WEAVE SPRINT . PAPER II

Eating & sleep-wake

The eating and the sleep-wake disorders in one document: anorexia and the refeeding syndrome that can kill during recovery, bulimia and binge eating disorder, ARFID and the atypical spectrum; then normal sleep architecture and the hypnogram, insomnia and the first-line place of CBT-I, narcolepsy and its tetrad, obstructive sleep apnoea, the parasomnias, and the circadian disorders, led by NICE, MARSIPAN and the BAP guidelines with the Indian position throughout.

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WEAVE . CENTRE FOR INTEGRATIVE PSYCHIATRY

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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*.

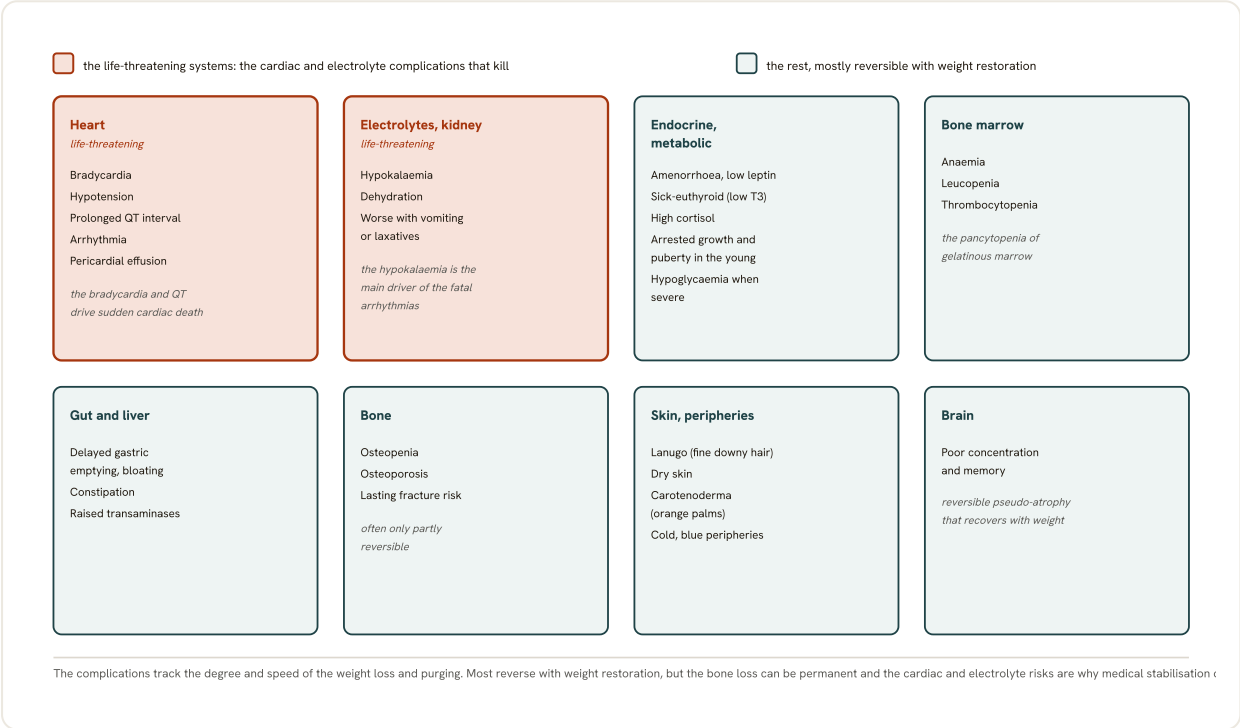

Fig 17.1 The medical complications of anorexia nervosa by body system. Starvation touches every organ. The cardiac and electrolyte complications (coral) are the ones that kill: bradycardia, hypotension, a prolonged QT interval and arrhythmia, and the hypokalaemia and dehydration made worse by vomiting 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.

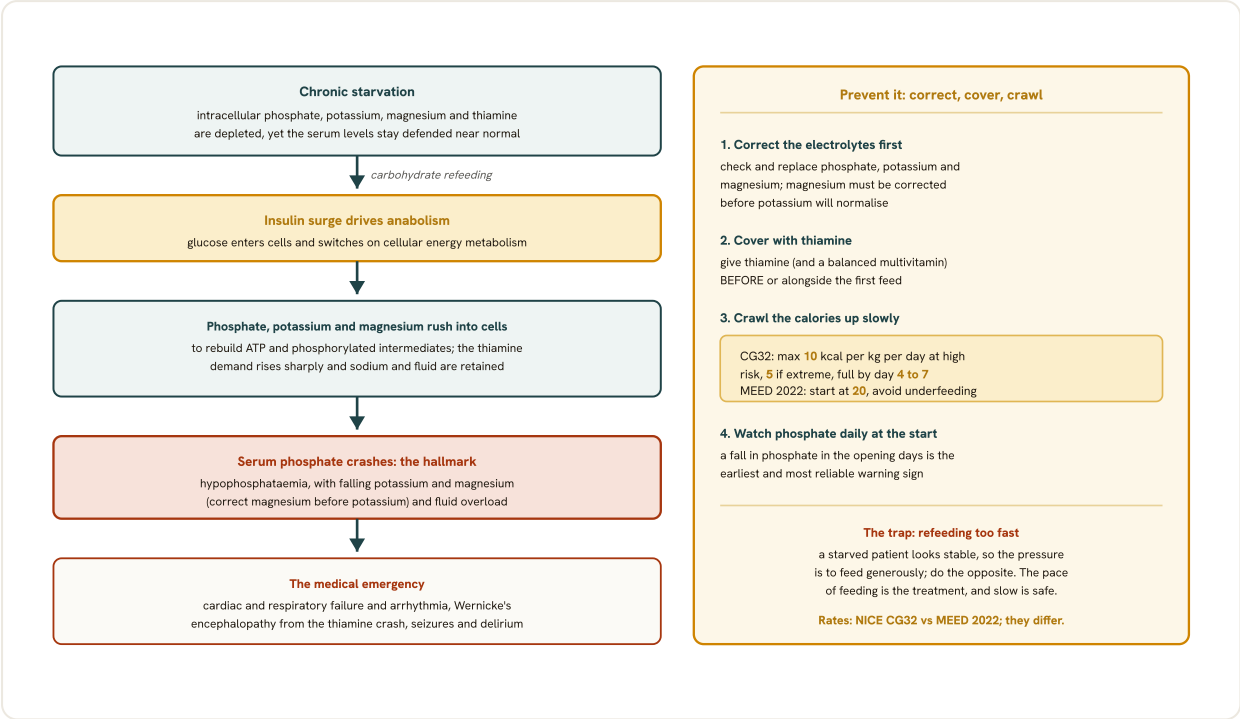

Fig 17.3 Refeeding syndrome. Starvation depletes the intracellular stores of phosphate, potassium, magnesium and thiamine while the serum levels stay defended near normal, so a routine blood test before feeding can look reassuring. When carbohydrate is reintroduced, the insulin surge switches anabolism 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*.

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
BINGE EPISODES FOR DIAGNOSIS, MINIMUM (PER WEEK, OVER THREE MONTHS)

3 of 5
ASSOCIATED FEATURES REQUIRED (WITH MARKED DISTRESS AND LOSS OF CONTROL)

50 to 70
LISDEXAMFETAMINE, THE ONLY LICENSED AGENT (MG PER DAY)

4 to 7
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.

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 RECOGNISED DRIVERS OF RESTRICTION	**4** CRITERION A CONSEQUENCES (AT LEAST ONE REQUIRED)	**14%** 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%	28–83%	57%	1994
AN TWIN-STUDY HERITABILITY RANGE	BN TWIN-STUDY HERITABILITY RANGE	BED HERITABILITY (UPPER TWIN ESTIMATE)	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	5%	28%	1 to 2%
MINIMUM SUGGESTED AGE FOR PICA (YEARS)	PICA PREVALENCE, SCHOOL-AGE CHILDREN	PICA IN PREGNANCY OR POSTPARTUM (META-ANALYSIS)	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 17.4 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.

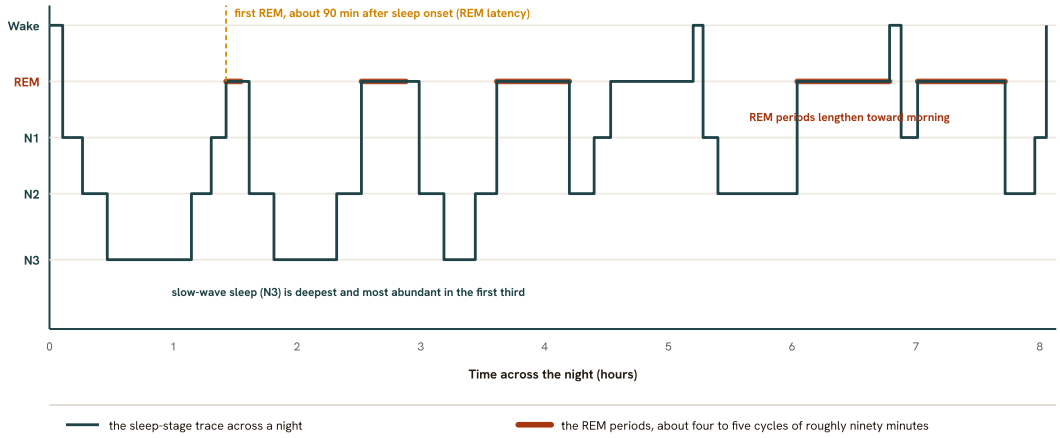

Fig 17.5 The sleep-architecture hypnogram. A normal night runs in four to five cycles of about ninety minutes, each descending from light N1 and N2 into deep slow-wave N3 sleep and then rising into a period of REM. The first REM period arrives about ninety minutes after sleep onset, the normal REM latency, 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.

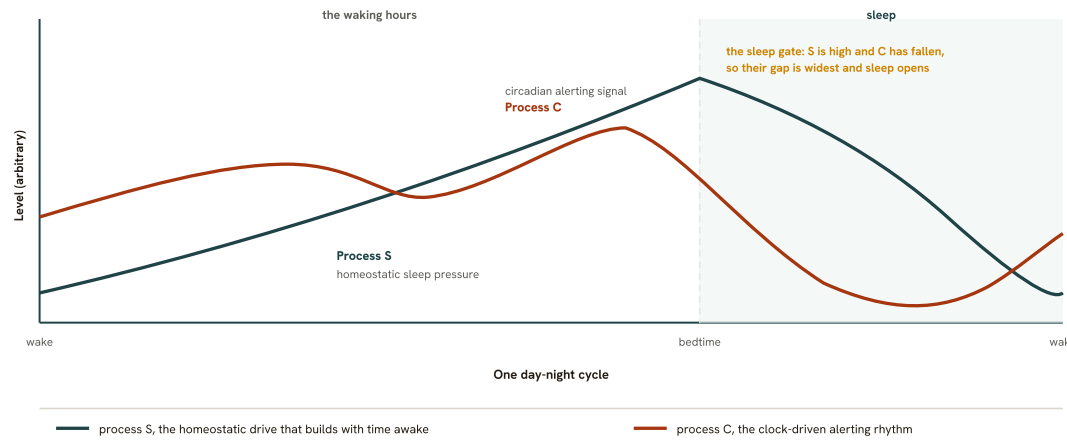

Fig 17.6 The two-process model of sleep regulation. Two independent processes set when we sleep. Process S is the homeostatic sleep pressure that builds steadily with every hour awake and discharges across sleep, so the longer the prior wakefulness the deeper the ensuing slow-wave sleep. Process C is 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.

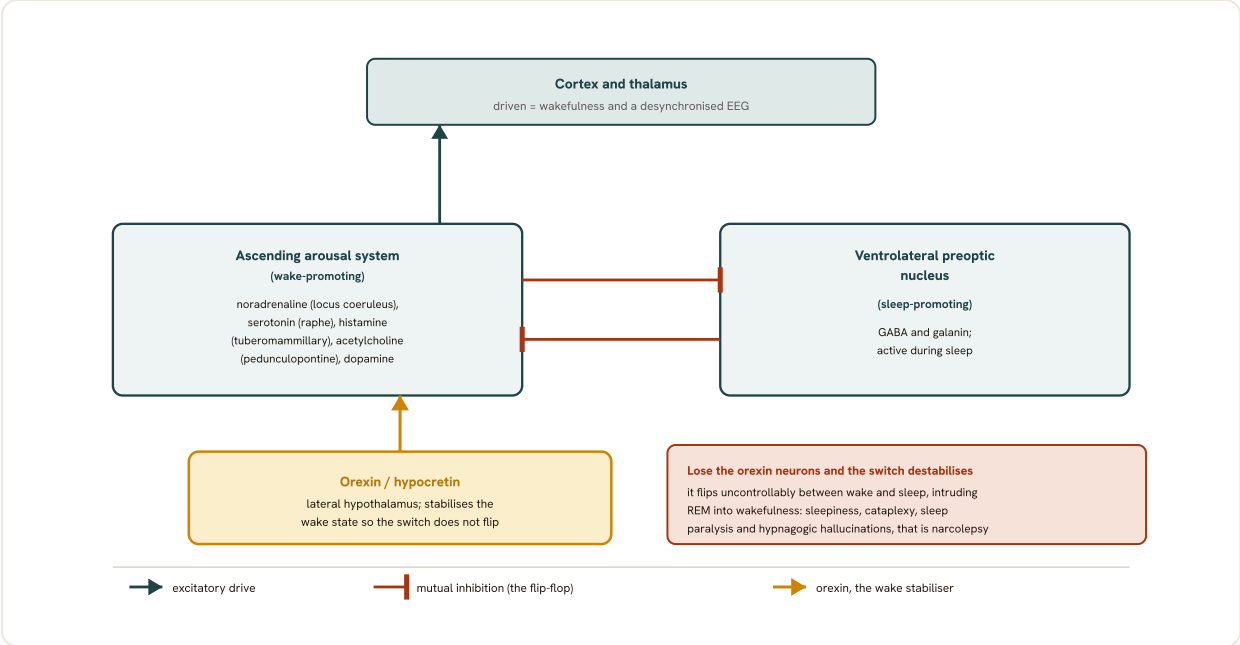

Fig 17.7 The ascending arousal system and the sleep-wake flip-flop switch. Wakefulness is driven by the ascending arousal system, a set of brainstem and hypothalamic monoaminergic and cholinergic nuclei that project up to the cortex; sleep is driven by the ventrolateral preoptic nucleus, which uses GABA 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 17.8 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	15 to 21	3	1 to 6
ISI SCREENING CUT-OFF FOR INSOMNIA (SCORE)	ISI MODERATE CLINICAL INSOMNIA BAND (SCORE)	CHRONIC INSOMNIA DURATION THRESHOLD (MONTHS)	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.

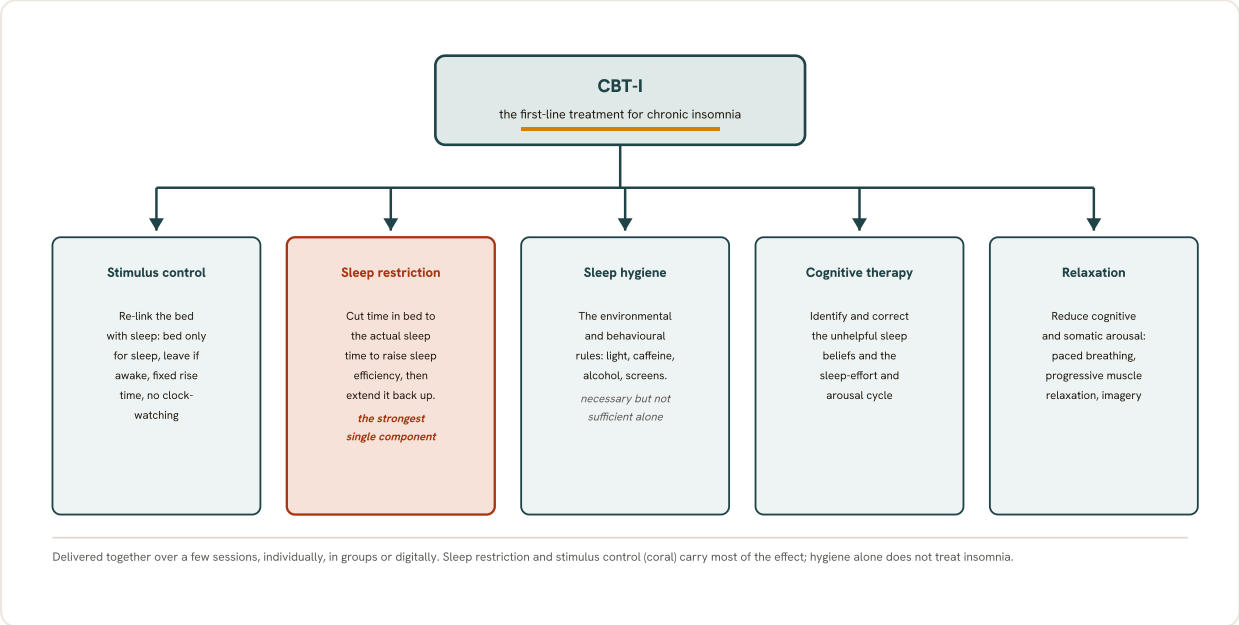

Fig 17.9 The components of cognitive behaviour therapy for insomnia. CBT-I, not a hypnotic, is the first-line treatment for chronic insomnia, and it is a package of five components. Stimulus control re-associates the bed with sleep; sleep restriction, the single most powerful element, curtails time in 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.

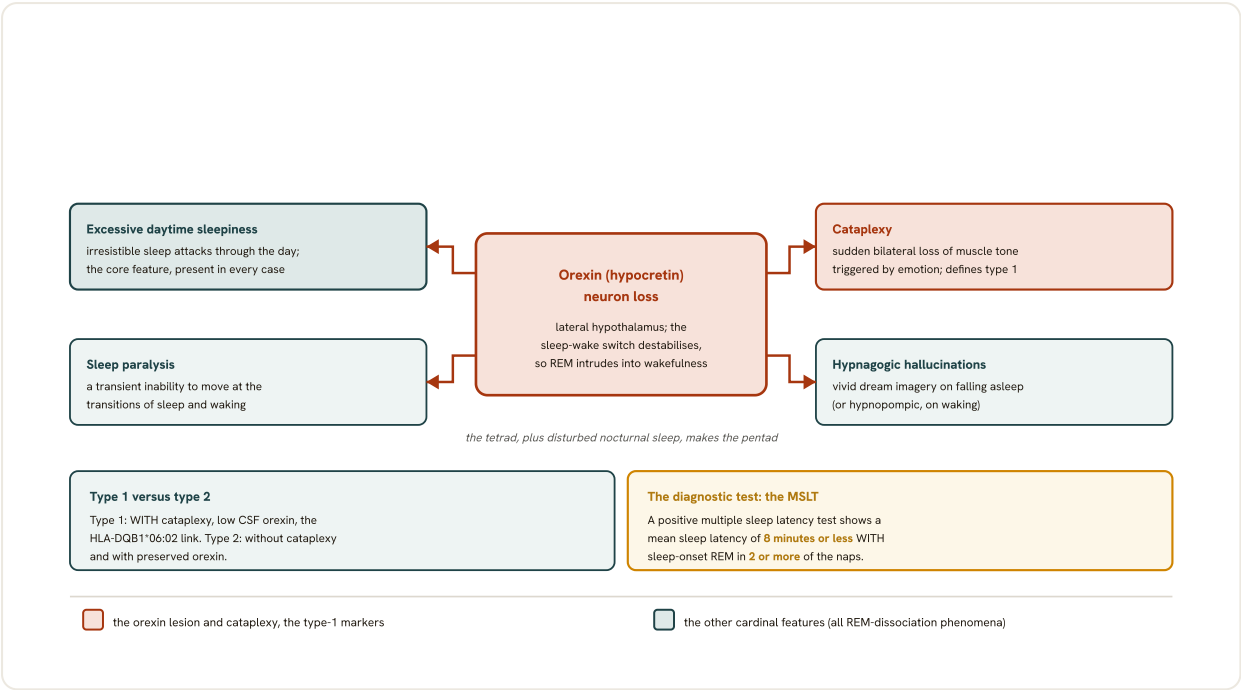

Fig 17.10 Narcolepsy: the orexin loss and the tetrad. The lesion is the loss of the orexin (hypocretin) neurons of the lateral hypothalamus, the peptide that stabilises the sleep-wake flip-flop switch, so the switch becomes unstable and the phenomena of REM sleep intrude into wakefulness. That produces 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, un-resistible 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.

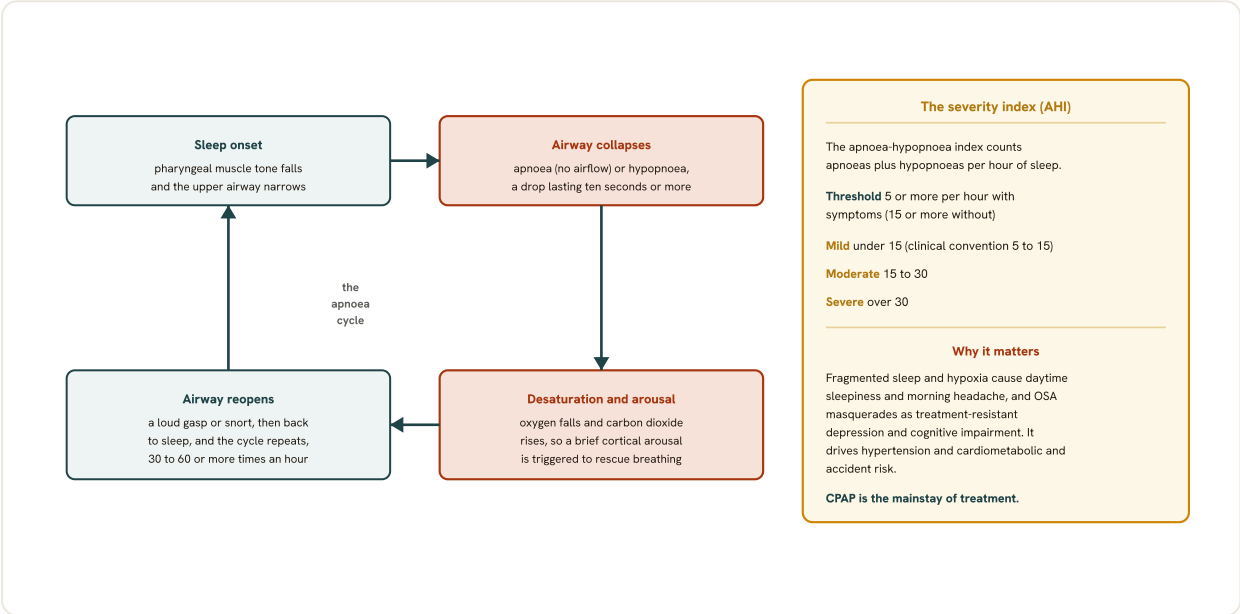

Fig 17.11 Obstructive sleep apnoea: the apnoea-hypopnoea cycle. In sleep the pharyngeal muscle tone falls and, in a susceptible airway, the upper airway collapses, so airflow stops (apnoea) or is sharply reduced (hypopnoea) for ten seconds or more. Oxygen falls and carbon dioxide rises until a brief 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 17.12 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.

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)
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.

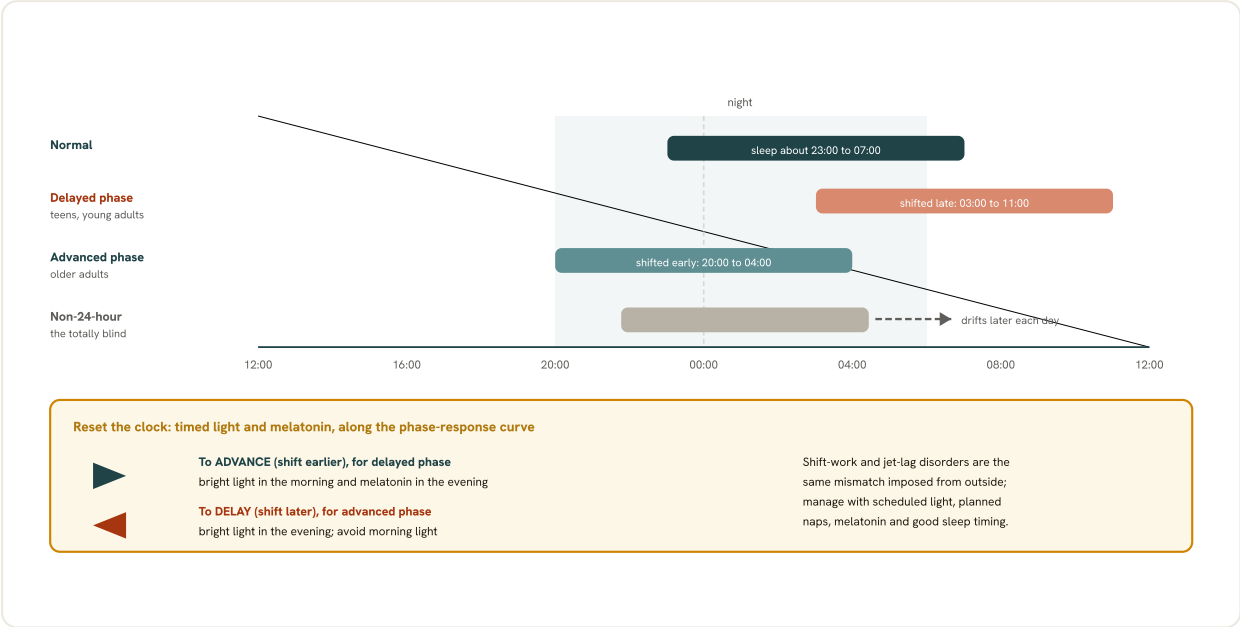

Fig 17.13 The circadian rhythm sleep-wake disorders. Each is a mismatch between the internal circadian clock and the desired or required sleep-wake schedule. In delayed sleep-wake phase disorder, common in adolescents, the whole sleep window is shifted late, so the person cannot fall asleep or wake at 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

SUBTYPE	TYPICAL POPULATION	SLEEP-WAKE PATTERN	FIRST-LINE MANAGEMENT (PER PHASE-RESPONSE CURVE)
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.

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	4	20 to 40	>80%
SERUM FERRITIN IRON-THERAPY THRESHOLD (MCG PER L)	ESSENTIAL RLS QUESTIONS, ALL MUST BE "YES"	INTER-MOVEMENT INTERVAL IN PLMS (SECONDS)	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**
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.

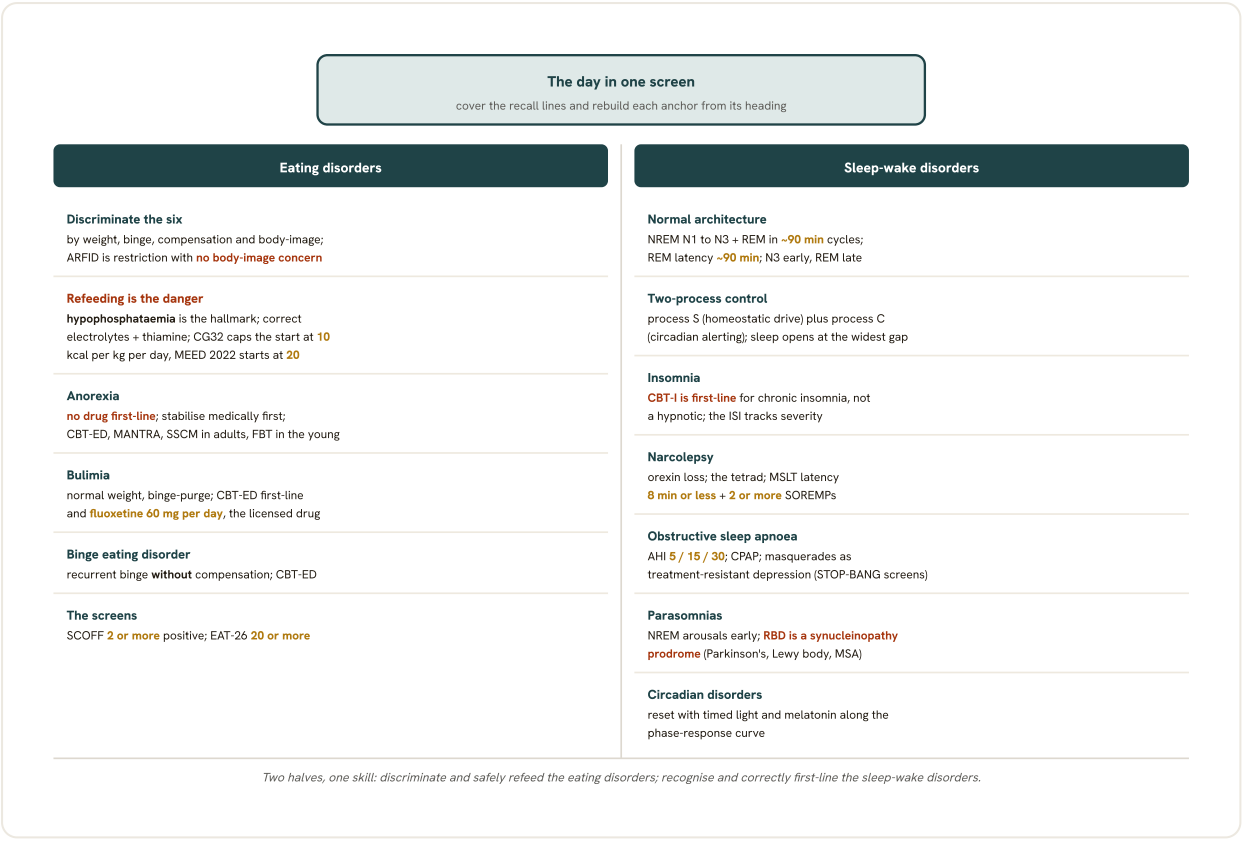

Fig 17.14 The chapter in one screen. The eating half reduces to four moves: discriminate the six presentations by weight, binge, compensation and body-image (with ARFID set apart by having no body-image disturbance); treat the refeeding syndrome as the lethal danger, marked by hypophosphataemia and prevented 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
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**

DISORDER	THE DISCRIMINATOR TO STATE (PROMPT, THEN CHECK)
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 REM SHARE OF THE NIGHT (% OF TOTAL SLEEP TIME)	**90** FIRST REM LATENCY AND NREM-REM CYCLE (MINUTES)	**≤8** MSLT POSITIVE MEAN SLEEP LATENCY (MINUTES)	**>30** 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**
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

OBJECT	THE DISCRIMINATOR AND NUMBER TO STATE (PROMPT, THEN CHECK)
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

- **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

- **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

- **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

- **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

- **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

- **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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