binges; and low-frequency or limited-duration bulimia and binge eating disorder fall below once weekly and/or under three months. A parallel high-yield variant is non-fat-phobic anorexia, in which the patient shows the weight loss and food refusal but does not voice the fear of fatness, rationalising the restriction as gastrointestinal discomfort, poor appetite or a religious fast. Both DSM-5-TR and ICD-11 keep the anorexia diagnosis when collateral or longitudinal evidence supports an underlying weight-shape drive. The single trap across all of these is the same sentence: "that is not a real eating disorder."

TRAP **Reading "subthreshold" or "non-fat-phobic" as "not anorexia".** Atypical anorexia and purging disorder carry the same electrolyte and cardiac risk as the full syndromes, and a large rapid loss can produce starvation physiology at a normal weight; the qualifier names the missing criterion, not a milder illness, so the medical work-up is unchanged.

GOOD TO REMEMBER

- **OSFED atypical anorexia (the criterion):** all anorexia criteria met, including significant weight loss and body-image overvaluation, but the weight is within or above the normal range (adult BMI about 19 kg per m squared or above); starvation physiology can still be present, so it is coded and treated as a serious eating disorder, and non-fat-phobic anorexia remains anorexia when weight-shape drive is inferable behind a somatic rationale.

INDIA

This is the most India-relevant slice of the differentiation. The **non-fat-phobic** presentation is comparatively common in Indian and other Asian patients, so an adolescent who has lost dangerous weight but frames it as "gas", "acidity" or "no appetite" is easily routed through gastroenterology rather than psychiatry, and eating disorders are widely under-recognised across Indian settings for exactly this reason, still stereotyped as a disease only of affluent urban young women when they occur across strata and in men. The remedy is the same low-friction discipline every time: weigh the patient, plot the trajectory, take collateral, and screen actively with the SCOFF rather than requiring the textbook sentence about fearing fatness before taking the illness seriously. The same instinct to somatise distress that hides anorexia here is developed in the Consultation-liaison, geriatric and transcultural psychiatry notes.

10.8 What the differentiation buys you: it changes the treatment

The label is not academic; it re-routes the management. Each fork above sends the patient down a different treatment path, and getting the discrimination right is what makes the plan right. *No drug is first-line for anorexia nervosa*: the primary treatment is nutritional rehabilitation plus a psychological therapy, and medication is at best adjunctive. Cross the weight fork into *bulimia nervosa* and the picture changes: a psychological therapy still leads (NICE NG69 guided self-help stepping up to CBT-ED), but here there is one licensed drug to know cold, fluoxetine at **60 mg per day**, above its 20 to 40 mg antidepressant dose and the medication of choice for bulimia (WFSBP 2023 first-line; brands in India include Prodep, Fludac and Flunil). Cross the compensation fork into *binge eating disorder* and the therapy is again psychological with weight loss explicitly *not* a target, and the only specifically licensed agent is lisdexamfetamine at 50 to 70

mg per day (US FDA, not marketed in India). *ARFID* has no first-line drug at all and is managed by a multidisciplinary team. The deep antidepressant pharmacology of fluoxetine at the high bulimia dose is developed in the Mood disorders: pharmacotherapy notes, and the general cognitive-behavioural technique in the Psychotherapies, clinical assessment, MSE and nosology notes.

RULE **The fork picks the drug.** No drug is first-line for anorexia; fluoxetine 60 mg per day is the one licensed drug for bulimia; lisdexamfetamine is the only agent specifically licensed for binge eating disorder; and ARFID has no first-line drug, so the differentiation directly selects the pharmacology.

GOOD TO REMEMBER

- **Treatment fork (the value anchor):** anorexia has *no* first-line drug (nutrition plus psychotherapy); bulimia's one licensed drug is *fluoxetine 60 mg per day* alongside CBT-ED; binge eating disorder's only specifically licensed agent is *lisdexamfetamine* (50 to 70 mg per day, US FDA) with weight loss not a therapy target; and ARFID has no first-line drug, managed by a multidisciplinary team.

2	60	18.5	19
SCOFF POSITIVE CUT-OFF (YES ANSWERS, OF FIVE)	FLUOXETINE DOSE FOR BULIMIA (MG PER DAY)	ICD-11 ANOREXIA WEIGHT THRESHOLD (ADULT BMI, KG PER M SQUARED)	ATYPICAL ANOREXIA WEIGHT FLOOR (ADULT BMI, KG PER M SQUARED)

WORKED EXAMPLE

Four young women present in one clinic. The first, BMI 15, eats tiny measured portions, exercises for hours, fears fatness, and has not binged or purged: anorexia nervosa, restricting subtype. The second, BMI 15, binges and vomits several times weekly: still anorexia nervosa, binge-purge subtype, because the low weight wins. The third, BMI 22, binges and vomits several times weekly with knuckle calluses and a potassium of 3.1: bulimia nervosa, the normal weight moving the label but not the danger. The fourth, BMI 34, binges alone at night, cannot stop, feels disgusted afterward, but never vomits, fasts or over-exercises: binge eating disorder. Same behaviours, four labels, decided by weight and by the presence of compensatory behaviour, and each label sends her down a different treatment path.

HOLD THIS

Differentiate the eating disorders on five axes, weight, binge, compensatory behaviour, body image and the medical picture: significantly low restriction-driven weight with intact body-image overvaluation is anorexia (restricting if no binge-purge, binge-purge if present), the same binge-purge behaviour at a normal-or-above weight is bulimia, recurrent binges without regular compensation is binge eating disorder, restricted eating with no body image disturbance is ARFID, and every-criterion-but-one presentations (atypical anorexia at a normal weight, non-fat-phobic anorexia, purging disorder) are OSFED and are never "not a real eating disorder"; the fork then picks the drug, no first-line agent for anorexia, fluoxetine 60 mg per day for bulimia, lisdexamfetamine for binge eating disorder, none for ARFID.

Sleep-wake disorders

11 · Normal sleep physiology, architecture and polysomnography

Every sleep-wake disorder in this band is read against a template, and that template is **normal sleep physiology**. Sleep is not the switching-off of the brain but an actively generated, tightly staged process that cycles through predictable states across the night; you cannot call insomnia, narcolepsy, obstructive sleep apnoea or a parasomnia abnormal until you know what the healthy **sleep architecture** looks like. This foundation topic is placed first for exactly that reason, and it earns its keep because the same handful of numbers, the stage percentages, the ninety-minute cycle, the normal REM latency and the polysomnographic indices, recur as the discriminators in every disorder that follows.

Why it matters, and how this note is framed. Two engines run underneath the whole topic. The first is the sleep switch and its drives: an **ascending arousal system** that holds the forebrain awake, a sleep-promoting nucleus that switches it off, and a two-drive timing model that decides when the flip happens. The second is the measurement layer: **polysomnography**, the EEG, EOG and EMG montage that lets you score the stages and read the apnoea-hypopnea and limb-movement indices. The sleep neurochemistry and the ascending-arousal anatomy are developed in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes and the Functional Neuroanatomy and Neurophysiology notes; the sleep architecture, the hypnogram and polysomnography are owned here. Every percentage and index below is drawn from the parsed sources, and the one value the library confirmed only in part, the exact split between the light stages, is flagged rather than dressed up.

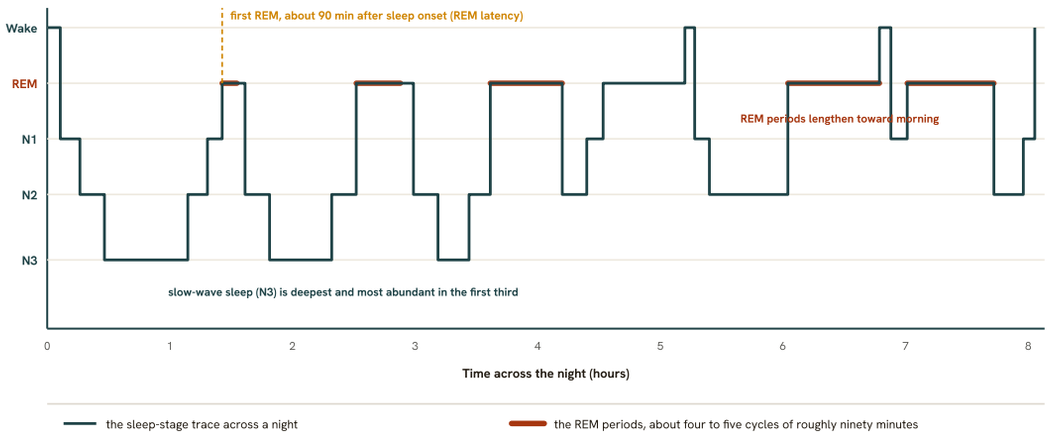


Fig 17.7 The sleep-architecture hypnogram. A normal night runs in four to five cycles of about ninety minutes, each descending from light N1 and N2 into deep slow-wave N3 sleep and then rising into a period of REM. The first REM period arrives about ninety minutes after sleep onset, the normal REM latency, and a shortened REM latency is a soft marker of depression and of narcolepsy. The architecture is not uniform across the night: slow-wave N3 sleep is deepest and most abundant in the first third and fades later, while the REM periods lengthen toward morning, so most deep sleep is early and most dreaming sleep is late. The stage percentages, the cycle length and the REM latency are the values to hold; the neurochemistry that generates these states is developed in the Neurochemistry, neuroendocrinology, neuroimaging and basic psychopharmacology notes and the Functional neuroanatomy and neurophysiology notes.

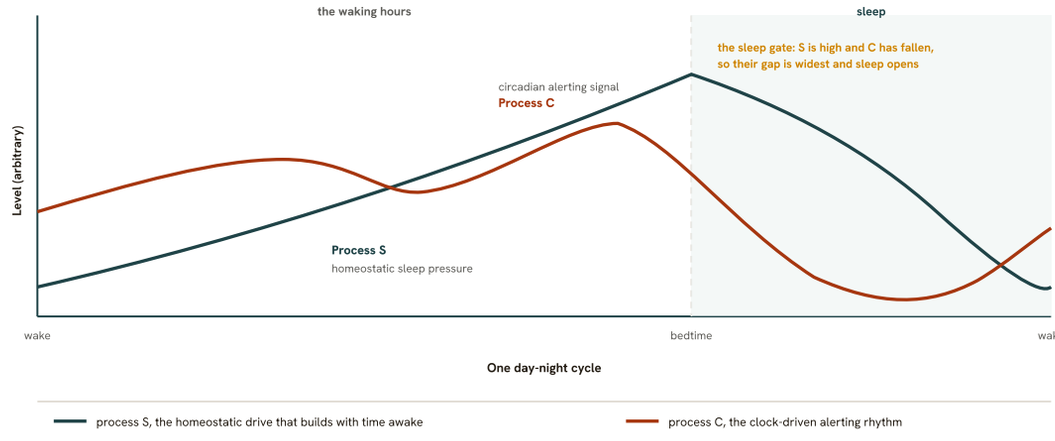


Fig 17.8 The two-process model of sleep regulation. Two independent processes set when we sleep. Process S is the homeostatic sleep pressure that builds steadily with every hour awake and discharges across sleep, so the longer the prior wakefulness the deeper the ensuing slow-wave sleep. Process C is the circadian alerting signal from the suprachiasmatic clock, which counter-intuitively rises through the evening (the wake-maintenance zone that makes it hard to fall asleep early) and then falls overnight to a trough in the early hours. Sleep opens when the gap between the two is widest, with S high and C fallen, and it consolidates through the night; the model explains why sleep deprivation deepens sleep, why the early-hours circadian trough is when lapses and errors peak, and why shifting the clock (the circadian disorders) or the drive (insomnia) disturbs sleep.

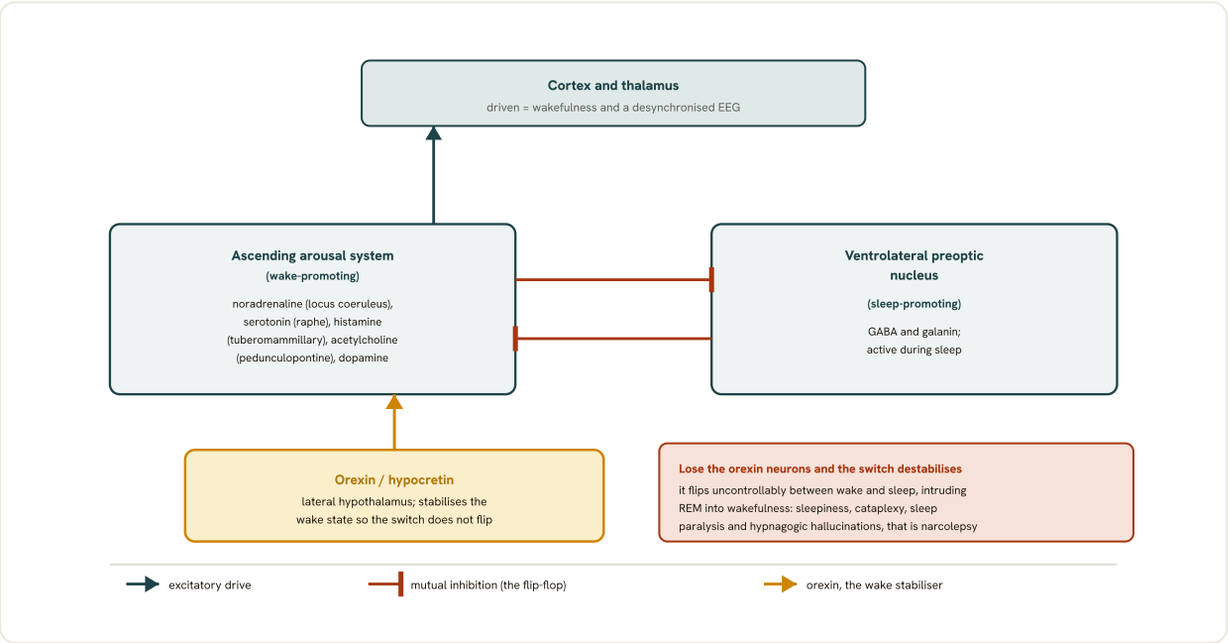


Fig 17.9 The ascending arousal system and the sleep-wake flip-flop switch. Wakefulness is driven by the ascending arousal system, a set of brainstem and hypothalamic monoaminergic and cholinergic nuclei that project up to the cortex; sleep is driven by the ventrolateral preoptic nucleus, which uses GABA and galanin. The two mutually inhibit each other, and mutual inhibition makes a flip-flop switch that snaps cleanly between wake and sleep and avoids dozing in between. The orexin (hypocretin) neurons of the lateral hypothalamus stabilise the wake side of the switch. When these neurons are lost, as in narcolepsy type 1, the switch becomes unstable and flips uncontrollably, so sleep and REM phenomena intrude into wakefulness as the tetrad of excessive sleepiness, cataplexy, sleep paralysis and hypnagogic hallucinations. The neurotransmitter systems themselves are developed in the Neurochemistry, neuroendocrinology, neuroimaging and basic psychopharmacology notes.

11.1 Two states of sleep: NREM and REM

The basic division. Human sleep alternates between two fundamentally different states: non-rapid eye movement (NREM) sleep, which has three stages of progressively deeper unconsciousness, and rapid eye movement (REM sleep), the dreaming, near-paralysed state whose brain activity resembles waking (New Oxford Textbook). Across a night the body passes in sequence through the NREM stages and then into REM, and repeats. Arousal threshold rises with each successive NREM stage while eye movement and muscle activity fall, so the depth of sleep is genuinely graded, not a single flat plateau.

Why the two states are taught apart. NREM and REM are opposites on almost every axis: NREM has high muscle tone, slow rolling or absent eye movements and slow high-voltage EEG, whereas REM has muscle atonia, bursts of rapid eye movement and a fast low-voltage EEG. Recalled, elaborate dreaming belongs to REM sleep; the fragmentary thought of NREM is rarely remembered. This split is the backbone of the parasomnia distinction later, where the NREM arousal disorders (sleepwalking, night terrors) arise out of slow-wave sleep and nightmare disorder and REM sleep behaviour disorder arise out of REM.

11.2 The NREM stages: N1, N2 and N3

The three stages and their EEG signatures. The **sleep stages** of NREM are defined electrographically. **N1** is the drowsy transition from wake, with attenuation and slowing of the waking alpha (8 to 12 Hz) rhythm and slow rolling eye movements. **N2** is the defining light-sleep stage, marked by two signature graphoelements, the sleep spindles (brief 11 to 16 Hz sigma

bursts) and the K-complexes, both appearing at the onset of stage 2 (New Oxford Textbook; Kaufman). **N3** is slow-wave sleep, dominated by high-amplitude slow delta (0.5 to 2 Hz) activity; it is the deepest, most restorative NREM stage and is the physical-recuperation portion of the night. The stage labels N1, N2 and N3 are the current names; older Rechtschaffen-and-Kales scoring split slow-wave sleep into stages 3 and 4.

NREM STAGE	EEG SIGNATURE	EOG / EMG AND ROLE
N1 (light transition)	Attenuation/slowing of alpha; low-voltage mixed frequency	Slow rolling eyes; muscle tone preserved; the doorway into sleep
N2 (light sleep)	**Sleep spindles (sigma bursts) and K-complexes**	Eyes slow or still; the single largest share of the night
N3 (slow-wave sleep)	**High-amplitude delta, 0.5 to 2 Hz**	Eyes still; deepest, most restorative; slow-wave parasomnias arise here

The three NREM stages by EEG signature; the load-bearing pairing is spindles and K-complexes marking N2 and delta activity marking N3.

GOOD TO REMEMBER

- **NREM stages (the identifiers):** N1 is alpha dropout with rolling eyes; *N2 is defined by sleep spindles and K-complexes*; N3 is slow-wave (delta) sleep, the deepest and most restorative stage. Spindles and K-complexes are the N2 signature and the commonest single graphoelements asked at the console.

11.3 REM sleep: the paradoxical state

Why it is called paradoxical. In REM sleep the EEG becomes low-voltage and fast, closely resembling the waking record, yet the sleeper is behaviourally asleep and the postural muscles are actively paralysed, hence paradoxical sleep. Bursts of rapid conjugate eye movement give the state its name, autonomic activity becomes irregular, and vivid narrative dreaming occurs. The muscle atonia is generated actively in the pons (the perilocus coeruleus region drives the eye movements, atonia and dreaming), and its failure is the substrate of REM sleep behaviour disorder, a synucleinopathy prodrome developed in the Dementias and neurocognitive disorders notes and the Neurology foundations for psychiatrists notes.

What REM does on the montage. On **polysomnography**, REM is scored from the triad of a wake-like low-voltage fast EEG, phasic rapid eye movements on the EOG, and a silent chin EMG reflecting atonia (Kaufman). That last channel matters clinically: loss of the normal REM atonia (REM sleep without atonia) is the polysomnographic marker of REM sleep behaviour disorder.

11.4 Sleep architecture: the stage percentages and the normal REM latency

How the night divides. In a healthy young adult the night distributes across the stages in a stable pattern. Light N2 sleep takes the largest single share, slow-wave N3 a smaller block loaded into the first third of the night, and REM sleep accounts for a verified **20 to 25%** of total sleep time, its proportion rising with each successive cycle (New Oxford Textbook). N1 is a thin transitional sliver. These proportions are the reference against which a disorder is called abnormal, for

example the reduced slow-wave sleep and increased N1 of ageing, or the elevated REM percentage of rebound after deprivation.

The normal REM latency. After sleep onset the sleeper descends through NREM and reaches the first REM period after roughly ninety minutes; this interval is the **REM latency**, and it starts at about **90 minutes** and lasts about ten minutes on that first pass (Kaufman). A shortened REM latency is a high-yield abnormality: a sleep-onset REM period, REM appearing within about fifteen minutes of falling asleep, points to narcolepsy, and a reduced REM latency is a classic (if non-specific) finding in major depression, cross-referenced to the mood chapters.

STAGE	SHARE OF TOTAL SLEEP (%)	NOTE
N1 (light transition)	about 5	Thin sliver; increases with age and with fragmentation
N2 (light sleep)	**about 45 to 55**	Largest single share of the night
N3 (slow-wave sleep)	about 15 to 20	Front-loaded into the first third; falls with age
REM sleep	**20 to 25**	Verified; proportion rises across successive cycles

Adult sleep architecture; the REM share (20 to 25%) is directly source-verified, and the N1/N2/N3 split is the standard sleep-medicine approximation (see gaps).

RULE **The first REM period comes at about ninety minutes.** Normal REM latency is roughly 90 minutes; a sleep-onset REM period (REM within about fifteen minutes) is pathological and points to narcolepsy, and a short REM latency is a recognised depression marker.

11.5 The NREM-REM cycle and the hypnogram

The ninety-minute cycle. The two states alternate in a regular cycle of roughly ninety minutes (the first cycle is a little shorter, 70 to 100 minutes; later cycles run 90 to 120 minutes), and four to five such cycles complete a night (New Oxford Textbook; Kaufman). The internal balance of the cycle shifts as the night goes on: slow-wave N3 dominates the early cycles and fades, while REM periods lengthen and cluster into the last third of the night. This is why the vivid dream a person recalls is usually the final one, and why early-morning waking exposes the REM-rich end of sleep.

Reading the hypnogram. Plotting the scored stage against clock time produces the hypnogram, the conventional single-page map of a night's **sleep architecture** (the accompanying figure). It shows the descent into slow-wave sleep early, the cyclic climbs into REM about every ninety minutes, the shrinking of N3 and the lengthening of REM towards morning. The hypnogram is the visual grammar of the whole band: insomnia flattens and fragments it, apnoea peppers it with arousals, and narcolepsy plants REM at the very start.

GOOD TO REMEMBER

- **The cycle and hypnogram (the values):** NREM and REM alternate about every *90 minutes*, with 4 to 5 cycles per night; slow-wave sleep front-loads the night and REM back-loads it. The hypnogram is the stage-versus-time map read in every sleep disorder.

11.6 The two-process model of sleep regulation

Two drives, one gate. The timing of sleep is explained by the two-process model, which sets a homeostatic drive against a circadian one (Borbely; New Oxford Textbook). **Process S**, the homeostatic sleep pressure, builds steadily the longer one is awake and dissipates during sleep; its likeliest chemical substrate is adenosine, which accumulates in the brain across waking and whose blockade by caffeine is why coffee fights sleepiness. **Process C**, the circadian drive for wakefulness, is generated by the suprachiasmatic nucleus and rises through the day to oppose the mounting sleep pressure, then falls in the evening. The figure alongside plots the two curves.

The sleep gate. We stay awake through the day because Process C climbs in step with Process S and cancels it; sleep is triggered in the evening when the circadian alerting signal drops away and the accumulated homeostatic pressure is suddenly unopposed, opening the sleep gate. The model explains ordinary phenomena directly: the post-lunch dip, the second-wind of the evening alerting zone, and the deep rebound of slow-wave sleep after a night of deprivation, when Process S is abnormally high.

GOOD TO REMEMBER

- **Two-process model (the principle):** sleep timing = *Process S* (homeostatic pressure, building with time awake, adenosine-mediated) opposed by *Process C* (circadian alerting, driven by the suprachiasmatic nucleus). Sleep starts when the circadian alerting signal falls and unmasks accumulated homeostatic drive.

11.7 The ascending arousal system and the sleep-wake flip-flop switch

The wake side. Wakefulness is actively maintained by the **ascending arousal system**, a set of projections rising from the upper brainstem to the thalamus, hypothalamus and cortex (New Oxford Textbook). One arm is cholinergic (pedunculopontine and laterodorsal tegmental nuclei) driving the thalamus; the other is a monoaminergic and peptidergic fan, noradrenaline from the locus coeruleus, serotonin from the raphe, dopamine from the ventral periaqueductal grey, histamine from the tuberomammillary nucleus, and orexin from the lateral hypothalamus. These wake-promoting cells fire fastest in waking, slow in NREM and fall nearly silent in REM.

The sleep side and the switch. Sleep is imposed by the ventrolateral preoptic nucleus (VLPO), which inhibits the whole arousal system through GABA and galanin. Because the arousal system inhibits the VLPO in turn, the two sides are mutually antagonistic, and this reciprocal inhibition behaves as a bistable flip-flop switch (the sleep switch): the brain is pushed firmly into wake or firmly into sleep with fast transitions and little time in between (the accompanying figure). The stabiliser of this switch is orexin (also called hypocretin), the lateral-hypothalamic peptide that reinforces the wake side and prevents unwanted flips. Loss of the orexin neurons destabilises the

switch and is the lesion of narcolepsy with cataplexy, which is why hypocretin and the flip-flop model are examined together.

TRAP **Over-reading normal age-related change as insomnia.** Healthy ageing brings reduced slow-wave sleep, more N1, earlier waking and lighter, more fragmented sleep. Read against the young-adult template these look pathological, but they are normal architecture for the age, not a disorder needing a hypnotic.

GOOD TO REMEMBER

- **Flip-flop switch (the mechanism):** reciprocal inhibition between the *ascending arousal system* (wake) and the *VLPO* (sleep) creates a bistable switch with sharp transitions; *orexin/hypocretin* stabilises it. Orexin-neuron loss destabilises the switch and produces narcolepsy with cataplexy.

11.8 The neurotransmitters of wake and sleep

Wake-promoting versus sleep-promoting. The chemistry maps onto the switch. Wake is promoted by acetylcholine, noradrenaline, serotonin, dopamine, histamine and orexin; sleep is promoted by GABA and galanin from the VLPO, by adenosine as the homeostatic signal, and by melatonin as the circadian darkness cue. REM is special: acetylcholine is highly active in REM as in waking, while the monoamines (noradrenaline and serotonin) go nearly silent, the reciprocal-interaction pattern behind REM generation (Kaufman). This is why anticholinergic and serotonergic antidepressants suppress and delay REM, and why antihistamines cause drowsiness.

STATE	PROMOTING TRANSMITTERS
Wakefulness	Acetylcholine, noradrenaline, serotonin, dopamine, histamine, orexin
NREM sleep	**GABA and galanin (VLPO); adenosine (homeostatic); reduced monoamines**
REM sleep	**Acetylcholine high; noradrenaline and serotonin nearly silent**

Wake and sleep neurochemistry; the REM row (cholinergic on, aminergic off) is the reciprocal-interaction pattern and the reason REM-suppressing drugs are aminergic/anticholinergic.

MNEMONIC

WAKE = A HANDO

Acetylcholine, **H**istamine, **A**drenergic (noradrenaline), **N**ot-quite (serotonin/5-HT), **D**opamine, **O**rexin: the six wake-promoting transmitters of the ascending arousal system. The deep hypnotic and wake-promoting pharmacology (melatonin, ramelteon, the dual orexin receptor antagonists, the z-drugs and the benzodiazepines) is developed in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes.

11.9 Polysomnography: the montage and sleep staging

The three core channels. Polysomnography (the overnight sleep study) scores sleep from a montage whose three defining channels are the EEG (brain rhythm, for staging), the EOG or electro-oculogram (eye movement, to catch the rolling eyes of drowsiness and the rapid eye

movements of REM), and the chin EMG or electromyogram (muscle tone, which falls to silence in REM atonia) (Kaufman). Around this core sit the physiological channels: ECG, nasal and oral airflow, thoracic and abdominal respiratory effort, pulse oximetry, a snore microphone, and an anterior tibialis EMG for leg movements. Sleep is scored in thirty-second epochs, and the sequence of epochs becomes the hypnogram.

What it is for, and its relatives. Polysomnography is indicated where the diagnosis turns on physiology rather than history: breathing-related sleep disorders, periodic limb movement disorder, REM sleep behaviour disorder and suspected narcolepsy (DSM-5-TR). Its daytime companion is the multiple sleep latency test (MSLT), which measures how quickly a person falls asleep across a series of scheduled naps and whether REM appears in them; the MSLT is positive for narcolepsy at a mean sleep latency of eight minutes or less with sleep-onset REM periods in two or more naps (DSM-5-TR, verified). Routine insomnia does not need polysomnography and is diagnosed clinically.

GOOD TO REMEMBER

- **Polysomnography montage (the essentials):** the three core channels are *EEG, EOG and EMG* (brain, eyes, muscle), scored in 30-second epochs; airflow, effort and oximetry are added for breathing disorders. The daytime *MSLT* quantifies sleepiness and traps sleep-onset REM (positive: mean sleep latency of 8 minutes or less plus REM in 2 or more naps).

11.10 The derived indices: the AHI and the periodic-limb-movement index

The apnoea-hypopnea index. Two numbers scored off the study run through the rest of the band. The apnoea-hypopnea index (AHI) counts apnoeas plus hypopneas per hour of sleep and grades the severity of obstructive sleep apnoea; an event is a breathing reduction of at least ten seconds (DSM-5-TR). The diagnostic threshold is five or more obstructive events per hour with symptoms (or fifteen or more without), and severity is banded as mild, moderate or severe. In the verified DSM-5-TR specifier the bands are an AHI under fifteen (mild), fifteen to thirty (moderate) and over thirty (severe); the clinical sleep-medicine convention labels an AHI of five to fifteen as mild, so the exact lower band edge depends on the scheme used and is flagged below.

The periodic-limb-movement index. The periodic limb movement index (PLMI) counts periodic limb movements per hour of sleep and is the polysomnographic sign of periodic limb movement disorder, which overlaps heavily with restless legs syndrome (about forty per cent of narcolepsy patients also show it). Its abnormal threshold in adults is conventionally taken as more than fifteen movements per hour, but the precise cut-off is scheme-dependent and is flagged rather than fixed here.

SEVERITY BAND	AHI (EVENTS PER HOUR)
Diagnostic threshold (with symptoms)	5 or more
Mild	under 15 (clinical convention: 5 to 15)
Moderate	**15 to 30**
Severe	**over 30**

Obstructive sleep apnoea severity by AHI (DSM-5-TR specifier, verified); the mild-band lower edge differs between DSM-5-TR (under 15) and the clinical convention (5 to 15).

20 to 25	90	>30	8
REM SHARE OF THE NIGHT (% OF TOTAL SLEEP TIME)	NORMAL REM LATENCY AND NREM-REM CYCLE (MINUTES)	SEVERE OBSTRUCTIVE SLEEP APNOEA AHI (EVENTS PER HOUR)	MSLT POSITIVE MEAN SLEEP LATENCY (MINUTES OR LESS)

11.11 The Indian frame

Physiology in a real clinic. The examinable physiology is universal, but its use in Indian practice has two edges. Sleep disorders are under-recognised and under-investigated: a full **polysomnography** lab sits mostly in tertiary and metropolitan centres, so the front-line diagnosis of insomnia and of ordinary sleepiness leans on a careful history, a sleep diary and validated questionnaires rather than the study itself, and the study is reserved for the physiological questions (apnoea, narcolepsy, the movement disorders) that history cannot settle.

The two practice pitfalls. The first is the routine prescription of a benzodiazepine for chronic insomnia, still common in Indian practice, when the normal-physiology teaching (that chronic insomnia is a conditioned, hyperarousal problem, not a hypnotic-deficiency state) argues for behavioural first-line care, developed in the insomnia and CBT-I notes. The second is over-reading normal, age-related architecture (lighter, shorter, more fragmented sleep with less slow-wave sleep) as a disease in the older patient. Knowing the healthy template is what protects an older person from an unnecessary long-term hypnotic.

INDIA

Two habits are worth naming. Polysomnography is concentrated in tertiary and metro sleep labs, so most sleep complaints in India are worked up clinically first, with the study reserved for suspected apnoea, narcolepsy or a movement disorder; and the reflex of a benzodiazepine for every sleepless night is over-used, when the physiology says chronic insomnia is a hyperarousal problem better met with sleep-hygiene and behavioural care. Good sleep hygiene, a regular rise time, light exposure, and cutting the late-evening screen and caffeine, is itself an application of the two-process model, strengthening the circadian signal and clearing homeostatic pressure honestly rather than pharmacologically.

HOLD THIS

Normal adult sleep cycles NREM to REM about every **90 minutes** across 4 to 5 cycles, with REM at **20 to 25%** of the night and a first REM period (normal REM latency) at about **90 minutes**; N2 is defined by spindles and K-complexes, N3 by delta, and REM by a wake-like EEG with atonia. Sleep is timed by the two-process model (homeostatic Process S versus circadian Process C) and switched by the arousal-system-versus-VLPO flip-flop that orexin stabilises, and it is measured by polysomnography (EEG, EOG, EMG) whose AHI grades apnoea (severe over 30 events per hour).

12 · Insomnia disorder: assessment and pharmacological management

Insomnia disorder is dissatisfaction with sleep quantity or quality, with difficulty initiating sleep, difficulty maintaining sleep or early-morning waking, that persists despite an adequate

opportunity to sleep and carries daytime consequences. It is the commonest sleep complaint the psychiatrist meets, and the examinable heart of it is a hybrid: first you must recognise insomnia disorder as a diagnosis in its own right and separate it cleanly from the circadian, breathing and movement disorders that mimic it, and then you must know that its treatment is led not by a tablet but by a psychological therapy.

Why it matters, and how this note is framed. Two anchors carry the topic. First, the modern nosology treats insomnia as an independent, treatable condition rather than a symptom secondary to something else, split into a **chronic insomnia** form (three months or longer) and a short-term form, and screened with the seven-item Insomnia Severity Index. Second, the whole of **pharmacological management of insomnia** sits downstream of one rule, that cognitive behavioural therapy for insomnia is first-line for chronic insomnia and a **hypnotic** is not. This is a management topic, so the drug half is built from the verified guideline rows for insomnia, leading with the British Association for Psychopharmacology position (BAP 2019) and then the Indian, NICE and VA/DoD guidance; every first-line named there is verified and is not contradicted here, and any dose or cut-off the parsed library could not confirm is flagged rather than invented.

INSOMNIA SEVERITY INDEX (TYPESET, NOT REPRODUCED)	VERIFIED STRUCTURE AND SEVERITY BANDS
Instrument	The Insomnia Severity Index, a brief self-report measure of perceived insomnia severity over the last two weeks (Morin, 1993), widely used to screen and to track response
Items	7 items, each scored 0 to 4, total range 0 to 28: the severity of sleep-onset difficulty, of sleep-maintenance difficulty and of early-morning waking; satisfaction with the current sleep pattern; interference with daytime functioning; how noticeable the impairment is to others; and the level of worry or distress about the sleep problem
Severity bands	**0 to 7 no clinically significant insomnia; 8 to 14 subthreshold insomnia; 15 to 21 clinical insomnia, moderate; 22 to 28 clinical insomnia, severe**
Use	a change of about 6 or more points marks a clinically meaningful response; it complements, and does not replace, the sleep history and the sleep diary

Insomnia Severity Index: typeset structure and verified bands; the form is copyright and its distribution is permission-sensitive, so it is presented here in typeset form rather than reproduced (Morin CM. Insomnia: Psychological Assessment and Management. New York: Guilford Press; 1993). For the free, non-commercial resident edition only.

Fig 17.10 The Insomnia Severity Index (ISI). A brief seven-item self-report of how severe the insomnia has been over the last two weeks: the severity of getting to sleep, staying asleep and waking too early, satisfaction with sleep, the daytime interference, how noticeable it is to others, and the distress it causes, each rated 0 to 4 for a total of 0 to 28. The bands run 0 to 7 no clinically significant insomnia, 8 to 14 subthreshold, 15 to 21 moderate clinical insomnia and 22 to 28 severe, and a fall of about six points or more marks a meaningful response to treatment. It is a screen and an outcome measure, not a substitute for the sleep history and the sleep diary.

12.1 What insomnia disorder is: the definition, and the shift to a diagnosis in its own right

The definition. The primary element is *dissatisfaction with sleep quantity or quality*, accompanied by at least one of difficulty initiating sleep, difficulty maintaining sleep (frequent awakenings or trouble returning to sleep) or early-morning waking with inability to return to sleep, occurring despite an adequate opportunity and adequate circumstances for sleep, and producing daytime consequences (fatigue, impaired attention or mood disturbance) (Kaplan and Sadock CTP 2024). The clause that adequate opportunity exists is what separates the disorder from simple sleep deprivation: the person is in bed with the chance to sleep and still cannot.

The paradigm shift. Where older systems called insomnia a symptom secondary to some underlying illness and told the clinician to treat only the cause, current thinking treats it as a possible independent condition and reframes the older secondary insomnias as *comorbid*, to be managed in their own right; nonrestorative sleep has moved out to hypersomnolence (Kaplan

and Sadock CTP 2024). The exam point is that treating the depression does not reliably fix the insomnia, so insomnia is targeted directly.

> **PEARL**
>
> Insomnia is no longer a secondary symptom to be ignored until its cause is fixed. It is a diagnosis in its own right and is treated directly, in parallel with any comorbid depression, anxiety or pain, not after it.

12.2 The DSM-5-TR and ICD-11 criteria, and the chronic-versus-short-term split

The eight criteria. DSM-5-TR evaluates insomnia against eight points: how it harms sleep, how it affects the following wakefulness, its frequency, its chronicity, whether it coexists with another sleep disorder, with medication or substance use, or with another psychiatric, neurological or medical condition, and, critically, that the difficulty occurs notwithstanding an adequate opportunity to sleep (Kaplan and Sadock CTP 2024). The two quantitative thresholds are the ones examiners test: the disturbance must occur at least three nights per week and be present for at least three months.

The chronic-versus-short-term distinction. All three systems now converge on the same divide. ICD-11, which moves the sleep-wake disorders out of the mental-disorders chapter into a dedicated sleep-wake chapter, separates chronic insomnia disorder (symptoms three or more nights per week for three months or longer) from short-term insomnia disorder (the same picture lasting under three months, usually with an identifiable stressor). The AASM classification (ICSD-3) draws the identical chronic-insomnia-disorder versus short-term-insomnia-disorder line (Kaplan and Sadock CTP 2024). The threshold matters because it decides the treatment lever: short-term insomnia is where a brief hypnotic has its legitimate place, whereas chronic insomnia is where the psychological therapy leads.

CRITERION	REQUIREMENT FOR INSOMNIA DISORDER
Core complaint	**Dissatisfaction with sleep quantity or quality**
Symptom (one or more)	Difficulty initiating sleep; difficulty maintaining sleep; early-morning waking with inability to return to sleep
Daytime consequence	Clinically significant distress or impairment (fatigue, attention, mood, function)
Frequency	**At least three nights per week**
Duration (chronic)	**Present for at least three months (short-term insomnia if under three months)**
Adequate opportunity	Occurs despite adequate opportunity and circumstances for sleep
Not better explained by	Another sleep-wake disorder, a substance, or a coexisting mental or medical disorder that fully accounts for it

DSM-5-TR insomnia disorder criteria; the load-bearing numbers are the frequency (three or more nights per week) and the duration (three months) that define the chronic form.

GOOD TO REMEMBER

- **Insomnia disorder (defining criterion):** dissatisfaction with sleep quantity or quality with difficulty initiating or maintaining sleep or early-morning waking, *despite adequate opportunity to sleep* and with daytime consequences; the diagnostic thresholds are at least three nights per week for at least three months, and that three-month mark is what separates chronic insomnia (psychological therapy leads) from short-term insomnia (a brief hypnotic has a place).

12.3 Assessment: the sleep history and the sleep diary

The sleep history. A sleep history should be a short but routine part of every psychiatric consultation, because a limited number of things go wrong with sleep and most sleep disorders can be diagnosed by clinical history alone (New Oxford Textbook of Psychiatry). The complaint reduces to sleeping too little, sleeping too much, sleeping at the wrong time of day, or things that go bump in the night, and a collateral history from the bed partner is often what reveals the snoring, the kicking or the abnormal nocturnal behaviour the patient cannot report. The history maps the day in full: the bedtime routine and environment, sleep-onset latency, awakenings and their causes, final waking time, daytime napping and function, and the substances, caffeine, alcohol, nicotine and stimulant medications, that erode sleep.

The sleep diary. Where the history is ambiguous, a prospective **sleep** diary kept over one to two weeks records bed and rise times, estimated latency, awakenings and daytime naps, exposing the mismatch between perceived and actual sleep and revealing a circadian pattern the single consultation misses; actigraphy extends this objectively where needed. Polysomnography is *not* routine for uncomplicated insomnia and is reserved for suspected sleep apnoea, narcolepsy or a periodic-limb-movement disorder (Kaplan and Sadock CTP 2024).

GOOD TO REMEMBER

- **Assessment (the first step):** take a routine *sleep history* (with a collateral account from the bed partner) and, where unclear, a one-to-two-week prospective sleep diary; most insomnia is diagnosed on history alone, and polysomnography is not routine, reserved for suspected obstructive sleep apnoea, narcolepsy or periodic limb movements.

12.4 The differential: the sleep-wake disorders that mimic insomnia

What must be excluded. Before insomnia disorder is settled, the sleep-wake mimics are cleared. A *circadian rhythm sleep-wake disorder* (delayed or advanced sleep phase, shift-work or jet-lag type) presents as insomnia only because the person is trying to sleep at the wrong biological time, and the tell is that sleep is normal when timed to the patient's own preferred schedule. A *breathing-related sleep disorder*, chiefly obstructive sleep apnoea, presents with un-refreshing sleep, snoring and witnessed apnoeas, and is screened with STOP-BANG. A *movement disorder*, restless legs syndrome or periodic limb movement disorder, delays or fragments sleep through an urge to move or repetitive kicking. Each is developed in its own note; the point here is that a hypnotic prescribed for what is actually apnoea or a phase disorder is both useless and, in apnoea, dangerous.

The comorbid causes. Insomnia is driven by comorbid psychiatric illness (depression, anxiety, mania, post-traumatic stress and substance use) and by medical disease (chronic pain, nocturia, cardiorespiratory disease, and endocrine disorders such as thyroid dysfunction). These are managed in parallel with the insomnia, not instead of it. The psychiatric relevance of these medical sleep disturbances is developed in the Somatic-symptom, sexual, impulse-control disorders and suicide notes and the Consultation-liaison, geriatric and transcultural psychiatry notes.

MIMIC OR DRIVER	THE DISCRIMINATING CLUE
Circadian rhythm sleep-wake disorder	**Sleep is normal in quantity and quality when allowed at the patient's own preferred timing**
Obstructive sleep apnoea (breathing-related)	Snoring, witnessed apnoeas, un-refreshing sleep, daytime sleepiness; screen with STOP-BANG
Restless legs / periodic limb movement	Evening urge to move the legs, relieved by movement; repetitive nocturnal kicking on collateral history
Comorbid psychiatric (depression, anxiety, mania)	**Insomnia tracks the mood or anxiety syndrome but is treated in its own right, not deferred**
Medical and substance causes	Pain, nocturia, cardiorespiratory or thyroid disease; caffeine, alcohol, nicotine, stimulants

The insomnia differential; the circadian and breathing-related disorders are the two that a hypnotic will fail, so both must be excluded before treating for insomnia disorder.

MNEMONIC

CIRCADIAN, BREATHE, MOVE, MOOD, MED

Before diagnosing insomnia disorder, clear the mimics: **Circadian** rhythm disorder (wrong-time sleep), **Breathe** (obstructive sleep apnoea), **Move** (restless legs / periodic limb movement), **Mood** (depression, anxiety, mania), and **Med**ical or substance causes. The five buckets a sleep history must exclude.

12.5 The Insomnia Severity Index and its severity bands

What the ISI is. The Insomnia Severity Index (ISI) is a seven-item self-report tool that captures the patient's own perception of their insomnia, its symptoms, its daytime effects, and how distressed or concerned they are by it. Each item is scored from zero to four, giving a maximum of twenty-eight, and a score of **10 or higher** identifies insomnia with high sensitivity and specificity as a screening threshold (Kaplan and Sadock CTP 2024). The VA/DoD guidance names the ISI (or the Athens Insomnia Scale) as the validated instrument to use in anyone presenting with a sleep complaint (VA/DoD 2025). This topic carries the ISI form on the accompanying scale plate; the daytime-sleepiness counterpart, the Epworth Sleepiness Scale, belongs to the hypersomnolence and narcolepsy assessment and is cross-referenced there.

The severity bands. The total is banded into four severity strata, verified against the max-of-twenty-eight scoring: 0 to 7 no clinically significant insomnia, 8 to 14 subthreshold insomnia, **15**

to 21 clinical insomnia of moderate severity, and 22 to 28 clinical insomnia, severe. These bands are what let the ISI double as an outcome measure: a fall of several points across treatment is the standard marker of response.

ISI TOTAL (SCORE)	SEVERITY BAND
0 to 7	No clinically significant insomnia
8 to 14	Subthreshold insomnia
15 to 21	**Clinical insomnia, moderate severity**
22 to 28	**Clinical insomnia, severe**

ISI severity bands (seven items, zero to four each, maximum twenty-eight); the screening cut-off of ten or higher and the moderate band of fifteen to twenty-one are the two most examinable numbers.

≥10 ISI SCREENING CUT-OFF FOR INSOMNIA (SCORE)	**15 to 21** ISI MODERATE CLINICAL INSOMNIA BAND (SCORE)	**3** CHRONIC INSOMNIA DURATION THRESHOLD (MONTHS)	**1 to 6** LOW-DOSE DOXEPIN FOR INSOMNIA (MG PER DAY)

GOOD TO REMEMBER

- **Insomnia Severity Index (the value):** seven items, each scored zero to four, maximum twenty-eight; a score of ten or higher screens positive with high sensitivity and specificity, and the clinical bands are 15 to 21 (moderate) and 22 to 28 (severe), with 0 to 7 no significant insomnia and 8 to 14 subthreshold; it doubles as the outcome measure across treatment.

12.6 The management rule: CBT-I is first-line for chronic insomnia, a hypnotic is not

The one rule. For **chronic insomnia**, cognitive behavioural therapy for insomnia (CBT-I), delivered as a multicomponent package that includes sleep restriction and stimulus control, is the first-line treatment, offered before drug treatment (BAP 2019; NICE 2023). BAP notes that face-to-face and digital CBT-I are both efficacious, so a shortage of therapists is not a reason to default straight to a tablet. The Indian Psychiatric Society CPG makes CBT-I the mainstay of chronic insomnia, delivered as an individualised package of sleep education, sleep hygiene, cognitive restructuring, stimulus control, sleep restriction and relaxation (IPS 2017), and VA/DoD explicitly prefers CBT-I over pharmacotherapy as first-line (VA/DoD 2025). The component detail of CBT-I is carried in its own note; here it is the anchor that pharmacology hangs off.

Where the drug fits. Pharmacology enters only where CBT-I fails, is unavailable, or the patient cannot engage with it, and even then it is short-term for chronic insomnia rather than an indefinite prescription (BAP 2019; IPS 2017). A crucial negative: BAP and VA/DoD both say antipsychotics such as quetiapine and olanzapine are *not* to be used as first-line for insomnia, and VA/DoD advises against benzodiazepines, diphenhydramine and trazodone for chronic insomnia.

RULE **CBT-I first, the tablet second.** Cognitive behavioural therapy for insomnia (with sleep restriction and stimulus control) is first-line for chronic insomnia; a hypnotic is not first-line and is a short-term measure for when CBT-I fails, is unavailable, or cannot be engaged with.

GOOD TO REMEMBER

- **First-line management (the principle):** CBT-I is first-line for chronic insomnia and is offered before any drug (BAP 2019, IPS 2017, NICE 2023, VA/DoD 2025); a hypnotic is second-line and short-term, and antipsychotics (quetiapine, olanzapine) are explicitly not first-line for insomnia.

12.7 Pharmacological management: the z-drugs and matching half-life to the pattern

When a hypnotic is used. When pharmacology is indicated, the workhorse short-term agents are the GABA positive allosteric modulators, the benzodiazepines and the z-drug hypnotics (zolpidem, zopiclone, eszopiclone, zaleplon) (BAP 2019). BAP frames the choice by kinetics: pick a short-acting agent for sleep-onset insomnia and a slightly longer-acting one for sleep-maintenance insomnia. NICE keeps this pragmatic, noting no compelling efficacy difference between zolpidem, zopiclone and the shorter-acting benzodiazepines, so the lowest-cost agent is chosen, prescribed short-term and strictly within the licensed indication, and a second one is not tried if the first has failed (NICE 2004).

Match the drug to the problem. The **pharmacological management of insomnia** is therefore a kinetics exercise: a short half-life for the patient who cannot fall asleep, a slightly longer one for the patient who wakes and cannot return. The deep hypnotic pharmacology, the mechanism at the GABA-A receptor, the half-life classification, the dependence liability and the taper, is developed in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes.

WORKED EXAMPLE

A patient who lies awake for two hours at the start of the night but sleeps through once asleep has sleep-onset insomnia, so a short-acting agent (for example zolpidem, sold in India as Zolfresh, or melatonin) fits. A patient who falls asleep readily but wakes at three in the morning and cannot return has sleep-maintenance insomnia, so a slightly longer-acting agent (for example zopiclone, or very low-dose doxepin for the late-night awakening) fits. Match the half-life to the pattern.

AGENT OR CLASS	PLACE IN INSOMNIA MANAGEMENT	GUIDELINE ANCHOR
CBT-I (with sleep restriction, stimulus control)	**First-line for chronic insomnia, before any drug**	BAP 2019; IPS 2017; NICE 2023; VA/DoD 2025
Z-drugs (zolpidem, zopiclone, eszopiclone, zaleplon)	**Short-term hypnotic; match half-life to onset vs maintenance**	BAP 2019; NICE 2004
Benzodiazepines (GABA-PAM)	Short-term only; avoided for chronic insomnia (dependence)	BAP 2019; VA/DoD 2025 (against, chronic)
Prolonged-release melatonin	Consider in adults over 55 for onset latency and quality	BAP 2019; IPS 2017
Low-dose doxepin (1 to 6 mg per day)	Selective antihistamine hypnotic, useful for late-night awakenings	BAP 2019

AGENT OR CLASS	PLACE IN INSOMNIA MANAGEMENT	GUIDELINE ANCHOR
Orexin antagonists (daridorexant, suvorexant, lemborexant)	**For long-term insomnia only after CBT-I fails or is unavailable**	NICE 2023; VA/DoD 2025
Sedating antidepressants (trazodone, mirtazapine)	Only when a comorbid mood disorder warrants; weak insomnia evidence	BAP 2019; IPS 2017
Antipsychotics (quetiapine, olanzapine)	Not first-line for insomnia; avoided	BAP 2019; VA/DoD 2025 (against)

The pharmacological ladder for insomnia; CBT-I sits above every drug, the z-drugs are the short-term workhorse matched by half-life, and antipsychotics are explicitly not first-line.

GOOD TO REMEMBER

- **Z-drug hypnotics (the value):** zolpidem, zopiclone, eszopiclone and zaleplon are short-term agents chosen by kinetics, short-acting for sleep-onset insomnia and slightly longer-acting for sleep-maintenance insomnia (BAP 2019); NICE finds no compelling efficacy difference, so pick the lowest-cost agent, prescribe short-term within licence, and do not try a second z-drug if the first has failed (NICE 2004).

12.8 Melatonin, the older adult, and the newer agents

Melatonin and the over-55 patient. Melatonin (in India, Meloset) is a weak hypnotic in its own right but a useful chronobiotic and sleep-promoter, and its licensed niche is the older adult: BAP and IPS both recommend prolonged-release melatonin in patients over 55 years to improve sleep-onset latency and sleep quality, at the licensed 2 mg prolonged-release dose taken before bedtime (BAP 2019; IPS 2017). Its favourable safety and absence of dependence make it the preferred first drug in the elderly, where z-drugs carry falls and confusion risk. The melatonin-receptor agonists ramelteon and agomelatine sit alongside it as chronobiotic options (IPS 2017).

The newer agents. The dual orexin receptor antagonists (daridorexant, suvorexant, lemborexant) block the wake-promoting orexin system and are positioned for long-term insomnia, but only after CBT-I has failed, is unsuitable or is unavailable; if daridorexant does not help enough it is stopped after three months, and if continued it is reviewed regularly (NICE 2023; VA/DoD 2025). Very low-dose doxepin (1 to 6 mg per day), a selective H1 antihistamine, is effective particularly for late-night awakenings (BAP 2019). The receptor pharmacology of melatonin, ramelteon, the orexin antagonists and the z-drugs is carried in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes, and the sleep neurotransmitter and ascending-arousal systems in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes and the Functional Neuroanatomy and Neurophysiology notes.

GOOD TO REMEMBER

- **Melatonin (the value):** a weak hypnotic but a useful chronobiotic; its licensed place is prolonged-release melatonin (2 mg) in the adult over 55 to improve sleep-onset latency and quality (BAP 2019, IPS 2017), preferred over a z-drug in the elderly for its safety and lack of dependence; the orexin antagonists (daridorexant) are reserved for long-term insomnia after CBT-I fails.

12.9 Discontinuation, the benzodiazepine trap, and the Indian frame

The taper. Hypnotics are used only as long as clinically indicated, and when they are stopped they are tapered slowly with concurrent CBT-I to improve the outcome and reduce rebound insomnia (BAP 2019). Because the z-drug and benzodiazepine dependence and the withdrawal taper are the deep story, the mechanism and the graded reduction are cross-referenced to the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes. The single most consequential caution stays here: do not start what you cannot stop, and do not run a hypnotic long-term for chronic insomnia when CBT-I is the treatment.

Sleep hygiene is not enough on its own. Sleep hygiene education is a component of CBT-I, but as a stand-alone treatment it is not effective for chronic insomnia and should not be offered alone (VA/DoD 2025). This is the quiet trap of the busy clinic: handing over a sleep-hygiene leaflet and calling it treatment.

TRAP **A long-term hypnotic (or a lone sleep-hygiene leaflet) instead of CBT-I.** Starting an indefinite z-drug or benzodiazepine for chronic insomnia, or offering sleep hygiene as a stand-alone, both substitute for the first-line treatment; CBT-I is what changes the trajectory, and the hypnotic is short-term only.

INDIA

The genuine Indian device here is *benzodiazepine over-use for insomnia*. With CBT-I therapists scarce and hypnotics cheap and easily obtained, long-term benzodiazepine and z-drug prescribing for chronic insomnia is widespread, and clonazepam and alprazolam are routinely used as sleeping tablets and drift into dependence. The corrective is not exotic: treat chronic insomnia with CBT-I as first-line (face-to-face or digital, both work), keep any hypnotic short-term and within licence, prefer prolonged-release melatonin in the older adult, and do not offer a sleep-hygiene leaflet as if it were the whole treatment. Correct sleep-hygiene practice is a component of CBT-I, not a substitute for it.

HOLD THIS

Insomnia disorder is dissatisfaction with sleep despite adequate opportunity, at least three nights per week for three months for the chronic form; screen with the ISI (positive at ten or higher, moderate band 15 to 21), clear the circadian, breathing and movement mimics, and treat chronic insomnia with CBT-I first-line, keeping z-drugs and other hypnotics short-term, kinetics-matched, and tapered, with prolonged-release melatonin for the over-55s and antipsychotics off the first line.

13 · Cognitive behaviour therapy for insomnia (CBT-I)

Cognitive behaviour therapy for insomnia is a structured, multicomponent psychological treatment that re-teaches the sleep system what the bed is for, compresses the night down to real sleep, and dismantles the worry that keeps the patient awake. It is the single most examinable fact in the whole sleep-treatment map, because the modern guidelines have moved the first move in chronic insomnia off the prescription pad and onto this therapy: CBT-I is first-line for chronic insomnia and a hypnotic is not.

Why it matters, and how this note is framed. This is a management topic, so the plan is built from the verified insomnia guideline rows, led by the British Association for Psychopharmacology position (BAP 2019) and then the Indian, NICE and VA/DoD guidance; every first-line named there is verified and is not contradicted here. The examinable spine is that CBT-I is a *package* of five components, that its two active behavioural ingredients are **sleep restriction** and **stimulus control**, that **sleep hygiene** is taught as necessary but not sufficient, and that the whole thing outlasts a tablet. The wider CBT technique and the general psychotherapy frame are developed in the Psychotherapies, clinical assessment, MSE and nosology notes; the deep hypnotic pharmacology that sits downstream of a CBT-I failure is carried in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes. The CBT-I components map is set out in the accompanying figure.

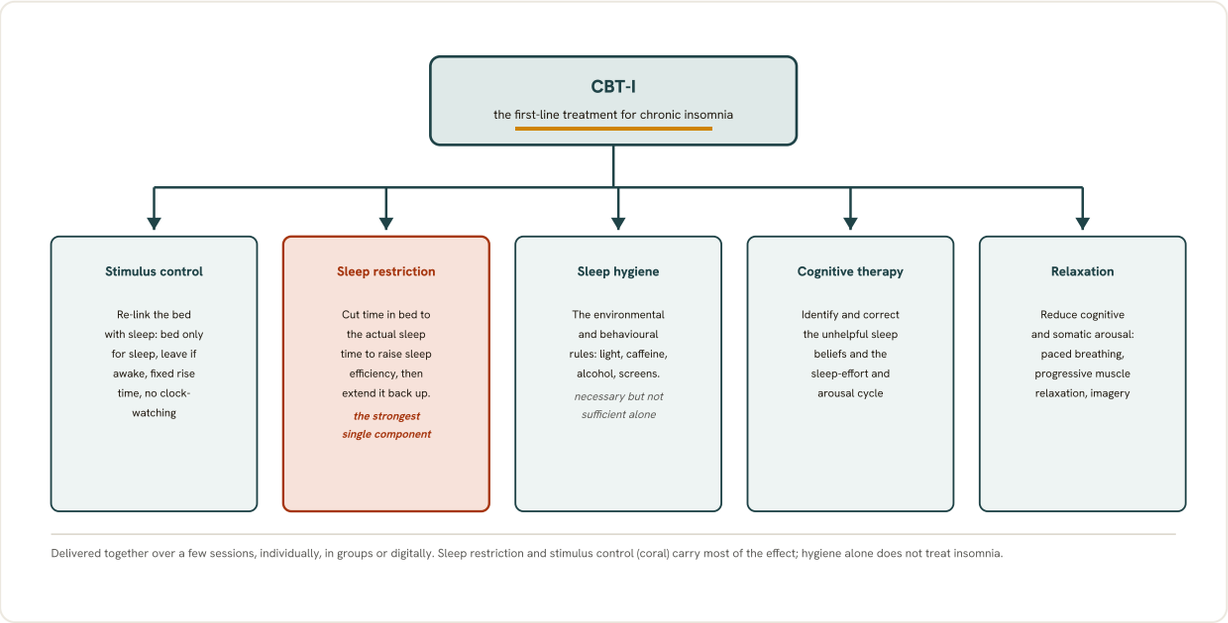


Fig 17.11 The components of cognitive behaviour therapy for insomnia. CBT-I, not a hypnotic, is the first-line treatment for chronic insomnia, and it is a package of five components. Stimulus control re-associates the bed with sleep; sleep restriction, the single most powerful element, curtails time in bed to the actual sleep time so that sleep consolidates and efficiency rises, then extends it back up; sleep hygiene sets the environmental and behavioural conditions but does not by itself treat insomnia; cognitive therapy corrects the unhelpful sleep beliefs and the effort-and-arousal cycle that keep a poor sleeper awake; and relaxation lowers the cognitive and somatic arousal. The wider cognitive behaviour therapy technique is developed in the Psychotherapies, clinical assessment, MSE and nosology notes.

13.1 What CBT-I is: a multicomponent package, not a single technique

The package. CBT-I begins with a careful clinical interview that maps the insomnia's aetiology, chronicity, severity and comorbidity, after which a treatment plan is assembled from relevant components: universal sleep hygiene, stimulus control therapy, sleep restriction therapy, relaxation therapies and biofeedback, cognitive therapy, and occasionally paradoxical intention (Kaplan and Sadock CTP 2024). It is delivered as an individualised multicomponent programme of sleep education, sleep hygiene, cognitive restructuring, stimulus control, sleep restriction and relaxation (IPS 2017). The point examiners test is that no single element is the treatment; the package works because it addresses the several variables that jointly perpetuate insomnia.

The two engines and the three supports. Not all components carry equal weight. The two behavioural interventions with the strongest independent evidence, and the two BAP names explicitly when it defines CBT-I, are sleep restriction and stimulus control (BAP 2019). Sleep hygiene, cognitive therapy and relaxation are the supporting components; sleep hygiene in particular is not a treatment on its own. Holding that hierarchy is what separates a real CBT-I referral from handing over a sleep-hygiene leaflet.

MNEMONIC

2 ENGINES + 3 SUPPORTS

The two behavioural **engines** of CBT-I are **sleep restriction** and **stimulus control** (the components BAP names when it defines the therapy). The three **supports** are **sleep hygiene**, **cognitive therapy** and **relaxation**. Five components, one package; the engines drive it, the supports steady it.

13.2 Stimulus control: re-associate the bed with sleep

The idea. Stimulus control therapy, a deconditioning paradigm developed by Richard Bootzin at the University of Arizona, aims to break the learned link between the bed and wakeful frustration and to rebuild the association between going to bed and falling asleep quickly (Kaplan and Sadock CTP 2024). The chronic insomniac has, in effect, conditioned the bedroom into a cue for lying awake; stimulus control undoes that conditioning through a small set of rules that must be followed consistently.

The rules. Go to bed only when sleepy. Use the bed for sleep only, so no television, reading, eating or phone in bed. If sleep does not come after a short while, get up (without watching the clock), go to another room, do something non-arousing, and return only when sleepy again, repeating this as often as needed. Rise at the same fixed time every morning regardless of how the night went, and do not nap. Results may take a few weeks to a month to appear, so the patient is warned not to abandon the method early.

GOOD TO REMEMBER

- **Stimulus control (the principle):** re-associate the bed with rapid sleep onset by four rules, bed only when sleepy, bed for sleep only, out of bed to another room if not asleep (return when sleepy, do not clock-watch), and a fixed rise time every morning with no napping; the effect builds over a few weeks, not overnight (Bootzin; BAP 2019).

13.3 Sleep restriction: curtail time in bed to consolidate sleep, then titrate up

The idea. Sleep restriction therapy, developed by Arthur Spielman, raises sleep efficiency, the proportion of time in bed actually spent asleep, by cutting back the time the patient lies in bed to match the time they truly sleep (Kaplan and Sadock CTP 2024). Compressing time in bed maximises homeostatic sleep drive and consolidates fragmented sleep; the mild sleep deprivation it induces is the therapeutic lever.

The titration. If a patient sleeps only about five hours of a scheduled eight in bed, time in bed is reduced towards the sleep time, but never set below roughly **four to five hours** per night, and the

patient is warned about the daytime sleepiness this deliberately provokes. Daytime sleep is avoided, though an older adult may take a short nap. Sleep efficiency is then monitored, and when it reaches **85%** averaged over **five nights**, time in bed is lengthened by **15 minutes**; the schedule is stepped up in this way until adequate sleep is reached, producing a gradual, steady fall in nocturnal wakefulness.

WORKED EXAMPLE

A patient records eight hours in bed but only about five asleep, a sleep efficiency near sixty-two per cent. Time in bed is set to around five and a half hours (not below the four-to-five-hour floor) with a fixed rise time. Over the following week the sleep the patient does get becomes solid and continuous; once efficiency averages 85% or more across five nights, time in bed is extended by fifteen minutes, and the process repeats until the patient is sleeping enough with little time awake in bed.

GOOD TO REMEMBER

- **Sleep restriction (the value):** curtail time in bed to the actual sleep time to raise sleep efficiency (time asleep as a proportion of time in bed), holding a floor of about four to five hours; when efficiency reaches 85% over five nights, add 15 minutes to time in bed and titrate up (Spielman; BAP 2019).

13.4 Sleep hygiene: the rules, taught as necessary but not sufficient

What it is. Sleep hygiene education targets the modifiable environmental and behavioural factors that erode sleep: a regular schedule, avoiding caffeine, nicotine and alcohol near bedtime, limiting daytime napping, keeping the bedroom dark, quiet and cool, and not exercising or eating heavily too close to sleep (Kaplan and Sadock CTP 2024). Because these are entrenched lifestyle habits, only one or two items are tackled at a time so the patient is not overwhelmed.

Why it is not enough on its own. The load-bearing exam point is that sleep hygiene education alone does not appear to improve chronic insomnia, and VA/DoD explicitly advises against using it as a stand-alone treatment for chronic insomnia disorder (VA/DoD 2025). It is a genuine component of the package and a reasonable universal measure, but calling a sleep-hygiene handout "treatment" is the quiet error of the busy clinic.

TRAP **Sleep hygiene is not CBT-I.** Handing over a sleep-hygiene leaflet and calling it done is not treatment; sleep hygiene alone does not improve chronic insomnia and must sit inside the full multicomponent package (VA/DoD 2025).

GOOD TO REMEMBER

- **Sleep hygiene (the caution):** the environmental and behavioural rules (regular schedule, no caffeine/nicotine/alcohol near bedtime, limit naps, cool dark quiet room, tackle one or two habits at a time) are necessary but not sufficient, and are not effective as a stand-alone treatment for chronic insomnia (VA/DoD 2025).

13.5 Cognitive therapy for insomnia: correct the beliefs and the sleep-effort cycle

What it targets. Cognitive therapy, adapted for insomnia (the cognitive therapy insomnia component of the package), targets the negative emotional response to an appraisal of the sleep situation, because that emotional arousal is what perpetuates the insomnia (Kaplan and Sadock CTP 2024). Patients hold maladaptive sleep-related beliefs, catastrophising ("there must be something wrong with me if I cannot fall asleep in forty minutes") and unrealistic expectations ("if I do not get eight hours the day is ruined"), which feed a self-defeating *sleep-effort and hyperarousal cycle*: the harder they try to sleep, the more aroused and awake they become.

How it works. The method is the standard cognitive sequence applied to sleep: first identify the dysfunctional sleep-related cognitions, then challenge their validity, then substitute more adaptive and realistic thoughts, which lowers the arousal that was keeping the patient awake. This dovetails with paradoxical intention, where the patient is asked to stop trying to sleep, removing the very sleep effort that fuels the arousal. The wider cognitive technique is developed in the Psychotherapies, clinical assessment, MSE and nosology notes.

GOOD TO REMEMBER

- **Cognitive therapy for insomnia (the principle):** identify, challenge and substitute the unhelpful sleep-related beliefs (catastrophising, rigid eight-hour expectations) that drive the sleep-effort and hyperarousal cycle; lowering that arousal is the mechanism, and paradoxical intention (stop trying to sleep) works the same lever (Kaplan and Sadock CTP 2024).

13.6 Relaxation: lower the somatic and cognitive arousal at bedtime

The techniques. Relaxation methods reduce the physiological and mental arousal that blocks sleep onset: progressive muscle relaxation (deliberately tensing a muscle group for a few seconds then releasing it for longer, working from head to feet, and noticing the contrast), guided imagery of a restful scene, slow abdominal breathing, self-hypnosis, and biofeedback of a physiological marker such as forehead muscle tension (Kaplan and Sadock CTP 2024). Progressive muscle relaxation suits the patient whose insomnia is driven by bodily tension.

How they are used. The crucial teaching point is that a relaxation technique must be over-learned and mastered in the daytime, away from bed, over several weeks before it is applied at bedtime, so that by the time it is used to fall asleep the skill is automatic and does not itself become associated with the failure to sleep. Relaxation combines readily with sleep hygiene and stimulus control and doubles as a distraction from the ruminations that fuel insomnia.

GOOD TO REMEMBER

- **Relaxation (the value):** progressive muscle relaxation, guided imagery, abdominal breathing and biofeedback lower the arousal that blocks sleep onset; they must be over-learned in the daytime, away from the bed, before being applied at bedtime so the skill is automatic and never linked to sleeplessness (Kaplan and Sadock CTP 2024).

13.7 The five components at a glance

The map. The table below sets the five components against what each one does and its core instruction; the two engines are marked, and it mirrors the CBT-I components map in the

accompanying figure. Paradoxical intention is added as the occasional sixth technique that shares the cognitive component's target.

COMPONENT	WHAT IT DOES	THE CORE INSTRUCTION
Stimulus control (engine)	**Re-associates the bed with rapid sleep onset (deconditioning)**	Bed only when sleepy; bed for sleep only; out of bed if awake; fixed rise time; no naps
Sleep restriction (engine)	**Consolidates sleep and raises sleep efficiency via mild sleep debt**	Cut time in bed to sleep time (floor about four to five hours); titrate up 15 min at 85% efficiency
Sleep hygiene (support)	Removes environmental and behavioural sleep-eroders	Regular schedule, no caffeine/nicotine/alcohol near bed, limit naps; one or two habits at a time
Cognitive therapy (support)	Corrects dysfunctional sleep beliefs and the arousal cycle	Identify, challenge and substitute catastrophic and rigid sleep thoughts
Relaxation (support)	Lowers somatic and cognitive arousal at bedtime	Master PMR, imagery or breathing in the daytime, then apply at bed
Paradoxical intention (occasional)	Removes the sleep effort that feeds hyperarousal	Stop trying to sleep; give up the effort to fall asleep

The CBT-I components; sleep restriction and stimulus control are the two behavioural engines, sleep hygiene, cognitive therapy and relaxation the supports, and sleep hygiene alone is not a treatment.

13.8 The evidence base: durable where a tablet is not

What the evidence shows. Studies repeatedly show significant, sustained improvement in sleep with CBT-I: shorter sleep-onset latency, fewer and briefer awakenings, and less time awake in bed, with no demonstrated adverse effects (Kaplan and Sadock CTP 2024). Its short-term benefit is broadly similar to that of hypnotic medication, but the decisive difference is durability, the gains persist long after treatment ends, even **36 months** out, whereas insomnia frequently returns when a hypnotic is stopped, sometimes with rebound insomnia.

The trade-off. The costs are that CBT-I takes longer to work than a tablet, needs an actively participating and motivated patient, and depends on trained therapists who are often scarce, which is exactly why the digital route matters. The desperate patient wants a quick fix; the clinician's task is to hold the line that the therapy that takes a few weeks is the one that lasts.

PEARL

CBT-I and a hypnotic look similar at the finish of a short course, but they diverge afterwards: stop the drug and the insomnia usually comes back, sometimes worse (rebound), whereas the CBT-I gains hold for years. Durability, not speed, is the reason CBT-I leads.

13.9 Delivery: individual, group, and digital CBT-I

The formats. CBT-I can be delivered one to one, in groups, or digitally with no in-person visit, and BAP is explicit that face-to-face and digital CBT-I (dCBT-I) are both efficacious, so the patient can be offered a choice among evidence-based options and a shortage of therapists is not a reason to default to a tablet (BAP 2019). Digital and app-based programmes extend reach where trained clinicians are few.

The brief variant. A condensed form, brief behavioural treatment for insomnia (BBT-I), distils the behavioural core (chiefly sleep restriction and stimulus control) into a shorter, more deliverable package, and VA/DoD lists it as a reasonable option for chronic insomnia disorder (VA/DoD 2025). The message across formats is the same: the components travel, whether delivered by a therapist, an app or a brief protocol.

GOOD TO REMEMBER

- **Delivery (the value):** CBT-I works individually, in groups and digitally, with face-to-face and digital CBT-I both efficacious (BAP 2019); brief behavioural treatment for insomnia (BBT-I) is a shorter behavioural-core option (VA/DoD 2025), so scarcity of therapists is not a reason to skip straight to a hypnotic.

13.10 First-line status, where the drug fits, and the Indian frame

The one rule. CBT-I, delivered as the multicomponent package including sleep restriction and stimulus control, is the first-line chronic insomnia treatment, offered before any drug (BAP 2019; NICE 2023). The Indian Psychiatric Society CPG makes CBT-I the mainstay of chronic insomnia (IPS 2017), and VA/DoD gives it a strong recommendation and explicitly prefers it over pharmacotherapy (VA/DoD 2025). Pharmacology enters only where CBT-I fails, is unavailable, or the patient cannot engage with it, and when a hypnotic is later discontinued it is tapered slowly with concurrent CBT-I to improve the outcome (BAP 2019). The deep hypnotic pharmacology is carried in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes.

The Indian frame. IPS 2017 sets CBT-I as the mainstay, but the practice reality is a shortage of clinicians trained to deliver it, so the default in much Indian practice slides towards a benzodiazepine or z-drug prescription and a sleep-hygiene handout, precisely the two moves the evidence cautions against as stand-alone treatment. Indian textbooks teach the sleep-hygiene rules, regular daytime exercise (not in the evening), minimising daytime naps, avoiding heavy meals and fluids before bed, and avoiding caffeine and alcohol as a sleep aid, but these are the necessary-but-not-sufficient support, not the therapy. The digital-CBT-I route is the realistic way to close the access gap.

RULE **CBT-I first, the tablet second.** Cognitive behaviour therapy for insomnia, with sleep restriction and stimulus control at its core, is first-line for chronic insomnia and is offered before any drug; a hypnotic is a short-term second-line measure for when CBT-I fails, is unavailable, or cannot be engaged with (BAP 2019, IPS 2017, NICE 2023, VA/DoD 2025).

TRAP **Do not start what you cannot stop.** Reaching for a long-term hypnotic in chronic insomnia when CBT-I is the treatment trades a durable fix for dependence and rebound; the tablet is short-term, and even then it is tapered with concurrent CBT-I.

INDIA

IPS 2017 names CBT-I the mainstay of chronic insomnia, yet trained CBT-I therapists are scarce across Indian services, so practice too often defaults to a benzodiazepine or z-drug plus a sleep-hygiene leaflet, the two moves the evidence flags as insufficient on their own. Digital CBT-I is the pragmatic way to widen access; the benzodiazepine habit is the pattern to unlearn.

85%	15	36
SLEEP-EFFICIENCY TARGET TO TITRATE TIME IN BED UP	TIME-IN-BED STEP-UP INCREMENT (MINUTES)	CBT-I BENEFIT SUSTAINED POST-TREATMENT (MONTHS)

HOLD THIS

CBT-I is a five-component package, sleep restriction and stimulus control the two engines, sleep hygiene, cognitive therapy and relaxation the supports, and it is first-line for chronic insomnia, offered before any drug because its gains outlast a hypnotic.

14 · Narcolepsy

Narcolepsy is a chronic disorder of the sleep-wake boundary in which the machinery of rapid-eye-movement sleep, its irresistible sleepiness and its muscle atonia and dreaming, intrudes into wakefulness. The examinable heart of it is a symptom cluster, a mechanism and a test: the **narcolepsy tetrad**, the loss of the hypothalamic orexin (hypocretin) neurons, and the multiple sleep latency test. It is a favourite of examiners precisely because it hides so well in psychiatric clinics, wearing the face of a treatment-resistant depression, of laziness, or of a seizure disorder, so that the patient who has been irresistibly sleepy since adolescence is treated for everything except the disorder they actually have.

Why it matters, and how this note is framed. This is a hybrid topic. The diagnostic half asks you to know the tetrad and its pentad extension, that cataplexy is the pathognomonic feature, the orexin loss and its HLA and autoimmune associations, the split into type 1 and type 2, the differential that traps the psychiatrist, and that the diagnosis is sealed on the mslt with its two verified numbers plus a low cerebrospinal-fluid orexin. The management half asks you to know the wake-promoting agents for the daytime sleepiness, sodium oxybate for cataplexy and the broken night, and the older antidepressants that suppress cataplexy, led by the sleep-medicine practice standard and with the Indian availability made explicit. Two structures accompany this topic and are flagged for drawing: the orexin sleep-switch and the tetrad schematic. Because narcolepsy sits outside the guideline database used for the eating and insomnia drugs, its criteria and doses are grounded in the parsed textbooks and the sleep classification, and any recommendation the library could not confirm is flagged rather than invented.

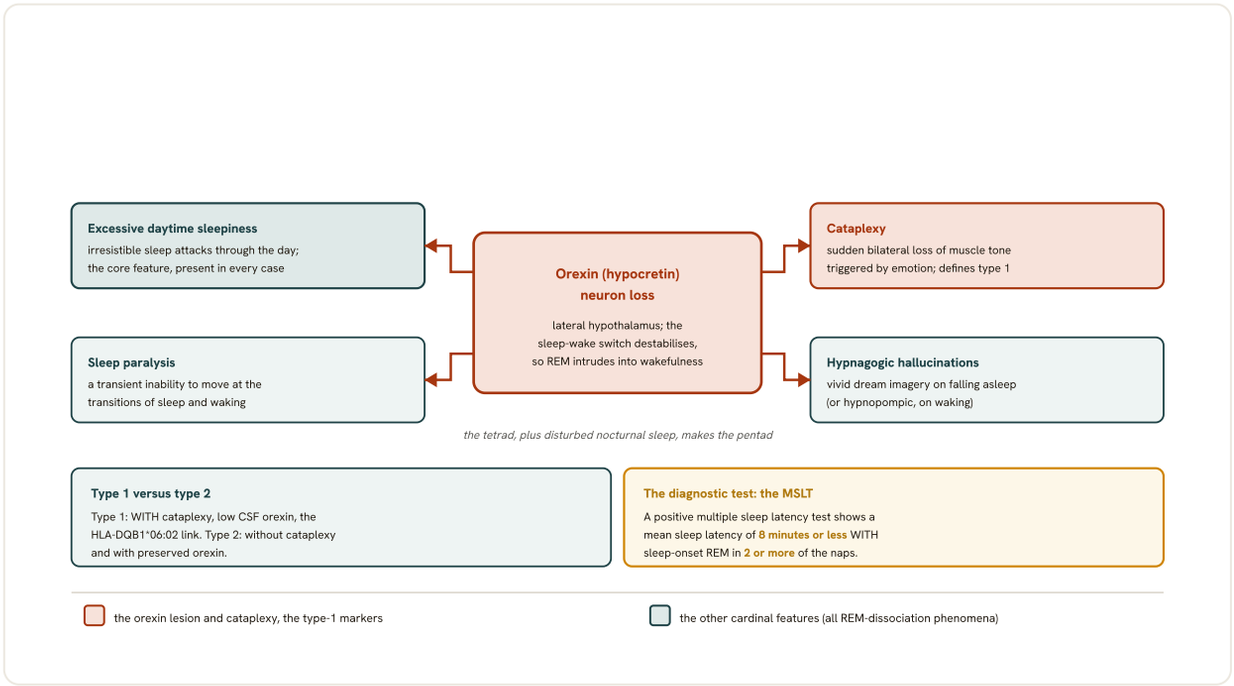


Fig 17.12 Narcolepsy: the orexin loss and the tetrad. The lesion is the loss of the orexin (hypocretin) neurons of the lateral hypothalamus, the peptide that stabilises the sleep-wake flip-flop switch, so the switch becomes unstable and the phenomena of REM sleep intrude into wakefulness. That produces the cardinal tetrad: excessive daytime sleepiness with irresistible sleep attacks (the core feature, present in every case), cataplexy (a sudden bilateral loss of muscle tone triggered by emotion, which is REM atonia breaking through and which defines type 1), sleep paralysis, and hypnagogic or hypnopompic hallucinations, with disturbed nocturnal sleep completing a pentad. Type 1 narcolepsy has cataplexy, a low cerebrospinal-fluid orexin and the HLA-DQB1*06:02 association; type 2 lacks cataplexy and has preserved orexin. The diagnosis is confirmed on the multiple sleep latency test by a mean sleep latency of eight minutes or less with sleep-onset REM periods in two or more naps. The wake-promoting and anti-cataplectic pharmacology is named in the topic and its detail cross-referenced to the anxiolytic and hypnotic pharmacology notes.

14.1 The tetrad, and the pentad

The four cardinal symptoms. For decades narcolepsy was defined by a tetrad (Kaplan and Sadock CTP 2024): irresistible **excessive daytime sleepiness** with sleep attacks, cataplexy, **sleep paralysis**, and **hypnagogic hallucinations** at sleep onset (or hypnopompic on waking). Excessive sleepiness is the constant and usually the presenting complaint: an overwhelming, unresistible urge to sleep that breaks through in the middle of a meal, a conversation or a task, with brief naps that are characteristically refreshing (unlike the un-refreshing naps of idiopathic hypersomnia). The other three are the REM-intrusion phenomena, the fragments of dream sleep leaking into wakefulness.

The pentad. Disturbed, fragmented nocturnal sleep is the fifth feature, which is why the paradox of narcolepsy is a patient who is desperately sleepy by day yet sleeps badly at night. The examinable trap embedded here is that only two of the four classic symptoms, the sleepiness and the cataplexy, are reasonably specific; sleep paralysis and hypnagogic hallucinations are nonspecific and occur in isolation in the general population, so they support but never make the diagnosis.

MNEMONIC

CHESS

Cataplexy; **H**ypnagogic (and hypnopompic) hallucinations; **E**xcessive daytime sleepiness (with sleep attacks); **S**leep paralysis; disturbed nocturnal **S**leep (the fifth, pentad, feature). Sleepiness is the constant; cataplexy is the specific one.

Sleep attack

An irresistible, overwhelming urge to sleep breaking through into wakefulness, with a brief and typically refreshing nap.

Cataplexy

Sudden bilateral loss of skeletal muscle tone triggered by strong emotion (classically laughter), with consciousness preserved.

Sleep paralysis

Transient inability to move or speak at sleep onset or on waking; the REM atonia persisting into wakefulness.

Hypnagogic hallucination

Vivid, often frightening dream-like perception at sleep onset (hypnopompic if on waking); dream imagery intruding into wake.

GOOD TO REMEMBER

- **Narcolepsy tetrad (the defining cluster):** excessive daytime sleepiness with irresistible sleep attacks, cataplexy, sleep paralysis and hypnagogic or hypnopompic hallucinations, with disturbed nocturnal sleep as the fifth (pentad) feature; the sleepiness is the constant and cataplexy is the specific member, while sleep paralysis and hypnagogic hallucinations are nonspecific and occur in isolation in healthy people.

14.2 Cataplexy: the pathognomonic feature

What it is. Cataplexy is the sudden loss of muscle tone, with continuing full or partial consciousness, precipitated by strong emotion and most commonly by laughter (Kaplan and Sadock CTP 2024). It ranges from the barely perceptible, a head nod, a sagging jaw, a slurring of speech or buckling knees, to a total collapse to the floor, and episodes last from a few seconds to a couple of minutes. It is bilateral, the patient is aware throughout, and reflexes are abolished during the attack, which is the neurological point that separates it from a psychogenic drop. In children, or in the first few months of onset, cataplexy can look atypical: spontaneous grimacing, jaw-opening with tongue thrusting, or a global hypotonia without a clear emotional trigger.

Why it matters. Cataplexy is the one feature specific enough to clinch the diagnosis and defines type 1 disease; it is REM-sleep atonia (the same paralysis that normally protects us from acting out dreams) discharging into wakefulness. It occurs in a majority of narcolepsy patients and is the strongest clinical marker of the orexin deficiency, which is why demonstrating cataplexy, like demonstrating a low cerebrospinal-fluid orexin, is enough to place the patient in type 1 without further testing.

PEARL

Ask specifically what happens when the patient laughs hard, is startled, or gets angry. The pathognomonic history is knees buckling or the head dropping at the punchline of a joke, with the patient fully aware and unable to move for a few seconds. That single question separates narcolepsy from every other cause of daytime sleepiness.

TRAP **Reading cataplexy or a sleep attack as a seizure.** An emotion-triggered fall with preserved awareness and abolished reflexes is cataplexy, not an atonic seizure, and repeated daytime "absences" that are really sleep attacks earn antiepileptics for years before the sleep history is finally taken.

14.3 The pathophysiology: loss of the orexin (hypocretin) neurons

The orexin system. A small population of neurons in the **lateral hypothalamus** makes the peptide neurotransmitter hypocretin, discovered independently by two groups and so carrying two names: hypocretin (for its hypothalamic location) and orexin (from the Greek *orexis*, appetite). These neurons normally excite the brainstem and hypothalamic arousal centres and hold wakefulness stable, stabilising the switch between the wake and sleep states; their peptides are orexin A and B (hypocretin 1 and 2). The accompanying figure sets this orexin sleep-switch against the tetrad so the leak of REM phenomena into wakefulness can be read off the failing switch.

The lesion. Narcolepsy with cataplexy is caused by the selective loss of these hypocretin-producing neurons, so the orexin signal that clamps the sleep-wake boundary is gone and REM sleep and its atonia intrude into the day (Kaplan and Sadock CTP 2024; Kaufman). The near-total loss (up to ninety per cent of the orexin neurons) is why the cerebrospinal-fluid orexin falls so low. The leading account of the lesion is autoimmune: the strong HLA-DQB1*06:02 association, the onset that often follows an infection, and the near-universal orexin loss in cataplectic disease all point to an immune-mediated destruction of the orexin neurons.

The HLA association. Narcolepsy with cataplexy is very tightly linked to the major-histocompatibility allele HLA-DQB1*06:02 on chromosome 6 (Kaufman). The examinable subtlety is that despite this tight association the allele is neither necessary nor sufficient: it is common in the healthy population, so it supports but cannot confirm the diagnosis, and its main use is as a screening or supportive test rather than a diagnostic one.

GOOD TO REMEMBER

- **Orexin loss (the mechanism):** narcolepsy type 1 is the selective loss of the lateral-hypothalamic orexin (hypocretin) neurons that normally stabilise wakefulness, most likely autoimmune, marked by the HLA-DQB1*06:02 association and a low or absent cerebrospinal-fluid orexin; the allele is neither necessary nor sufficient, so it screens but never seals the diagnosis.

14.4 Type 1 versus type 2

The split. The classification (ICSD-3; DSM-5-TR 2022) divides narcolepsy by the orexin lesion. **Type 1** (narcolepsy with cataplexy, or hypocretin deficiency) is the classic orexin-deficient

disease: cataplexy is present and, or, the cerebrospinal-fluid orexin is low; this is the form with the tight HLA link and the autoimmune story. **Type 2** (narcolepsy without cataplexy and without demonstrated hypocretin deficiency) has the daytime sleepiness and the MSLT findings but no cataplexy and a normal or unmeasured orexin; it is a more heterogeneous, less well-understood group, and a proportion later declare cataplexy and reclassify as type 1.

The load-bearing rule. Either cataplexy or a low cerebrospinal-fluid orexin is sufficient to call it type 1, which is why the bedside question about emotion-triggered weakness carries as much diagnostic weight as a lumbar puncture. Both types still require the objective sleepiness on the MSLT; the type distinction is about the orexin lesion, not about the sleepiness.

FEATURE	TYPE 1 (WITH CATAPLEXY OR LOW OREXIN)	TYPE 2 (WITHOUT EITHER)
Cataplexy	**Present (or low CSF orexin)**	Absent
CSF orexin (hypocretin-1)	**Low or absent (under 110 pg per mL)**	Normal or not measured
HLA-DQB1*06:02	Strongly associated	Less strongly associated
MSLT (sleepiness plus SOREMPs)	Required	Required
Underlying lesion	Orexin-neuron loss (autoimmune)	Heterogeneous, orexin often intact

Type 1 versus type 2 narcolepsy; either cataplexy or a low cerebrospinal-fluid orexin defines type 1, both types share the objective MSLT sleepiness, and some type 2 patients later declare cataplexy and reclassify.

GOOD TO REMEMBER

- **Type 1 versus type 2 (the discriminator):** type 1 narcolepsy is defined by cataplexy or a low cerebrospinal-fluid orexin (under 110 pg per mL) and carries the HLA-DQB1*06:02 and autoimmune story; type 2 has the sleepiness and the MSLT findings but no cataplexy and a normal or unmeasured orexin; both require objective MSLT sleepiness, so the type is about the orexin lesion, not the sleepiness.

14.5 The differential: the masquerades that trap the psychiatrist

The sleep-medicine differential. Idiopathic hypersomnia is the closest mimic: genuine excessive daytime sleepiness, but with long, un-refreshing naps and sleep drunkenness, no cataplexy, and an MSLT that shows a short mean sleep latency without the two sleep-onset REM periods and without orexin deficiency. Chronic insufficient sleep (behaviourally induced sleep deprivation), the commonest cause of daytime sleepiness of all, is excluded by a sleep diary or actigraphy and by the sleepiness resolving when sleep is extended; obstructive sleep apnoea is excluded on the preceding-night polysomnogram.

The psychiatric masquerades. Two errors are the examinable ones. The first is the **treatment-resistant depression** masquerade: the young adult with pervasive daytime sleepiness, low energy, poor concentration and secondary low mood is labelled depressed, and antidepressant after antidepressant is stacked while the untreated narcolepsy drives the fatigue (and, worse,

several antidepressants blunt REM and mask the cataplexy that would have made the diagnosis). The second is the **epilepsy** masquerade: cataplectic drops and sleep attacks are mistaken for atonic or absence seizures, and the patient collects antiepileptics and a normal electroencephalogram for years. The corrective in both is the same, take a sleep history and ask about emotion-triggered weakness.

MIMIC	DISCRIMINATOR FROM NARCOLEPSY
Idiopathic hypersomnia	**Long un-refreshing naps, no cataplexy, MSLT without 2 SOREMPs, normal orexin**
Chronic insufficient sleep	Short habitual sleep; resolves when sleep is extended (diary or actigraphy)
Obstructive sleep apnoea	Snoring and witnessed apnoeas; excluded on preceding-night polysomnogram
Treatment-resistant depression	**Sleepiness and fatigue precede or exceed the mood; ask for cataplexy**
Epilepsy (atonic or absence)	**Cataplexy has an emotional trigger, preserved awareness and a normal EEG**

The narcolepsy differential; idiopathic hypersomnia is the closest sleep-medicine mimic, while the depression and epilepsy masquerades are the errors that keep the psychiatrist from the diagnosis for years.

RULE **Ask for cataplexy before you escalate.** In a young adult with pervasive daytime sleepiness that outweighs the mood, ask about emotion-triggered weakness and take a sleep history before labelling a "treatment-resistant depression" or an epilepsy, because narcolepsy is the diagnosis those two masquerades hide.

14.6 Diagnosis: the MSLT and the low cerebrospinal-fluid orexin

The confirmatory tests. The diagnosis is built on an overnight polysomnogram followed the next morning by the multiple sleep latency test (MSLT), five short scheduled nap opportunities across the day that quantify how fast the patient falls asleep and whether REM sleep appears abnormally early (Kaufman; Tasman Psychiatry). The overnight study serves two purposes: it excludes another cause of the sleepiness (apnoea, poor prior-night sleep) and it can itself catch an abnormally short REM latency.

The two MSLT numbers. The MSLT criterion for narcolepsy is a mean sleep latency of eight minutes or less *and* two or more sleep-onset REM periods (SOREMPs) across the naps (Kaufman; DSM-5-TR 2022). A short REM latency of fifteen minutes or less on the preceding-night polysomnogram can count as one of the two required SOREMPs. These are the load-bearing figures: the short latency proves the pathological sleepiness, and the two SOREMPs prove the REM-intrusion that is the signature of narcolepsy (normal REM latency is about ninety to a hundred minutes, so REM within a nap is grossly abnormal).

The orexin test. The most specific single finding in narcolepsy with cataplexy is a low or absent cerebrospinal-fluid orexin (hypocretin-1), conventionally under 110 pg per mL, while the serum level stays normal (Kaufman). It is diagnostic of type 1 but is not routinely available in most

clinical settings, so in practice cataplexy plus the MSLT carries the diagnosis and the lumbar puncture is reserved for the difficult case.

≤8	≥2	<110	200 to 400
MSLT MEAN SLEEP LATENCY (MINUTES)	SLEEP-ONSET REM PERIODS ON MSLT (COUNT)	CSF OREXIN CUT-OFF, TYPE 1 (PG PER ML)	MODAFINIL DOSE IN NARCOLEPSY (MG PER DAY)

TEST	DIAGNOSTIC FINDING
MSLT mean sleep latency	**8 minutes or less**
MSLT sleep-onset REM periods	**2 or more across the naps**
Preceding-night PSG REM latency	15 minutes or less can count as one SOREMP
CSF orexin (hypocretin-1)	**Low or absent (under 110 pg per mL); defines type 1**

The diagnostic thresholds for narcolepsy; the MSLT needs a mean sleep latency of eight minutes or less plus two or more sleep-onset REM periods, and a low cerebrospinal-fluid orexin is the most specific but least available test.

GOOD TO REMEMBER

- **Diagnosis (the confirmatory step):** an overnight polysomnogram followed by the MSLT, which is positive at a mean sleep latency of eight minutes or less *and* two or more sleep-onset REM periods (a preceding-night REM latency of fifteen minutes or less can serve as one SOREMP); a cerebrospinal-fluid orexin under 110 pg per mL is the most specific finding and defines type 1 but is rarely available.

14.7 Management: the wake-promoting agents for daytime sleepiness

The practice-standard position. Narcolepsy is not curable, so treatment targets the two problems separately: the excessive daytime sleepiness and the cataplexy. The sleep-medicine practice standard (the American Academy of Sleep Medicine clinical practice guideline, and the sleep-medicine texts) places the wake-promoting agents first for the daytime sleepiness, with scheduled short naps and firm sleep hygiene as the non-drug backbone alongside them. Because narcolepsy is not covered by the day's guideline supplement, the drug detail here is grounded in the parsed prescribing texts, and the exact strength-of-recommendation grading of the AASM 2021 guideline is flagged for confirmation against the primary source rather than reproduced.

The wake-promoting drugs. Modafinil (in India, Modalert or Provake) is the first-line agent for the excessive daytime sleepiness of narcolepsy, a wakefulness-promoting dopamine-transporter inhibitor that is not classed as a psychostimulant, given at 200 to 400 mg per day, and it improves the sleepiness but does *not* benefit cataplexy (Kaplan and Sadock CTP 2024). Its longer-acting R-enantiomer armodafinil is an alternative. Two newer agents extend the class: solriamfetol, a norepinephrine-dopamine reuptake inhibitor, and pitolisant, a histamine H3-receptor antagonist and inverse agonist that raises brain histamine and, unusually for a wake-promoter, also reduces cataplexy (Stahl). The traditional psychostimulants (methylphenidate, dexamphetamine) remain a second line for residual sleepiness, with their tolerance and dependence liability.

GOOD TO REMEMBER

- **Wake-promoting agents (the value):** modafinil (Indian brands Modalert or Provake), a dopamine-transporter inhibitor at 200 to 400 mg per day, is first-line for the excessive daytime sleepiness of narcolepsy but does not touch cataplexy; armodafinil, solriamfetol and pitolisant extend the class, pitolisant (an H3 antagonist) uniquely also reducing cataplexy, with the amphetamines held as a dependence-prone second line.

14.8 Sodium oxybate for cataplexy, and the anticataplectic antidepressants

Sodium oxybate. Sodium oxybate (the sodium salt of gamma-hydroxybutyrate, GHB) is the most effective single treatment for cataplexy and is the one agent that also consolidates the broken night and improves the daytime sleepiness, taken in divided doses across the night because it enhances slow-wave sleep (Kaufman; Tasman Psychiatry; Stahl). It also blunts sleep paralysis and the hypnagogic and hypnopompic hallucinations. Its liability is its identity: GHB is a drug of abuse (the "date-rape drug"), so it is a tightly controlled substance dispensed through restricted programmes, and it must never be combined with alcohol or other central-nervous-system depressants.

The older anticataplectic drugs. Before sodium oxybate and pitolisant, cataplexy was suppressed with REM-suppressing antidepressants, and these remain a practical option: an SSRI or an SNRI (venlafaxine), or the tricyclic clomipramine (also imipramine or protriptyline). They work by suppressing REM sleep and its atonia, and abrupt withdrawal can cause a rebound of cataplexy (status cataplecticus), so they are tapered rather than stopped. They do little for the daytime sleepiness, which is why cataplexy and sleepiness are usually treated with two different drugs.

WORKED EXAMPLE

A woman in her twenties has been irresistibly sleepy since her mid-teens and has failed three antidepressants for a presumed depression. Asked directly, she describes her knees buckling whenever she laughs hard, fully aware throughout. An overnight polysomnogram excludes apnoea, and the next-morning MSLT shows a mean sleep latency of five minutes with three sleep-onset REM periods. The diagnosis is narcolepsy type 1. She is started on modafinil (Modalert) for the sleepiness, and because the cataplexy is disabling and her nights are fragmented, sodium oxybate is added where available; where it is not, venlafaxine is used for the cataplexy instead.

GOOD TO REMEMBER

- **Cataplexy treatment (the value):** sodium oxybate (GHB) is the most effective drug for cataplexy and the only one that also consolidates the broken night and improves daytime sleepiness, but it is an abuse-liable controlled substance never combined with alcohol; the older option is a REM-suppressing antidepressant (an SSRI/SNRI or clomipramine), tapered rather than stopped to avoid rebound cataplexy.

14.9 The Indian picture: drug availability and the missed diagnosis

What is available, and what is not. The genuine Indian device in narcolepsy is drug availability. Modafinil is widely and cheaply available in India as Modalert, Provake and other brands, and

armodafinil is available too, so the first-line treatment of the daytime sleepiness is entirely practical. The problem is the rest of the ladder: sodium oxybate is not marketed in India (it is a controlled substance dispensed only through restricted programmes abroad), and the newer agents pitolisant and solriamfetol are not routinely available either. The practical consequence is that Indian cataplexy is usually managed not with sodium oxybate but with an SSRI, an SNRI or clomipramine, the older anticataplectic antidepressants, which is exactly the substitution the worked example makes.

The under-recognition. Narcolepsy is under-diagnosed in India for the same reasons the eating disorders are: sleep laboratories and the MSLT are scarce and out-of-pocket, cerebrospinal-fluid orexin testing is essentially unavailable, and the young adult who has been sleepy since adolescence is far more likely to be told to sleep more, try harder, or take an antidepressant than to be sent for a sleep study. The cheap corrective is clinical, ask every chronically sleepy young patient about emotion-triggered weakness, because cataplexy alone can carry the diagnosis to treatment even where the confirmatory tests cannot be had.

INDIA

Modafinil (Modalert, Provake) and armodafinil are freely available and make first-line treatment of the daytime sleepiness straightforward, but sodium oxybate, pitolisant and solriamfetol are not marketed in India, so cataplexy is managed with the older anticataplectic antidepressants (an SSRI or SNRI, or clomipramine) instead. Layered on this is under-recognition: MSLT-capable sleep labs are scarce and orexin assay is essentially unavailable, so the sleepy adolescent is treated for laziness or depression for years. The corrective costs nothing, ask about knees buckling at a joke, because cataplexy alone places the patient in type 1 and gets them treated.

HOLD THIS

Narcolepsy is REM sleep intruding into wakefulness from loss of the hypothalamic orexin (hypocretin) neurons: the tetrad is excessive daytime sleepiness with sleep attacks, cataplexy, sleep paralysis and hypnagogic hallucinations (disturbed nocturnal sleep the pentad fifth), cataplexy plus a low CSF orexin (under 110 pg per mL) defines type 1, and it is confirmed on the MSLT at a mean sleep latency of eight minutes or less with two or more sleep-onset REM periods; treat the sleepiness with modafinil (Modalert or Provake) and the cataplexy with sodium oxybate or, in India, an SSRI/SNRI or clomipramine.

15 · Obstructive sleep apnoea and its psychiatric relevance

Obstructive sleep apnoea is a breathing-related sleep disorder in which the upper airway repeatedly collapses during sleep, so that ventilation stops or falls, the blood oxygen dips, and the brain briefly rouses to reopen the airway, hundreds of times a night. It is included here not as respiratory trivia but because it is the medical disorder the psychiatrist most reliably misses: it masquerades as depression, as cognitive impairment, and as a stubborn **treatment-resistant depression**, and the man snoring in the waiting room with a collar-size neck and a low mood is more often carrying obstructive sleep apnoea (OSA) than a pure mood disorder.

Why it matters, and how this note is framed. This is a hybrid topic. The diagnostic half asks you to know the pathophysiology (the airway collapse, the apnoea and hypopnoea, the oxygen desaturation, the arousal and the sleep fragmentation), the clinical picture, the **apnoea-hypopnoea index** and its verified severity bands, and that the diagnosis is sealed on polysomnography. The management half asks you to know that continuous positive airway pressure is the mainstay, with weight, positional, oral-appliance and surgical options behind it, and to screen at the bedside with STOP-BANG and its verified risk bands. Two figures accompany this topic and are flagged for drawing: the apnoea-hypopnoea and oxygen-desaturation trace, and the STOP-BANG scale plate. Because this disorder sits outside the guideline database used for the eating and insomnia drugs, its numbers are grounded in the parsed textbooks and a verified primary source, and any figure the library could not confirm is flagged rather than invented.

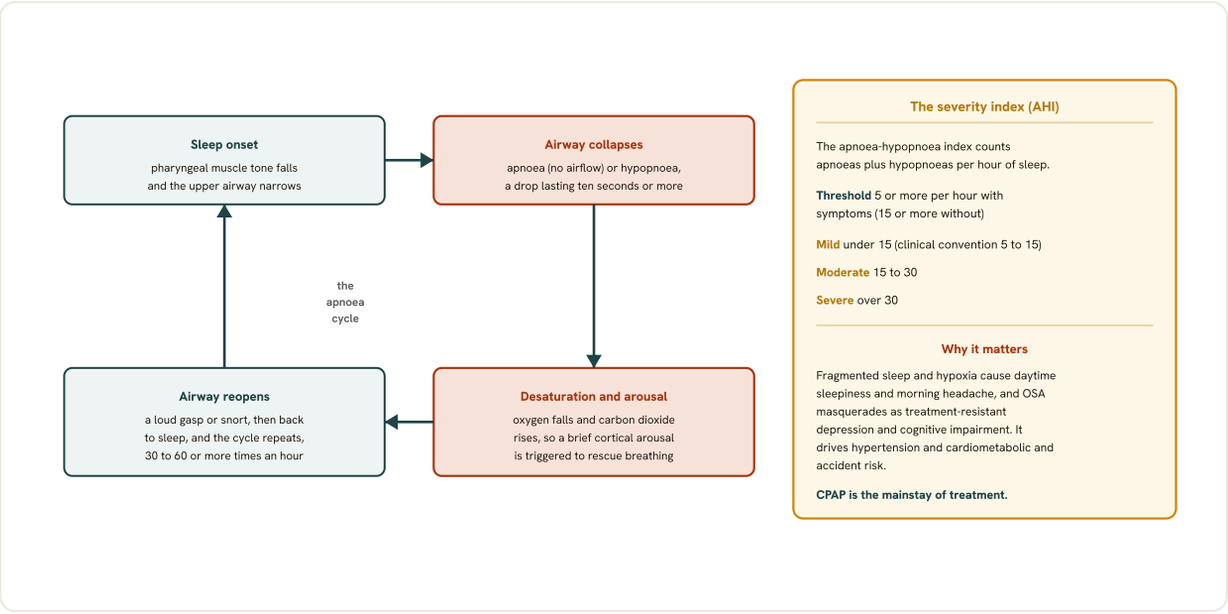


Fig 17.13 Obstructive sleep apnoea: the apnoea-hypopnoea cycle. In sleep the pharyngeal muscle tone falls and, in a susceptible airway, the upper airway collapses, so airflow stops (apnoea) or is sharply reduced (hypopnoea) for ten seconds or more. Oxygen falls and carbon dioxide rises until a brief cortical arousal restores muscle tone and the airway reopens with the characteristic loud gasp or snort, whereupon the person returns to sleep and the whole cycle repeats, thirty to sixty or more times an hour. The result is a night shredded into fragments with repetitive hypoxia. Severity is graded by the apnoea-hypopnoea index, the number of events per hour, diagnostic at five or more with symptoms and banded mild, moderate (fifteen to thirty) and severe (over thirty), with the lower edge of the mild band differing between the DSM-5-TR specifier and the clinical convention. The importance for psychiatry is that the daytime sleepiness and cognitive dulling masquerade as treatment-resistant depression, so it is a diagnosis to exclude; continuous positive airway pressure is the mainstay of treatment.

STOP-BANG, THE OSA SCREEN (REPRODUCED)	SCORE ONE POINT FOR EACH "YES"
S noring	Do you snore loudly (louder than talking, or loud enough to be heard through a closed door)?
T iredness	Do you often feel tired, fatigued or sleepy during the daytime?
O bserved	Has anyone observed you stop breathing during your sleep?
P ressure	Do you have, or are you being treated for, high blood pressure?
B MI	Body mass index more than 35 kg per square metre?
A ge	Age over 50 years?
N eck	Neck circumference greater than 40 cm?
G ender	Male?
Risk bands	**0 to 2 low risk; 3 to 4 intermediate risk; 5 to 8 high risk of moderate-to-severe obstructive sleep apnoea**

STOP-BANG: reproduced from Chung F, Yegneswaran B, Liao P, et al. Anesthesiology. 2008;108:812-821. A widely used, freely available OSA screen; for the free, non-commercial resident edition only.

Fig 17.14 The STOP-BANG obstructive sleep apnoea screen. An eight-item yes-or-no screen whose name is its mnemonic: Snoring, Tiredness, Observed apnoea and Pressure (blood pressure), then BMI over 35, Age over 50, Neck over 40 cm and male Gender. One point is scored for each yes, and the total stratifies risk as low (0 to 2), intermediate (3 to 4) or high (5 to 8) for moderate-to-severe obstructive sleep apnoea, so a high score prompts referral for sleep study. It is quick and sensitive, which is exactly what a psychiatric clinic needs to catch the sleep apnoea hiding behind a picture of treatment-resistant depression or daytime fatigue.

15.1 The pathophysiology: repetitive upper-airway collapse, apnoea and hypopnoea, desaturation and arousal

The mechanism. During sleep the muscles that hold the pharynx open lose tone, and in the vulnerable airway (a crowded, narrow or fat-loaded pharynx behind the tongue and soft palate) the negative pressure of inspiration sucks the walls shut despite continued respiratory effort. A total collapse for **ten seconds** or longer is an *apnoea*; a partial collapse that reduces airflow for ten seconds or longer is a *hypopnoea* (Kaplan and Sadock CTP 2024). The obstruction is what distinguishes it from central apnoea, where effort itself ceases: in the obstructive event the chest and abdomen keep straining against a shut airway.

The cascade. Each obstructed breath produces a fall in blood oxygen, an **oxygen desaturation**, and the rising carbon dioxide and effort trigger a brief cortical arousal that restores airway tone, often ended by a loud snort or gasp, after which the person falls back asleep and the cycle repeats. The consequence is twofold: intermittent nocturnal hypoxaemia, and a sleep so fragmented by micro-arousals that it never consolidates, which is why the patient sleeps a full night yet wakes unrefreshed and is sleepy throughout the day. The accompanying figure sets the airflow trace, the

effort trace and the falling oxygen saturation against one another so the apnoea, the hypopnoea and the arousal can be read off a single polysomnographic strip.

Apnoea

Cessation of airflow for ten seconds or longer during sleep (obstructive when respiratory effort continues against a collapsed airway).

Hypopnoea

A reduction in airflow for ten seconds or longer, scored when it produces a defined oxygen desaturation or a cortical arousal.

Oxygen desaturation

The dip in blood oxygen saturation each obstructed breath produces; a hypopnoea is conventionally scored at a fall of a few percent or more.

Arousal and fragmentation

The brief cortical waking that reopens the airway; repeated hundreds of times, it shatters sleep continuity and drives the daytime sleepiness.

> **PEARL**
>
> The daytime sleepiness of obstructive sleep apnoea is not from too little sleep but from sleep that is repeatedly broken. The patient may spend eight hours in bed and still be pathologically sleepy, because every obstructed breath buys an arousal and no stretch of sleep is ever allowed to deepen and consolidate.

15.2 The clinical picture and the risk factors

What the patient and the bed partner report. The cardinal triad is loud habitual **snoring**, witnessed apnoeas, and excessive daytime sleepiness. The snoring of obstructive sleep apnoea is characteristically loud enough to be heard outside the bedroom door, the pauses are long, typically twenty to thirty seconds rather than a few, and each is broken by a snort or a start (New Oxford Textbook of Psychiatry). The bed partner is the key informant, because the patient is asleep for the apnoeas and the snorting and often denies both. Add un-refreshing sleep, a dry mouth, a morning headache, nocturia, irritability and impaired concentration, and, in the psychiatrist's chair, low mood and cognitive slowing.

Who is at risk. The dominant risk factor is obesity, and specifically central and upper-body fat that loads the pharynx, so a large neck circumference is a better bedside marker than weight alone. Male sex, older age, a retrognathic or micrognathic jaw and other craniofacial crowding of the airway, tonsillar hypertrophy, and alcohol or sedatives (which relax the airway further) all raise the risk. The examinable point is that the sleepy, snoring, thick-necked, hypertensive middle-aged man is the archetype, and that in India a lean patient with craniofacial crowding can carry it without the obesity that would prompt the thought.

DOMAIN	FEATURE
Nocturnal	**Loud habitual snoring, witnessed apnoeas, snorting or gasping arousals, choking**
Nocturnal (other)	Restless un-refreshing sleep, nocturia, dry mouth, sweating
Daytime	**Excessive daytime sleepiness, morning headache, poor concentration, low mood, irritability**
Risk factors	Obesity, large neck circumference, male sex, older age, craniofacial or retrognathic crowding, alcohol and sedatives

The clinical picture of obstructive sleep apnoea; snoring, witnessed apnoeas and daytime sleepiness are the triad, and the neck circumference is the single most useful bedside risk marker.

15.3 The apnoea-hypopnoea index and its severity bands

What the index is. The apnoea-hypopnoea index (AHI) is the number of apnoeas plus hypopnoeas per hour of sleep, and it is the number that both diagnoses the disorder and grades its severity (Kaplan and Sadock CTP 2024). A related index, the oxygen desaturation index, counts the desaturation dips per hour and tracks the hypoxic burden. The AHI is what every treatment decision and every examination question hangs off.

The verified severity bands. An AHI **under five per hour** is normal. Five to fifteen is mild, **fifteen to thirty** is moderate, and **above thirty** is severe (the AASM and ICSD-3 clinical bands). The DSM-5-TR severity specifier for obstructive sleep apnoea hypopnoea writes the same ceiling: mild is an AHI less than fifteen, moderate is fifteen to thirty, and severe is greater than thirty, its mild band starting at the diagnostic floor of five (DSM-5-TR 2022; Kaplan and Sadock CTP 2024). The moderate and severe thresholds are the load-bearing numbers, because they are where continuous positive airway pressure becomes the clear treatment of choice.

AHI (EVENTS PER HOUR)	SEVERITY BAND
Under 5	Normal (no obstructive sleep apnoea)
5 to 15	Mild
15 to 30	**Moderate**
Greater than 30	**Severe**

AHI severity bands (apnoeas plus hypopnoeas per hour of sleep); DSM-5-TR labels the mild band as an AHI under fifteen because diagnosis requires at least five events, and the moderate (15 to 30) and severe (over 30) cut-offs are identical across the classifications.

10	15 to 30	>30	5 to 8
MINIMUM APNOEA OR HYPOPNOEA EVENT (SECONDS)	AHI MODERATE BAND (EVENTS PER HOUR)	AHI SEVERE-OSA THRESHOLD (EVENTS PER HOUR)	STOP-BANG HIGH-RISK BAND (SCORE)

GOOD TO REMEMBER

- **Obstructive sleep apnoea (defining criterion):** repetitive obstructive apnoeas and hypopnoeas (airflow stopping or falling for ten seconds or longer despite continued effort) quantified as the apnoea-hypopnoea index; the disorder is graded normal (AHI under 5), mild (5 to 15), moderate (15 to 30) and severe (over 30), and DSM-5-TR writes mild as an AHI under fifteen with the diagnostic floor at five.

15.4 Diagnosis by polysomnography

The confirmatory test. The diagnosis is made on polysomnography, the overnight sleep study that records airflow, respiratory effort, oxygen saturation, the electroencephalogram, eye movements and muscle tone, from which the apnoeas, hypopnoeas and arousals are counted and the AHI computed; a validated home sleep apnoea test can substitute in the appropriately selected patient (Kaplan and Sadock CTP 2024). Polysomnography is not routine for uncomplicated insomnia, but it is exactly what suspected obstructive sleep apnoea earns.

The diagnostic thresholds. In the absence of symptoms an AHI of fifteen or more (predominantly obstructive events) establishes the diagnosis; with relevant symptoms, an AHI of five or more suffices (Kaplan and Sadock CTP 2024). The symptoms and comorbidities that license the lower threshold are the psychiatrically loaded ones: daytime sleepiness, witnessed apnoeas, gasping or habitual snoring, and hypertension, a mood disorder, cognitive dysfunction, coronary disease, stroke, heart failure, atrial fibrillation or type 2 diabetes. Note that a coexisting mood disorder is itself listed among the conditions that lower the diagnostic bar, which is the nosology quietly telling you that depression and apnoea travel together.

SETTING	AHI REQUIRED (EVENTS PER HOUR)
No symptoms or comorbidity	15 or more, predominantly obstructive
With symptoms or relevant comorbidity	5 or more, predominantly obstructive

Polysomnographic diagnostic thresholds for obstructive sleep apnoea; a mood disorder and cognitive dysfunction are among the comorbidities that lower the bar to an AHI of five.

GOOD TO REMEMBER

- **Diagnosis (the confirmatory step):** obstructive sleep apnoea is confirmed on polysomnography (or a validated home sleep apnoea test), diagnostic at an AHI of fifteen or more without symptoms or five or more with symptoms or relevant comorbidity; a coexisting mood disorder is one of the comorbidities that licenses the lower five-per-hour threshold.

15.5 The STOP-BANG screen and its verified risk bands

What STOP-BANG is. STOP-BANG is an eight-item yes/no bedside screen for the risk of obstructive sleep apnoea, developed to flag the patients who need a sleep study (Tasman Psychiatry). Each positive item scores one point, giving a total out of eight. The mnemonic itself carries the four symptom questions and the four demographic-and-examination items, which is why it is so examinable. This topic carries the STOP-BANG form on the accompanying scale plate; the Epworth Sleepiness Scale, the eight-item self-rating of daytime sleepiness (scored out

of twenty-four, sleepiness above ten), quantifies the sleepiness that STOP-BANG's first items ask about and is cross-referenced with the hypersomnolence and narcolepsy assessment.

The verified risk bands. The total stratifies risk into three bands: zero to two is low risk, three to four is intermediate risk, and **five to eight** is high risk of obstructive sleep apnoea. These item cut-offs and risk bands are taken from the primary STOP-BANG literature (Chung and colleagues), because the parsed textbooks name the instrument and its general risk factors but do not print the banded scoring; the exact bands are therefore flagged as source-verified rather than textbook-derived.

MNEMONIC

STOP-BANG

Snoring (loud); **T**iredness or daytime sleepiness; **O**bserved apnoeas; high blood **P**ressure; **B**MI over 35 (kg per m squared); **A**ge over 50 years; **N**eck circumference over 40 cm; male **G**ender. One point each, out of eight; three or more is a positive screen worth a sleep study, five or more is high risk.

STOP-BANG ITEM	POSITIVE IF
Snoring	Loud, heard through a closed door
Tiredness	Excessive daytime sleepiness or fatigue
Observed apnoea	**Bed partner has witnessed you stop breathing**
Pressure	Hypertension (treated or untreated)
BMI	Over 35 (kg per m squared)
Age	Over 50 years
Neck	**Circumference over 40 cm**
Gender	Male

The eight STOP-BANG items (Chung and colleagues); each yes scores one point out of eight, with three or more flagging a patient for polysomnography.

STOP-BANG TOTAL (SCORE)	RISK OF OBSTRUCTIVE SLEEP APNOEA
0 to 2	Low risk
3 to 4	Intermediate risk
5 to 8	**High risk**

STOP-BANG risk bands; five or more of eight is high risk and should prompt a sleep study, and the screen is less sensitive in women and older patients.

GOOD TO REMEMBER

- **STOP-BANG (the value):** eight yes/no items (Snoring, Tiredness, Observed apnoea, Pressure, BMI over 35, Age over 50, Neck over 40 cm, male Gender), one point each out of eight; 0 to 2 is low risk, 3 to 4 intermediate and 5 to 8 high risk of obstructive sleep apnoea, so three or more warrants a sleep study; less sensitive in women and the elderly.

15.6 The psychiatric relevance: the depression and cognitive masquerade, the treatment-resistant-depression trap, and the cardiometabolic risk

The masquerade. The **psychiatric relevance** of obstructive sleep apnoea is that its daytime face, fatigue, low mood, anhedonia, poor concentration, memory complaints and irritability, is indistinguishable at a glance from a depressive episode or an early neurocognitive disorder. Obstructive sleep apnoea is independently associated with depressed mood, an association stronger in women than in men (Tasman Psychiatry), and the intermittent nocturnal hypoxaemia and fragmented sleep produce genuine deficits of attention, executive function and memory that can be mistaken for a dementia. It sits, alongside thyroid disease and vitamin deficiency, on the list of reversible medical conditions to exclude before a depression or a cognitive decline is accepted as primary (Companion to Psychiatric Studies).

The treatment-resistant-depression trap. The consequential error is to keep switching and stacking antidepressants in a depression that will not lift, when the fatigue, the un-refreshing sleep and the concentration loss are being driven by an untreated apnoea. Untreated obstructive sleep apnoea blunts antidepressant response, and the sleepy, snoring, obese, hypertensive depressive who is called treatment-resistant should be screened for it before the regimen is escalated again. The cardiometabolic tail sharpens the stakes: obstructive sleep apnoea drives hypertension, atrial fibrillation and other nocturnal dysrhythmias, heart failure, myocardial infarction and stroke, and carries insulin and leptin resistance, so the sleepiness is also a cardiovascular warning, and the daytime sleepiness itself causes motor-vehicle and workplace accidents.

RULE **Screen before you escalate.** Before a fatigued, poorly concentrating "treatment-resistant" depression is escalated again, screen the sleepy, snoring, thick-necked, hypertensive patient for obstructive sleep apnoea with STOP-BANG; a mood disorder is itself a comorbidity that lowers the diagnostic threshold.

WORKED EXAMPLE

A man in his fifties is referred with a depression that has failed three antidepressants. He is overweight, hypertensive, and complains of no energy and no focus. His wife, asked directly, says he snores loudly and she has watched him stop breathing and gasp awake. His STOP-BANG is six of eight (snoring, tiredness, observed apnoea, pressure, neck over 40 cm, male). The next step is not a fourth antidepressant but polysomnography; the AHI is thirty-four, severe, and continuous positive airway pressure lifts both the sleepiness and much of the mood.

15.7 Management: continuous positive airway pressure is the mainstay

The one treatment. Continuous positive airway pressure (CPAP) is the gold standard and most effective treatment for obstructive sleep apnoea (Kaplan and Sadock CTP 2024; Stahl). A blower delivers air through a nasal or oronasal mask at a titrated positive pressure that acts as a pneumatic splint, holding the collapsing pharynx open; it abolishes the apnoeas, lifts the oxygen desaturation, restores sleep continuity, and improves daytime sleepiness, cognition and cardiovascular outcomes, and even the most severe apnoea is usually controlled once the pressure

is set correctly. Variants exist for the patient who cannot exhale against a fixed pressure (bi-level, BPAP) or who needs an adapting pressure (auto-titrating, APAP), but the principle is one.

The one real problem is adherence. The therapeutic challenge is not efficacy but nightly use, and the striking psychiatric point is that depression is a key predictor of poor CPAP compliance (Kaplan and Sadock CTP 2024), so the untreated mood disorder and the untreated apnoea sabotage each other, and treating the depression helps the patient wear the mask. Wake-promoting agents, modafinil (in India, Modalert or Provake), armodafinil and solriamfetol, are licensed only for the residual sleepiness that persists in a minority of otherwise well-treated, adherent patients; they do nothing for the airway obstruction itself and are never a substitute for CPAP.

GOOD TO REMEMBER

- **Continuous positive airway pressure (the value):** CPAP is the mainstay and gold-standard treatment for obstructive sleep apnoea, splinting the airway open to abolish apnoeas, correct the oxygen desaturation and restore sleep; its one real limitation is adherence, and depression is a key predictor of poor compliance, so a wake-promoting agent (modafinil) treats only residual sleepiness and never replaces CPAP.

15.8 The weight, positional, oral-appliance and surgical options

The options behind CPAP. Weight loss genuinely reduces the AHI and is recommended for every overweight patient, though it is slow and unreliable and cannot be relied on alone. A mandibular-advancement oral appliance, which holds the jaw and tongue forward, is a reasonable alternative in mild-to-moderate disease or for the patient who cannot tolerate CPAP. Positional therapy (preventing supine sleep with a wedge, a back-worn bumper or an alarm) helps the patient whose apnoea is confined to lying on the back. Surgery is reserved for the patient with a correctable anatomical obstruction who has failed or declined CPAP: uvulopalatopharyngoplasty benefits perhaps a third to a half of selected patients, maxillomandibular advancement suits retrognathic anatomy, and a hypoglossal nerve stimulator (the implanted "pacemaker" that protrudes the tongue) is the newer option, with tracheostomy the historical last resort.

Alcohol and sedatives. A quiet but examinable adjunct is to remove what worsens the airway: alcohol in the evening and sedative-hypnotics both relax the pharyngeal muscles and deepen the desaturations, so they are avoided, and this is precisely where the disorder collides with everyday Indian prescribing.

OPTION	PLACE IN MANAGEMENT
CPAP (and BPAP, APAP)	**Mainstay and gold standard for moderate-to-severe disease**
Weight loss	Recommended for every overweight patient; adjunct, not sole therapy
Mandibular-advancement oral appliance	Alternative in mild-to-moderate disease or CPAP intolerance
Positional therapy	For supine-predominant apnoea
Surgery (UPPP, maxillomandibular, hypoglossal stimulator)	**Selected anatomical obstruction after CPAP fails or is declined**
Avoid alcohol and sedatives	They relax the airway and worsen desaturation

The management ladder for obstructive sleep apnoea; CPAP leads, the other options fit selected patients, and evening alcohol and sedatives are removed because they worsen the airway.

GOOD TO REMEMBER

- **The options behind CPAP (the value):** weight loss lowers the AHI in every overweight patient but is adjunct, not sole therapy; a mandibular-advancement oral appliance is the alternative in mild-to-moderate disease or CPAP intolerance; positional therapy suits supine-predominant apnoea; surgery (uvulopalatopharyngoplasty, maxillomandibular advancement, hypoglossal nerve stimulator) is reserved for a correctable anatomical obstruction after CPAP fails or is declined; and evening alcohol and sedative-hypnotics are stopped because they relax the airway and deepen the oxygen desaturation.

TRAP **A hypnotic for the "insomniac" who actually has apnoea.** Prescribing a benzodiazepine or Z-drug to a snoring, sleepy patient whose real problem is obstructive sleep apnoea is useless and dangerous: the sedative relaxes the airway further, deepens the oxygen desaturation, and worsens the disorder you failed to diagnose.

INDIA

The genuine Indian device here is under-recognition compounded by the benzodiazepine reflex. Obstructive sleep apnoea is common and badly under-diagnosed, sleep laboratories are scarce and CPAP machines are a real out-of-pocket cost, so the snoring, sleepy, thick-necked patient is too often handed a benzodiazepine or Z-drug for "insomnia", the very drug class that relaxes the airway and worsens the apnoea. Two correctives are cheap and exam-worthy: use STOP-BANG at the bedside to catch the high-risk patient before reaching for a hypnotic, and do not treat presumed insomnia in a loud snorer with a sedative until apnoea has been excluded. The lean patient with craniofacial crowding, who lacks the obesity that would prompt the thought, is the one most often missed.

HOLD THIS

Obstructive sleep apnoea is repetitive upper-airway collapse causing apnoeas and hypopnoeas (airflow gone for ten seconds or longer), oxygen desaturation and arousal, graded by the apnoea-hypopnoea index (mild 5 to 15, moderate 15 to 30, severe over 30) and confirmed on polysomnography; it masquerades as depression, cognitive impairment and treatment-resistant depression, so screen the sleepy snorer with STOP-BANG (high risk 5 to 8 of 8), and treat with CPAP as the mainstay, never a hypnotic.

16 · Parasomnias: NREM and REM including RBD

A parasomnia is an unwanted physical or experiential event that intrudes into sleep or the transitions in and out of it: not a disorder of *how much* a person sleeps but of *what happens* while they do. Kaplan and Sadock frame the whole class as a series of **state-boundary violations**, one of the three basic states (wakefulness, NREM sleep, REM sleep) leaking into another. That single idea organises the entire topic. When wakeful motor behaviour erupts out of deep NREM sleep you get the **disorders of arousal**; when the paralysis of REM sleep fails and the sleeper acts out the dream you get REM sleep behaviour disorder; when REM atonia persists into waking you get sleep paralysis.

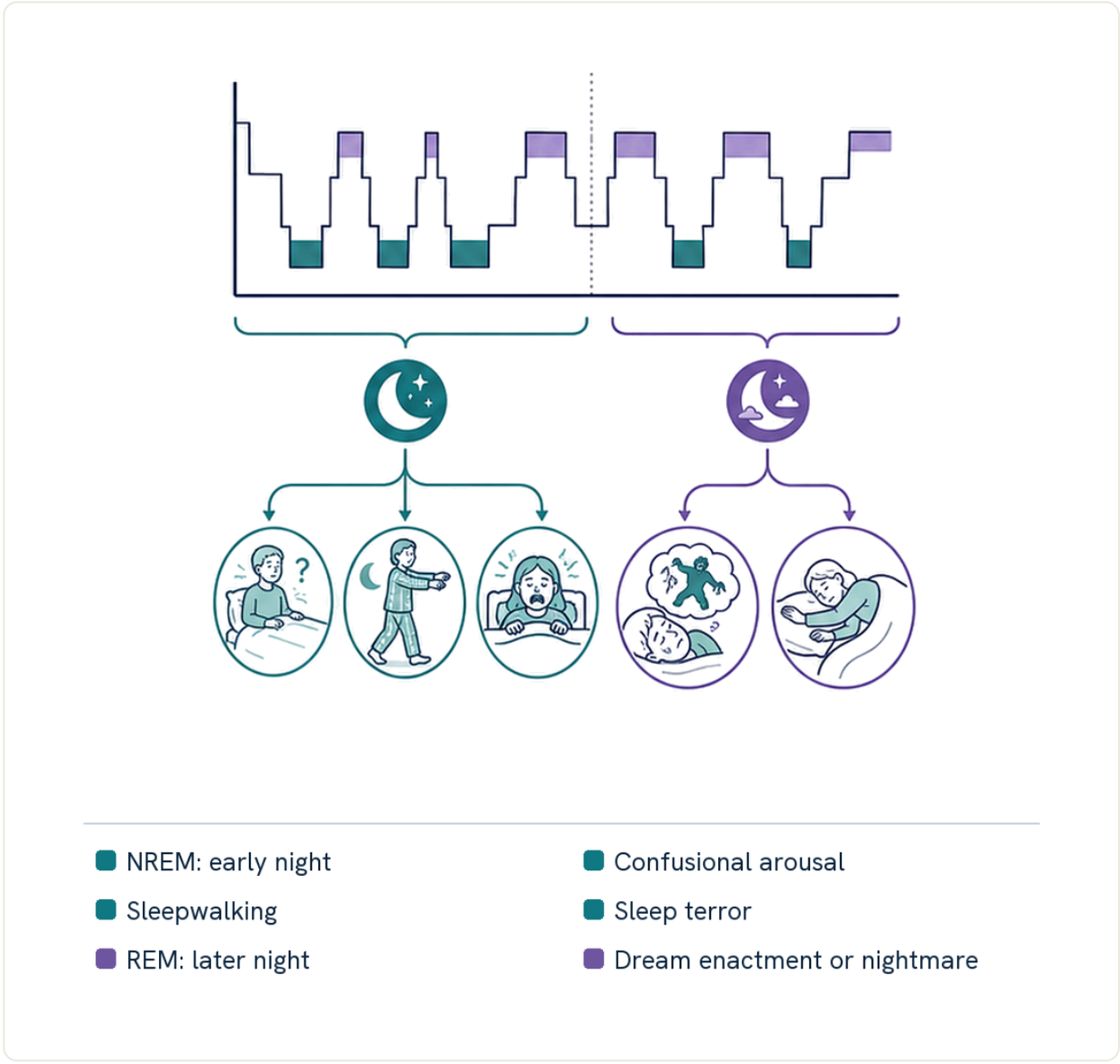


Fig 17.15 Parasomnias map onto sleep state: confusional arousals, sleepwalking and sleep terrors arise from NREM sleep, often earlier in the night, while nightmares and REM sleep behaviour disorder arise from REM sleep, often later.

Why it matters, and how this note is framed. This is a teaching-the-diagnosis topic, and the examinable spine is the split between two families: the NREM parasomnia disorders of arousal (confusional arousal, sleepwalking and sleep terror) that rise from slow-wave sleep in the first third of the night in amnestic, hard-to-rouse children, and the REM parasomnias (nightmare disorder, sleep paralysis and RBD) that belong to the dream-rich second half of the night. The one point the examiner will not let you miss, and the reason this topic outgrows its size, is that RBD is not a benign curiosity of an old man's bedroom but the strongest clinical prodrome of a synucleinopathy, cross-referenced to the Dementias and neurocognitive disorders notes and the Neurology foundations for psychiatrists notes. Two traps are set for you: reading a dream-enacting RBD as a simple nightmare (and missing the neurodegenerative risk), and reading a sleepwalking or sleep-terror event as a nocturnal seizure. The accompanying figure sets the two families against the hypnogram so the timing tells you the family.

16.1 Two families, read off the sleep stage

The organising split. Every parasomnia in the syllabus falls into one of two boxes defined by the sleep state it erupts from (Kaplan and Sadock CTP 2024). The **NREM disorders of arousal** are momentary wakeful behaviours breaking out of slow-wave (deep NREM, stage N3) sleep, so they cluster in the first third of the night when N3 is richest, and the sleeper is caught between sleep and wake with reduced awareness and dense amnesia. The **REM parasomnias** belong to REM sleep, which dominates the last third of the night, and they carry the signature of dreaming: vivid recall, and in RBD the motor enactment of the dream because the atonia has failed.

Why the timing is diagnostic. The stage a parasomnia comes from is not a detail, it is the discriminator. A frightening nocturnal event early in the night in an amnestic child who cannot be roused is almost certainly a disorder of arousal; the same story late in the night in an older man who wakes and describes fighting off an attacker is RBD until proven otherwise. Learn the two families as a pair and most single-best-answer questions on parasomnias answer themselves.

> **PEARL**
>
> Ask two questions of any nocturnal event: *when in the night* and *does the patient remember it*. First third of the night with amnesia points to a NREM disorder of arousal; last third with full dream recall (and enactment) points to a REM parasomnia. Time and recall sort the two families before you reach for a sleep study.

16.2 The NREM disorders of arousal: the shared signature

The common phenotype. The three NREM parasomnias share one pathophysiology, an incomplete arousal from slow-wave sleep, and therefore one clinical signature (DSM-5-TR 2022; Kaplan and Sadock CTP 2024). The DSM-5-TR criterion A is recurrent episodes of incomplete awakening from sleep, usually during the **first third of the major sleep episode**, with either sleepwalking or sleep-terror behaviour; the eyes are typically open, the events last one to ten minutes, there is **no or little dream imagery** recalled (criterion B), and there is **amnesia** for the episode (criterion C). The person is difficult to rouse, and if woken is confused and disoriented before full recovery.

The demographic and the triggers. These are disorders of childhood: prevalence peaks young and they usually remit spontaneously after adolescence, they run in families, and they are provoked by anything that deepens or fragments slow-wave sleep, above all sleep deprivation, but also fever, a full bladder, noise, and CNS-depressant use or withdrawal. Persistence into or new onset in adult life is less benign and raises the question of a comorbid disorder (obstructive sleep apnoea in particular) or an underlying stressor. Confusional arousals are the mild end of this same continuum, sleepwalking the middle, sleep terrors the florid extreme.

FEATURE	VALUE IN THE NREM DISORDERS OF AROUSAL
Sleep stage of origin	**Slow-wave (deep NREM, N3) sleep**
Timing in the night	**First third of the major sleep episode**
Episode duration	1 to 10 minutes (occasionally longer)

FEATURE	VALUE IN THE NREM DISORDERS OF AROUSAL
Eyes	Typically open
Dream recall	**No or little imagery recalled**
Memory for the event	**Amnesia**
Rousability	Difficult to rouse; confused if woken
Age and family history	Childhood peak, remits after adolescence, familial

The shared signature of the NREM disorders of arousal; deep-NREM origin, first-third timing, amnesia and no dream recall are what separate the whole family from the REM parasomnias.

RULE **Timing plus amnesia diagnoses the family.** A nocturnal event out of the first third of the night, in a child who is hard to rouse and has no memory of it and recalls no dream, is a NREM disorder of arousal, no sleep study needed to make that call.

GOOD TO REMEMBER

- **NREM disorders of arousal (the defining criterion):** recurrent incomplete awakenings from slow-wave (deep NREM) sleep in the first third of the night, with the eyes typically open, no or little dream imagery recalled and amnesia for the event (DSM-5-TR criteria A to C); peak in childhood, remit after adolescence, run in families, and are provoked by sleep deprivation, fever and CNS depressants.

16.3 Confusional arousals

The mild end. A confusional arousal (ICSD-3; Kaplan and Sadock CTP 2024) is the gentlest disorder of arousal: the sleeper, usually a young child, partially wakes and sits up confused, may mumble or look about, but characteristically lies back down and resumes sleep without leaving the bed and without the autonomic storm of a sleep terror. It is thought to sit on a continuum with sleepwalking and sleep terrors, sharing their slow-wave origin and their amnesia. In adults an equivalent is the disoriented, sometimes belligerent partial waking on being roused from deep sleep (classically sleep drunkenness or morning confusion), and it can be exacerbated by sedatives and sleep deprivation.

GOOD TO REMEMBER

- **Confusional arousal (the defining criterion):** a partial awakening from slow-wave sleep with confusion but *without* ambulation and without the intense fear and autonomic surge of a sleep terror; the mildest of the disorders of arousal, common in young children, on a continuum with sleepwalking and sleep terrors, and amnesic like the rest of the family.

16.4 Sleepwalking (somnambulism)

What it is. Sleepwalking, or **somnambulism**, is arising from bed and ambulating without fully awakening, out of slow-wave sleep, characteristically toward the end of the first or second slow-wave episode (Kaplan and Sadock CTP 2024). The DSM-5-TR requires repeated episodes of rising and walking with a blank, staring face, relative unresponsiveness, and great difficulty being awakened. Behaviour ranges from sitting up and fumbling to complex semi-purposeful sequences; the sleepwalker can often navigate the environment yet may interact with it

dangerously (stepping through an upstairs window, walking into a road), and injury and even acts of violence occur. It is very common in children, with peak prevalence between four and eight years of age, and rare and more clinically significant in adults, where it is familial and often secondary to another sleep disorder such as apnoea.

The management principle in one line. Do not grab, shake or shout at a sleepwalker; in their confused state they may believe they are being attacked and lash out. Guide them gently back to bed. Two clinical variants are subsumed here as the sleepwalking type: sleep-related eating (nocturnal eating with amnesia and weight gain) and sleep-related sexual behaviour (**sexsomnia**), which the DSM-5-TR classes as a NREM arousal disorder, not a REM phenomenon.

GOOD TO REMEMBER

- **Sleepwalking (the defining criterion):** repeated episodes of rising from bed and walking during slow-wave sleep with a blank stare, unresponsiveness and difficulty being awakened, and amnesia afterwards (DSM-5-TR); childhood peak four to eight years, familial, provoked by sleep deprivation; sleep-related eating and sexsomnia are its specifiers, and it is managed by gently returning the walker to bed, never by forcibly waking them.

16.5 Sleep terrors (pavor nocturnus)

What it is. A sleep terror is a sudden arousal from slow-wave sleep with intense fear, often opening with a piercing scream, in a child who sits up unresponsive, inconsolable, with florid autonomic arousal (mydriasis, tachycardia, rapid breathing, sweating) and who is amnesic for the event afterwards (DSM-5-TR 2022; Kaplan and Sadock CTP 2024). Unlike a nightmare there is no unfolding dream narrative, at most a single fragmentary frightening image, and the child cannot be comforted during the episode and does not remember it in the morning. Also called **pavor nocturnus** or night terror, it is familial, provoked by sleep deprivation, fever and CNS-depressant withdrawal, and psychopathology is seldom associated in children (though a history of trauma or psychiatric problems is more often comorbid when it presents in adults).

The two things it is confused with. A sleep terror is confused with a nightmare (a REM event with full recall, from the second half of the night, no autonomic storm and the child is easily consoled and remembers it) and, more dangerously, with a nocturnal seizure. For children whose sleep terrors are frequent and distressing, scheduled awakenings shortly before the usual time of the event can pre-empt them (Rutter).

COMMON TRAP

The screaming, thrashing, inconsolable child who is amnesic in the morning is a sleep terror, a NREM parasomnia, not a nightmare and not necessarily an epileptic event, but seizure sits at the top of the differential for *any* stereotyped nocturnal behaviour. The discriminators: a sleep terror varies in content and is provoked by sleep deprivation and fever, whereas nocturnal seizures tend to repeat the same stereotyped semiology and are captured on the polysomnogram's EEG leads. When in doubt, the study that settles it is a polysomnogram with seizure montage.

GOOD TO REMEMBER

- **Sleep terror (the defining criterion):** abrupt arousal from slow-wave sleep with a panicky scream, intense fear and marked autonomic arousal, inconsolable during the event, no or only a fragmentary image recalled, and amnesia afterwards (DSM-5-TR); distinguished from a nightmare (REM, full recall, consolable) and from a nocturnal seizure (stereotyped, EEG-positive) which is the discriminator that must not be missed.

16.6 The REM parasomnias: nightmare disorder and sleep paralysis

Nightmare disorder. Nightmares are frightening, elaborate dreams that arise in REM sleep and therefore in the last third of the night, growing more terrifying until they awaken the dreamer, who comes fully awake and **remembers the dream in detail** (Kaplan and Sadock CTP 2024). That full recall and rapid return to alert orientation is the single feature that separates a nightmare from a sleep terror. Nightmares are common in children aged three to six and rare in adults, and become nightmare disorder when they are frequent and distressing enough to breed a fear of sleeping. Post-traumatic nightmares are a special case; the evidence-supported drug there is prazosin, a central alpha-1 antagonist, for PTSD-related nightmares.

Sleep paralysis. Isolated sleep paralysis is the persistence of REM atonia into the wake transition: for seconds to a couple of minutes at sleep onset or on waking the person is conscious and aware of the surroundings but unable to move or speak, often with a frightening sense of a presence in the room (Kaplan and Sadock CTP 2024). It is common and usually benign in isolation, but recurrent sleep paralysis with hypnagogic hallucinations should prompt a thought about narcolepsy, cross-referenced to the sleep neurotransmitter systems in the Functional Neuroanatomy and Neurophysiology notes.

GOOD TO REMEMBER

- **Nightmare disorder (the defining criterion):** repeated frightening, well-remembered dreams arising from REM sleep in the second half of the night, with rapid full awakening and detailed recall (the opposite of the amnestic, autonomic sleep terror), causing distress or fear of sleep; sleep paralysis is REM atonia intruding into waking, benign in isolation but a pointer to narcolepsy when it recurs with hypnagogic hallucinations.

16.7 REM sleep behaviour disorder: the atonia fails and the dream is enacted

The mechanism. During normal REM sleep the perilocus-coeruleus nuclei impose atonia (flaccid quadriparesis and areflexia) so that the vivid dreaming brain cannot move the body (Kaufman; Kaplan and Sadock CTP 2024). In **RBD** that atonia fails, intermittently or completely, and the sleeper enacts the dream: punching, kicking, leaping, running, shouting and screaming, frequently injuring themselves or the bed partner. Because it is a REM phenomenon it belongs to the last half of the night, the eyes are closed, and when woken the patient is quickly alert and can recount the dream, which characteristically involved defending against or fleeing an attack. The contrast with sleepwalking is instructive: a sleepwalker may calmly open a window and step out, whereas a person with RBD, acting on the dream sensorium rather than the real room, would dive through it believing it a lake.

Who gets it. RBD is overwhelmingly a disorder of **older men (usually over 65 years)** (Kaufman). It may be idiopathic, drug-induced (many antidepressants, the SSRIs, SNRIs such as venlafaxine, tricyclics and MAOIs, can precipitate or unmask it by allowing muscle activity during REM), or, most importantly for the examiner, the harbinger of a neurodegenerative disease.

FEATURE	NREM DISORDER OF AROUSAL	RBD (REM PARASOMNIA)
Sleep stage	Slow-wave (deep NREM, N3)	**REM sleep**
Time of night	First third	**Last half**
Typical age and sex	Children, either sex	**Men over 65 years**
Eyes	Typically open	Closed
Dream recall	None or a fragment; amnesic	**Recalls the dream; alert on waking**
Behaviour	Ambulation, navigates the real room	**Dream enactment, acts on the dream**
Ominous association	Benign; remits	**Prodrome of a synucleinopathy**

The load-bearing comparison; the NREM disorders of arousal and RBD differ on stage, timing, age, eyes, recall and, decisively, on what they portend.

GOOD TO REMEMBER

- **RBD (the defining criterion):** loss of the normal REM-sleep atonia so that the patient enacts the dream (punching, kicking, leaping, shouting) with the eyes closed in the last half of the night, is quickly alert on waking and recalls a dream of being attacked; typically a man over 65, in contrast to the amnestic, eyes-open, first-third NREM disorders of arousal.

16.8 The critical point: RBD is a prodrome of a synucleinopathy

The link that makes this topic examinable. RBD is now regarded as the single strongest clinical prodrome of the **synucleinopathies**, the neurodegenerative diseases in which alpha-synuclein aggregates: Parkinson's disease, dementia with Lewy bodies and multiple system atrophy (Kaufman; Tasman Psychiatry). The dream-enactment often precedes the motor or cognitive disease by years, so an older man with new RBD is carrying a warning of neurodegeneration well before the tremor, the parkinsonism or the fluctuating cognition and visual hallucinations appear. The relationship is close and specific: RBD develops in the course of Parkinson's disease in roughly 30 to 50 per cent of cases and in dementia with Lewy bodies in 50 to 80 per cent, and conversely a synucleinopathy declares itself in as many as half of RBD patients within three to ten years of the sleep disorder (longer follow-up pushes the conversion figure higher still). The point is cross-referenced to the Dementias and neurocognitive disorders notes and the Neurology foundations for psychiatrists notes.

The discriminating subtlety. This specificity is diagnostic gold. RBD tracks with the *synucleinopathies* and only rarely complicates the tauopathies such as Alzheimer's disease and frontotemporal dementia (Kaufman). So dream enactment in a patient with an evolving dementia

is itself a pointer toward Lewy body disease rather than Alzheimer's. It is diagnostically weighty enough that when RBD is comorbid with a known synucleinopathy the DSM-5-TR does not even require polysomnographic confirmation of the REM sleep.

MNEMONIC

RBD warns of PDM

Parkinson's disease, **D**ementia with Lewy bodies, **M**ultiple system atrophy: the three synucleinopathies RBD heralds. If you see dream enactment in an older man, think alpha-synuclein and watch for parkinsonism and fluctuating cognition, not for the tau of Alzheimer's disease.

TRAP **Filing RBD under nightmare disorder.** An older man who "acts out his nightmares" is not having a nightmare disorder (which has no motor enactment and no neurodegenerative risk); he has RBD, and calling it a nightmare buries the single most important thing about him, that he is showing a prodrome of a synucleinopathy years before the neurology declares itself.

1 to 10	≈50	>65	1
NREM AROUSAL EPISODE LENGTH (MINUTES)	RBD PHENOCONVERTING WITHIN 3 TO 10 YEARS (PER CENT)	TYPICAL RBD ONSET AGE (YEARS)	CLONAZEPAM BEDTIME DOSE IN RBD (MG)

16.9 Safety first, then the drugs: managing the parasomnias

Safety is the shared first step. Across both families the first intervention is not a drug but the bedroom: because the injury risk is real in sleepwalking and considerable in RBD, the environment is made safe (locking windows and doors, removing sharp or breakable objects, moving the mattress low, and for RBD the bed partner often sleeping separately) (Kaplan and Sadock CTP 2024; Kaufman). For the NREM disorders of arousal the mainstay is reassurance of the family, protecting slow-wave sleep by **avoiding sleep deprivation**, treating the provokers (obstructive sleep apnoea, fever, a full bladder, offending sedatives), and, for a child with frequent sleep terrors, scheduled awakenings; drugs are seldom needed and are reserved for severe, injurious or highly disruptive cases, where a bedtime benzodiazepine (clonazepam) or an antidepressant may be used.

The pharmacology of RBD. RBD, by contrast, usually does need a drug because of the injury risk, and here two agents carry the topic (Kaufman). Clonazepam at a low bedtime dose (the parsed texts quote 1 mg at night, in practice titrated in the 0.5 to 2 mg range) reduces or abolishes the episodes so reliably that a good response supports the diagnosis. High-dose melatonin at night is the other mainstay and is often preferred first, particularly in the very patients who have RBD, older people and those with parkinsonism or dementia, where clonazepam's sedation, confusion and fall risk are least welcome. Both agents, and the wider question of the benzodiazepines, the z-drugs and melatonin, belong to the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes; a further step is to review and, where possible, stop any antidepressant that may be unmasking the RBD.

RULE **Secure the bedroom before you reach for a prescription.** In every parasomnia the first-line intervention is environmental safety and, for the NREM disorders of arousal, protecting sleep and removing triggers; medication (clonazepam or melatonin) is for RBD and for the severe, injurious NREM case, not the routine one.

INDIA

The genuine Indian problem in parasomnias is on the RBD side. Polysomnography and the sleep laboratories that would confirm the loss of REM atonia are scarce and out-of-pocket, so an older man's dream enactment is far more likely to be dismissed as "just nightmares" than recognised, and the neurodegenerative warning it carries is lost. Layered on this is India's liberal use of benzodiazepines: clonazepam is among the most freely prescribed drugs in the country, which cuts two ways, it means an effective RBD treatment is cheap and available, but it also means a benign childhood NREM parasomnia that needed only reassurance and a safe bedroom can end up medicated with a benzodiazepine it never required. Sleep hygiene, and specifically protecting sleep against the deprivation that provokes the disorders of arousal, is the cheapest and most under-used intervention of all.

GOOD TO REMEMBER

- **Management (the value):** environmental safety and trigger removal come first in every parasomnia; the NREM disorders of arousal need reassurance, protected sleep and (for children with sleep terrors) scheduled awakenings rather than drugs, while RBD is treated pharmacologically with clonazepam (about 1 mg at bedtime) or high-dose melatonin (often preferred in the elderly and in those with parkinsonism to avoid clonazepam's falls and sedation), alongside stopping any antidepressant that may be unmasking it.

HOLD THIS

Read a parasomnia off the sleep stage: NREM disorders of arousal (confusional arousal, sleepwalking, sleep terror) rise from slow-wave sleep in the first third of the night in amnestic, hard-to-rouse, eyes-open children who recall no dream and are managed with safety and reassurance; REM parasomnias belong to the dream-rich second half, and REM sleep behaviour disorder (lost REM atonia, dream enactment, eyes closed, dream recalled, men over 65) is the one that matters most because it is a prodrome of a synucleinopathy (Parkinson's disease, dementia with Lewy bodies, multiple system atrophy), phenoconverting in up to half of patients within three to ten years, and is treated with clonazepam (about 1 mg at night) or high-dose melatonin.

17 · Circadian rhythm sleep-wake disorders

A **circadian rhythm sleep-wake disorder** is a persistent mismatch between the endogenous circadian clock and the sleep-wake schedule the person wants or is required to keep. The clock itself, the suprachiasmatic nucleus, is intact and keeps near-perfect time; the fault is one of *alignment*, so the patient sleeps well but at the wrong hour of the twenty-four. That single idea is the examinable heart of the group, and it is also the diagnostic discriminator: when a person with one of these disorders is allowed to sleep on their own preferred schedule, the quantity and quality of sleep are normal.

Why it matters, and how this note is framed. The circadian disorders are the mimic that a hypnotic will fail, so recognising them cleanly saves the patient a pointless sleeping tablet, and their management is a small, elegant piece of physiology rather than a drug ladder: light therapy and melatonin are timed against the phase-response curve to push the clock the right way. This is a management topic, so the treatment half leads with the British Association for Psychopharmacology position (BAP 2019), whose consensus statement explicitly covers circadian rhythm disorders, and then states what the sleep-medicine guideline recommends per type; the specific per-type light-and-melatonin timings and doses are not in the verified drug supplement, so they are grounded on the American Academy of Sleep Medicine clinical practice guideline (AASM 2015, Auger et al.) and Kaplan and Sadock and flagged as such. The one rule to carry throughout is the direction: advance the clock with morning light, delay it with evening light, and treat melatonin as the mirror image.

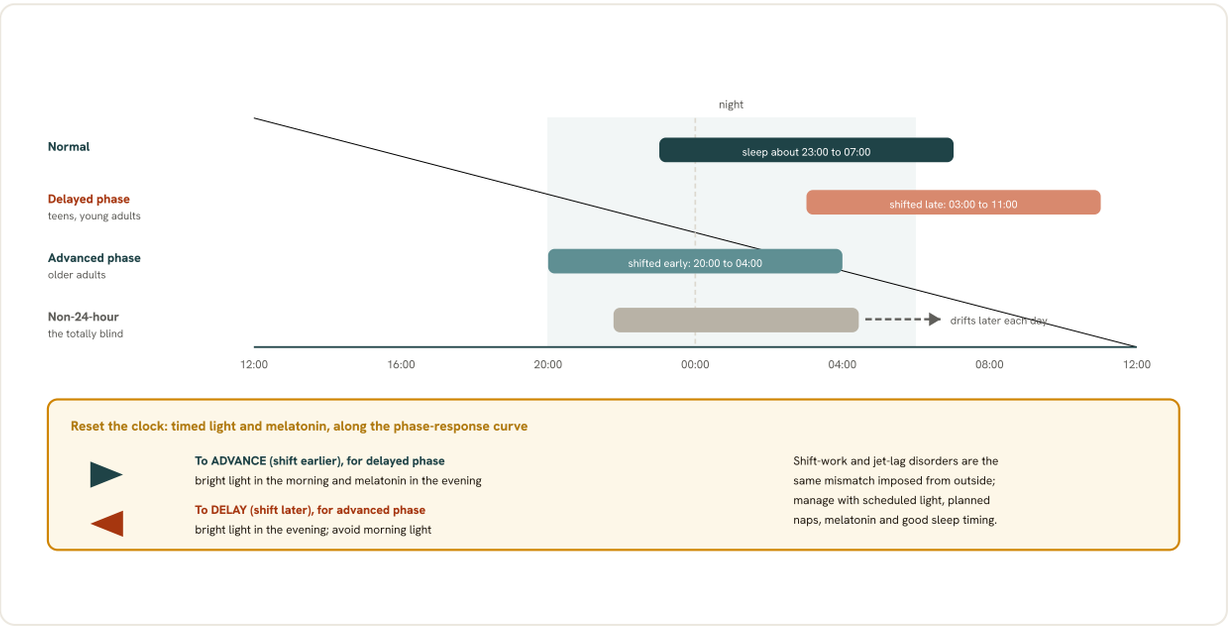


Fig 17.16 The circadian rhythm sleep-wake disorders. Each is a mismatch between the internal circadian clock and the desired or required sleep-wake schedule. In delayed sleep-wake phase disorder, common in adolescents, the whole sleep window is shifted late, so the person cannot fall asleep or wake at conventional times; in advanced sleep-wake phase disorder, seen in older adults, it is shifted early, with evening sleepiness and pre-dawn waking; in non-24-hour disorder, typical of the totally blind who lack light entrainment, the clock drifts a little later each day; and shift-work and jet-lag disorders are the same misalignment imposed from outside. Management resets the clock along the phase-response curve: morning bright light and evening melatonin advance the clock (for the delayed type), while evening light delays it (for the advanced type), supported by scheduled light, planned naps and consistent sleep timing.

17.1 The concept: a clock that keeps good time but points the wrong way

The shared mechanism. Every disorder in this collection shares one aetiology: desynchrony between the internal circadian pacemaker and the desired sleep-wake cycle (Kaplan and Sadock CTP 2024). The pacemaker sits in the suprachiasmatic nucleus, oscillates with a period of about twenty-four hours, and its output tracks the daily swing in core body temperature. The mismatch can arise three ways, an improper phase relationship between the clock and the schedule (the phase is shifted early or late), travel across time zones, or an intrinsic fault in the rhythm itself (the clock free-runs or is flattened).

The discriminator from insomnia. The tell that separates a circadian disorder from insomnia disorder is that sleep is normal in amount and quality when it is timed to the patient's own biological schedule; the delayed-phase adolescent who cannot fall asleep at a socially normal hour sleeps perfectly well if left to sleep late (Kaplan and Sadock CTP 2024). Insomnia persists whenever the person tries to sleep; a circadian disorder resolves the moment the schedule is allowed to follow the clock.

> **PEARL**
>
> Ask the single screening question: "when you are on holiday and can sleep and wake whenever you like, is your sleep normal?" A yes points to a circadian rhythm sleep-wake disorder, not insomnia. The clock is not broken, it is simply set to the wrong time.

17.2 The diagnostic criteria, the subtypes, and the duration specifiers

The three criteria. To diagnose a circadian rhythm sleep-wake disorder there must be (a) an alteration of, or a misalignment between, the endogenous circadian rhythm and the required sleep-wake schedule, (b) sleep disruption producing insomnia, excessive daytime sleepiness, or both, and (c) resulting distress or social, occupational or other functional impairment (DSM-5-TR; Kaplan and Sadock CTP 2024). ICD-11 moves the whole sleep-wake group out of the mental-disorders chapter into its own dedicated chapter, reflecting that the pathophysiology is a body-clock problem rather than a primary psychiatric one.

The subtypes and the specifiers. DSM-5-TR codes six subtypes: delayed sleep-wake phase type, advanced sleep-wake phase type, irregular sleep-wake type, non-24-hour sleep-wake type, shift work type, and unspecified type. The jet lag type is listed in the sleep-medicine classification (ICSD-3) but was *not* carried into DSM-5-TR, a favourite examiner point. Each is further specified by course: episodic (symptoms one month or more but under three months), persistent (three months or longer), or recurrent (two or more episodes within a one-year period) (Kaplan and Sadock CTP 2024).

ELEMENT	REQUIREMENT FOR A CIRCADIAN RHYTHM SLEEP-WAKE DISORDER
Core mechanism	**Alteration of, or misalignment between, the endogenous circadian rhythm and the required sleep-wake schedule**
Sleep disruption	Insomnia, excessive daytime sleepiness, or both
Consequence	Clinically significant distress or functional impairment
Discriminator	**Sleep is normal in amount and quality when timed to the patient's own preferred schedule**
Subtypes (DSM-5-TR)	Delayed phase, advanced phase, irregular, non-24-hour, shift work, unspecified (jet lag is ICSD-3, not DSM-5-TR)
Course specifier	Episodic (one to under three months), persistent (three months or longer), recurrent (two or more episodes in a year)

The circadian rhythm sleep-wake disorder criteria; the load-bearing points are the misalignment mechanism, the normal-sleep-on-own-schedule discriminator, and the persistent-versus-episodic three-month line.

GOOD TO REMEMBER

- **Circadian rhythm sleep-wake disorder (defining criterion):** a persistent misalignment between the endogenous circadian clock and the required sleep-wake schedule that produces insomnia or excessive sleepiness plus impairment, with the discriminating feature that sleep is *normal when timed to the patient's own preferred schedule*; DSM-5-TR codes six subtypes and specifies persistent when three months or longer, and jet lag sits in ICSD-3 but not DSM-5-TR.

17.3 Delayed sleep-wake phase type: the adolescent night owl

The picture. In the **delayed sleep-wake phase** type the biological clock runs slower than twenty-four hours or is set later than the desired schedule, producing a phase delay in the sleepiness-alertness cycle. These patients are alert late into the evening, fall asleep only in the small hours (habitual onset around two to six in the morning) and wake late (around ten in the morning to one in the afternoon), and are the classic "night owls" (Kaplan and Sadock CTP 2024). It is the commonest of the intrinsic circadian disorders, affecting roughly two percent of the population, and is overrepresented in adolescents and young adults because puberty-related changes in sleep homeostasis push the clock later just as inflexible school and college timings demand an early start (New Oxford Textbook of Psychiatry). Forced to a conventional schedule they present as sleep-onset insomnia with morning grogginess; left to their own timing they sleep normally.

GOOD TO REMEMBER

- **Delayed sleep-wake phase type (the criterion):** a stable, later-than-desired sleep window (onset around two to six in the morning) with normal sleep on the patient's own schedule; the population is the adolescent and young adult night owl, prevalence about two percent, and management advances the clock with morning light plus early-evening melatonin.

17.4 Advanced sleep-wake phase type: the early-rising older adult

The picture. The **advanced sleep-wake phase** type is the mirror: the clock is set earlier, so the patient becomes sleepy in the early evening (sleep onset around six to nine in the evening) and wakes several hours before they wish to (around two to five in the morning), unable to return to sleep (New Oxford Textbook of Psychiatry). The complaint is therefore early-morning waking and an inability to stay awake for evening social life. Prevalence rises with age, and an age-related phase advance, a shortened endogenous circadian period, and increased retinal sensitivity to morning light are the proposed mechanisms. A familial advanced-phase form shows autosomal dominant inheritance, classically a PER2 gene mutation causing hypophosphorylation of the PER2 protein, and a missense mutation in casein kinase I (Kaplan and Sadock CTP 2024).

GOOD TO REMEMBER

- **Advanced sleep-wake phase type (the criterion):** an earlier-than-desired sleep window (onset around six to nine in the evening, waking around two to five in the morning) presenting as early-morning waking; the population is the older adult, the familial form is autosomal dominant with a PER2 mutation, and management delays the clock with timed evening bright light.

17.5 Non-24-hour and irregular sleep-wake types: the blind and the neurologically impaired

Non-24-hour type. Here the pacemaker has a cycle length different from twenty-four hours and, crucially, is not reset each morning, so it free-runs and drifts steadily out of phase with the environment (Kaplan and Sadock CTP 2024). It is the disorder of the *totally blind*, in whom the loss of light input to the suprachiasmatic nucleus removes the daily photic reset. Sleep-onset insomnia and daytime sleepiness worsen progressively until clock and environment are furthest apart, then briefly normalise as they realign, giving the characteristic cycling that earned the older name periodic insomnia and periodic excessive sleepiness.

Irregular sleep-wake type. In the irregular type the circadian rhythm is absent or pathologically diminished, so sleep is scattered into three or more short episodes across the twenty-four hours with no consolidated night; the total sleep amount is normal but its temporal organisation is lost (Kaplan and Sadock CTP 2024). It is seen with neurodegeneration and dementia and with neurodevelopmental disorder, where the driving oscillator and the daily zeitgebers are both weak. Long daytime naps alternate with inappropriate nocturnal wakefulness, and daily function is significantly impaired.

GOOD TO REMEMBER

- **Non-24-hour type (the criterion):** a free-running clock not reset each morning, the disorder of the *totally blind*, giving cyclically worsening then briefly normalising insomnia and sleepiness; managed with timed melatonin or the melatonin agonist tasimelteon.
- **Irregular sleep-wake type (the criterion):** an absent or flattened rhythm with *three or more* fragmented sleep bouts across the day and no consolidated night (total sleep normal), typical of dementia and neurodevelopmental disorder; managed with morning bright light and a structured daily routine.

17.6 Shift work and jet lag types: the extrinsic, socially imposed disorders

Shift work type. This is diagnosed by the history of recent **shift work**: the person must sleep and stay awake against their circadian rhythm, giving insomnia when they try to sleep by day and excessive sleepiness when they must work by night, with profound cumulative sleep deprivation (Kaplan and Sadock CTP 2024). The natural low point of the sleep-wake rhythm falls at about three to five in the morning, which is precisely when transport and industrial accidents cluster, so the disorder is a safety issue as much as a sleep complaint. Weekend reversion to a day schedule keeps the underlying rhythm stubbornly unshifted.

Jet lag type. Rapid travel across several time zones induces a transient misalignment: eastward travel requires a phase advance and westward travel a phase delay (Kaplan and Sadock CTP 2024). Eastward flights are harder to adjust to (and harder still for "owls") because advancing the clock is intrinsically difficult. Healthy people re-entrain at roughly one to two time zones per day, so an eight-hour translocation takes about four or more days to settle. Jet lag disorder is classified in ICSD-3 but, again, is not a DSM-5-TR subtype.

GOOD TO REMEMBER

- **Shift work type (the criterion):** insomnia and sleepiness driven by a work schedule that opposes the circadian rhythm (recent shift-work history is the anchor), with the circadian nadir and accident window at about three to five in the morning; managed with planned napping, timed melatonin and modafinil for the sleepiness.
- **Jet lag type (the criterion):** transient misalignment after crossing multiple time zones, eastward travel needing a phase advance (harder) and westward a phase delay, re-entraining at about one to two zones per day; an ICSD-3 entity absent from DSM-5-TR.

17.7 Assessment: the sleep diary, actigraphy, and the phase markers

The two workhorse tools. The diagnosis is largely clinical, confirmed by a prospective **sleep** diary kept over one to two weeks, which exposes the stable early or late timing that a single consultation misses, supplemented by actigraphy, a wrist accelerometer that objectively logs the rest-activity pattern over the same period (Kaplan and Sadock CTP 2024; New Oxford Textbook of Psychiatry). Together they show that the sleep is normal in structure but consistently mistimed.

The phase markers that let you time treatment. To give light and melatonin correctly the clinician must estimate the patient's circadian phase, and the two most robust markers are the core body temperature nadir and the dim light melatonin onset (DLMO), the rise in endogenous melatonin measured under dim light (New Oxford Textbook of Psychiatry). As a rule of thumb the temperature nadir falls about two hours before habitual waking, and the DLMO begins about two to three hours before habitual sleep onset. These are not academic: light and melatonin shift the clock in opposite directions on either side of these markers, so mis-estimating the phase means pushing the clock the wrong way.

Zeitgeber

An external time cue that entrains the clock; light is the most powerful.

Phase advance

Shifting the sleep-wake cycle *earlier* (the fix for delayed phase).

Phase delay

Shifting the sleep-wake cycle *later* (the fix for advanced phase).

DLMO

Dim light melatonin onset, the endogenous melatonin rise about two to three hours before habitual sleep onset; a key phase marker.

Core temperature nadir

The lowest point of core body temperature, about two hours before habitual waking; the pivot of the light phase-response curve.

GOOD TO REMEMBER

- **Assessment (the first step):** a one-to-two-week prospective sleep diary plus actigraphy establishes the mistimed-but-normal pattern; the dim light melatonin onset (about two to three hours before habitual sleep onset) and the core body temperature nadir (about two hours before waking) are the two phase markers used to time light and melatonin correctly.

17.8 Management: the phase-response curve, and timed light therapy

The governing rule. The clock is reset each day chiefly by bright light acting as the dominant zeitgeber, and the *timing* of light decides the direction of the shift. Referenced to the core body temperature nadir, light given *after* the nadir advances the clock, while light given *before* the nadir delays it (Kaplan and Sadock CTP 2024). Translated to the clinic this is the one thing to memorise: morning bright light advances the clock and treats the delayed-phase patient, and evening bright light delays the clock and treats the advanced-phase patient (New Oxford Textbook of Psychiatry). Therapeutic intensity is roughly two thousand five hundred to ten thousand lux, and the blue spectrum is the most potent ingredient.

What the guidelines say, by type. BAP 2019 endorses appropriately timed light and melatonin for the circadian disorders as the core approach (BAP 2019). The sleep-medicine guideline is type-specific: it supports timed evening bright light for advanced sleep-wake phase disorder; for delayed sleep-wake phase disorder in adults it could not recommend light as a stand-alone on current evidence quality, though it endorses it combined with behavioural measures in children and adolescents; and for the irregular type in older adults with dementia it supports morning bright light while advising against sleep-promoting medication (AASM 2015). The types, populations and first-line levers are set out in the figure alongside and the table below.

RULE **Morning light advances, evening light delays.** Timed bright light is the primary tool; give it in the morning to phase-advance a delayed clock and in the evening to phase-delay an advanced clock. Relative to the core temperature nadir: light after the nadir advances, light before it delays.

MNEMONIC

AM ADVANCES, PM POSTPONES

AM light **A**dvances the clock (earlier sleep, treats delayed phase); **P**M light **P**ostpones it (later sleep, treats advanced phase). Melatonin is the mirror image, so flip the timing for the tablet.

SUBTYPE	TYPICAL POPULATION	SLEEP-WAKE PATTERN	FIRST-LINE MANAGEMENT (PER PHASE-RESPONSE CURVE)
Delayed sleep-wake phase	**Adolescents, young adults ("night owls")**	Onset around 2 to 6 in the morning, wake around 10 in the morning to 1 in the afternoon	**Advance: morning bright light plus early-evening melatonin (0.5 to 3 mg)**
Advanced sleep-wake phase	Older adults; autosomal dominant familial form (PER2)	Onset around 6 to 9 in the evening, wake around 2 to 5 in the morning	**Delay: timed evening bright light**
Non-24-hour	The totally blind	Sleep drifts progressively later, cycling insomnia and sleepiness	Timed melatonin, or the melatonin agonist tasimelteon
Irregular sleep-wake	Dementia, neurodevelopmental disorder	Three or more fragmented sleep bouts, no consolidated night	Morning bright light plus a structured routine and daytime activity
Shift work	Night and rotating-shift workers	Daytime insomnia, night-time sleepiness; nadir 3 to 5 in the morning	Planned napping, timed melatonin, modafinil for sleepiness
Jet lag (ICSD-3)	Multi-time-zone travellers	Transient misalignment; eastward is the harder advance	Timed light and melatonin toward destination time; adapts about 1 to 2 zones per day

The six circadian subtypes with population and first-line management; the pattern to hold is that delayed phase is advanced (morning light, evening melatonin) and advanced phase is delayed (evening light).

2	2500 to 10000	0.5 to 3	1.5 to 6.5
DELAYED SLEEP-WAKE PHASE PREVALENCE (%)	THERAPEUTIC BRIGHT-LIGHT INTENSITY (LUX)	MELATONIN DOSE, DELAYED PHASE (MG)	MELATONIN TIMING BEFORE BEDTIME (HOURS)

GOOD TO REMEMBER

- **Light therapy (the value):** timed bright light (about two thousand five hundred to ten thousand lux, blue spectrum most potent) is the primary lever; *morning light advances* the clock for delayed phase and *evening light delays* it for advanced phase (relative to the core temperature nadir, after the nadir advances and before it delays); BAP 2019 endorses timed light, and AASM 2015 supports evening light for advanced phase and morning light for the irregular type in dementia.

17.9 Management: timed melatonin, modafinil, chronotherapy, and the Indian frame

Melatonin, the mirror of light. Melatonin (in India, Meloset) is the internal signal of darkness: endogenous levels rise at dusk and stay high until dawn, and bright light suppresses them (Kaplan and Sadock CTP 2024). Its phase-response curve is roughly the mirror image of light's, so *evening* melatonin advances the clock while *morning* melatonin delays it. For delayed sleep-wake phase disorder, small doses (0.5 to 3 mg) taken about one and a half to six and a half hours

before habitual bedtime, that is before the dim light melatonin onset, reliably advance the clock; earlier timing within that window works best (New Oxford Textbook of Psychiatry). Combined with morning bright light, this is the standard advance package for the delayed-phase adolescent, and the sleep-medicine guideline recommends timed melatonin for delayed phase across adults, children and adolescents (AASM 2015). For the non-24-hour disorder of the totally blind, timed melatonin, or the melatonin agonist tasimelteon, re-entrains the free-running clock; tasimelteon is licensed for this abroad but is not marketed in India, so only the generic is named here.

Modafinil and chronotherapy. For the excessive sleepiness of shift work type, the wake-promoter modafinil (in India, Modalert or Provake) is approved to treat the sleepiness, alongside planned napping and melatonin (Kaplan and Sadock CTP 2024). Chronotherapy, progressively phase-delaying bedtime by two to three hours each night until the clock reaches the desired schedule, exploits the natural human tendency to drift later and so is easier than forcing an advance, but it is cumbersome, disrupts a week of school or work, and has been largely overshadowed by light and melatonin. The receptor pharmacology of melatonin, ramelteon and the hypnotics is developed in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes, and the ascending-arousal and sleep neurotransmitter systems in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes and the Functional Neuroanatomy and Neurophysiology notes.

WORKED EXAMPLE

A seventeen-year-old cannot fall asleep before three in the morning, sleeps soundly until noon at weekends, but is failing college for chronic lateness and morning grogginess. This is delayed sleep-wake phase disorder, and the clock needs advancing. The plan advances it from both ends: bright light (or simply bright morning sunlight) on waking to phase-advance, and low-dose melatonin (0.5 to 3 mg) in the early evening, before the dim light melatonin onset, to advance from the other side, with the wake time then held steady. A hypnotic would be the wrong tool entirely.

TRAP **A hypnotic for a clock problem, or the timing reversed.** Prescribing a sleeping tablet for what is a circadian misalignment treats the symptom and not the cause, and giving light or melatonin at the wrong time pushes the clock the wrong way; the patient who sleeps normally on their own schedule needs the clock re-timed, not sedated.

INDIA

The genuine Indian device here is *shift work type*. India's large IT, business-process-outsourcing and healthcare workforces run night and rotating shifts, often serving Western time zones, so shift-work disorder is common and under-recognised, dismissed as ordinary tiredness. Two practical Indian realities shape management: melatonin (Meloset) is inexpensive and available over the counter, so timed low-dose melatonin is easy to deploy, while formal light boxes are scarce, which makes free, abundant morning sunlight the most accessible phototherapy for advancing a delayed clock. The trap to avoid is the widespread habit of using benzodiazepines to force sleep at the wrong biological hour, which sedates without re-timing and drifts into dependence; the fix is scheduling, timed light and melatonin, not a hypnotic.

HOLD THIS

A circadian rhythm sleep-wake disorder is a clock that keeps good time but points the wrong way, so sleep is normal on the patient's own schedule; recognise it, phase it with a sleep diary, actigraphy and the melatonin and temperature markers, then re-time the clock with the phase-response curve, morning light advances and evening light delays, with melatonin as the mirror (evening melatonin advances), modafinil for shift-work sleepiness, and never a hypnotic for what is a timing problem.

18 · Restless legs syndrome and PLMD

Two movement disorders of the night, one waking and one asleep. **Restless legs syndrome** (restless legs syndrome, RLS, historically Ekbom syndrome) and **periodic limb movement disorder** (periodic limb movement disorder, PLMD) are the two sleep-related movement disorders an examiner expects you to separate cleanly. RLS is a *waking* clinical diagnosis: a sensory urge to move the legs that comes on at rest and in the evening and eases with movement, diagnosed from the history alone. PLMD is an *asleep* polysomnographic diagnosis: repetitive stereotyped leg jerks recorded on the sleep study that fragment sleep and cause daytime sleepiness. They overlap heavily but are not the same thing, and the discrimination is the whole examinable point.

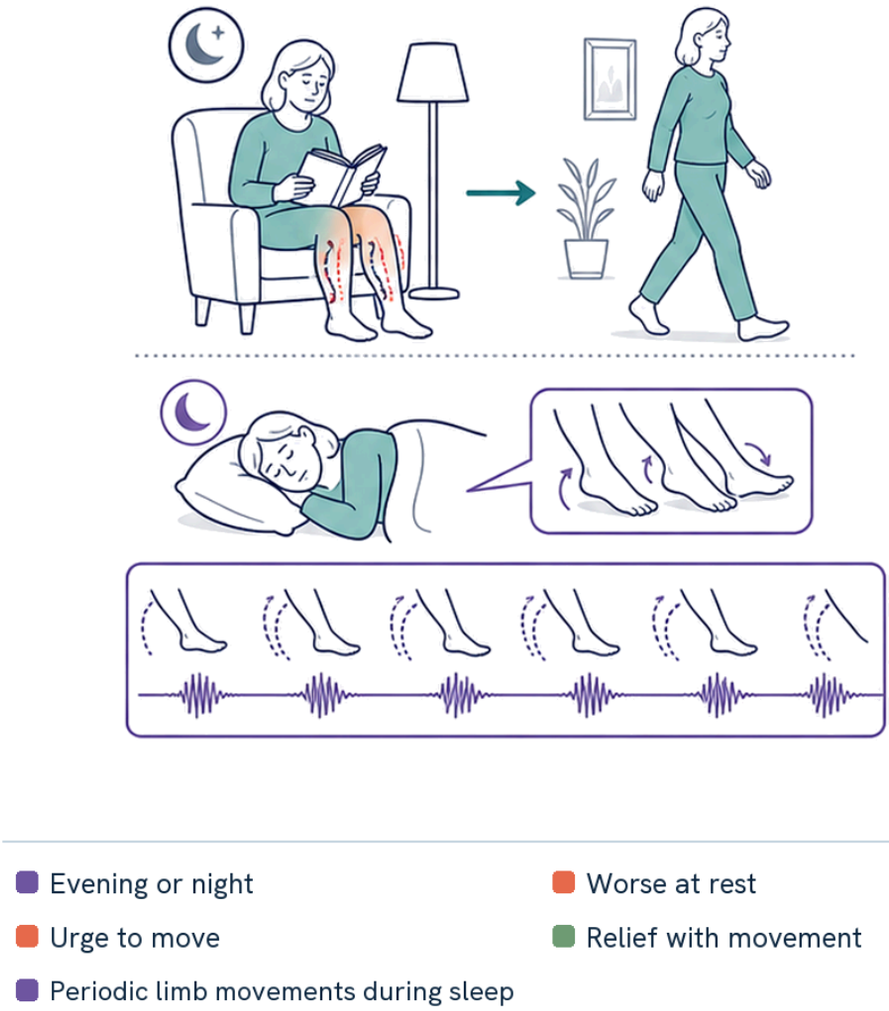


Fig 17.17 Restless legs syndrome is an urge to move with unpleasant sensations, worse at rest and in the evening and relieved by movement. Periodic limb movements are stereotyped recurrent movements during sleep.

Why the pair matters. Both are common, both rise with age, both are easily missed or mislabelled, and both have a specific, cheap first move that the viva rewards: check the ferritin. RLS is under-recognised, frequently dismissed as cramp or anxiety or a peripheral neuropathy, and its treatment carries a signature drug trap (augmentation) that is worth a mark on its own. The **dopaminergic** systems and iron stores that underlie RLS are laid out further in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes and the Functional Neuroanatomy and Neurophysiology notes; this note owns the diagnosis, the discrimination and the guideline-anchored management.

18.1 Restless legs syndrome: the sensory urge and the four essential questions

The clinical picture. RLS is a sleep-related movement disorder defined by an urge to move the legs, usually accompanied or caused by an uncomfortable or unpleasant sensation, classically described as a "creepy-crawly", crawling or restless feeling deep in the calves. It comes on during relaxed wakefulness and inactivity, is worse in the evening or at night, and is transiently relieved

by movement such as walking or stretching. The sensation, not the movement, is the engine: the patient moves to relieve the urge.

Four questions, all "yes". The diagnosis is purely clinical, built on four essential features. All four must be present, and other causes of leg discomfort must be excluded first (Kaplan & Sadock, CTP 2024).

ESSENTIAL DIAGNOSTIC FEATURE (ALL FOUR REQUIRED)	URGE CUE
An urge to move the legs, usually with an uncomfortable or unpleasant leg sensation	**U**: Urge
The urge begins or worsens during rest or inactivity (sitting, lying down)	**R**: Rest worsens
The urge is partially or wholly relieved by movement, for as long as movement continues	**G**: Gets better on moving
The urge is worse in the evening or at night, or occurs only then	**E**: Evening/night

The four essential clinical criteria for restless legs syndrome; the diagnosis needs all four and is made on history alone, with no role for polysomnography (Kaplan & Sadock, CTP 2024; the accompanying scale plate typesets the Cambridge-Hopkins diagnostic questionnaire).

MNEMONIC

URGE

Urge to move (with an unpleasant sensation), worse on **R**est/inactivity, **G**ets better with movement, worse in the **E**vening or at night. Four features, all four needed. Structured questionnaires (the Cambridge-Hopkins diagnostic questionnaire, the RLS Diagnostic Index) map onto exactly these.

RULE **RLS is a bedside diagnosis, not a sleep-lab one.** All four URGE features present, mimics excluded, and you have restless legs syndrome; polysomnography has no role in diagnosing RLS itself.

GOOD TO REMEMBER

- **Restless legs syndrome (the syndrome):** an urge to move the legs with an uncomfortable sensation that (1) worsens at rest, (2) is relieved by movement, and (3) is worse in the evening or at night; all four features are required, mimics (neuropathy, cramps, arthritis, vascular insufficiency, positional discomfort) are excluded, and the diagnosis is clinical with no polysomnography needed.

18.2 Ferritin, iron and the comorbidities

Iron is the reversible cause you must not miss. The pathophysiology of RLS involves both central and peripheral abnormalities, with iron stores and **dopaminergic** transmission both implicated (low brain iron impairs dopamine synthesis). So every patient gets a serum ferritin: supplemental iron is recommended when the ferritin is **≤75 mcg per L**, because correcting the iron deficiency frequently reduces both the paraesthesiae and the movements (Kaplan & Sadock,

CTP 2024; Kaufman's Clinical Neurology). Ferritin is an acute-phase reactant, so a "low-normal" value can still reflect true depletion.

The company RLS keeps. Secondary RLS clusters with iron deficiency, end-stage renal disease (dialysis patients), pregnancy, peripheral neuropathy, diabetes, fibromyalgia, multiple sclerosis and Parkinson disease; the Parkinson and neurodegenerative associations are drawn out further in the Neurology foundations for psychiatrists notes. Prevalence in older adults runs **9% to 20%** and rises with age, and RLS is about twice as common in older women as in older men (Kaplan & Sadock, CTP 2024).

WORKED EXAMPLE

A woman on maintenance haemodialysis complains that every evening her calves feel "crawly" and she has to get up and pace; the feeling settles while she walks and returns the moment she sits. There is no daytime problem. This is textbook secondary RLS on end-stage renal disease. Send a ferritin before reaching for any drug: if it is at or below the threshold, iron replacement may be all she needs.

GOOD TO REMEMBER

- **The ferritin check (the value):** in restless legs syndrome, measure serum ferritin and give supplemental iron when it is **≤75 mcg per L**; low iron stores are the commonest reversible driver, and end-stage renal disease, pregnancy and neuropathy are the other secondary causes to screen for.

18.3 Treatment: dopaminergic and alpha-2-delta agents, and the augmentation trap

Two first-line drug classes. Once iron is corrected and any provoking drug removed, two classes are both considered first-line for RLS and PLMD (Kaplan & Sadock, CTP 2024). The non-ergot **dopaminergic** agonists pramipexole, ropinirole and transdermal rotigotine suppress the sensations and the movements. The **alpha-2-delta** calcium-channel ligands (gabapentin enacarbil, pregabalin and gabapentin) are equally first-line and are often preferred where there is pain, anxiety or a history of impulse-control problems.

Remove the aggravators. Several psychiatric drugs worsen RLS and should be stopped where possible: antidepressants, antipsychotics (neuroleptics) and sedating antihistamines, including the ones hidden in over-the-counter sleep aids and allergy remedies. Sleep deprivation also aggravates it.

Dopaminergic (non-ergot dopamine agonists)

Pramipexole, ropinirole, rotigotine transdermal patch; effective but carry the augmentation risk with long-term use.

Alpha-2-delta ligands

Gabapentin enacarbil, pregabalin, gabapentin; equally first-line, no augmentation, useful when pain or anxiety coexists.

Iron

Replace when ferritin is at or below threshold; can be the only treatment secondary RLS needs.

TRAP **Augmentation.** Long-term **dopaminergic** therapy can paradoxically make RLS worse: symptoms start earlier in the day, become more intense, and spread to the arms and trunk as the dose is pushed up. Do not chase it with more drug; reduce or split the dose, or switch to an alpha-2-delta agent.

GOOD TO REMEMBER

- **RLS pharmacotherapy (the value and the caution):** non-ergot dopamine agonists (pramipexole, ropinirole, rotigotine) and alpha-2-delta ligands (gabapentin enacarbil, pregabalin, gabapentin) are both first-line; the signature complication is dopaminergic augmentation (earlier, more intense, spreading symptoms on rising doses), managed by dose reduction, dose-splitting or a switch away from the dopamine agonist.

18.4 Periodic limb movement disorder: PLMS on polysomnography and the index

The movements, then the disorder. Periodic limb movements in sleep (PLMS) are periodic, repetitive, stereotyped limb movements during sleep, most often a brief upward dorsiflexion of both feet lasting roughly half a second to a few seconds, recurring in trains typically separated by **20 to 40 seconds**, predominantly in NREM sleep and recorded from the anterior tibialis on polysomnography. PLMS are extremely common on their own and are seen in more than **80%** of RLS patients who undergo a sleep study; up to **45%** of community-dwelling adults over 65 have them (Kaplan & Sadock, CTP 2024). The movements alone are not a disorder.

When PLMS becomes PLMD. The burden is quantified by the **periodic-limb-movement index**, the number of periodic limb movements per hour of sleep. Periodic limb movement disorder (PLMD) is diagnosed only when polysomnography-confirmed PLMS are combined with a clinical complaint of disturbed, non-restorative sleep or excessive daytime sleepiness, and other sleep disorders have been excluded. PLMD is explicitly a polysomnographic diagnosis, and it is undefined as a category in DSM-5 (Kaufman's Clinical Neurology); the International Classification of Sleep Disorders, third edition (ICSD-3) places both RLS and PLMD among the sleep-related movement disorders.

GOOD TO REMEMBER

- **Periodic limb movement disorder (the criterion):** periodic limb movements in sleep (PLMS) on polysomnography (stereotyped leg jerks, roughly **20 to 40 seconds** apart, mainly in NREM) quantified by the periodic-limb-movement index, *plus* disturbed sleep or daytime sleepiness, with other sleep disorders excluded; PLMS without any sleep consequence is not PLMD, and PLMD needs the sleep study whereas RLS does not.

18.5 Telling RLS from PLMD (and from the mimics)

One discriminator settles it. Is there a conscious sensory urge? RLS has a waking urge and paraesthesia that drive the movement; PLMS/PLMD are purely sleep movements that arise at regular intervals and are *not* a response to any urge or sensation, of which the patient is usually unaware (the bed partner reports the kicking, or the patient reports only unrefreshing sleep). The figure alongside places the waking urge of RLS against a sleep tracing of rhythmic anterior tibialis contractions.

FEATURE	RESTLESS LEGS SYNDROME (RLS)	PERIODIC LIMB MOVEMENT DISORDER (PLMD)
Core phenomenon	Waking sensory urge to move the legs	Repetitive stereotyped leg jerks in sleep
State when it occurs	Relaxed wakefulness, at rest, evening/night	**Only during sleep (mainly NREM)**
Sensory urge / paraesthesia	**Present and drives the movement**	Absent; movements are not a response to an urge
How diagnosed	Clinical, history alone; the four URGE features	**Polysomnography showing PLMS (plus sleep complaint)**
Patient awareness	Aware of the urge	Usually unaware; bed partner reports the jerks
Overlap	Over 80% have PLMS on the sleep study	Frequently coexists with RLS

Restless legs syndrome versus periodic limb movement disorder; the discriminator is the waking sensory urge (present in RLS, absent in PLMD) and whether polysomnography is required (yes for PLMD, no for RLS) (Kaplan & Sadock, CTP 2024; Kaufman's Clinical Neurology).

75 SERUM FERRITIN IRON-THERAPY THRESHOLD (MCG PER L)	**4** ESSENTIAL RLS QUESTIONS, ALL MUST BE "YES"	**20 to 40** INTER-MOVEMENT INTERVAL IN PLMS (SECONDS)	**>80%** OF RLS PATIENTS SHOW PLMS ON POLYSOMNOGRAPHY

INDIA

Three Indian realities converge here. First, iron deficiency is highly prevalent, so the ferritin check is unusually high-yield and often curative when RLS is secondary to depleted stores, especially in menstruating women and in pregnancy. Second, RLS is widely under-recognised and easily mislabelled as cramp, "gas", anxiety or a peripheral neuropathy, so evening leg restlessness relieved by walking is worth asking about directly. Third, patients presenting with this kind of broken, non-restorative sleep are commonly started on a benzodiazepine for "insomnia", which treats neither the iron deficiency nor the movement disorder and adds a dependence risk; the wider problem of benzodiazepine over-use for insomnia and the safer taper are covered in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes. Check a ferritin before you write a hypnotic.

HOLD THIS

Restless legs syndrome is a clinical, waking diagnosis (urge to move the legs, worse at rest and in the evening, relieved by movement, all four URGE features) whose first move is a ferritin (replace iron if ≤75 mcg per L) and whose first-line drugs are dopaminergic agonists or alpha-2-delta ligands, the dopaminergic ones risking augmentation; PLMD is its polysomnographic cousin, periodic limb movements in sleep plus a sleep complaint, with no waking urge.

Apply and consolidate

19 · Worked cases

Worked cases is where the whole arc pays off. The eating and sleep-wake topics were taught one machine at a time, the criteria on one page and the management on another; the exam presents them fused, as a patient in front of you. This closer drills that fusion through four **vignette**-driven worked examples, each reasoned aloud, so the moves become reflex: an anorexia nervosa case taken through medical-risk assessment, stabilisation and safe refeeding; a bulimia nervosa case read off its purging footprint and its potassium; a chronic-insomnia case that chooses a therapy over a tablet; and a narcolepsy work-up rescued from a mislabelled depression.

How to use this closer. Every case is worked in two disciplined halves, the same split the day was built on. First you *name the syndrome* from its defining criterion and its discriminator, then you *manage it* by the guideline anchor, the one first-line, the one value. The verified numbers, the refeeding rate, the fluoxetine dose, the multiple-sleep-latency thresholds, the scale cut-offs, are carried here exactly as the source topics established them; nothing new is invented, and any figure a source could not confirm was already flagged upstream. The reasoning flow for the four cases is set out in the accompanying figure, and each screening instrument named sits on the accompanying scale plate.

19.1 Reading an eating or sleep case: name the syndrome, then treat it

The two-halves discipline. The commonest way a candidate loses a station is by lunging at treatment before the diagnosis is secure, prescribing a hypnotic for what is actually a phase disorder, or a drug to make an anorexic patient gain weight. The corrective is a fixed order: settle the diagnosis on its criterion and its one discriminator, stage the medical risk, and only then reach for the management ladder. On these notes the discriminators are cheap and verbal, the significantly low weight from restriction, the binge-purge cycle at a normal weight, the sleep complaint despite adequate opportunity, and the emotion-triggered muscle weakness of cataplexy.

What each case tests. The eating cases test whether you can hold a hierarchy, that no drug is first-line in anorexia and that fluoxetine is the one licensed drug in bulimia, and whether you can refeed without killing the patient. The sleep cases test whether you lead chronic insomnia with a therapy rather than a tablet, and whether you can pull a narcolepsy out from behind a treatment-resistant depression. Hold the order and each case answers itself.

> **PEARL**
>
> Diagnose before you treat, and treat by the anchor, not the impulse. In every one of these cases the marks are split evenly between naming the syndrome from its one discriminator and choosing the one first-line move; a candidate who reaches for a drug before the diagnosis is settled loses half the station whatever they prescribe.

19.2 Case one, anorexia nervosa: the vignette and the medical-risk read

The vignette. The presentation is the sickest patient of the four, and the one where the danger is easy to under-read because the bloods can look bland.

WORKED EXAMPLE

case: a 19-year-old woman is brought by her family for fainting and six months of amenorrhoea. She eats tiny measured portions and exercises for hours, but attributes the poor intake to "gas and bloating and no appetite" and denies any wish to be thin. She is emaciated with fine downy **lanugo** on the forearms and cold peripheries; the body mass index is 14.6, the pulse 42 with a lying-to-standing blood-pressure drop, the serum potassium 2.9 and the electrocardiogram QTc prolonged.

Naming it, despite the absent fear. This is anorexia nervosa, restricting subtype: a restriction-driven, significantly low weight held by persistent weight-defeating behaviour, with disturbed recognition of the danger. The trap embedded in the vignette is the absent stated fear of fatness, the non-fat-phobic presentation; the diagnosis still stands, because the criterion accepts persistent behaviour that blocks weight gain in place of a voiced fear, and the significantly low weight from restriction is the non-negotiable anchor. By the DSM-5-TR band she is extreme (a body mass index under **15**); ICD-11 calls this significantly low body weight, with dangerously low body weight (the deeper grade) reserved for a body mass index under **14**, just below where she sits.

The medical-risk assessment. Before any feeding, the body is staged: weight, orthostatic blood pressure, pulse, temperature and peripheral circulation, with bloods for full blood count, urea and electrolytes weighted to **potassium**, magnesium and **phosphate**, liver function, glucose, an electrocardiogram for the QTc, and bone density by DEXA if the underweight is prolonged. Her bradycardia, postural drop, hypokalaemia and prolonged QTc place her, on the MARSIPAN risk stratification, in the high-risk zone that mandates emergency medical admission rather than outpatient refeeding. The neuroendocrine axes behind her amenorrhoea and bradycardia (the hypothalamic-pituitary-gonadal, thyroid and adrenal changes of starvation) are developed in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes.

INDIA

This vignette is deliberately Indian in shape. The **non-fat-phobic** presentation, restriction rationalised as gastrointestinal discomfort or loss of appetite rather than a wish to be thin, is relatively more common in Indian and other Asian patients, and calling such a starving patient "not anorexia" for want of a stated fear is the classic error. Layered on it is under-recognition: eating disorders are widely missed in India and stereotyped as an affluent-urban-female problem, so the severely underweight patient reaches care late and profoundly starved, exactly the one most endangered by refeeding. The corrective is a low threshold to screen (the SCOFF at the bedside) and always to attach a weight, orthostatic vitals, a potassium and an electrocardiogram QTc.

GOOD TO REMEMBER

- **Anorexia nervosa (defining criterion):** restriction of intake driving a *significantly low body weight*, plus a fear of weight gain *or* persistent behaviour that blocks it, plus disturbed body-image or non-recognition of the danger; the defining criterion is the restriction-driven low weight, and the fear need not be voiced, so a non-fat-phobic case: still meets the diagnosis.

19.3 Case one, the management: stabilise, refeed low-and-slow, then the therapy

Correct first, then feed cautiously. Once admitted, the sequence is correct the electrolytes, cover with **thiamine** before or with the first carbohydrate, and crawl the calories up. Feeding begins low, and the two numeric sources disagree about how low: NICE CG32 1.4.8 caps the high-risk start at **10 kcal per kg per day** and uses only **5 kcal per kg per day** in the extreme case such as a body mass index under 14, increasing slowly to meet or exceed full needs within **4 to 7 days**, whereas RCPsych MEED CR233 (2022) starts at **20 kcal per kg per day**, never below the immediate pre-admission intake, and warns against underfeeding; NICE NG69 gives no figure and refers to MEED, so quote the source the question names. Either way, daily phosphate, magnesium, potassium and thiamine supplementation runs alongside. The hallmark to pre-empt is refeeding syndrome, whose biochemical signature is hypophosphataemia.

Watch the phosphate early. Electrolytes are monitored daily through the first week, when the insulin-driven intracellular shift is steepest, and the phosphate trend over the first three days is the earliest warning that the syndrome is developing; a near-normal admission panel does not exclude the risk. Only once she is medically stable does the primary treatment proper begin: weight restoration to a healthy body mass index as the goal, delivered inside a multidisciplinary plan, plus a manualised psychological therapy chosen with her, CBT-ED, MANTRA or SSCM, all first-line and equivalent in adults. Medication treats comorbidity only; the general cognitive-behavioural technique behind CBT-ED is developed in the Psychotherapies, clinical assessment, MSE and nosology notes.

SOURCE AND CLINICAL STATE	STARTING RATE (KCAL PER KG PER DAY)	SUPPLEMENT AND MONITOR
NICE CG32 1.4.8, high risk of refeeding problems	**maximum 10**	Phosphate, magnesium, potassium, thiamine daily; full needs within 4 to 7 days
NICE CG32 1.4.8, extreme (body mass index under 14)	**only 5**	**Slowest start; electrolytes daily, phosphate watched over the first three days**
RCPsych MEED CR233 2022, eating disorders	**start at 20**	Never below the pre-admission intake; build up swiftly, avoid underfeeding

The two numeric refeeding positions for this case, and they disagree: NICE CG32 caps the high-risk start at 10 kcal per kg per day, RCPsych MEED CR233 2022 opens at 20, and NICE NG69 sets no rate and refers to MEED. Quote whichever source the question names; the exact electrolyte thresholds and the full monitoring schedule are owned by the refeeding-syndrome topic and its diagram.

MNEMONIC

CORRECT, COVER, CRAWL

Correct the electrolytes proactively (phosphate, potassium, magnesium); **Cover** with thiamine before or with the first feed; **Crawl** the calories up from a low start. The three prevention moves, applied to this admission in that order.

TRAP **Refeeding too fast, and trusting a bland admission panel.** Piling on calories to correct a dangerously low weight drives phosphate, potassium and magnesium into the cells; the falling phosphate of the opening days precipitates cardiac failure and arrhythmia. Start low, replace electrolytes, give thiamine first, and re-check daily through the first week.

GOOD TO REMEMBER

- **Refeeding syndrome (defining criterion):** the fatal fluid-and-electrolyte cluster of reintroducing feeding into a starved body, defined biochemically by *hypophosphataemia* with falling potassium and magnesium and fluid retention; the discriminator is that serum ions look near-normal until feeding drives them intracellularly.
- **Safe refeeding (the value):** *correct the electrolytes, give thiamine, start slow*, opening at a maximum of 10 kcal per kg per day on NICE CG32 (only 5 if extreme) or at 20 on RCPsych MEED CR233 2022, which warns against underfeeding, quoting whichever the question names, with daily supplementation and electrolytes monitored daily through the first week; the rate is the one variable fully under the clinician's control.
- **Anorexia therapy (the value):** the primary treatment is *weight restoration plus a manualised psychological therapy* (adults: CBT-ED, MANTRA or SSCM, all first-line; young people: family-based treatment), and *no drug is first-line*, medication treats comorbidity only.

19.4 Case two, bulimia nervosa: the binge-purge cycle and the potassium

The vignette, and the diagnosis you make by asking. This case hides in plain sight, because the weight is normal and nothing on inspection announces the disorder.

WORKED EXAMPLE

case: a 22-year-old woman of normal weight (body mass index 22) discloses, only when asked directly, that she binges secretly several evenings a week and then makes herself vomit. On examination there are knuckle calluses (Russell's sign), bilateral parotid fullness and eroded dental enamel; the serum potassium is 3.0 with a borderline-prolonged QTc.

Naming it. This is bulimia nervosa: the recurrent **binge-purge cycle**, binge eating with loss of control plus recurrent inappropriate compensatory behaviour (here self-induced vomiting), with self-evaluation hostage to weight and shape, at a normal or above-normal weight. That normal weight is the whole diagnostic point, and the reason it is missed; the discriminators from its neighbours are that the binges are objective, the purging recurrent, and the weight not significantly low (were it low, the label would become anorexia nervosa, binge-purge type). The frequency-and-duration threshold to state is at least once weekly for **three months** in DSM-5-TR (one month in ICD-11).

Reading the footprint of purging. The body records the vomiting: Russell's sign, parotid swelling and dental erosion are the visible residue, and the biochemistry is a hypochloraemic, hypokalaemic metabolic alkalosis. The two urgent findings that must be actively sought are marked hypokalaemia and a prolonged QTc, because a low potassium drives fatal arrhythmia; her potassium of 3.0 and borderline QTc make the ECG and the electrolyte correction the first, non-negotiable move, normal weight notwithstanding.

PEARL

Bulimia nervosa is the eating disorder you diagnose by *asking*, not by looking. The weight is normal, so the tells are the secret binge-purge cycle on history and Russell's sign, parotid fullness, eroded teeth and a quietly low potassium on examination. Screen with the SCOFF (positive at two), and always attach a potassium and an electrocardiogram.

GOOD TO REMEMBER

- **Bulimia nervosa (defining criterion):** the recurrent *binge-purge cycle*, binge eating with loss of control plus recurrent compensatory behaviour (most often self-induced vomiting) at least once weekly for three months (one month in ICD-11), with self-evaluation unduly influenced by weight and shape; the discriminating fact in this case: is that the weight is *normal or above*, not low, and the potassium and QTc are the emergency to seek.

19.5 Case two, the management: CBT-ED, and fluoxetine at 60 mg per day

Stabilise, then therapy first. The plan is to correct and monitor the potassium and arrange an electrocardiogram, then lead with a psychological therapy, never a drug alone. Under NICE NG69 the adult first step is bulimia-nervosa-focused guided self-help, stepping up to individual **CBT-ED** if that is unacceptable or ineffective; CBT-ED is the best-evidenced treatment and targets the machinery of the cycle directly, installing regular eating and dismantling the compensatory behaviours and the over-evaluation of shape and weight.

The one drug, at the antibinge dose. If a medication is added for the bingeing and purging, the answer is fluoxetine, the medication of choice and the one licensed drug for bulimia nervosa, titrated to **60 mg per day**, a dose *above* its 20 to 40 mg antidepressant range, and effective whether or not the patient is depressed. In India fluoxetine is cheap and widely available (brands Prodep, Fludac, Flunil), given alongside the therapy, not instead of it; its deep pharmacology (the high bulimia dose, the long half-life, the interactions) is carried in the Mood disorders: pharmacotherapy notes. The drug to avoid is the exam trap, bupropion is contraindicated because it lowers the seizure threshold in purging patients.

RULE **Fluoxetine 60 mg per day is THE licensed drug for bulimia.** The one antibulimic dose to memorise is fluoxetine *60 mg per day*, the antibinge dose sitting above the 20 to 40 mg antidepressant range; it is the medication of choice, given *with* a psychological therapy (guided self-help then CBT-ED), never instead of it, and never bupropion.

GOOD TO REMEMBER

- **CBT-ED (the value):** eating-disorder-focused CBT is the *best-evidenced treatment* for bulimia nervosa; NICE NG69 leads with guided self-help stepping up to CBT-ED, a psychological therapy is primary and medication is never the sole treatment.
- **Fluoxetine (the value):** the one value is *60 mg per day*, above the antidepressant dose, as the licensed antibinge drug (works whether or not the patient is depressed), given with the therapy; bupropion is *contraindicated* for its seizure-threshold effect in purging patients.

19.6 Case three, chronic insomnia: choosing CBT-I over a long-term hypnotic

The vignette. The third case is the one where the easy move is the wrong one, and the patient is actively asking for it.

WORKED EXAMPLE

case: a 46-year-old man reports six months of difficulty staying asleep, waking around three in the morning at least four nights a week, unable to return, with daytime fatigue and poor concentration, all despite going to bed with an adequate chance to sleep. His Insomnia Severity Index is 18 (moderate). He has been taking nightly alprazolam obtained from a pharmacy and asks for a stronger dose.

Naming it, after clearing the mimics. This is insomnia disorder, chronic form: dissatisfaction with sleep with difficulty maintaining it, *despite adequate opportunity*, at least three nights a week for at least three months, with daytime consequences. Before settling it, the sleep-wake mimics are cleared, a circadian phase disorder (sleep normal when timed to his own preference), obstructive sleep apnoea (snoring, witnessed apnoeas), and a movement disorder (an evening urge to move the legs); a hypnotic prescribed for apnoea would be useless and, in apnoea, dangerous. His ISI of 18 sits in the moderate clinical band (**15 to 21**), and the screening threshold is **10 or higher**.

The management move that defines the case. The whole station turns on one decision: cognitive behaviour therapy for insomnia (CBT-I), delivered as the multicomponent package with **sleep restriction** and **stimulus control** at its core, is first-line for chronic insomnia and is offered before any drug, because its gains outlast a tablet. The correct plan is to start CBT-I (face-to-face or digital, both efficacious) and to taper his alprazolam slowly with concurrent CBT-I, not to escalate a hypnotic he cannot then stop. The z-drug and benzodiazepine dependence and the withdrawal taper are developed in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes; the ISI form sits on the accompanying scale plate.

INDIA

The genuine Indian device here is *benzodiazepine over-use for insomnia*. With CBT-I therapists scarce and hypnotics cheap and easily obtained over the counter, long-term alprazolam and clonazepam prescribing for chronic insomnia is widespread and drifts into dependence, exactly this patient's trajectory. The corrective is not exotic: lead with CBT-I as first-line (digital where a therapist is out of reach), keep any hypnotic short-term and within licence, prefer prolonged-release melatonin in the older adult, and remember that correct sleep-hygiene practice is a *component* of CBT-I, not a substitute handed over as a leaflet.

RULE **CBT-I first, the tablet second, and do not start what you cannot stop.** Cognitive behaviour therapy for insomnia, with sleep restriction and stimulus control at its core, is first-line for chronic insomnia and precedes any drug; a hypnotic is short-term only, and an existing long-term benzodiazepine is tapered *with* concurrent CBT-I, not escalated.

GOOD TO REMEMBER

- **Insomnia disorder (defining criterion):** dissatisfaction with sleep with difficulty initiating or maintaining it or early waking, *despite adequate opportunity to sleep*, with daytime consequences; the thresholds are at least three nights a week for at least three months for the chronic form, and the mimics (circadian, breathing, movement) are cleared before the label is fixed.
- **CBT-I (the value):** CBT-I is *first-line for chronic insomnia, before any drug* (sleep restriction and stimulus control are its two engines); a hypnotic is not first-line, and a long-term benzodiazepine is tapered with concurrent CBT-I rather than escalated.

19.7 Case four, the narcolepsy work-up: the tetrad and the MSLT

The vignette, hiding behind a diagnosis. The last case is the one the psychiatrist meets already mislabelled, and the whole diagnosis turns on a single question no one has asked.

WORKED EXAMPLE

case: a 21-year-old man has been irresistibly sleepy since his mid-teens and has failed three antidepressants for a presumed "treatment-resistant depression". Asked directly what happens when he laughs hard, he describes his knees buckling and his head dropping while fully aware, lasting a few seconds (cataplexy); he also reports occasional inability to move on waking (sleep paralysis) and vivid dream-like images at sleep onset (hypnagogic hallucinations).

Naming it from the tetrad. The cluster is the **narcolepsy tetrad**: excessive daytime sleepiness with sleep attacks, cataplexy, sleep paralysis and hypnagogic hallucinations (disturbed nocturnal sleep is the fifth, pentad, feature). Sleepiness is the constant and the presenting complaint; cataplexy is the pathognomonic member, the emotion-triggered bilateral loss of muscle tone with preserved awareness, and it is REM-sleep atonia discharging into wakefulness from loss of the hypothalamic orexin (hypocretin) neurons. Cataplexy alone places him in type 1.

Sealing it on the test. The work-up is an overnight polysomnogram (to exclude apnoea) followed by the multiple sleep latency test, and his result is diagnostic: a mean sleep latency of five minutes with three sleep-onset REM periods across the naps. The MSLT criterion is a mean sleep

latency of **eight minutes or less** and **two or more** sleep-onset REM periods; a low cerebrospinal-fluid orexin (under **110 pg per mL**) is the most specific finding and defines type 1 but is rarely available.

TEST	DIAGNOSTIC FINDING IN THIS CASE
MSLT mean sleep latency	**8 minutes or less (his was 5)**
MSLT sleep-onset REM periods	**2 or more across the naps (his was 3)**
CSF orexin (hypocretin-1)	Low or absent (under 110 pg per mL); defines type 1, rarely available

The confirmatory thresholds applied to case four; the MSLT needs a mean sleep latency of eight minutes or less plus two or more sleep-onset REM periods, both of which he exceeds.

MNEMONIC

CHESS

Cataplexy; **H**ypnagogic hallucinations; **E**xcessive daytime sleepiness; **S**leep paralysis; disturbed nocturnal **S**leep (the pentad fifth). In this case: the sleepiness is the constant that got him labelled depressed; the cataplexy is the specific member that makes the diagnosis.

TRAP **Narcolepsy hiding as a treatment-resistant depression (or an epilepsy).** Pervasive daytime sleepiness that outweighs the mood earns antidepressant after antidepressant, and emotion-triggered drops get read as atonic seizures; the corrective is one question, ask about knees buckling at a joke, before escalating either label.

GOOD TO REMEMBER

- **Narcolepsy (defining cluster and confirmatory step):** the tetrad is excessive daytime sleepiness with sleep attacks, cataplexy, sleep paralysis and hypnagogic hallucinations (disturbed nocturnal sleep the pentad fifth), from loss of the orexin (hypocretin) neurons; *cataplexy is the pathognomonic member* and defines type 1, and the diagnosis is sealed on the MSLT at a mean sleep latency of eight minutes or less with two or more sleep-onset REM periods.

19.8 Case four, the management: the wake-promoting agent and sodium oxybate for cataplexy

Treat the two problems separately. Narcolepsy is not curable, so the daytime sleepiness and the cataplexy are targeted with different drugs on the sleep-medicine practice standard, with scheduled short naps and firm sleep hygiene as the non-drug backbone. For his sleepiness, the first-line wake-promoting agent is modafinil (in India, Modalert or Provake), a dopamine-transporter inhibitor at **200 to 400 mg per day** that improves the sleepiness but does *not* touch cataplexy; armodafinil, solriamfetol and pitolisant extend the class.

The cataplexy, and the Indian substitution. For the disabling cataplexy and his fragmented nights, sodium oxybate (the sodium salt of gamma-hydroxybutyrate) is the most effective single agent and the only one that also consolidates the broken night and improves daytime sleepiness, but it is an abuse-liable controlled substance dispensed through restricted programmes and never combined with alcohol. Where it is unavailable, cataplexy is suppressed with a REM-suppressing

antidepressant, an SSRI or SNRI (venlafaxine) or the tricyclic clomipramine, tapered rather than stopped to avoid rebound cataplexy. The sleep neurotransmitter and ascending-arousal systems behind these agents are developed in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes and the Functional Neuroanatomy and Neurophysiology notes.

INDIA

The genuine Indian device in narcolepsy is *drug availability*. Modafinil (Modalert, Provake) and armodafinil are freely and cheaply available, so first-line treatment of the daytime sleepiness is entirely practical; but sodium oxybate, pitolisant and solriamfetol are not marketed in India, so cataplexy is usually managed with the older anticataplectic antidepressants (an SSRI or SNRI, or clomipramine) instead, exactly the substitution this case: makes. Under-recognition compounds it: MSLT-capable sleep labs are scarce and orexin assay essentially unavailable, so the sleepy adolescent is treated for laziness or depression for years. The corrective costs nothing, ask every chronically sleepy young patient about emotion-triggered weakness.

GOOD TO REMEMBER

- **Wake-promoting agent (the value):** *modafinil* (Modalert or Provake), a dopamine-transporter inhibitor at 200 to 400 mg per day, is first-line for the excessive daytime sleepiness of narcolepsy but does *not* benefit cataplexy; armodafinil, solriamfetol and pitolisant extend the class.
- **Sodium oxybate (the value):** the most effective single drug for *cataplexy* and the only one that also consolidates the broken night, but an abuse-liable controlled substance never combined with alcohol; where it is unavailable (as in India) cataplexy is managed with a REM-suppressing antidepressant (SSRI or SNRI, or clomipramine), tapered to avoid rebound cataplexy.

19.9 The four cases at a glance, and the through-line

One grid to hold them. Read side by side, the four cases rehearse the day's spine: the eating pair drills that no drug is first-line in anorexia while fluoxetine is the one licensed drug in bulimia, and that refeeding is prevented, not chased; the sleep pair drills that chronic insomnia leads with a therapy and that a narcolepsy is only ever found by asking about cataplexy. The through-line across all four is the same discipline, name the syndrome from its one discriminator, then treat by the anchor.

CASE	WORKING DIAGNOSIS	THE ONE DISCRIMINATOR	THE FIRST MANAGEMENT MOVE
One (case:)	**Anorexia nervosa, restricting**	Significantly low weight from restriction (fear need not be voiced)	Stabilise and refeed low-and-slow (CG32 caps at 10 kcal per kg per day, MEED 2022 opens at 20), then therapy; no drug first-line
Two (case:)	**Bulimia nervosa**	Binge-purge cycle at a *normal* weight; low potassium and Russell's sign	**Correct potassium; CBT-ED, and fluoxetine 60 mg per day if a drug is added**

CASE	WORKING DIAGNOSIS	THE ONE DISCRIMINATOR	THE FIRST MANAGEMENT MOVE
Three (case:)	**Insomnia disorder, chronic**	Sleep complaint despite adequate opportunity, three-plus nights a week for three months	**CBT-I first-line; taper the benzodiazepine, do not escalate a hypnotic**
Four (case:)	**Narcolepsy, type 1**	Cataplexy (emotion-triggered atonia, aware); MSLT sleepiness plus SOREMPs	Modafinil for sleepiness; sodium oxybate (or, in India, an SSRI/SNRI/clomipramine) for cataplexy

The four worked cases side by side; each is settled on one discriminator and opened on one management anchor, the discipline these notes are built to install.

10 or 20 CASE ONE REFEEDING START (KCAL PER KG PER DAY): CG32 CAPS AT 10, MEED 2022 OPENS AT 20	**60** CASE TWO FLUOXETINE DOSE FOR BULIMIA (MG PER DAY)	**≤8** CASE FOUR MSLT MEAN SLEEP LATENCY (MINUTES)	**≥2** CASE FOUR MSLT SLEEP-ONSET REM PERIODS (COUNT)

HOLD THIS

Four cases, one discipline: name the syndrome from its discriminator, then treat by the anchor. Anorexia is restriction-driven low weight (fear need not be voiced), stabilised and refed low-and-slow to pre-empt hypophosphataemia with no drug first-line; bulimia is the binge-purge cycle at a normal weight with a dangerous potassium, treated with CBT-ED and fluoxetine 60 mg per day; chronic insomnia is a sleep complaint despite adequate opportunity, led by CBT-I not a long-term hypnotic; and narcolepsy is the tetrad with cataplexy, sealed on the MSLT (mean sleep latency eight minutes or less with two or more sleep-onset REM periods) and treated with modafinil for sleepiness and sodium oxybate, or in India an SSRI/SNRI or clomipramine, for cataplexy.

20 · Consolidation

The whole eating and sleep-wake block now sits in front of you, and this closing topic is not new content but the machinery that turns reading into recall. The examination does not reward the candidate who has *read* the eating and sleep chapter; it rewards the one who can, cold and under pressure, state the discriminating criterion for each syndrome, name the one first-line move for each management problem, quote the refeeding rate and the scale bands to the number, and recite the narcolepsy tetrad and the AHI cut-offs in a single line. Those retrievals are what this topic drills. The single most robust finding in the science of learning is that **active recall** (pulling an answer out of an empty page) and **retrieval practice** (repeatedly testing yourself rather than re-reading) beat every passive method for durable memory, so the design here is deliberately answer-sparse: prompts first, and the answers held elsewhere in the chapter so you must fetch them.

Why it matters, and what this note owns. This note owns three things: the **mnemonic** that ties the chapter together, paid off from where it was seeded at the anorexia opener; a set of answer-free prompt lists for **active recall** across the eating-disorder discriminators, the refeeding thresholds and the drug anchors, the sleep architecture and the two-process model, the CBT-I

rule, and the narcolepsy tetrad, the AHI bands and the parasomnia-neurodegeneration link; and a whole-chapter **synthesis map** you are meant to **redraw from memory**. It re-derives no criteria, doses or bands of its own; every number quoted here is a pointer back to the topic that verified it. The deep pharmacology behind the drug names lives in the Mood disorders: pharmacotherapy notes (the SSRI at the high bulimia dose) and the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes (the hypnotics, their dependence and the taper); the sleep neurotransmitter systems and the neuroendocrine axes that fail in starvation sit in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes and the Functional Neuroanatomy and Neurophysiology notes; the general CBT frame sits in the Psychotherapies, clinical assessment, MSE and nosology notes. The synthesis map that carries the whole block is drawn in the accompanying figure, framed cover-the-branches so it can be used as a retrieval test.

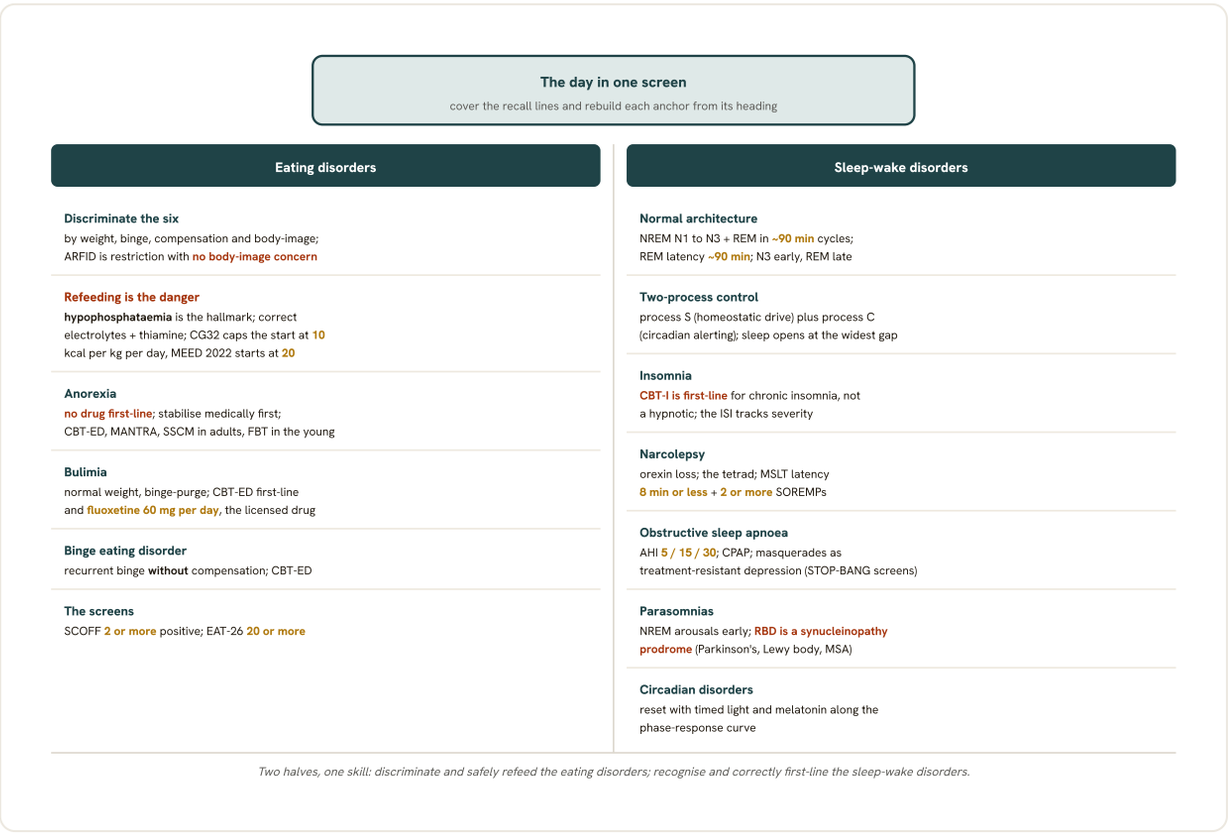


Fig 17.18 The chapter in one screen. The eating half reduces to four moves: discriminate the six presentations by weight, binge, compensation and body-image (with ARFID set apart by having no body-image disturbance); treat the refeeding syndrome as the lethal danger, marked by hypophosphataemia and prevented by correcting the electrolytes, giving thiamine and crawling the calories up; hold the two management anchors, that no drug is first-line for anorexia and that fluoxetine at sixty milligrams a day is the licensed drug for bulimia; and screen with SCOFF and the EAT-26. The sleep half reduces to the normal architecture and the two-process model as the foundation, then the disorders: cognitive behaviour therapy for insomnia first-line rather than a hypnotic, narcolepsy from orexin loss confirmed on the multiple sleep latency test, obstructive sleep apnoea graded by the apnoea-hypopnoea index and masquerading as resistant depression, REM sleep behaviour disorder as a prodrome of a synucleinopathy, and the circadian disorders reset with timed light and melatonin. Cover the recall lines and rebuild each anchor from its heading.

20.1 The master mnemonic, paid off: STARVED

Seeded at the opener, closed here. The chapter opened by seeding the master mnemonic **STARVED** at anorexia nervosa, and this is where it is paid off, because it is the single most

examined slice of the whole eating half: the physical complications of starvation, the ones that make anorexia the most lethal psychiatric disorder. **S**kin (lanugo, dry skin, carotenaemia); **T**hyroid (sick-euthyroid pattern) and low Temperature; **A**menorrhoea and the hypothalamic-pituitary-gonadal axis; **R**ate and rhythm (bradycardia, hypotension, prolonged QTc); **V**alues, the electrolytes (hypokalaemia, hyponatraemia); **E**rythron and marrow (pancytopenia); **D**igestive and Density (delayed gastric emptying, low bone mineral density). Riding alongside STARVED are the four working mnemonics the chapter leans on and you should be able to expand blind: **CORRECT, COVER, CRAWL** for refeeding prevention, the four-question **Weight? Binge? Compensation? Body-image?** for the eating differentiation, **CHESS** for the narcolepsy pentad, and **RBD warns of PDM** for the parasomnia-neurodegeneration link.

MNEMONIC

STARVED

The chapter master mnemonic, seeded at the anorexia opener and paid off here. **S**kin (lanugo, carotenaemia); **T**hyroid sick-euthyroid and low Temperature; **A**menorrhoea (hypothalamic-pituitary-gonadal axis); **R**ate and rhythm (bradycardia, hypotension, prolonged QTc, arrhythmia); **V**alues (hypokalaemia, hyponatraemia, dehydration); **E**rythron and marrow (pancytopenia); **D**igestive and Density (delayed gastric emptying, constipation, low bone mineral density). If you can expand STARVED and the four ride-along mnemonics without looking, you hold the spine of both halves of the chapter.

RULE **Learn each half by its one anchor.** The eating half hangs off two management anchors (no drug is first-line for anorexia; fluoxetine is the licensed drug for bulimia) and one danger (refeeding); the sleep half hangs off one therapy anchor (CBT-I first for chronic insomnia) and three test objects (the two-process model, the narcolepsy tetrad with the MSLT, and the AHI bands); everything else hangs off those.

20.2 Active-recall prompt list: the eating-disorder discriminators

Cover the answers and pull each one. This is a **retrieval practice** grid, not a summary. The four-question tool settles almost every eating differentiation in order (weight, then binge, then compensation, then body-image); for each row, state the discriminator before you check it against the topic that verified it. The instrument forms (the SCOFF, EAT-26, EDE-Q) are typeset centrally, so work from the accompanying scale plate rather than any pasted questionnaire.

DISORDER	THE DISCRIMINATOR TO STATE (PROMPT, THEN CHECK)
Anorexia nervosa	**Low weight from restriction plus overvaluation of shape/weight and a dread of fatness; the fear need not be spoken (the atypical, non-fat-phobic presentation still counts); severity by BMI band**
Bulimia nervosa	Recurrent binges with recurrent *compensation* (vomiting, laxatives, fasting, exercise) at a *normal or above-normal* weight; DSM-5-TR floor once weekly over three months

DISORDER	THE DISCRIMINATOR TO STATE (PROMPT, THEN CHECK)
Binge eating disorder	**Loss-of-control binges *without* regular compensation; three or more of the five features (rapid, alone, guilt, until uncomfortably full, not hungry); weight loss is not a therapy target**
ARFID	**Avoidant or restrictive intake driven by low interest, sensory aversion or fear of aversive consequence, *with no* body-image disturbance; that missing overvaluation is what separates it from anorexia**
Atypical anorexia / OSFED	Full anorexia cognition and behaviour but weight in or above the normal range (atypical anorexia), or subthreshold pictures (purging disorder, night-eating syndrome); a real disorder, not a mild one

The eating-disorder discriminator grid: the block is carved by weight, then binge, then compensation, then body-image, with ARFID marked by the absent overvaluation (verified against the anorexia, bulimia, binge-eating, ARFID and atypical/OSFED topics).

GOOD TO REMEMBER

- **Anorexia nervosa:** the defining criterion is low weight maintained by restriction *plus* overvaluation of shape and weight; the discriminator you must hold is that the dread of fatness can be unspoken and the weight can even be normal in atypical anorexia, so a non-fat-phobic presentation is still anorexia.
- **Bulimia versus binge eating disorder:** the one question that separates them is *compensation*, present in bulimia and absent in binge eating disorder; and the one question that separates both from ARFID is *body-image overvaluation*, present in the eating disorders proper and absent in ARFID.

20.3 Active-recall prompt list: refeeding thresholds and the two drug anchors

These are the values examiners want verbatim. The eating half turns on one danger and two prescribing rules, and each carries a number to pull cold. Retrieve the refeeding hallmark and rate, then the anorexia rule, then the bulimia dose, before checking against the refeeding, anorexia-management and bulimia topics.

- **The refeeding hallmark.** Prompt: the signature electrolyte, and its two companions? (Hypophosphataemia is the hallmark of refeeding syndrome, with falls in potassium and magnesium as insulin drives them intracellularly; a near-normal admission panel does *not* exclude risk.)
- **The refeeding prevention and rate.** Prompt: the three moves and the starting numbers? (CORRECT the electrolytes, COVER with thiamine before or with the first feed, CRAWL the calories up, and know that the two numeric sources disagree: NICE CG32 1.4.8 caps the high-risk start at **10 kcal per kg per day** and uses only **5 kcal per kg per day** when the BMI is under 14, reaching full needs within **4 to 7 days**, whereas RCPsych MEED CR233 2022 starts at **20 kcal per kg per day** and warns against underfeeding; NICE NG69 2017 gives no

figure and refers to MEED, so quote the source the question names. Monitor electrolytes daily through the first week.)

- **The anorexia drug rule.** Prompt: what is first-line, and what medication is *not*? (No drug is first-line: nutritional restoration plus a psychological therapy is primary, CBT-ED, MANTRA or SSCM for adults and family therapy FT-AN for young people; medication is never the sole treatment and olanzapine is not for routine use; NICE NG69 2017.)
- **The bulimia drug anchor.** Prompt: the one licensed drug and its dose? (Fluoxetine (Prodep, Fludac, Flunil) at **60 mg per day**, the anti-binge dose above the 20 to 40 mg antidepressant range, given *alongside* a psychological therapy such as guided self-help then CBT-ED, never instead of it; WFSBP 2023.)

10	20	60	2
NICE CG32 MAXIMUM REFEEDING START AT HIGH RISK (KCAL PER KG PER DAY)	RCPSYCH MEED CR233 2022 REFEEDING START (KCAL PER KG PER DAY)	FLUOXETINE, THE LICENSED DRUG FOR BULIMIA (MG PER DAY)	SCOFF POSITIVE SCREEN (YES ANSWERS)

TRAP **Refeeding a starved patient too fast, or reading a normal phosphate as safety.** The exam-fatal error is to correct the calorie deficit briskly or to be reassured by a near-normal admission electrolyte panel; the phosphate falls only once feeding starts, so the danger is missed until the patient arrests. Start low, replace phosphate *while* feeding continues, and monitor daily through the opening days.

GOOD TO REMEMBER

- **Refeeding syndrome:** the one value is that hypophosphataemia is the hallmark; the one principle is correct-electrolytes, give-thiamine, start-slow (NICE CG32 caps the high-risk start at 10 kcal per kg per day and uses only 5 when the BMI is under 14, full needs within 4 to 7 days, while RCPsych MEED CR233 2022 starts at 20 and warns against underfeeding, so quote the source the question names), and the single strongest lever against the syndrome is the feeding rate, the one variable fully under your control.
- **The two eating drug anchors:** no drug is first-line for anorexia (nutrition plus psychological therapy is primary), and fluoxetine 60 mg per day is the one licensed drug for bulimia, always adjunctive to therapy, never the sole treatment for either disorder.

INDIA

Eating disorders are widely **under-recognised in India**, partly because the classic Western fat-phobic script is often absent: the presentation is frequently the **atypical, non-fat-phobic anorexia** in which restriction is rationalised as loss of appetite, gastric discomfort or religious fasting rather than a stated fear of fatness, so the low weight is investigated medically for months before the eating disorder is named. Holding the rule that the dread of fatness can be unspoken, and that atypical anorexia at a normal weight is still a disorder, is what turns a missed case into a diagnosed one.

20.4 Active-recall prompt list: sleep architecture and the two-process model

The physiology is pure structure, so drill the structure. The sleep half opens on normal architecture, and the two most-asked objects are the proportions of a night and the model that

governs when it happens. Retrieve each before checking against the normal sleep physiology topic.

- **The stages.** Prompt: the NREM ladder and its markers? (NREM runs N1, light transition with alpha dropout; N2, defined by sleep spindles and K-complexes; N3, slow-wave delta sleep, the deepest and most restorative; then REM with its atonia and dreaming.)
- **The proportions.** Prompt: the REM share and the cycle length? (REM is about **20 to 25%** of total sleep time in a young adult; the NREM-REM cycle and the first REM latency are around **90 minutes**; N3 dominates the first half of the night and REM lengthens across the second half.)
- **The two-process model.** Prompt: the two processes and what opens the sleep gate? (Borbely's **two-process model**: Process S is the homeostatic sleep pressure that builds with time awake; Process C is the circadian alerting signal; sleep is triggered in the evening when Process C falls away and the accumulated Process S is suddenly unopposed.)

20 to 25	90	≤8	>30
REM SHARE OF THE NIGHT (% OF TOTAL SLEEP TIME)	FIRST REM LATENCY AND NREM-REM CYCLE (MINUTES)	MSLT POSITIVE MEAN SLEEP LATENCY (MINUTES)	SEVERE OBSTRUCTIVE SLEEP APNOEA (AHI, EVENTS PER HOUR)

GOOD TO REMEMBER

- **Sleep architecture:** the number to hold is REM at 20 to 25% of total sleep time on a roughly 90-minute cycle; the principle is that N3 slow-wave sleep loads the first half of the night and REM loads the second, and the two-process model explains the timing (homeostatic Process S against circadian Process C), the frame the deep sleep-neurotransmitter systems build on in the Functional Neuroanatomy and Neurophysiology notes.

20.5 Active-recall prompt list: the insomnia and CBT-I rule

State the first-line before any drug. Chronic insomnia has the cleanest management rule in the sleep half, and it should be the first sentence of any answer: the therapy comes before the tablet. Retrieve the rule and the scale bands before checking against the insomnia and CBT-I topics.

- **The first-line.** Prompt: what is offered before any drug, and its two engines? (CBT-I, the multicomponent package, is first-line for chronic insomnia and is offered before any hypnotic; its two behavioural engines are *sleep restriction* and *stimulus control*, steadied by sleep hygiene, cognitive therapy and relaxation; BAP 2019, NICE 2023.)
- **The drug place.** Prompt: where does a hypnotic sit, and which is a maintenance-insomnia option? (Not first-line; a short-term, second-line measure for when CBT-I fails, is unavailable or cannot be engaged with, matching the agent's half-life to the pattern; low-dose doxepin at **1 to 6 mg per day** suits late-night awakenings, and daridorexant is the licensed long-term option after CBT-I.)
- **The scale.** Prompt: the insomnia scale and its cut-off? (The Insomnia Severity Index; a score of **10 or higher** screens positive, with the moderate clinical band at 15 to 21; chronic insomnia needs symptoms three or more nights a week for three months.)

RULE **CBT-I is first-line for chronic insomnia; a hypnotic is not.** The therapy is offered before any drug and is the only intervention that durably resets the sleep, matching the twist across the whole management block: the non-pharmacological move leads, and the drug, when used, is time-limited and second.

GOOD TO REMEMBER

- **CBT-I (the first-line management step):** the one value is that CBT-I, not a hypnotic, is first-line for chronic insomnia and is offered before any drug; its two engines are sleep restriction and stimulus control, and the signature caution is that a hypnotic is a short-term second-line measure to be tapered slowly with concurrent CBT-I, never the long-term answer.

INDIA

Two Indian reflexes work against this rule. First, structured CBT-I is scarce, so practice defaults to a **benzodiazepine or z-drug plus a sleep-hygiene leaflet**, precisely the two moves the evidence flags as insufficient on their own (sleep hygiene is not a stand-alone treatment). Second, the resulting **long-term hypnotic over-use for insomnia** is entrenched and dependence-forming; the corrective, and the exam answer, is CBT-I first with the hypnotic reserved, time-limited and tapered, the wider taper being developed in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes.

20.6 Active-recall prompt list: the narcolepsy tetrad, the AHI bands and RBD

Three sleep-disorder objects, three sets of numbers. The remaining high-yield sleep retrievals are the narcolepsy picture and its investigation, the sleep-apnoea severity bands, and the one parasomnia that carries a neurodegenerative warning. Retrieve each before checking against the narcolepsy, obstructive sleep apnoea and parasomnias topics.

OBJECT	THE DISCRIMINATOR AND NUMBER TO STATE (PROMPT, THEN CHECK)
Narcolepsy tetrad	**Excessive daytime sleepiness with sleep attacks, cataplexy, sleep paralysis, hypnagogic hallucinations (CHESS pentad adds disturbed nocturnal sleep); sleepiness is the constant, cataplexy the specific one**
Narcolepsy mechanism and confirmation	Loss of hypothalamic orexin (hypocretin) neurons; CSF orexin under 110 pg per mL defines type 1; the MSLT confirms at a mean sleep latency of 8 minutes or less with two or more sleep-onset REM periods
Narcolepsy treatment	**Modafinil (Modalert, Provake) 200 to 400 mg per day first-line for the sleepiness (does not touch cataplexy); sodium oxybate for cataplexy but not marketed in India, so an SSRI/SNRI or clomipramine is used there**

OBJECT	THE DISCRIMINATOR AND NUMBER TO STATE (PROMPT, THEN CHECK)
Obstructive sleep apnoea (AHI)	Mild 5 to 15, moderate 15 to 30, severe over 30 events per hour (DSM-5-TR writes the mild band as under 15 with the diagnostic floor at five); STOP-BANG three or more screens positive, five or more is high risk; CPAP is first-line, not sole, therapy
REM sleep behaviour disorder	**Dream enactment with lost REM atonia; a *prodrome of a synucleinopathy* (Parkinson's disease, dementia with Lewy bodies, multiple system atrophy), not a nightmare disorder**

The three high-yield sleep-disorder objects and their verified numbers: the narcolepsy tetrad with the MSLT and orexin cut-off, the AHI severity bands, and RBD as a synucleinopathy prodrome (verified against the narcolepsy, obstructive sleep apnoea and parasomnias topics).

TRAP **Missing narcolepsy behind "treatment-resistant depression", or reading RBD as a nightmare.** Sleepiness and amotivation get labelled depression and the cataplexy is never asked about; and dream-enactment violence in an older man gets called a nightmare disorder, missing that REM sleep behaviour disorder heralds a synucleinopathy, the neurodegenerative link developed in the Dementias and neurocognitive disorders notes and the Neurology foundations for psychiatrists notes.

GOOD TO REMEMBER

- **Narcolepsy:** the defining criterion is excessive daytime sleepiness plus cataplexy from orexin loss; the confirming values are an MSLT mean sleep latency of 8 minutes or less with two or more sleep-onset REM periods, and a CSF orexin under 110 pg per mL for type 1.
- **Narcolepsy management (the value anchor):** modafinil 200 to 400 mg per day is first-line for the sleepiness but does nothing for cataplexy, and because sodium oxybate is not marketed in India the cataplexy is treated there with an SSRI, SNRI or clomipramine.
- **Obstructive sleep apnoea:** the AHI bands are mild 5 to 15, moderate 15 to 30 and severe over 30 events per hour, and CPAP is first-line but adjunct, never sole, therapy.

20.7 The whole-chapter synthesis map, framed cover-the-branches

Build the tree, then redraw it blind. The strongest single revision act for this block is to **redraw from memory** a **synthesis map** that hangs every disorder off its trunk. The map has two trunks. The *eating disorders* are carved by the four questions (weight, binge, compensation, body-image) and governed by two management anchors and one danger (no drug first for anorexia, fluoxetine for bulimia, refeeding to fear). The *sleep-wake disorders* are carved by the presenting complaint (cannot sleep, too sleepy, moves in sleep, sleeps at the wrong time) and governed by one therapy rule (CBT-I first) and three test objects (the two-process model, the narcolepsy tetrad with the MSLT, and the AHI bands). From each trunk hang the disorders, and from each disorder hang its one discriminator, its scale or investigation, and its first-line move. The accompanying figure draws this map; use it as a retrieval test by covering all the branches and regenerating them, then uncovering to score yourself, exactly the cover-the-branches method that makes **retrieval practice** work.

How to use it. First pass: cover everything but the two trunks and name the disorders under each. Second pass: for each disorder, cover the discriminator and pull it. Third pass: cover the treatment column and recite the first-line move and the one number. A branch you cannot regenerate is a branch to re-read in its own topic; a branch you can is one you can leave. Repeated over spaced sittings, the map converts the chapter from something you recognise into something you can produce.

PEARL

The chapter collapses to two trunks, four anchors and one danger. Trunk one, the *eating disorders*, are carved by weight, binge, compensation and body-image, and answer to psychological therapy first, with no drug first-line for anorexia and fluoxetine 60 mg per day for bulimia; the danger threaded through it is refeeding syndrome, whose hallmark is hypophosphataemia and whose antidote is correct-electrolytes, give-thiamine, start-slow. Trunk two, the *sleep-wake disorders*, are carved by the complaint, and turn on CBT-I first for chronic insomnia, the narcolepsy tetrad confirmed by the MSLT, the AHI bands for apnoea, and RBD as the synucleinopathy prodrome. Cover the branches, redraw the tree, and you own the block.

HOLD THIS

Redraw the two-trunk synthesis map from memory: the eating disorders (carved by weight, binge, compensation and body-image, psychological therapy first, no drug first-line for anorexia, fluoxetine 60 mg per day for bulimia, and refeeding syndrome as the hypophosphataemic danger you crawl the calories to avoid) and the sleep-wake disorders (carved by the complaint, CBT-I first for chronic insomnia, the narcolepsy tetrad confirmed on the MSLT at 8 minutes or less with two or more sleep-onset REM periods, the AHI bands of mild 5 to 15 / moderate 15 to 30 / severe over 30, and RBD heralding a synucleinopathy).

Previously asked

This territory is examined at two levels at once: the syndrome (its clinical features, its differential diagnosis and its course) and the treatment (which first-line agent, how to refeed safely, which psychological therapy). The reliable pattern is that bulimia nervosa carries the most weight in the eating section and is asked at both essay and short-note length, that binge eating disorder recurs as a standalone note, and that insomnia is the dominant sleep essay while the circadian rhythm disorders recur as a note. Every long essay ends in a management plan, so the guideline-anchored first-line recommendation and the reason for it must be exam-ready: no drug is first-line for anorexia, fluoxetine is the licensed drug for bulimia, and cognitive behaviour therapy for insomnia is first-line for chronic insomnia. The genuine questions on record are grouped by band below. The deep hypnotic and antidepressant pharmacology these disorders are treated with is developed in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes and the Mood disorders: pharmacotherapy notes; the sleep neurochemistry and the ascending-arousal anatomy in the Neurochemistry, neuroendocrinology, neuroimaging and basic psychopharmacology notes and the Functional neuroanatomy and neurophysiology notes; while cognitive behaviour therapy for insomnia, the eating-disorder cognitive behaviour therapy and

family-based treatment, and the sleep architecture, the hypnogram and polysomnography are owned here.

? HOW THIS TERRITORY IS ACTUALLY TESTED

Q **Bulimia nervosa, the dominant eating essay.** "Etiopathogenesis, clinical features and management of bulimia nervosa" has come as a full 25-mark essay, and "Bulimia nervosa" recurs as a standalone 10-mark note. Prepare the binge-purge cycle and the compensatory behaviours, the medical complications (the hypokalaemia and metabolic alkalosis, Russell's sign, dental erosion), and the guideline-anchored management (cognitive behaviour therapy for eating disorders and guided self-help first-line, fluoxetine as the licensed drug at the high dose). MUHS 2014, 2016

Q **Binge eating disorder as a standalone note.** "Binge eating disorder" has come as a 10-mark note. Prepare the recurrent binge eating with loss of control but without the regular compensatory behaviour that defines bulimia, the association with obesity, and the cognitive-behaviour-therapy-first management. MUHS 2014

Q **Anorexia nervosa and safe refeeding.** Anorexia and its refeeding are high-yield across the exams even where the exact wording varies: prepare the diagnostic criteria (the significantly-low-weight criterion and the dropping of amenorrhoea in DSM-5), the medical complications by body system, the refeeding syndrome (hypophosphataemia as the hallmark, correct the electrolytes, give thiamine and start slow), and the management (no drug first-line, the psychological therapies and family-based treatment). Refeeding and its electrolyte monitoring are exactly the kind of dose-and-value detail an examiner probes. high-yield across sittings

Q **Insomnia, the dominant sleep essay.** "Classify insomnia and discuss its evaluation, management and prognosis" has come as a 10-mark question. Prepare the classification and the sleep-history assessment, and above all the management: cognitive behaviour therapy for insomnia is first-line for chronic insomnia, a hypnotic is not first-line and is short-term, and the pharmacology and its taper are cross-referenced. MUHS 2013

Q **The circadian rhythm sleep-wake disorders as a note.** "Circadian sleep-wake rhythm" has recurred as a short answer within the sleep and organic material. Prepare the phase types (delayed and advanced sleep-wake phase, non-24-hour, shift-work and jet lag) and the management with timed light and melatonin according to the phase-response curve. MUHS 2012, 2015

Q **Normal sleep, narcolepsy and the parasomnias, prepared for the spot and the note.** Normal sleep architecture and the hypnogram, the narcolepsy tetrad and the multiple sleep latency test, obstructive sleep apnoea and its psychiatric masquerade, and REM sleep behaviour disorder as a prodrome of a synucleinopathy are all examinable as spotters and short notes. Prepare each to be recognised and named, with the values (the sleep-architecture percentages, the AHI severity bands) exam-ready. spot and short note

Prepare this material to be diagnosed and treated, not just recited: the eating-disorder differentiation grid, the refeeding electrolyte thresholds and rate, the scale cut-offs (SCOFF, the ISI bands, the ESS and STOP-BANG cut-offs) and the AHI severity bands, and the value-anchored good-to-remember boxes in these notes are built to the way the block is examined. The deep hypnotic pharmacology lives in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes; the deep antidepressant pharmacology in the Mood disorders: pharmacotherapy notes; the sleep neurochemistry and the ascending-arousal anatomy in the Neurochemistry notes and the Functional neuroanatomy notes; and REM sleep behaviour disorder as a neurodegenerative prodrome is followed up in the Dementias and neurocognitive disorders notes and the Neurology foundations for psychiatrists notes.

Quick review

THE EATING DISORDERS: DISCRIMINATE, REFEED SAFELY, FIRST-LINE	
THE DISCRIMINATOR	Separate the six by **weight** (low in anorexia, normal or above in bulimia, often raised in binge eating disorder), the presence of a **binge**, the **compensatory behaviour**, and the **weight-shape overvaluation**. ARFID is the trap: food restriction with weight loss but **no body-image concern**, so it is not anorexia.
ANOREXIA NERVOSA	Significantly low weight, fear of weight gain, body-image disturbance (amenorrhoea dropped in DSM-5). Complications by system; the cardiac (bradycardia, long QT, arrhythmia) and electrolyte (hypokalaemia) ones kill. **No drug is first-line**: medical stabilisation, nutritional rehabilitation, and the psychological therapies (CBT-ED, MANTRA, SSCM in adults; family-based treatment in the young).
REFEEDING SYNDROME	The lethal danger of recovery. Starvation depletes intracellular phosphate, potassium and magnesium; carbohydrate refeeding drives an insulin surge that crashes the serum, with **hypophosphataemia the hallmark**. Prevent it: correct electrolytes (magnesium before potassium), give **thiamine before feeding**, and start slow, remembering that the two numeric sources differ: NICE CG32 caps the high-risk start at **10 kcal per kg per day** (only **5 if BMI is under 14**) with full needs within **4 to 7 days**, while RCPsych MEED CR233 2022 starts at **20 kcal per kg per day** and warns against underfeeding. NG69 gives no figure.
BULIMIA AND BINGE EATING DISORDER	Bulimia: normal weight, binge-purge cycle, hypokalaemia, Russell's sign, dental erosion; **CBT-ED first-line** plus **fluoxetine 60 mg per day**, the licensed drug. Binge eating disorder: recurrent binge **without** regular compensation, associated with obesity; CBT-ED and guided self-help first-line.
ARFID, OSFED, THE FEEDING DISORDERS	ARFID: restriction from sensory aversion, low interest or fear of aversive consequences, no weight-shape overvaluation. OSFED and atypical anorexia meet some but not all criteria; the non-fat-phobic presentation is common in India. Pica and rumination are the feeding disorders.
THE SCREENS	**SCOFF**: five items, **two or more** positive is a positive screen. **EAT-26**: total **20 or more** prompts assessment. Both are screens, not diagnoses.

NORMAL SLEEP AND THE DISORDERS OF TOO LITTLE OR TOO MUCH	
NORMAL ARCHITECTURE	NREM stages N1, N2 (spindles and K-complexes) and N3 (slow-wave), plus REM, cycling every **about 90 minutes** across four to five cycles. First REM at a latency of **about 90 minutes**; N3 dominates the first third of the night and REM lengthens toward morning. Regulated by process S (homeostatic drive) and process C (circadian rhythm), with a wake-promoting ascending arousal system and a sleep-promoting VLPO in a flip-flop switch stabilised by orexin.
INSOMNIA AND CBT-I	Difficulty initiating or maintaining sleep with daytime consequences despite adequate opportunity. **CBT-I is first-line for chronic insomnia**, not a hypnotic: stimulus control, sleep restriction (the engine), sleep hygiene, cognitive therapy and relaxation. Hypnotics (z-drugs, melatonin in the older adult) are short-term; the **ISI** bands run 0 to 7, 8 to 14, 15 to 21 and 22 to 28.
NARCOLEPSY	Loss of the orexin neurons destabilises the sleep-wake switch, so REM intrudes into wake: the **tetrad** of excessive daytime sleepiness, cataplexy (defines type 1), sleep paralysis and hypnagogic hallucinations. Type 1 has cataplexy, low CSF orexin and HLA-DQB1*06:02. Diagnosed on the **MSLT: mean sleep latency 8 minutes or less with 2 or more sleep-onset REM periods**. Treat with modafinil, solriamfetol or pitolisant, and sodium oxybate for cataplexy.
SLEEP APNOEA, THE PARASOMNIAS, AND THE CIRCADIAN DISORDERS	
OBSTRUCTIVE SLEEP APNOEA	Repetitive upper-airway collapse with apnoea or hypopnoea, desaturation and arousal. Graded by the apnoea-hypopnoea index: **mild 5 to 15, moderate 15 to 30, severe over 30** events per hour. It masquerades as treatment-resistant depression and cognitive impairment, so exclude it; **CPAP** is the mainstay. The **STOP-BANG** screen stratifies risk 0 to 2, 3 to 4 and 5 to 8.
PARASOMNIAS	NREM parasomnias (confusional arousals, sleepwalking, sleep terrors) arise from deep slow-wave sleep in the **first third of the night**, with amnesia and a childhood peak. REM parasomnias include **REM sleep behaviour disorder**, the loss of REM atonia with dream enactment, which is a **prodrome of a synucleinopathy** (Parkinson's disease, dementia with Lewy bodies, multiple system atrophy). Do not mistake it for a nightmare disorder.
CIRCADIAN AND MOVEMENT DISORDERS	Circadian disorders are a clock-schedule mismatch (delayed phase in teens, advanced phase in the elderly, non-24 in the blind, shift-work, jet lag); reset with **timed light and melatonin** along the phase-response curve (morning light and evening melatonin advance; evening light delays). Restless legs syndrome: the urge to move worse at rest and in the evening, relieved by movement; check **ferritin**, use dopaminergic or alpha-2-delta agents, and watch for dopaminergic augmentation.

THE NUMBERS TO HOLD	
REFEEDING START RATE	The two sources disagree. NICE CG32 1.4.8: maximum **10** kcal per kg per day at high risk, only **5** if BMI is under 14, full needs within **4 to 7 days**. RCPsych MEED CR233 2022: start at **20**, never below the pre-admission intake, avoid underfeeding. NG69 gives no figure. Quote whichever the question names.
BULIMIA DRUG	**Fluoxetine 60 mg per day**, the licensed drug (higher than the antidepressant dose)
EATING SCREENS	SCOFF **two or more** positive; EAT-26 total **20 or more**
ISI SEVERITY	0 to 7 none, 8 to 14 subthreshold, 15 to 21 moderate, 22 to 28 severe
ESS AND PSQI	Epworth **over 10** is excessive daytime sleepiness; PSQI global **over 5** is poor sleep quality
NARCOLEPSY MSLT	Mean sleep latency **8 minutes or less** with **2 or more** sleep-onset REM periods
SLEEP APNOEA AHI	Mild **5 to 15**, moderate **15 to 30**, severe **over 30** events per hour; STOP-BANG high risk **5 to 8**
SLEEP ARCHITECTURE	Cycle and REM latency **about 90 minutes**; N3 early, REM late; four to five cycles a night

Prepare this material to be diagnosed and treated, not just recited: the eating-disorder differentiation, the refeeding electrolyte thresholds and rate, the scale cut-offs and the AHI severity bands are the value-anchored points an examiner probes, and the first-line rules (no drug for anorexia, fluoxetine for bulimia, CBT-I for chronic insomnia) are the ones a wrong answer is marked down for. The deep hypnotic and antidepressant pharmacology lives in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes and the Mood disorders: pharmacotherapy notes; the sleep neurochemistry in the Neurochemistry, neuroendocrinology, neuroimaging and basic psychopharmacology notes; and REM sleep behaviour disorder as a neurodegenerative prodrome in the Dementias and neurocognitive disorders notes.

References

The eating-disorder and sleep-wake content is grounded in the standard psychiatry and sleep-medicine sources (the source hierarchy below), with Kaplan and Sadock and the New Oxford Textbook as the depth bar for the phenomenology, the neurobiology and the sleep architecture, Kaufman's Clinical Neurology for Psychiatrists for the polysomnography and the sleep-stage detail. The management is guideline-first: the eating half is led by the NICE eating-disorders guideline (NG69) with the MARSIPAN guidance for the medical management and refeeding of the severely ill patient, and the sleep half by the British Association for Psychopharmacology consensus on insomnia, parasomnias and circadian-rhythm disorders, with NICE, the Indian Psychiatric Society, the American Academy of Sleep Medicine and the United States VA/DoD. Every refeeding value, drug dose, scale cut-off, AHI band and first-line recommendation was checked against these sources and the adversarial content pass; anything that could not be confirmed, including the exact serum electrolyte thresholds and the lower edge of the mild-apnoea band, was flagged rather than guessed. Each journal PMID below was verified against PubMed this build and matched on title, first author and year; identifiers that resolved to the

wrong paper were corrected or dropped. Books and guideline documents are cited by author, title and year without a PMID.

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Index

Every entry jumps to its page. Numbers are the page of first mention.

A

actigraphy 241
adenosine 234
advanced sleep-wake phase 280
affective instability 23
alternative hybrid model 11
alternative model 20
alternative model of personality disorders 20
Anankastia 19
anankastic personality disorder 73
anorexia nervosa 160
anorexia-nervosa-focused family therapy 184
antisocial personality disorder 11
apnoea-hypopnea index 236
apnoea-hypopnoea index 262
applied behaviour analysis 216
arcuate nucleus 208
ARFID 201
Armand Loranger 96
armodafinil 259
atypical anorexia nervosa 215
autism spectrum disorder 204
avoidant personality disorder 29

B

BAP 161
Bateman and Fonagy 65
BDNF 207
BED 17
Binge eating disorder 160
binge eating disorder 160
binge-purge subtype 162
biosocial theory 86
bipolar II disorder 52
BMI 27
Bootzin 248
borderline pattern qualifier 20
borderline personality disorder 11
borderline personality organisation 119
Bratman 221
brief behavioural treatment for insomnia 252
bulimia nervosa 160
Bulimia nervosa 160
bulimia-nervosa-focused guided self-help 191
bupropion 193
Bupropion 193

C

cataplexy 231
categorical approach 36
categorical versus dimensional 36
CBT-ED 178
CBT-I 160
chair work 113
chronic emptiness 23
Chronotherapy 286
circadian rhythm sleep-wake disorder 241
Clarkin and Kernberg 115
Cleckley 61
clomipramine 260
clonazepam 246
Clonazepam 246
cognitive behaviour therapy for insomnia 160
cognitive behavioural therapy for insomnia 238
complex post-traumatic stress disorder 52
conduct disorder 29
confusional arousal 272
consultation team 92
Continuous positive airway pressure 262
CPAP 262
crisis management 130
crisis plan 126
cyproheptadine 205

D

delayed sleep-wake phase 280
dependent personality disorder 20
Detachment 16
dialectical behaviour therapy 11
Diaphragmatic breathing 216
digital CBT-I 243
dim light melatonin onset 284
dimensional approach 36
Disinhibition 19
dissocial personality disorder 59
Dissociality 19
distress tolerance 19
dopamine 206
DSM-5 11

DSM-5-TR 13

E

early maladaptive schema 109
EAT-26 163
EDE-Q 169
EDNOS 217
Ekbom syndrome 288
Emil Coccaro 88
emotion regulation 19
Epworth Sleepiness Scale 242
ESS 11
euthyroid sick syndrome 168

F

family-based treatment 161
ferritin 289
five-factor model 37
Fluoxetine 126
fluoxetine 126

G

gabapentin 291
Gerald Russell 189
ghrelin 206
Ghrelin 206
grandiose narcissism 66
guided self-help 187

H

Hare 25
Hare Psychopathy Checklist-Revised 62
harm avoidance 207
HCR-20 141
histrionic personality disorder 29
HLA-DQB1*06:02 254
HTR1D 207
hypnogram 161
hypocretin 231
hypoglossal nerve stimulator 269
hypokalaemia 162
hypomagnesaemia 171
hypomania 81
hypophosphataemia 167
Hypophosphataemia 167

I

ICD-10 20
ICD-11 11
ICD-11 dimensional model 11
ICSD-3 256
ideas of reference 28
Identity disturbance 23
imagery rescripting 113
Indian Psychiatric Society 12
insomnia 160
insomnia disorder 237
Insomnia Severity Index 238
insula 210
integrated management of borderline personality disorder 128
intellectual developmental disorder 215
International Personality Disorder Examination 93
Interoception 206
interpersonal effectiveness 19
IPDE 93
ISI 12

J

Jeffrey Young 108
jet lag 281

K

K-complexes 232
Kernberg 55
Kurt Schneider 14

L

Larry Siever 88
leptin 162
Leptin 162
Level of Personality Functioning 24
light therapy 280
Limited reparenting 109
Linehan 11
Lisdexamfetamine 199
lisdexamfetamine 199
loss of control eating 195

M

magical thinking 28
MANTRA 182
Marsha Linehan 86
MARSIPAN 161
Mary Zanarini 99
Maudsley Anorexia Nervosa Treatment for Adults 182
melanocortin-4 receptor 208
melatonin 161
Melatonin 161
Mentalisation-based therapy 115

mentalisation-based treatment 52
mentalising 115
mild personality disorder 22
mindfulness 19
mirtazapine 205
mnemonic 19
modafinil 259
Modafinil 259
moderate personality disorder 22
mslt 236
MSLT 236
multiple sleep latency test 236

N

narcissistic personality disorder 29
narcolepsy 160
Narcolepsy 160
Negative affectivity 19
neuropeptide Y 208
NICE 12
NICE NG69 171
nightmare disorder 231
non-24-hour 280
non-fat-phobic 160
NREM parasomnia 272

O

obese 190
object relations 108
obsessive-compulsive disorder 73
obsessive-compulsive personality disorder 29
obstructive sleep apnoea 160
olanzapine 125
oral appliance 269
orexin 161
Orlistat 192
orthorexia 217
Orthorexia nervosa 217
orthorexia nervosa 217
OSFED 189
osteoporosis 162
other specified feeding or eating disorder 214
Otto Kernberg 55

P

paranoid personality disorder 28
parasomnia 160
parotid 187
pathological trait domains 24
Perfectionism 21
periodic limb movement disorder 236
Periodic limb movement disorder 236
periodic limb movement index 236
Periodic limb movements in sleep 292
personality difficulty 17
personality disorder 11
Personality Inventory for DSM-*5* 93
Peter Fonagy 89
phone coaching 92
pica 16
Pica 16
PID-5 93
pitolisant 259
polysomnography 161
Polysomnography 161
pramipexole 291
pregabalin 291
progressive muscle relaxation 247
PSQI 315
psychopathy 59
purging disorder 190

R

radical acceptance 103
ramelteon 161
Refeeding syndrome 160
refeeding syndrome 160
Relaxation 243
REM 12
REM latency 229
REM sleep 160
REM sleep behaviour disorder 160
restless legs syndrome 236
restricting subtype 162
rhabdomyolysis 173
RLS 165
ropinirole 291
rotigotine 291
rumination disorder 212
Rumination disorder 212
Russell's sign 187

S

schema modes 91
schema therapy 11
schizoid personality disorder 28
schizotypal disorder 48
schizotypal personality disorder 28
SCID-5-PD 93
SCOFF 163
self-induced vomiting 166
serotonin 12

sertraline 126
severe personality disorder 22
shift work 281
short-term crisis 124
Sir William Gull 162
sleep efficiency 247
sleep history 239
sleep hygiene 237
Sleep hygiene 237
sleep paralysis 231
Sleep restriction 243
sleep restriction 243
sleep spindles 231
sleep terror 272
sleepwalking 231
slow-wave sleep 230
social anxiety disorder 29
sodium oxybate 253
Sodium oxybate 253
Soloff 124
solriamfetol 259
Specialist Supportive Clinical Management 182
Spielman 248
splitting 54
SSCM 182
stepped care 129
Stimulus control 243
stimulus control 243
STOP-BANG 241
Structured Clinical Interview for DSM-5 Personality Disorders 93
structured psychological therapy 12
suprachiasmatic nucleus 234
synucleinopathy 161

T

Temperament and Character Inventory 207
thiamine 167
topiramate 125
Topiramate 125
trait domain 11
transference-focused psychotherapy 11
two-process model 230

U

Unspecified feeding or eating disorder 214
uvulopalatopharyngoplasty 269

V

ventrolateral preoptic nucleus 231
vulnerable narcissism 66

W

Wernicke's encephalopathy 171
WFSBP 12

Y

Young 63

Z

z-drug 161
ZAN-BPD 93
Zanarini 56
Zanarini Rating Scale for Borderline Personality Disorder 93
zolpidem 244
zopiclone 244

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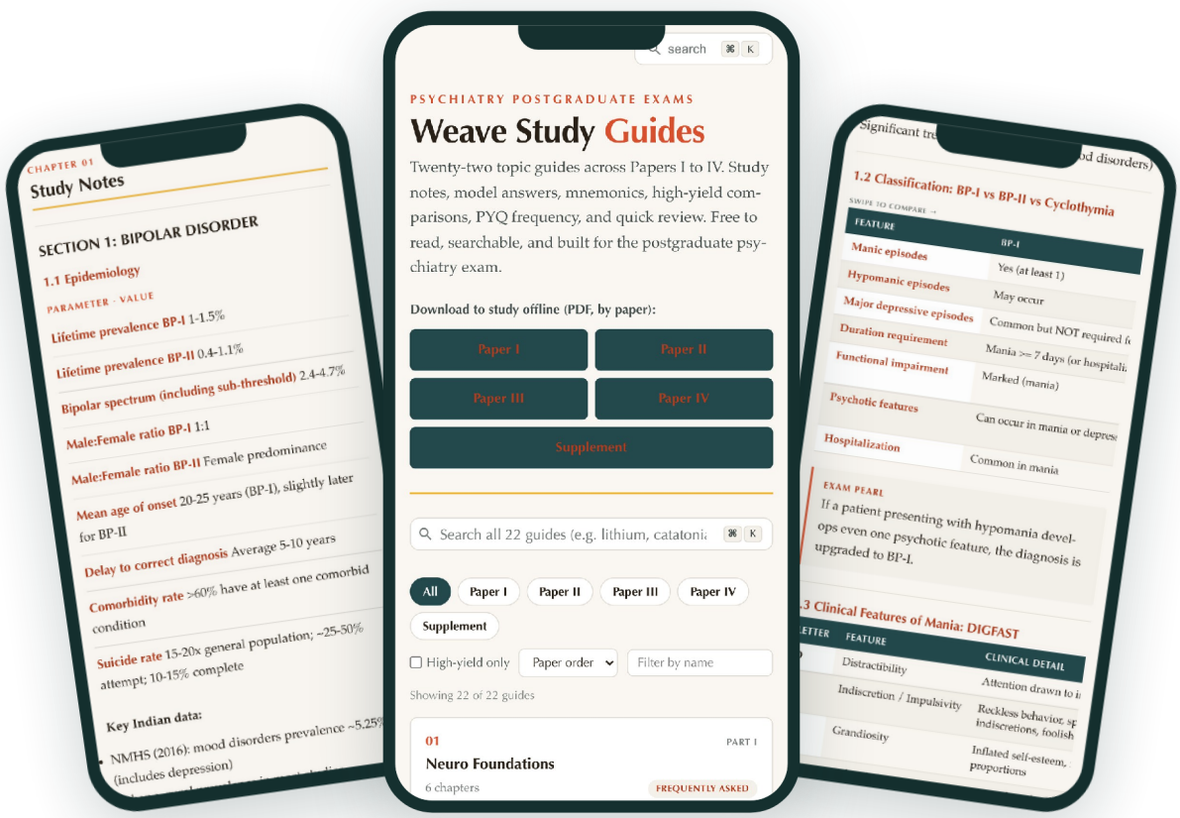
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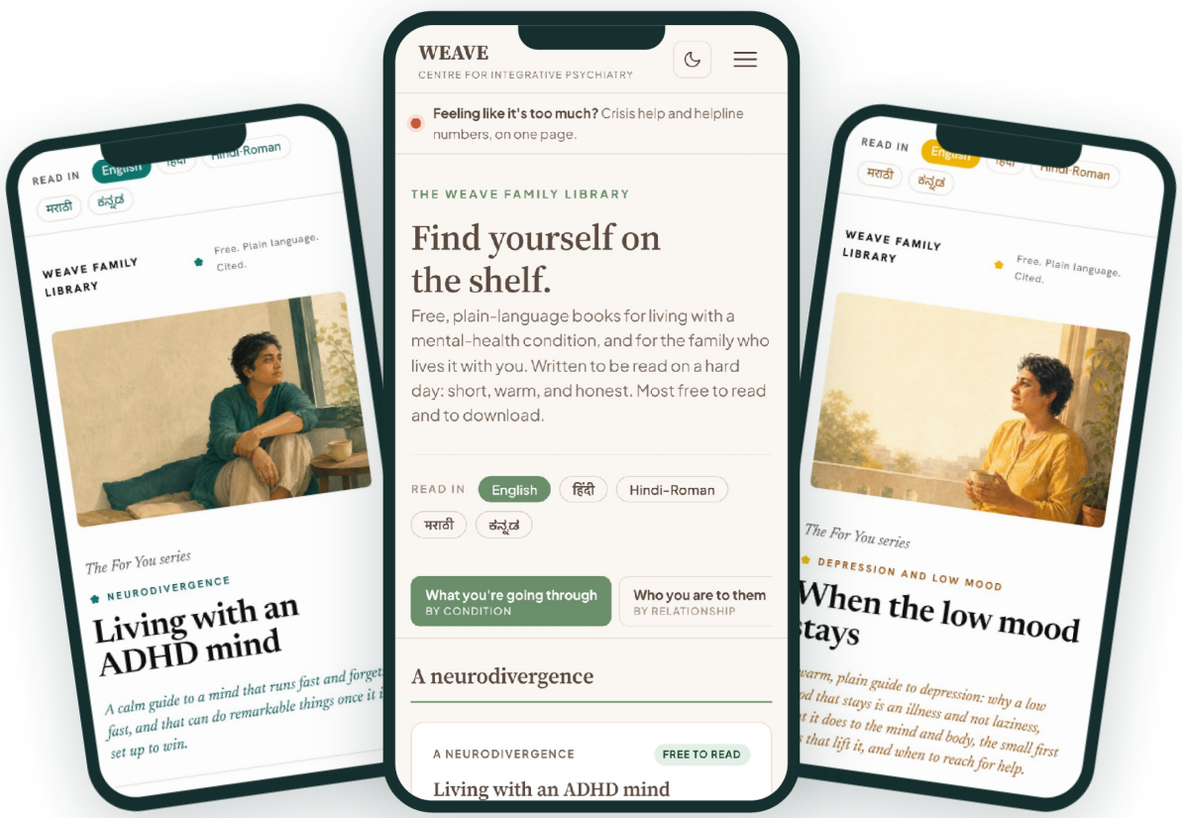
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