WEAVE SPRINT . PAPER II

Somatic, sexual & suicide

The somatic, sexual, impulse-control and suicide-related disorders in one document: the somatic-symptom nosology and its positive criterion, factitious disorder and malingering; the sexual response cycle, the male and female dysfunctions, gender incongruence and Dhat; the impulse-control disorders; and the suicide and self-harm band under strict safe-messaging, from structured risk assessment and safety planning to means restriction and the anti-suicidal evidence, led by the guidelines and the Indian position throughout.

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About this insert. Weave Sprint is educational outreach from Weave, the psychiatry vertical of Trijog. Therapy is provided through Trijog.

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Concept / rule Key number Trap

PAPER II

Somatic-symptom, sexual, impulse-control disorders and suicide

Five sections . one complete notes document

This material gathers the somatic-symptom, sexual, impulse-control and suicide-related disorders into one document, by syndrome and by management. The first band is **the somatic symptom and related disorders**: somatic symptom disorder and the somatoform-to-SSD nosology shift with its positive psychological criterion, illness anxiety disorder, conversion or functional neurological symptom disorder placed on the spectrum, factitious disorder and malingering, and the management of medically unexplained symptoms. The second band is **the sexual dysfunctions, paraphilias and gender**: the normal sexual response cycle, the male and female sexual dysfunctions and their management, the paraphilic disorders, gender incongruence and the affirmative-care pathway, Dhat and the culture-bound presentations, and medication-induced sexual dysfunction. The third band is **psychosomatic medicine and consultation-liaison**, taught here as the psychosomatic concept with the service model and disease-specific aspects cross-referenced. The fourth band is **the impulse-control disorders**. The fifth band is **suicide and self-harm**, the high-stakes clinical core, written throughout with strict safe-messaging: the structured risk assessment and the balance of risk and protective factors, the management of the suicidal patient and collaborative safety planning, deliberate self-harm, prevention through means restriction and gatekeeper training, and the anti-suicidal pharmacology. The examinable skill is the safe, guideline-anchored assessment and management of each: the positive criterion behind a somatic presentation, factitious versus malingering, the first-line for erectile dysfunction, the affirming frame for gender incongruence, and the structured, compassionate assessment and safety planning of a person at risk.

HOW TO USE THESE NOTES

Read the five bands as one continuous account of how these disorders are assessed and managed. The **somatic symptom and related** band builds the nosology: the shift from the somatoform disorders to somatic symptom disorder with its positive psychological criterion, illness anxiety disorder, and the discrimination of the somatic disorders from factitious disorder and malingering by voluntariness and external incentive. The **sexual, paraphilias and gender** band teaches the sexual response cycle, the male and female dysfunctions and their guideline-anchored management, the paraphilic disorders, gender incongruence taught in the affirming ICD-11 frame with the Indian legal position, and Dhat as a culture-bound presentation. The **psychosomatic and consultation-liaison** band teaches the psychosomatic concept compactly. The **impulse-control** band teaches the disorders of urge and control. The **suicide and self-harm** band is the core: the structured risk assessment and the balance of risk and protective factors, the limits of individual-risk prediction, the compassionate non-judgemental assessment of self-harm, collaborative safety planning, and prevention through means restriction, all written with strict safe-messaging, stating the burden without method specifics and framing means restriction, help-seeking and safety planning positively. Every management recommendation is guideline-anchored, led by NICE for the self-harm and suicide pathway with the WHO LIVE LIFE frame, ISSM and BSSM for the sexual dysfunctions and WPATH SOC-8 for gender, 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 first move. 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; every suicide figure is conceptual and non-sensational, with no method depiction. The rating scales are reproduced as the actual instruments where the licence permits, the Columbia scale the key plate, credited to their sources, or typeset from their structure where it does not. Several boundaries are stated up front: conversion or functional neurological symptom disorder is taught in full in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes and cross-referenced here; the consultation-liaison service model and the disease-specific psychiatric aspects in the Consultation-liaison, geriatric and transcultural psychiatry notes; body dysmorphic disorder in the Anxiety, OCD and trauma-related disorders notes; and the deep antidepressant, antipsychotic and the lithium and clozapine pharmacology in the Mood disorders: pharmacotherapy notes and the Schizophrenia: management, antipsychotics and adverse effects notes. The somatic nosology, the factitious and malingering discrimination, the sexual and gender syndromes, Dhat, the impulse-control disorders and the whole suicide and self-harm band are owned and fully taught here. This is doctor-facing revision, so the full technical vocabulary is used, brand names lead with the Indian brand, and every dose, scale cut-off, criterion and legal provision is verified against a source or omitted and flagged.

Somatic symptom and related disorders

1 · Somatic symptom disorder and the somatoform nosology shift

This is the opening topic of a chapter that runs from the body that *complains* to the body that *desires*, then to the urge that *acts* and finally to the life that must be *protected*; the whole arc is

stitched together by one master mnemonic seeded here and paid off at consolidation. It opens with **somatic symptom disorder** because that entity is the product of the single largest reclassification in this part of the syllabus: the DSM-IV and ICD-10 **somatoform disorders**, a scattered family diagnosed by what could *not* be found on investigation, have collapsed forward into one positively defined diagnosis. Examiners test this **nosology shift** two ways, and you must be fluent in both: a straight "what replaced what" crosswalk, and a discrimination task where a vignette must be placed in the right modern category and defended against its neighbour.

How to use this page. Fix the three DSM-5-TR criteria first, then unpack Criterion B (the **positive psychological criterion**) because that clause carries most of the marks, then internalise the one conceptual break, that being **medically unexplained** is no longer a requirement, so a symptom may be fully medically explained and still qualify. After that, learn the ICD-10 family that was folded in, the DSM-5 specifiers, and the parallel ICD-11 move to **bodily distress disorder**; finish with the assessment scales and the guideline-anchored first moves in management. The cardinal error the chapter is built to prevent is calling this diagnosis "medically unexplained".

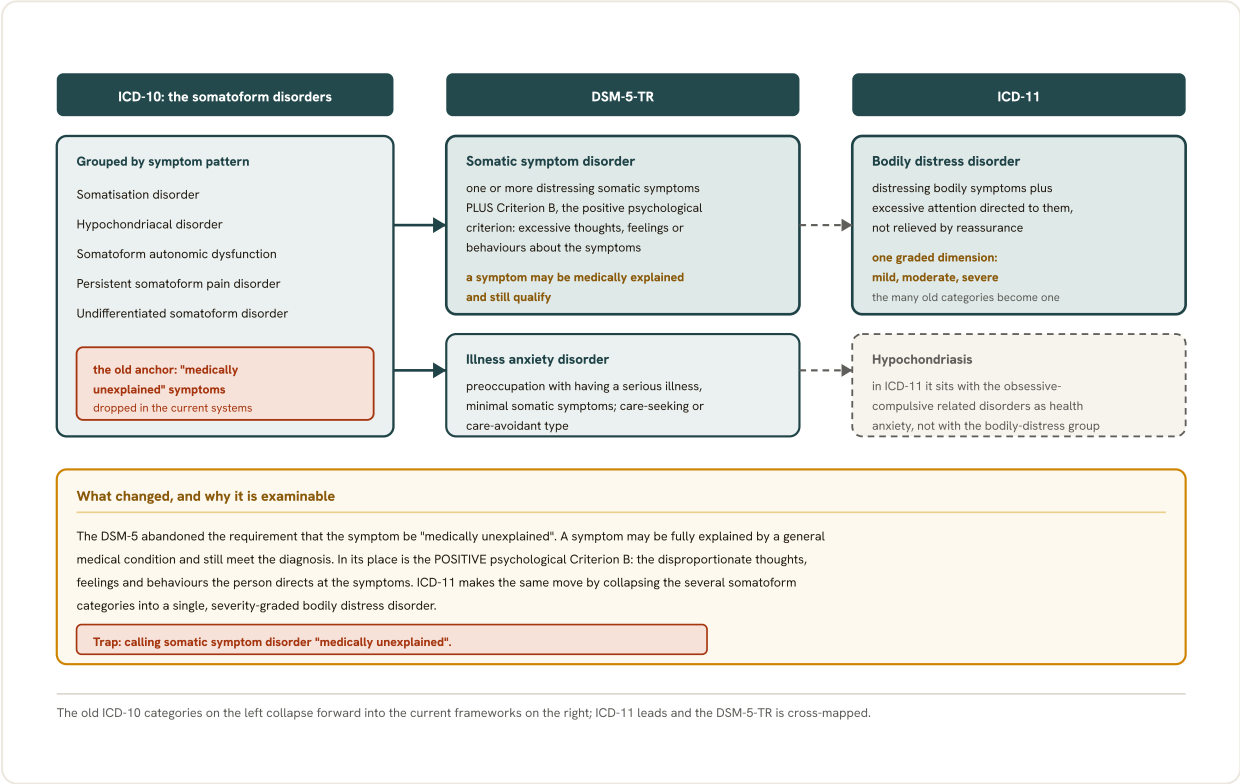

Fig 18.1 The somatic-symptom nosology shift. The ICD-10 somatoform disorders, grouped by the pattern of symptoms, collapse forward into a much simpler current picture. The DSM-5-TR replaces the whole group with somatic symptom disorder, which turns on a POSITIVE psychological criterion (excessive thoughts, feelings and behaviours about the symptoms) rather than on the symptom being medically unexplained, so a medically explained symptom may still qualify; hypochondriasis becomes illness anxiety disorder. ICD-11 goes further and folds the categories into a single, severity-graded bodily distress disorder, while placing hypochondriasis with the obsessive-compulsive related disorders as health anxiety. The examinable move, and the classic trap, is the retirement of "medically unexplained" in favour of the positive criterion.

Patient Health Questionnaire-15: Physical Symptoms

During the last 4 weeks, how much have you have been bothered by any of the following problems? Please place a check mark in the box to indicate your answer.

	Not bothered	Bothered a little	Bothered a lot
	0	1	2
1. Stomach pain	☐	☐	☐
2. Back pain	☐	☐	☐
3. Pain in your arms, legs, or joints (knees, hips, etc)	☐	☐	☐
4. Menstrual cramps or other problems with your periods [women only]	☐	☐	☐
5. Headaches	☐	☐	☐
6. Chest pain	☐	☐	☐
7. Dizziness	☐	☐	☐
8. Fainting spells	☐	☐	☐
9. Feeling your heart pound or race	☐	☐	☐
10. Shortness of breath	☐	☐	☐
11. Pain or problems during sexual intercourse	☐	☐	☐
12. Constipation, loose bowels, or diarrhea	☐	☐	☐
13. Nausea, gas, or indigestion	☐	☐	☐
14. Feeling tired or having low energy	☐	☐	☐
15. Trouble sleeping	☐	☐	☐
TOTAL SCORE =			

The PHQ-15: 15 common somatic symptoms, each scored 0 to 2 over the last four weeks (total out of a possible 30); the bands are 0 to 4 minimal, 5 to 9 low, 10 to 14 medium and 15 to 30 high somatic-symptom burden.

PHQ-15 (Kroenke, Spitzer and Williams, 2002); PHQ family, no permission required to reproduce; free non-commercial educational use.

SSD-12 (SOMATIC SYMPTOM DISORDER - B CRITERIA SCALE)	STRUCTURE AND THE VERIFIED CUT-OFF
What it measures	the DSM-5 Criterion B psychological burden (the thoughts, feelings and behaviours directed at the symptoms), not the somatic symptoms themselves
Structure	12 items across three subscales (cognitive, affective, behavioural), each item scored 0 to 4
Total and cut-off	**out of a possible 48; the validated cut-off is 23 and above (Toussaint 2016); a lower primary-care threshold of 13 and above has been proposed (Cao 2020)**

SSD-12 (Toussaint et al, 2016); copyright the authors and Mapi Research Trust, permission required, so typeset here in summary with the verified cut-off rather than reproduced.

Fig 18.2 The somatic-symptom assessment scales. The PHQ-15 grades the burden of the somatic symptoms themselves: fifteen common complaints, each scored 0 to 2 over four weeks for a total out of a possible 30, banded 0 to 4 minimal, 5 to 9 low, 10 to 14 medium and 15 to 30 high. The SSD-12 measures the other half of the modern diagnosis, the positive Criterion B, rating the excessive thoughts, feelings and behaviours the person directs at the symptoms across twelve items for a total out of a possible 48, with a validated cut-off of 23 and above. The PHQ-15 is shown as its actual freely reproducible form; the SSD-12 is permission-required and is summarised from its verified structure.

1.1 Somatic symptom disorder: the three DSM-5-TR criteria

One entity, defined by symptoms plus a psychological response. Somatic symptom disorder (DSM-5-TR) is built on three criteria (DSM-5-TR 2022). **Criterion A** requires one or more somatic symptoms that are distressing or result in significant disruption of daily life; the symptom may be specific, such as localised pain, or non-specific, such as fatigue, and it may or may not accompany a diagnosable medical condition. **Criterion B** is the heart of the diagnosis: excessive thoughts, feelings or behaviours related to the somatic symptoms or the associated health concerns. **Criterion C** requires that, although any single symptom need not be continuously present, the *state of being symptomatic* is persistent, typically for more than six months. The suffering is authentic whether or not it is medically explained; the diagnosis rests on the person's disproportionate response to bodily symptoms, not on the symptoms being unfounded.

GOOD TO REMEMBER

- **Somatic symptom disorder (the diagnosis):** Criterion A, one or more distressing or disruptive somatic symptoms; Criterion B, excessive thoughts, feelings or behaviours about the symptoms (the positive psychological criterion); Criterion C, persistent symptomatic state, typically more than six months. The single discriminator from every predecessor diagnosis is that Criterion B, not the absence of a medical explanation, defines it.

1.2 Criterion B: the positive psychological criterion

Three ways the excessive response can show, any one of which suffices. The **positive psychological criterion** is met by at least one of three manifestations (DSM-5-TR 2022): first, *disproportionate and persistent thoughts* about the seriousness of one's symptoms; second, a *persistently high level of anxiety* about health or symptoms; third, *excessive time and energy* devoted to the symptoms or health concerns, such as repeated bodily checking, help-seeking and reassurance-seeking. Only one of the three is required for the diagnosis, though the count of them then drives the severity specifier. This is the criterion that shifts the diagnostic gaze from the investigation report to the patient's cognitive, affective and behavioural relationship with the body, which is exactly why it can be measured directly by a Criterion-B scale rather than by cataloguing negative test results.

PEARL

Hold Criterion B as **T-A-B**: excessive **T**houghts (disproportionate and persistent), high **A**nxiety about health, and excessive **B**ehaviour and time devoted to symptoms. Any one of the three, running alongside a distressing somatic symptom, is enough; the number present grades the severity.

RULE **Diagnose on the positive criterion, not on a normal work-up.** Somatic symptom disorder is established by the excessive thoughts, feelings and behaviours of Criterion B, present alongside a distressing somatic symptom; whether the symptom is medically explained is irrelevant to whether the criteria are met.

1.3 The cardinal break: "medically unexplained" is gone

A medically explained symptom can still qualify. The defining conceptual move of the shift is the deletion of the old **medically unexplained** symptoms requirement (DSM-5-TR 2022; Kaplan and Sadock CTP 2024). Under DSM-IV and ICD-10, a somatoform diagnosis demanded that the symptoms lack an adequate physical explanation, which forced clinicians into an unreliable and often adversarial hunt to *exclude* disease before they could name distress. DSM-5 abolished that. The diagnosis now turns on the presence of the positive psychological features, so a patient whose symptom is entirely accounted for by a genuine medical condition, an uncomplicated myocardial infarction, say, still meets criteria if their thoughts, anxiety and behaviour about that symptom are clearly excessive and disabling. Somatic symptoms without a medical explanation are, on their own, *not* sufficient for the diagnosis; the psychological response is what is required. This single change also dissolves the old mind-body dualism the somatoform label carried, letting the diagnosis sit comfortably alongside, rather than in place of, medical disease.

WORKED EXAMPLE

A man is fully rehabilitated after an uncomplicated myocardial infarction with a normal ejection fraction, yet several months on he remains housebound, checks his pulse dozens of times a day, has abandoned work and exercise, and cannot be reassured by repeated normal reviews. His chest symptoms are medically explained, but his response is grossly disproportionate; he meets Criterion A (distressing symptom), Criterion B (all three manifestations) and Criterion C (persistent), so he has somatic symptom disorder *with* established cardiac disease. Under the old rules he would have been un-diagnosable because nothing was "unexplained".

TRAP **Calling somatic symptom disorder "medically unexplained".** That is the abolished DSM-IV and ICD-10 criterion. The modern diagnosis is defined by the positive psychological criterion; a medically explained symptom with an excessive response qualifies, and an unexplained symptom without that response does not.

1.4 The forward collapse: what DSM-5 moved where

Many old boxes, one new home, with a few relocations. DSM-5 replaced the DSM-IV "somatoform disorders" chapter with "somatic symptom and related disorders", and folded most of the old members forward into somatic symptom disorder (Kaplan and Sadock CTP 2024). DSM-IV somatisation disorder, undifferentiated somatoform disorder and pain disorder are all now captured by somatic symptom disorder, the last of them by its predominant-pain specifier. Hypochondriasis was *split*: roughly three-quarters of former cases, those with prominent somatic symptoms, become somatic symptom disorder, and the remainder, with high health anxiety but few or no somatic symptoms, become the new illness anxiety disorder. Two members left the

chapter's core in different directions: conversion disorder was retained but renamed functional neurological symptom disorder, and body dysmorphic disorder migrated to the obsessive-compulsive and related disorders. Factitious disorder was, conversely, brought *into* this chapter. The positive signs and management of the conversion presentation are developed in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes, and body dysmorphic disorder on the obsessive-compulsive spectrum in the Anxiety, OCD and trauma-related disorders notes.

DSM-IV SOMATOFORM MEMBER	DSM-5-TR DESTINATION	THE ONE-LINE REASON
Somatisation disorder	Somatic symptom disorder	**Positive Criterion B replaces the multi-system symptom count**
Undifferentiated somatoform disorder	Somatic symptom disorder	**Same positive-criterion home, no symptom threshold to meet**
Pain disorder	Somatic symptom disorder, with predominant pain	**Becomes a specifier, not a separate diagnosis**
Hypochondriasis	Split: somatic symptom disorder / illness anxiety disorder	**Somatic symptoms present goes to the former; health anxiety with few symptoms to the latter**
Conversion disorder	Functional neurological symptom disorder (retained, renamed)	**Kept, because it still needs incompatibility with disease, not a psychological criterion**
Body dysmorphic disorder	Obsessive-compulsive and related disorders	**Repetitive checking and preoccupation place it on the OCD spectrum**

The forward collapse of the DSM-IV somatoform disorders into DSM-5-TR, with the two relocations shown in the nosology-shift diagram alongside (Kaplan and Sadock CTP 2024, DSM-5-TR 2022).

ICD-10 TO DSM-5: WHAT CHANGED

ICD-10 (F45) **kept a family of separate somatoform diagnoses**, each requiring symptoms not fully explained by physical disorder: somatisation disorder (F45.0), undifferentiated somatoform disorder (F45.1), hypochondriacal disorder (F45.2), somatoform autonomic dysfunction (F45.3) and persistent somatoform pain disorder (F45.4), with neurasthenia sitting nearby at F48.0. DSM-5 **discarded both the family structure and the medically-unexplained rule**, replacing the lot with one positively defined somatic symptom disorder (plus a separate illness anxiety disorder for the health-anxiety remnant). The old names still get tested because clinicians and ICD-10-using services still speak them, so you must read fluently in both the old family and the new single diagnosis.

1.5 The ICD-10 somatoform family that was folded in

Five members, each with its own discriminator. The ICD-10 F45 group is worth holding intact, both because ICD-10 is still in service and because the members are the classic viva names (ICD-10). Somatisation disorder (F45.0), historically Briquet syndrome, needs multiple, recurrent and

frequently changing physical symptoms across several body systems for at least two years, with refusal to accept reassurance and resulting social impairment. Undifferentiated somatoform disorder (F45.1) is the sub-threshold version, the same picture without the full symptom load or duration. Hypochondriacal disorder (F45.2) is a persistent preoccupation with the possibility of having one or more serious progressive physical disorders, the focus being on the feared *diagnosis* rather than the symptoms themselves. Somatoform autonomic dysfunction (F45.3) presents symptoms as though arising from an autonomically innervated organ, with objective autonomic arousal (palpitations, sweating, tremor, flushing) plus subjective organ-referred complaints; its cardiovascular subtype is the eponymous Da Costa syndrome or cardiac neurosis. Persistent somatoform pain disorder (F45.4) is severe, distressing pain not fully explained physiologically, arising with emotional or psychosocial conflict.

ICD-10 SOMATOFORM MEMBER	CORE REQUIREMENT	THE DISCRIMINATOR TO HOLD
Somatisation disorder (F45.0)	Multiple, changing, multi-system symptoms for at least two years	**Symptom multiplicity plus the two-year duration; Briquet syndrome**
Undifferentiated somatoform (F45.1)	Same picture below the full threshold	**Fewer symptoms or shorter than two years**
Hypochondriacal disorder (F45.2)	Preoccupation with having a serious disease	**Focus is the feared diagnosis, not the symptom itself**
Somatoform autonomic dysfunction (F45.3)	Organ-referred symptoms with objective autonomic arousal	**Autonomic signs plus a named organ system; Da Costa for the heart**
Persistent somatoform pain (F45.4)	Persistent severe pain with emotional or psychosocial conflict	**Pain is the whole presentation, tied to psychosocial context**

The ICD-10 F45 somatoform family, all now subsumed forward into somatic symptom disorder (or, for the health-anxiety type, into illness anxiety disorder) under DSM-5-TR (ICD-10).

GOOD TO REMEMBER

- **Somatisation disorder / Briquet syndrome (the ICD-10 diagnosis):** multiple, recurrent, frequently changing physical symptoms across several systems for at least two years, with refusal to accept reassurance; the discriminator from hypochondriacal disorder is that the focus is the symptoms, not the fear of a specific disease.
- **Somatoform autonomic dysfunction / Da Costa syndrome (the ICD-10 diagnosis):** objective autonomic arousal plus subjective symptoms referred to a single autonomically innervated organ system; the discriminator is the presence of true autonomic signs attached to a named organ.

1.6 The specifiers of somatic symptom disorder

Two descriptive specifiers and a severity grade counted from Criterion B. DSM-5-TR lets the diagnosis be qualified in three ways (DSM-5-TR 2022). The *with predominant pain* specifier (formerly the separate pain disorder) is used when the somatic symptoms predominantly involve pain. The *persistent* specifier marks a course characterised by severe symptoms, marked impairment and long duration, running beyond six months. Severity is then graded directly by how many of the Criterion-B manifestations are fulfilled: *mild* when only one is present, *moderate*

when two or more are present, and *severe* when two or more are present together with multiple somatic complaints or one very severe somatic symptom. This is a neat, examinable structure because severity is read off the positive psychological criterion itself rather than off symptom counts, which keeps the whole diagnosis anchored to Criterion B.

SEVERITY GRADE	CRITERION-B MANIFESTATIONS FULFILLED	ADDITIONAL REQUIREMENT
Mild	Only one	**None**
Moderate	Two or more	**None**
Severe	Two or more	**Plus multiple somatic complaints, or one very severe somatic symptom**

DSM-5-TR severity is graded from the count of Criterion-B manifestations, not from the number of somatic symptoms (DSM-5-TR 2022).

GOOD TO REMEMBER

- **Specifiers (the diagnostic qualifiers):** *with predominant pain* replaces the old pain disorder; *persistent* marks severe, impairing symptoms beyond six months; severity is mild (one Criterion-B feature), moderate (two or more), or severe (two or more plus multiple or one very severe somatic symptom). The rule to hold is that severity counts Criterion B, not symptoms.

1.7 The ICD-11 parallel: bodily distress disorder

ICD-11 made the same positive-criterion move under a different name. ICD-11 discarded the ICD-10 somatoform family and created the grouping "disorders of bodily distress and bodily experience", whose principal member is bodily distress disorder (6C20), sitting beside the newly introduced body integrity dysphoria (6C21) (ICD-11 CDDR; Kaplan and Sadock CTP 2024). The **icd-11 bodily distress** disorder is characterised by bodily symptoms that are distressing to the individual together with *excessive attention* directed toward them, shown as persistent preoccupation with the symptoms' severity or as repeated health-care contacts substantially in excess of what is medically necessary, and this attention persists despite appropriate examination, investigation and reassurance. Symptoms must be present on most days for at least several months, roughly three months or more. Like DSM-5, ICD-11 defines the disorder by these positive psychological features and *not* by the absence of a medical explanation, and it grades severity as mild, moderate or severe by impact on functioning. The two systems therefore converge on mechanism while differing on detail.

Where they still diverge. Two differences are examinable. First, the duration floor: DSM-5 speaks of a symptomatic state typically beyond six months, whereas ICD-11 sets its threshold at several months, giving the example of three months or more. Second, and more important, the health-anxiety patient is placed differently: DSM-5 keeps illness anxiety disorder *within* this chapter, whereas ICD-11 moves hypochondriasis *out*, into the obsessive-compulsive and related disorders, on the logic that its repetitive checking and reassurance-seeking are compulsion-like. In ICD-11 the bodily distress patient is preoccupied with the symptoms themselves and their

impact on life; the hypochondriasis patient is driven instead by the fear that the symptoms signify a serious progressive illness.

FEATURE	ICD-11	DSM-5-TR
Umbrella diagnosis	Bodily distress disorder (6C20)	**Somatic symptom disorder**
Defining basis	Distressing symptoms plus excessive attention	**Distressing symptoms plus excessive thoughts, feelings, behaviours (Criterion B)**
Medically-unexplained rule	Dropped	**Dropped**
Duration floor	Most days for at least several months (about three)	**Persistent state, typically beyond six months**
Health-anxiety type	Hypochondriasis, moved to obsessive-compulsive and related disorders	**Illness anxiety disorder, kept within this chapter**
Severity grading	Mild / moderate / severe by functional impact	**Mild / moderate / severe by Criterion-B count**

The ICD-11 to DSM-5-TR cross-map: convergent on the positive-criterion logic, divergent on duration and on where the health-anxiety patient sits (ICD-11 CDDR, DSM-5-TR 2022).

GOOD TO REMEMBER

- **Bodily distress disorder (the ICD-11 diagnosis):** distressing bodily symptoms with excessive attention (preoccupation with severity, or repeated medically unnecessary health-care contacts), persisting despite appropriate examination and reassurance, present on most days for at least several months (about three); graded mild, moderate or severe by functioning. The two discriminators to hold are that it is positively defined, not unexplained-based, and that ICD-11 hypochondriasis sits with the obsessive-compulsive and related disorders, not here.

1.8 Epidemiology and course

Common, female-predominant, and heavily concentrated in general medical settings. Population-based studies using DSM-5 criteria place the general adult prevalence of somatic symptom disorder at roughly 4 to 6 percent, well above the under-1-percent rate of the old, more restrictive DSM-IV somatisation disorder (DSM-5-TR 2022). The disorder is markedly commoner in general medical than in psychiatric settings: a twelve-month prevalence around 10 to 20 percent is plausible among primary-care attenders, rising to between 40 and 60 percent in services that specialise in psychosomatic or functional disorders. Women report more somatic symptoms than men, so the prevalence is higher in women. Onset is typically early, often in adolescence or before the age of thirty, and the course is characteristically chronic and fluctuating, its trajectory shaped by symptom number, age, degree of impairment and comorbidity; lower harm-avoidance and greater cooperativeness predict a shorter time to remission. Because these patients present to physicians rather than to mental-health services and may meet the suggestion of psychiatric referral with surprise or refusal, the disorder is easily missed and heavily over-investigated.

1.9 Assessment: the somatic-symptom scales

Two scales measure the symptom burden, one measures the positive criterion. Structured instruments grade severity and track change alongside the clinical interview; the reproduced forms sit in the accompanying scale plate, and only the verified bands matter here. The PHQ-15, the Patient Health Questionnaire-15, rates fifteen common somatic symptoms, each scored 0 to 2, summing to a total out of a possible 30, with cut-points at 5, 10 and 15 marking low, medium and high somatic-symptom severity (Kroenke and Spitzer). The SSS-8, the Somatic Symptom Scale-8, is its brief eight-item derivative, each item scored 0 to 4 over the past seven days, summing to a total out of a possible 32, banded as none-to-minimal (0 to 3), low (4 to 7), medium (8 to 11), high (12 to 15) and very high (16 to 32) (Gierk). Both of these read Criterion A, the somatic burden. The SSD-12, the twelve-item Somatic Symptom Disorder-B Criteria Scale, is different in kind: its twelve items, each 0 to 4 for a total out of a possible 48, measure the cognitive, affective and behavioural aspects of Criterion B itself, so it is the instrument that captures the positive psychological criterion rather than the symptoms. The clean pairing to remember is a symptom-burden scale (PHQ-15 or SSS-8) plus a Criterion-B scale (SSD-12).

15	16	4-6%
PHQ-15 HIGH-SEVERITY CUT-OFF (0 TO 30 SCALE)	SSS-8 VERY-HIGH BAND, LOWER BOUND (0 TO 32 SCALE)	SOMATIC SYMPTOM DISORDER, GENERAL-ADULT PREVALENCE

GOOD TO REMEMBER

- **PHQ-15 (the scale):** fifteen somatic symptoms, each 0 to 2, total out of a possible 30; cut-points at 5, 10 and 15 for low, medium and high somatic-symptom severity; it measures Criterion A, the symptom burden.
- **SSD-12 (the scale):** twelve items, each 0 to 4, total out of a possible 48; it measures Criterion B, the excessive thoughts, feelings and behaviours, and so is the direct read-out of the positive psychological criterion.

1.10 The guideline-anchored first moves in management

Physician-centred care first, psychological therapy next, drugs only for a comorbidity. Management is led by non-pharmacological, physician-centred care (IPS_CPG 2020, psychotherapies for somatoform disorders): build a therapeutic alliance, validate that the symptoms and the distress are real, restrict unnecessary investigations and referrals, treat any concurrent depression or anxiety early, and shift the focus from symptoms toward functioning, seeing stable patients about once every one to two months. Cognitive behaviour therapy is the first-line psychotherapy, targeting the catastrophic misinterpretation of bodily sensations and rebuilding activity, with a brief package of psychoeducation, relaxation and reattribution as an alternative first-line approach. The pharmacological rule is restrictive and heavily tested: do *not* prescribe anti-anxiety or antidepressant medicines for medically unexplained somatic complaints unless a specialist advises it (WHO mhGAP 2016); antidepressants are reserved for a genuinely comorbid depressive or anxiety disorder, or for a dominant chronic-pain component, where an SNRI such as duloxetine or a low-dose tricyclic such as amitriptyline is preferred for its analgesic action. Drugs supplement, and never replace, physician-centred and psychological care.

GOOD TO REMEMBER

- **Physician-centred care (the management step):** the guideline first-line (IPS_CPG 2020), being therapeutic alliance, validation of real symptoms, restriction of unnecessary tests and referrals, early treatment of comorbid mood or anxiety, and a shift toward functioning; the first move is the alliance, not a prescription.
- **Antidepressants in somatic symptom disorder (the drug principle):** the guideline rule (WHO mhGAP 2016) is not to prescribe anti-anxiety or antidepressant drugs for medically unexplained somatic symptoms unless a specialist advises; reserve them for comorbid depression or anxiety, or for a dominant chronic-pain component, where an SNRI (duloxetine) or low-dose amitriptyline is preferred.

MNEMONIC

SEED

The master mnemonic for the whole chapter, seeded here and reaped at consolidation. **S** is the *Somatic* room, somatic symptom disorder and bodily distress, where the body complains. **E** is *Eros*, the sexual dysfunctions and gender incongruence, where the body desires and identity is affirmed. **E** is the *Explosive urges*, the impulse-control disorders, where the urge acts. **D** is the *Defence of life*, suicide risk assessment, prevention and the protective, compassionate stance the chapter closes on. Four rooms, one house: S-E-E-D.

INDIA

Somatic presentation is the Indian norm rather than the exception: psychological distress is very commonly expressed and first brought to the clinician as bodily symptoms, so a large share of primary-care and general-hospital attenders sit in this diagnostic space, and the Indian evidence base is anchored by the IPS_CPG 2020 guideline on psychotherapies for somatoform disorders and by indigenous tools such as the Scale for Assessment of Somatic Symptoms (SASS). The most examinable India-specific presentation is the culture-bound Dhat syndrome, a somatic and anxious preoccupation attributed to perceived loss of vital fluid, which must be read through a cultural formulation rather than pathologised on its surface content; its wider transcultural frame and the consultation-liaison service model that most often meets these patients are developed in the Consultation-liaison, geriatric and transcultural psychiatry notes.

HOLD THIS

Somatic symptom disorder is diagnosed by the POSITIVE psychological criterion (Criterion B, excessive thoughts, feelings or behaviours about the somatic symptoms), not by the symptoms being medically unexplained, so a medically explained symptom can still qualify; DSM-5 collapsed the DSM-IV somatoform disorders into it, splitting hypochondriasis into somatic symptom disorder and illness anxiety disorder and renaming conversion as functional neurological symptom disorder, while ICD-11 made the parallel move to bodily distress disorder (6C20, positively defined, present on most days for at least several months) but sent its hypochondriasis to the obsessive-compulsive and related disorders.

2 · Illness anxiety disorder

Hypochondriasis, renamed and re-sorted. **Illness anxiety disorder** is the modern label for the health-anxious patient whose problem is the *fear of illness* rather than the *burden of symptoms*: a preoccupation with having or acquiring a serious illness, held with **minimal or no somatic symptoms** and a high level of **health anxiety**. It is what DSM-5 carved out of the old **hypochondriasis** once the term was retired for its stigma, and the examiner's favourite hook is the split it created: the same clinical tradition now flows into two boxes, illness anxiety disorder where the body is quiet and the mind is frightened, and somatic symptom disorder where distressing bodily symptoms dominate.

This note owns the diagnosis and the discrimination: the reconceptualisation of hypochondriasis, the DSM-5-TR criteria with the **care-seeking type** and **care-avoidant type** specifiers and the six-month duration, the ICD-11 position that kept the name and moved it onto the obsessive-compulsive spectrum, the sharp line against somatic symptom disorder, and the management, where the discipline of limited reassurance, first-line CBT, and treating comorbid anxiety and depression carries the whole load. The somatic symptom disorder that sits on the other side of the split is developed in the somatic symptom disorder topic in these notes; body dysmorphic disorder, its close obsessive-compulsive relative, is in the Anxiety, OCD and trauma-related disorders notes; and the wider consultation-liaison and transcultural frame is in the Consultation-liaison, geriatric and transcultural psychiatry notes.

2.1 The reconceptualisation of hypochondriasis

One old diagnosis, split in two. Because of the pejorative weight the word carried, DSM-5 removed the classical term hypochondriasis and redistributed its patients (Kaplan and Sadock 2024). The majority, roughly three-quarters, carry prominent bodily symptoms and are now diagnosed as somatic symptom disorder. The minority, about one in five (roughly 20%), have relatively minor somatic symptoms and are instead preoccupied with the fear that they will get sick or already harbour an undiagnosed disease; these become illness anxiety disorder. The line between the two boxes is simply the *severity of the somatic symptoms*.

ICD-11 kept the word but changed its home. ICD-11 retained the label hypochondriasis, added *health anxiety disorder* as an official alternative name, and, crucially, placed it not among the somatic disorders but within the obsessive-compulsive and related disorders grouping, on the strength of the shared phenomenology (intrusive health-related preoccupations plus repetitive checking or reassurance behaviours) and a similar treatment response (ICD-11 CDDR). In ICD-11 the presence of somatic symptoms is explicitly *not* an essential feature. The classic triad to name is **disease conviction**, **disease fear**, and **bodily preoccupation**.

RULE **Symptom load decides the box.** Prominent, distressing somatic symptoms mean somatic symptom disorder; a fear-of-illness preoccupation with minimal or no somatic symptoms and high health anxiety means illness anxiety disorder.

2.2 DSM-5-TR criteria and the two specifiers

The criteria to state. DSM-5-TR defines illness anxiety disorder as a preoccupation with having or acquiring a serious illness, in which somatic symptoms are *absent or only mild*; if a real medical condition or genuine risk is present, the preoccupation is clearly excessive or disproportionate (DSM-5-TR 2022). There is a high level of anxiety about health and the person is easily alarmed about their health status. They either perform excessive health-related behaviours (repeatedly checking the body) or show maladaptive avoidance (staying away from appointments and hospitals). The preoccupation has lasted at least six months, although the specific feared illness may change over that time, and it is not better explained by another mental disorder.

Then choose the specifier. DSM-5-TR asks the clinician to specify one of two behavioural phenotypes. In the care-seeking type, medical care (physician visits, tests and procedures) is frequently used. In the care-avoidant type, medical care is rarely used because contact with doctors and health information is itself frightening. The two look opposite at the surface yet spring from the same engine of health anxiety, which is the point examiners test.

DSM-5-TR CRITERION (ILLNESS ANXIETY DISORDER)	WHAT IT REQUIRES
A. Preoccupation	**Preoccupation with having or acquiring a serious illness**
B. Somatic symptoms	**Somatic symptoms absent or only mild; if a condition or risk exists, the worry is disproportionate**
C. Health anxiety	High anxiety about health; easily alarmed about personal health status
D. Behaviour	Excessive health-related behaviours (checking, reassurance-seeking) or maladaptive avoidance
E. Duration	**Illness preoccupation present at least six months (the specific feared illness may change)**
F. Exclusion	Not better explained by another mental disorder

The DSM-5-TR criteria for illness anxiety disorder; the discriminating pair is Criterion B (somatic symptoms absent or mild) and the six-month duration, then specify care-seeking type or care-avoidant type (DSM-5-TR 2022).

GOOD TO REMEMBER

- **Illness anxiety disorder (the syndrome):** preoccupation with having or acquiring a serious illness, with somatic symptoms absent or only mild, a high level of health anxiety, and either excessive health behaviours or maladaptive avoidance, present at least six months; the defining criterion is the fear-of-illness preoccupation on a body that is largely symptom-free, and the mandatory specifier is care-seeking type versus care-avoidant type.

2.3 ICD-11 hypochondriasis and its insight specifiers

The ICD-11 essential features. ICD-11 requires a persistent preoccupation or fear about the possibility of having one or more serious, progressive or life-threatening illnesses, accompanied by *either* repetitive and excessive health-related behaviours (repeated body-checking, exhaustive searching for information, repeated reassurance-seeking and multiple consultations) *or*

maladaptive avoidance related to health (avoiding medical appointments), with resulting significant distress or impairment (ICD-11 CDDR). The catastrophic misinterpretation of ordinary bodily sensations, for example reading a tension headache as a brain tumour, is the additional clinical hallmark.

Insight is coded, not assumed. ICD-11 attaches an insight specifier: *hypochondriasis with fair to good insight* (6B23.0), where the person can much of the time entertain that their belief may be untrue, and *hypochondriasis with poor to absent insight* (6B23.1), where they are convinced most or all of the time and, in a small minority, hold the belief with near-delusional conviction. Because insight can swing with the level of anxiety, it is judged over a period long enough to allow for fluctuation, a few days or a week. Onset is typically in early to mid-adulthood, and the course is chronic and relapsing.

FEATURE	ILLNESS ANXIETY DISORDER (DSM-5-TR)	HYPOCHONDRIASIS / HEALTH ANXIETY DISORDER (ICD-11)
Term retained	Renamed from hypochondriasis	**Term hypochondriasis kept; health anxiety disorder is the alternative name**
Classification home	Somatic symptom and related disorders	**Obsessive-compulsive and related disorders**
Somatic symptoms	Must be absent or only mild (Criterion B)	Not an essential feature; may be present or absent
Behavioural specifier	Care-seeking type / care-avoidant type	Excessive health behaviours or maladaptive avoidance built into the criteria
Insight specifier	Poor-insight / with-delusional-beliefs qualifiers	**Fair-to-good (6B23.0) vs poor-to-absent (6B23.1)**

The same construct across the two systems; the crunchy contrast is where it is filed, somatic-symptom-and-related in DSM-5-TR versus obsessive-compulsive-and-related in ICD-11, and whether somatic symptoms are required at all (DSM-5-TR 2022; ICD-11 CDDR).

GOOD TO REMEMBER

- **Hypochondriasis (ICD-11, health anxiety disorder):** persistent preoccupation or fear of having a serious, progressive or life-threatening illness plus *either* excessive health-related behaviours *or* maladaptive avoidance, causing distress or impairment; filed under obsessive-compulsive and related disorders, somatic symptoms are not required, and insight is coded as fair-to-good (6B23.0) or poor-to-absent (6B23.1).

2.4 Illness anxiety disorder versus somatic symptom disorder

The examiner's discriminator. Both patients are anxious about their health, so the split turns on the *weight of somatic symptoms*. In illness anxiety disorder the body is largely quiet: symptoms are absent or mild and the suffering is the fear itself, the conviction or dread of a disease not yet found. In somatic symptom disorder the patient carries one or more genuinely distressing somatic symptoms with disproportionate thoughts, feelings and behaviours about them; the symptoms drive the picture. DSM-5 also made somatic symptom disorder a *positive* psychological

diagnosis, resting on the excessive health-related thoughts, feelings and behaviours, rather than the old requirement that symptoms be medically unexplained.

DISCRIMINATOR	ILLNESS ANXIETY DISORDER	SOMATIC SYMPTOM DISORDER
Somatic symptoms	**Absent or only mild**	**One or more, prominent and distressing**
Core suffering	Fear of having or acquiring a disease	Burden of the symptoms and their meaning
Basis of the diagnosis	Health preoccupation despite reassurance	Positive psychological criterion (excessive thoughts, feelings, behaviours)
Behavioural axis	Care-seeking type or care-avoidant type	High health-related anxiety and effort directed at symptoms
Share of old hypochondriasis	About one in five (roughly 20%)	About three-quarters

Illness anxiety disorder against somatic symptom disorder; the one line to hold is that illness anxiety disorder is fear on a quiet body while somatic symptom disorder is a loud body (Kaplan and Sadock 2024; DSM-5-TR 2022).

TRAP **Do not diagnose on "medically unexplained".** Somatic symptom disorder now rests on a *positive* psychological criterion, not on symptoms being unexplained; a symptom can have a full medical explanation and the diagnosis still stand, and illness anxiety disorder is missed if you demand unexplained symptoms it does not require.

2.5 Epidemiology, comorbidity and course

Common and easily missed. Health anxiety is frequent: it may affect about **4% to 6%** of all patients, with primary-care rates around 3% to 7% (Kaplan and Sadock 2024), and DSM-5-TR puts the 6-month to one-year prevalence in ambulatory medical populations at roughly 2.2% to 8% (weighted mean about 3%), rising to nearly one-fifth in specialty clinics; it is about equally common in men and women (DSM-5-TR 2022). Onset is usually early to mid-adulthood, and identification is often delayed because these patients present to physicians rather than to mental-health services.

The company it keeps. Psychiatric comorbidity is the rule: depression in about **40%**, panic disorder in 10% to 20%, and generalised anxiety disorder in 5% to 10%, alongside obsessive-compulsive disorder (Kaplan and Sadock 2024). The course tends to be chronic and relapsing, with more than 60% still symptomatic after several years of follow-up. That chronicity, plus the fact that comorbid mood and anxiety often carry the distress, is exactly why the management targets the comorbidity as hard as the health anxiety.

PEARL

Two facts reframe the whole encounter. First, most of these patients sit in medical clinics, not psychiatric ones, so the diagnosis is made by whoever is willing to look past the presenting organ. Second, depression rides along in about four in ten, and it is frequently the treatable driver of the health anxiety, so screening for and treating a comorbid depressive or anxiety disorder is often the single most useful move.

2.6 Management: reassurance limits, CBT, treat the comorbidity

Reassurance is a trap if it is unlimited. The paradox of health anxiety is that reassurance relieves the fear for minutes and then feeds it, so that endless testing and "one more scan" become part of the disorder. The workable stance mirrors physician-centred care for somatic symptom disorder: a single trusted clinician, regular scheduled appointments that are not triggered by each new symptom, a genuine but *bounded* acknowledgement that the symptoms and fear are real, and a deliberate restraint on repeat investigations and referrals once an adequate work-up is done. Reassurance is offered once, clearly, and then the focus shifts from the question "am I ill" to living well despite the uncertainty.

CBT is first-line, and it is what works. Cognitive-behavioural therapy is the prototype, evidence-based first-line treatment, targeting the catastrophic misinterpretation of bodily sensations and the reassurance-seeking and checking that maintain the cycle (Kaplan and Sadock 2024; Barsky and Ahern 2004). India's own guidance is aligned: the IPS Clinical Practice Guidelines recommend, for hypochondriacal or illness-anxiety presentations, CBT with cognitive restructuring and exposure and response prevention as first line, with mindfulness-based cognitive therapy or acceptance and commitment therapy as second line (IPS_CPG 2020).

Medication treats the comorbidity, chiefly. Where a comorbid depressive or anxiety disorder is present, an SSRI is reasonable and the health anxiety often improves in parallel. The best single-drug evidence is for fluoxetine (Prozac), where a double-blind placebo-controlled trial at **20 to 80 mg per day** showed a moderate, well-tolerated benefit (Fallon and colleagues). The guideline caution to keep in view is that drugs are not started reflexively for medically unexplained somatic complaints without a clear comorbid indication or specialist advice (WHO mhGAP 2016); psychological care leads, and medication supplements it.

GOOD TO REMEMBER

- **First-line treatment (guideline):** CBT is the prototype first-line therapy; IPS_CPG 2020 specifies CBT with cognitive restructuring and exposure and response prevention first line for illness-anxiety presentations, with MBCT or ACT second line.
- **Reassurance principle:** unlimited reassurance and repeat testing maintain the disorder; offer a single trusted clinician, regular scheduled visits, a bounded validation of the distress, and restraint on further investigations once an adequate work-up is complete.
- **Pharmacotherapy (guideline):** an SSRI is used mainly for comorbid depression or anxiety; fluoxetine 20 to 80 (mg per day) has the best trial evidence; do not prescribe reflexively for medically unexplained symptoms without a clear indication (WHO mhGAP 2016).

COMMON TRAP

Chasing the fear with tests. Each new investigation buys a few minutes of calm and then raises the next question, deepening the care-seeking cycle and risking iatrogenic harm and doctor-shopping. The corrective is not to withhold care but to move from symptom-driven testing to scheduled, relationship-based follow-up, and to treat the health anxiety and any comorbid depression directly rather than through the radiology department.

6	~20%	20 to 80	3 to 8%
MINIMUM MONTHS OF PREOCCUPATION (DSM-5-TR)	OF OLD HYPOCHONDRIASIS NOW ILLNESS ANXIETY DISORDER	FLUOXETINE DOSE (MG PER DAY)	HEALTH ANXIETY IN AMBULATORY CARE

INDIA

Health anxiety fits the Indian care landscape closely. Most patients present to physicians and specialists rather than to psychiatry, and a fragmented, largely out-of-pocket fee-for-service system makes doctor-shopping and repeated investigation easy, so the **care-seeking type** is readily and iatrogenically reinforced by the very system meant to reassure. National guidance is clear and usable: the IPS Clinical Practice Guidelines anchor CBT with cognitive restructuring and exposure and response prevention as first line for illness-anxiety and hypochondriacal presentations (IPS_CPG 2020). The related culture-bound presentation of Dhat, and the wider transcultural handling of health anxiety, are developed in the Consultation-liaison, geriatric and transcultural psychiatry notes.

WORKED EXAMPLE

A 34-year-old man attends his fourth physician in six months, each time with a new fear, a heart attack, then a brain tumour, then bowel cancer, prompted by a normal sensation such as a skipped beat or a headache. Examinations and an extensive work-up are repeatedly normal, yet reassurance calms him only briefly before the next worry arrives, and he arranges further scans himself. He has no substantial ongoing bodily symptoms; the suffering is the dread. This is illness anxiety disorder, care-seeking type: preoccupation with acquiring a serious illness, somatic symptoms minimal, high **health anxiety**, over six months. Management is a single named clinician with scheduled visits, bounded reassurance, no further tests without a fresh indication, first-line CBT with exposure and response prevention, and screening for the comorbid depression that so often sits underneath.

HOLD THIS

Illness anxiety disorder is DSM-5's carve-out of hypochondriasis for the fear-of-illness patient whose body is quiet, preoccupation with having or acquiring a serious illness, somatic symptoms absent or mild, high health anxiety, at least six months, specify care-seeking type or care-avoidant type; somatic symptom disorder is the loud-body sibling with prominent symptoms and a positive psychological criterion; ICD-11 keeps the name hypochondriasis and files it under obsessive-compulsive and related disorders; and management is bounded reassurance plus first-line CBT with exposure and response prevention (IPS_CPG 2020), with an SSRI such as fluoxetine reserved for comorbid depression or anxiety.

3 · Conversion and functional neurological symptom disorder

The neurology-facing member of the family. **Conversion disorder**, renamed **functional neurological symptom disorder** in DSM-5, is the diagnosis given when a patient has a genuine neurological symptom, a motor, sensory or seizure-like disturbance, that is demonstrably *incompatible* with any recognised neurological disease. It sits on the **somatic spectrum** as the one condition defined not by how much the patient worries about a symptom but by an examination

finding, which is why examiners like it: the whole diagnosis turns on a sign you can elicit at the bedside. Across these notes the disorder is abbreviated fnd.

This note does one job, and does it precisely: it *places* functional neurological symptom disorder in the somatic nosology map, states the two shifts DSM-5 made (dropping the requirement for a preceding psychological stressor, and moving from a diagnosis of exclusion to a **positive signs** rule-in), and names the cardinal trap of the whole topic, missing an organic disease behind a presentation labelled "conversion". The actual bedside **positive signs** (Hoover's sign, the tremor entrainment test, the give-way weakness pattern) and the full management are taught in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes and are cross-referenced there rather than re-taught here. What follows is how to recognise the disorder and where it belongs. The term "conversion" is Charcot-era and psychoanalytic, the idea that unconscious conflict is "converted" into a bodily deficit; the modern label keeps the phenomenon and drops the theory.

3.1 Where it sits on the somatic spectrum, and the DSM versus ICD split

One disorder, two homes. The **somatic spectrum** groups conditions in which bodily symptoms dominate the presentation and are first met in medical rather than psychiatric settings. Within it, conversion / functional neurological symptom disorder is the pole defined by *loss or alteration of a function* (weakness, abnormal movement, sensory loss, non-epileptic attacks) rather than by distressing symptoms plus excessive health thoughts. Its neighbours on the somatic spectrum are these: somatic symptom disorder (positive distressing symptoms with disproportionate cognition), illness anxiety disorder (health anxiety with few or no symptoms), factitious disorder (deliberately produced signs, no external incentive) and, outside this chapter, body dysmorphic disorder, which belongs on the obsessive-compulsive spectrum and is developed in the Anxiety, OCD and trauma-related disorders notes.

The two classifications disagree on the address. This is a favourite discriminator. DSM-5-TR files the disorder inside *Somatic Symptom and Related Disorders* as functional neurological symptom disorder. ICD-11 instead places it among the *dissociative disorders*, as dissociative neurological symptom disorder (code 6B60), sitting beside dissociative amnesia and trance and possession disorders (ICD-11 CDDR). Both describe the same clinical picture, motor, sensory, seizure-like or speech symptoms not consistent with a recognised nervous-system disease; they simply differ on whether the mechanism is best framed as somatic or dissociative.

FRAMEWORK	NAME AND CODE	CHAPTER IT LIVES IN
DSM-5-TR	Functional neurological symptom disorder (conversion disorder)	**Somatic Symptom and Related Disorders**
ICD-11	Dissociative neurological symptom disorder (6B60)	**Dissociative Disorders**
ICD-10 (historical)	Dissociative (conversion) disorders (F44)	Neurotic, stress-related and somatoform disorders

The same disorder, two homes: DSM-5-TR keeps it on the somatic spectrum, ICD-11 files it under the dissociative disorders as 6B60, and the coexisting motor / sensory / seizure specifiers run in parallel across both.

3.2 The DSM-5-TR criteria and the two shifts

Four criteria, A to D. DSM-5-TR requires (A) one or more symptoms of altered voluntary motor or sensory function; (B) clinical findings that provide evidence of *incompatibility* between the symptom and recognised neurological or medical conditions; (C) the symptom is not better explained by another medical or mental disorder; and (D) it causes clinically significant distress or impairment, or warrants medical evaluation (DSM-5-TR 2022). Symptom-type specifiers name the presentation (with weakness or paralysis, abnormal movement, non-epileptic attacks or seizures, anaesthesia or sensory loss, speech or special-sensory symptoms), and course is specified as acute episode or persistent.

The two things DSM-5 changed, and both are examinable. First, it became a *rule-in* diagnosis: Criterion B demands positive evidence of incompatibility, so the disorder is no longer diagnosed by exclusion, no longer requires normal investigations, and can be made even in a patient who also has a recognised neurological disease. Second, DSM-5 *dropped the requirement for a preceding psychological stressor*. A stressor may be specified when present, but it is not necessary, precisely because an identifiable stressor is absent in up to **50%** of patients, and the diagnosis is not withheld for want of one.

RULE **Positive incompatibility, not normal scans.** The diagnosis rests on a positive examination finding of internal inconsistency (a sign present one way and gone another), never on "all the tests came back normal" or on the symptom looking bizarre; it is a rule-in diagnosis that can coexist with genuine neurological disease.

GOOD TO REMEMBER

- **Functional neurological symptom disorder (the syndrome):** altered voluntary motor or sensory function whose *defining* criterion is positive clinical evidence of incompatibility with recognised neurological disease (DSM-5-TR Criterion B); it is a rule-in, not an exclusion, diagnosis, needs no preceding stressor, and may be diagnosed alongside a real neurological disorder.

3.3 The positive signs, named here and taught elsewhere

What "incompatibility" looks like at the bedside. The demonstration is *internal inconsistency*: a deficit that changes when attention is redirected or when the same movement is tested a different way. The three the examiner wants named are Hoover's sign (functional hip-extension weakness that returns to normal power when the opposite hip is flexed against resistance), the tremor entrainment test (a functional tremor that entrains to, is suppressed by, or cannot copy a rhythm tapped by the other hand), and the give-way, or collapsing, weakness pattern (power that gives way suddenly rather than yielding smoothly). These, and the dissociative (non-epileptic) attack features, plus the full management, are worked through in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes; the point for this note is only that such **positive signs** exist and carry the diagnosis.

Two old signs that do not carry it. La belle indifférence (a bland lack of concern about a disabling deficit) and the presence of "secondary gain" were once treated as diagnostic; both are non-specific, occur in organic disease too, and must *not* be used to make the diagnosis (DSM-5-TR 2022). The diagnosis is built on the overall clinical picture and the positive signs, not on a single feature and not on the patient's affect.

GOOD TO REMEMBER

- **Positive signs (the discriminator):** Hoover's sign, the tremor entrainment test and give-way weakness demonstrate internal inconsistency and rule the diagnosis in; la belle indifférence and "secondary gain" are non-specific and do not diagnose it. Full teaching sits in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes.

WORKED EXAMPLE

A 26-year-old woman develops sudden left-leg weakness after a family crisis and cannot lift the leg on the couch. On examination the strength of left hip extension is absent when tested directly but returns to full power the moment she flexes the opposite hip against your hand: a positive **Hoover's sign**. She is oddly untroubled by the disability. The positive sign rules the diagnosis in as functional neurological symptom disorder; the flat affect (la belle indifférence) is a footnote, not the evidence, and you still document a neurological screen because fnd can sit on top of real disease.

3.4 The cardinal trap: missing organic disease

The reassuring modern number, and why it does not license complacency. Eliot Slater's influential but methodologically flawed 1965 study suggested that up to two-thirds of patients labelled "hysteria" in fact had misdiagnosed neurological disease, and it wrongly persuaded a generation that the diagnosis was unsafe. The evidence since is very different: a systematic review pooling 27 outcome studies found a misdiagnosis rate of under **5%** from 1970 onward, in the era before MRI and video-telemetry (New Oxford Textbook of Psychiatry). Functional neurological symptoms are, in fact, the second commonest reason to see a neurologist, so a confident positive diagnosis is both possible and expected.

But the traps are specific and worth memorising. Comorbid neurological disease is common: around **one in five** patients with dissociative (non-epileptic) seizures *also* have epilepsy (DSM-5-TR 2022), so finding a functional attack never excludes a real one. Misreading true disease as fnd is most likely with an isolated gait disorder that examines normally on the couch, with frontal-lobe epilepsy (which can look odd and show a normal surface EEG), and with autoimmune encephalitis or the prodrome of a degenerative disease such as Parkinson's (New Oxford Textbook of Psychiatry). The discipline is to base the diagnosis on positive signs, and to re-examine if the picture becomes progressive.

TRAP **"Tests are normal, so it is conversion."** Missing organic disease behind a "conversion" label is the cardinal error; diagnose on positive incompatibility, remember functional and organic disease coexist (one in five non-epileptic-seizure patients also have epilepsy), and re-examine a progressive course.

4	5-15%	<5%	1 in 5
DSM-5-TR CRITERIA (A TO D)	OF NEUROLOGY-CLINIC PATIENTS HAVE FND	MISDIAGNOSIS RATE ON FOLLOW-UP SINCE 1970	NON-EPILEPTIC-SEIZURE PATIENTS ALSO HAVE EPILEPSY

INDIA

Conversion and dissociative presentations, pseudoseizures (dissociative attacks), functional weakness and possession or trance states, are among the commonest reasons a young or rural patient reaches Indian psychiatric services, often through a physician or neurologist first. ICD-11's placement of the disorder among the dissociative disorders, beside trance and possession disorder, fits this clinical reality well. The wider transcultural framing of possession and trance is developed in the Consultation-liaison, geriatric and transcultural psychiatry notes; the applied lesson here is to demonstrate the positive sign and rule out organic disease before attributing an attack to "hysteria" or a spiritual cause.

HOLD THIS

Conversion / functional neurological symptom disorder is a genuine neurological symptom shown by positive signs (Hoover's sign, tremor entrainment, give-way weakness) to be internally inconsistent and incompatible with recognised disease; DSM-5 made it a rule-in diagnosis and dropped the required stressor, ICD-11 files it as dissociative neurological symptom disorder (6B60) rather than on the somatic spectrum, and the cardinal trap is missing organic disease, so diagnose on the positive sign, not on normal scans, remembering that fnd and real neurological disease coexist.

4 · Factitious disorder and Munchausen syndrome

The one diagnosis defined by a deception. **Factitious disorder** is the intentional falsification or induction of physical or psychological signs and symptoms, driven not by any outside reward but by the internal need to occupy the **sick role**, to be seen and cared for as a patient. That single motive is what makes it examinable: it sits at the tip of a three-way spectrum of illness deception, distinguished from the unconscious somatic disorders on one side (where the symptom is not deliberately produced) and from malingering on the other (where the production is deliberate but the incentive is external). DSM-5 moved it into the somatic symptom and related disorders chapter precisely because these patients present with somatic complaints, most often first to the general physician.

How to use this page. Fix the DSM-5-TR criteria and the internal-versus-external incentive rule first, because that rule carries most of the marks. DSM-5-TR names two forms, factitious disorder imposed on self (historically **Munchausen syndrome**) and factitious disorder imposed on another (Munchausen by proxy); this note abbreviates them factitious imposed on self and factitious imposed on another. Then learn Munchausen syndrome as the severe chronic phenotype, factitious imposed on another as a form of child abuse whose first duty is safeguarding, and Ganser syndrome as a brief borderland name. The chapter closes on the therapeutic, non-punitive stance that management demands.

4.1 The core: intentional falsification, the sick role, no external incentive

Four criteria, A to D, for factitious imposed on self. DSM-5-TR defines factitious disorder imposed on self (F68.10) by four criteria (DSM-5-TR 2022): (A) falsification of physical or psychological signs or symptoms, or induction of injury or disease, associated with identified deception; (B) the individual presents to others as ill, impaired or injured; (C) the deceptive behaviour is evident even in the absence of obvious external rewards; and (D) it is not better explained by another mental disorder such as a delusional or psychotic disorder. The course is specified as a single episode or as recurrent episodes, the latter meaning two or more events of falsifying illness or inducing injury. The patient may feign symptoms (report chest pain that is not felt), simulate signs (manipulate a thermometer), or genuinely induce disease (self-inject a contaminant), and often carries a detailed, superficial medical vocabulary and a history of multiple procedures.

The whole diagnosis turns on Criterion C. The behaviour is conscious and goal-directed, but the goal is *internal*, the psychological gratification of being a patient, not money, drugs, disability or escape from duty. This is what separates it from malingering, and it is why the ICD codes it as a genuine mental disorder (6D50 imposed on self, 6D51 imposed on another) whereas malingering is not a disorder at all. Note the nuance that examiners like: the presence of some external reward does not automatically exclude factitious disorder, because the two can coexist, but the deception must be evident even when no external reward is on offer.

Symptom production

Conscious and deliberate in factitious disorder and malingering; unconscious in somatic symptom and conversion disorders.

Motivation

Internal, to assume the sick role, in factitious disorder; external and tangible in malingering; absent or unconscious in the somatic disorders.

Deception

Identified deception is required for factitious disorder (Criterion A); the somatic disorders involve no deception at all.

GOOD TO REMEMBER

- **Factitious disorder imposed on self (the syndrome):** intentional falsification or induction of signs or symptoms with identified deception (Criterion A), self-presented as ill (B), evident *even in the absence of obvious external rewards* (C, the defining criterion), and not better explained by another mental disorder (D); the internal motive is to occupy the sick role, coded 6D50 in ICD-11.

4.2 Factitious disorder versus malingering, and versus the unconscious somatic disorders

Two axes settle every vignette. Place the presentation on two questions: is the symptom produced *deliberately*, and is the motive *internal or external*. Somatic symptom disorder and conversion (functional neurological symptom disorder) answer "no" to deliberate production, the suffering is authentic and unwilled. Factitious disorder answers "yes, deliberate" and "internal motive" (the sick role). Malingering answers "yes, deliberate" and "external motive" (compensation,

narcotics, avoiding work or legal jeopardy). Only the middle column is defined by a lie told for its own psychological sake.

PRESENTATION	SYMPTOM PRODUCTION	MOTIVATION	STATUS
Somatic symptom / conversion disorder	Unconscious, not deliberate	None conscious; symptom is unwilled	**A mental disorder; genuine distress**
Factitious disorder	Intentional, with deception	Internal: to assume the sick role	**A mental disorder (ICD-11 6D50 / 6D51)**
Malingering	Intentional, with deception	External: money, drugs, escape from duty	**Not a disorder; a focus of clinical attention (ICD-11 QC30)**

The illness-deception spectrum: deliberate production plus internal motive is factitious disorder; deliberate production plus external reward is malingering; neither is present in the unconscious somatic disorders (DSM-5-TR 2022; Kaplan and Sadock CTP 2024).

RULE **Internal motive is factitious; external reward is malingering.** Both produce symptoms deliberately and deceptively; the discriminator is the pay-off, the psychological sick role versus a tangible external gain, and only factitious disorder is a mental disorder.

TRAP **Calling a factitious presentation "malingering."** Naming the deception is not enough; you must locate the motive. If there is no external incentive, the label is factitious disorder, not malingering, and the patient needs psychiatric care rather than discharge.

4.3 Munchausen syndrome: the severe, chronic, wandering form

The severe end of factitious imposed on self. Munchausen syndrome is the term Asher applied in his 1951 Lancet paper to the most chronic and dramatic patients, the "hospital addicts," "hospital hoboes" and "professional patients" who repeatedly simulate or induce disease for the sole purpose of obtaining medical attention. Two features beyond simple simulation define it: pseudologia fantastica, fluent, plausible, self-aggrandising lying in which the tale often contains a kernel of truth, and peregrination, the tendency to travel widely from hospital to hospital, discharging against advice and beginning the performance anew elsewhere. Antisocial traits are common, evidence of earlier surgery accumulates as multiple scars (the classic "gridiron abdomen"), and the mortality of severe induced disease is real.

But the commoner phenotype does not wander. A second, more prevalent form, sometimes called common or non-peregrinating factitious disorder, confines the behaviour to one locality and a narrow set of complaints. Its prototypical patient is younger, more often female, and frequently has a health-care background. The teaching point is that Munchausen syndrome is the memorable extreme, not the whole of factitious disorder, and the eponym is often wrongly stretched to cover every case.

4 DSM-5-TR CRITERIA (A TO D), IMPOSED ON SELF	**2+** FALSIFICATION EVENTS DEFINE RECURRENT EPISODES	**1951** ASHER NAMES MUNCHAUSEN SYNDROME (LANCET)	**1977** MEADOW DESCRIBES IT BY PROXY (CHILD ABUSE)

WORKED EXAMPLE

A woman in her thirties presents to a fourth hospital in a year with dramatic haematemesis and a spellbinding account of nursing in a war zone; the notes are thin, the story shifts, and a laparotomy scar suggests earlier surgery elsewhere. No compensation claim, no controlled-drug request and no legal case are in play. Recurrent presentations, **pseudologia fantastica** and **peregrination**, with deception and no external reward, point to Munchausen syndrome (severe factitious imposed on self), not malingering, and she needs a psychiatric alliance rather than another endoscopy.

PEARL

The eponym is a double misnomer worth knowing. It honours Baron von Munchausen, the fictional teller of tall tales, so it captures the *storytelling* but not the *self-harm*; and because the label was so widely misapplied to milder cases, DSM-5-TR retired it in favour of the plain descriptor factitious disorder imposed on self. Reserve "Munchausen" for the chronic, peregrinating, pseudologia-rich extreme.

GOOD TO REMEMBER

- **Munchausen syndrome (the syndrome):** the severe chronic form of factitious imposed on self, its two discriminators being pseudologia fantastica (fluent self-aggrandising lies with a kernel of truth) and peregrination (wandering hospital to hospital); Asher, 1951; the commoner non-peregrinating phenotype is younger, more often female and health-care linked.

4.4 Factitious disorder imposed on another (Munchausen by proxy): abuse, not curiosity

The illness is fabricated in someone else. Factitious disorder imposed on another (F68.A; ICD-11 6D51), the entity Meadow named Munchausen by proxy in 1977, applies its four criteria to a perpetrator who falsifies or induces illness in a victim, most often a preverbal child, sometimes a dependent elder or disabled adult, and presents that victim to others as ill (DSM-5-TR 2022). The reward the perpetrator seeks is still internal, the vicarious sick role and the attention of clinicians, with no obvious external incentive. The single most tested fact is the direction of the diagnosis: **the perpetrator, not the victim, receives the diagnosis**, and the perpetrator is most commonly the mother. It is formally recognised as a form of abuse, and, like any physical abuse, the diagnosis does not depend on proving the caregiver's motive.

Safeguarding is the first and overriding step. The moment factitious imposed on another is suspected, protection of the victim outranks every diagnostic nicety: the induced illness itself can be lethal, and the child cannot self-report. Contemporaneous, factual documentation, separation of the fabricated history from verified findings, senior and multidisciplinary involvement and referral into the statutory child-protection pathway are the priorities. Confronting the caregiver alone, without safeguards in place, can escalate risk to the victim and is not the first move.

INDIA

Two India-specific realities matter. First, a fragmented, largely out-of-pocket, multi-provider health system with no unified medical record makes peregrination and repeated fresh presentations easy and hard to detect, so a suspicious index and a request for old records across facilities do real work. Second, when factitious imposed on another is suspected, safeguarding runs through India's child-protection framework: the child is a "child in need of care and protection" under the Juvenile Justice (Care and Protection of Children) Act 2015, with reporting to the Child Welfare Committee, alongside the consultation-liaison team that most often first meets these patients. The wider consultation-liaison service model is developed in the Consultation-liaison, geriatric and transcultural psychiatry notes.

GOOD TO REMEMBER

- **Factitious imposed on another / Munchausen by proxy (the syndrome):** falsification or induction of illness in another (usually a preverbal child), with deception and no obvious external reward; the perpetrator (commonly the mother), not the victim, receives the diagnosis; it is a recognised form of abuse, so safeguarding the victim is the first and overriding step (DSM-5-TR; Meadow, 1977).

4.5 Ganser syndrome: the factitious and dissociative borderland

The syndrome of approximate answers. Ganser syndrome, described by Ganser in the 1890s and historically labelled "prison psychosis," is defined by vorbeireden, the giving of approximate answers, responses that are wrong but wrong in a near-miss way that betrays that the question was fully understood (asked how many legs a horse has, the patient answers "three"). It classically runs with a clouded or twilight state, may carry conversion-like somatic features and pseudohallucinations, and is often followed by amnesia for the episode. It has never had a settled home, sitting at the borderland of dissociation, factitious behaviour and malingering; ICD-11 files it among the dissociative disorders, and it earns a place here because the approximate answer looks feigned yet, unlike malingering, seeks no external reward. It is grouped under the dissociative disorders as hysterical pseudodementia.

GOOD TO REMEMBER

- **Ganser syndrome (the syndrome):** the syndrome of approximate answers, vorbeireden, where replies are wrong but near-miss in a way that proves the question was understood, classically with a clouded/twilight state and historically seen in prison inmates ("prison psychosis"); it sits at the dissociative/factitious/malingering borderland and is classed by ICD-11 among the dissociative disorders.

4.6 Management: a therapeutic, non-punitive stance

No specific drug; treat the person, not the deception. There is no standard pharmacological treatment for factitious disorder itself; medication is directed only at a comorbid psychiatric disorder, such as a depressive, anxiety or personality disorder, or at the emotional distress that precipitated the illness behaviour (Kaplan and Sadock CTP 2024). The core of management is a stance: build a therapeutic alliance, adopt a supportive, face-saving and explicitly non-confrontational approach that addresses the patient's genuine emotional needs rather than the

fabricated ones, and steer toward psychiatric care without aggressive direct exposure. Because these patients split teams and generate intense negative feeling, care is coordinated through a multidisciplinary team with regular meetings to contain countertransference and reduce staff conflict, and iatrogenic harm is minimised by restricting unnecessary investigations and procedures.

Confrontation cautions. Blunt accusatory confrontation typically ruptures the alliance and drives the patient to abscond and re-present elsewhere, so it is avoided; when the deception is addressed it is done gently and truthfully. There is no role for the clinician's own deception either, no disguised placebos and no misrepresented tests; the double-bind manoeuvre (framing a treatment such that improvement is expected if the complaint is genuine, giving a face-saving exit) is a described technique to be used sparingly and honestly, never as a trap. Where the presentation is factitious imposed on another, this therapeutic stance is subordinate to safeguarding: protecting the victim comes first, always.

GOOD TO REMEMBER

- **Management of factitious disorder (the principle):** no specific drug treatment, treat only the comorbidity; the first line is a supportive, face-saving, non-confrontational therapeutic alliance, not exposure; coordinate through a multidisciplinary team, manage countertransference and splitting, minimise iatrogenic investigation, and for factitious imposed on another let safeguarding override everything (Kaplan and Sadock CTP 2024).

HOLD THIS

Factitious disorder is the deliberate, deceptive falsification or induction of signs or symptoms with an *internal* motive (the sick role) and *no* external incentive, which is exactly what separates it from malingering (external reward) and from the unconscious somatic disorders (no deliberate production); Munchausen syndrome is its severe wandering form (pseudologia fantastica plus peregrination), factitious imposed on another / Munchausen by proxy is a form of abuse in which the perpetrator, not the victim, is diagnosed and safeguarding comes first, and management is a non-confrontational therapeutic stance with no specific drug, treating only the comorbidity.

5 · Malingering and its differentiation from factitious disorder

This is the discrimination step of the somatic chapter, the point where every disorder met so far is separated by asking two questions rather than one: is the symptom *produced*, and if so, *why*. The genuine **somatic disorders** answer the first question with no, the symptom is real and not deliberately made; malingering and factitious disorder both answer yes, the symptom is feigned, and then part company on the second question, the motive. Examiners love this space precisely because it is a two-axis grid and a vignette can be walked through it cleanly, so the whole topic reduces to holding one figure in the head.

How to use this page. Fix malingering first, its definition, its status as a condition rather than a disorder, and the clinical pointers that raise suspicion. Then recall factitious disorder in a single frame so the contrast is sharp. Then learn the two discriminators the paper actually tests: the **voluntariness**-by-incentive grid that separates the two feigned conditions from each other and

from the genuine disorders, and the fuller five-way map that places somatic symptom disorder, illness anxiety disorder, conversion and these two side by side. The single error the whole topic is built to prevent is confusing factitious disorder, where no external incentive is present, with malingering, where one is.

Disorder	Core feature	Symptom production	External incentive	The discriminator
Somatic symptom d.	distressing somatic symptom(s) plus excessive thoughts, feelings or behaviours (Criterion B)	genuine, not feigned	**No**	the positive psychological criterion; the symptom may be medically explained
Illness anxiety d.	preoccupation with having a serious illness; minimal actual somatic symptoms	genuine, not feigned	**No**	the fear is of the illness, not the somatic burden
Conversion / FND	a neurological symptom incompatible with disease; positive signs on examination	not consciously feigned	**No**	positive functional signs (Hoover, entrainment); taught in the Dissociative notes
Factitious disorder	signs or symptoms deliberately produced or falsified	consciously feigned	**No** (internal sick role)	feigned but NO external gain; the motive is the sick role
Malingering	symptoms feigned or exaggerated	consciously feigned	**Yes** (external gain)	feigned WITH an external incentive; not a disorder

Read down the external-incentive column: it is the single line that separates factitious disorder (feigned, no external gain) from malingering (feigned, external gain).

Fig 18.3 The somatic and related disorders discriminated. Somatic symptom disorder and illness anxiety disorder are genuine, not feigned, and carry no external incentive; they are told apart by whether the problem is the symptom burden with a positive psychological criterion or a preoccupation with having an illness. Conversion or functional neurological symptom disorder is also not consciously feigned and is recognised by its positive signs on examination, taught in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes. The two feigned conditions are separated by one thing only: factitious disorder is feigned for the internal reward of the sick role with no external gain, whereas malingering is feigned for an external incentive and is not a mental disorder at all.

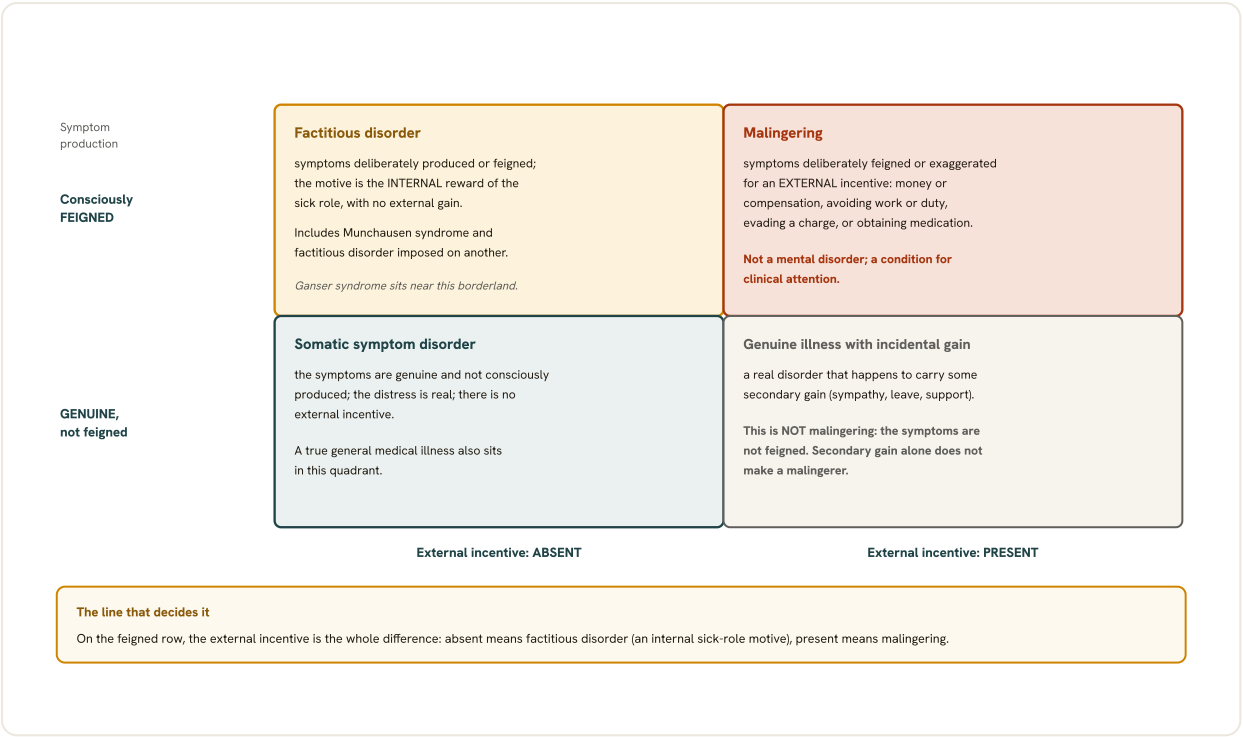

Fig 18.4 Feigning by incentive. Two questions place any of these presentations. First, are the symptoms consciously feigned or genuine? Somatic symptom disorder and a true medical illness are genuine and sit on the lower row; factitious disorder and malingering are feigned and sit on the upper row. Second, is there an external incentive? On the feigned row this is the entire distinction: factitious disorder is feigned for the internal reward of occupying the sick role, with no external gain, while malingering is feigned for an external incentive such as money, avoiding duty or evading a charge, and is not a mental disorder. The lower-right quadrant is the common trap: a genuine illness that carries some incidental secondary gain is not malingering, because nothing is being feigned.

5.1 Malingering: feigning driven by an external incentive

Not a disorder, a condition, defined by the motive behind the feigning. The essential feature of malingering is the intentional production of false or grossly exaggerated physical or psychological symptoms, motivated by an **external incentive** such as avoiding military duty, avoiding work, obtaining financial compensation, evading criminal prosecution, or obtaining drugs (DSM-5-TR 2022; Kaplan and Sadock CTP 2024). Two features anchor it. First, the symptoms are under voluntary control and the **feigning** is conscious and deliberate; the person is aware of the presentation as a strategy. Second, and decisively, the motive is external: some tangible reward is sought, or some obligation avoided. Because of that motive it is coded in DSM-5-TR as Z76.5, a condition that may be a focus of clinical attention and expressly *not* a mental disorder. It may even be adaptive in the right context, the classic example being a captive who feigns illness while held by an enemy in wartime, which is why it is described neutrally as illness behaviour rather than psychopathology.

GOOD TO REMEMBER

- **Malingering (the condition):** intentional production of false or grossly exaggerated physical or psychological symptoms motivated by an **external incentive** (avoiding work or service, financial compensation, evading prosecution, obtaining drugs); coded Z76.5 as a condition for clinical attention, *not* a mental disorder. The single defining criterion is the external incentive; remove it and the label cannot stand.

5.2 The pointers that raise suspicion

Suspect, do not diagnose, on a combination of pointers. DSM-5-TR states that malingering should be *strongly suspected* when any combination of four features is present (DSM-5-TR 2022). These are a medicolegal context (the person is referred by an attorney, or self-refers while litigation or criminal charges are pending); a marked discrepancy between the claimed distress or disability and the objective findings; a lack of cooperation with the evaluation and with the prescribed treatment; and the presence of antisocial personality disorder. The word to hold is *suspected*: no single pointer confirms anything, and the diagnosis requires firm, documented evidence gathered across time, examiners and settings, with collateral information weighed carefully. The clinician's role here is medical, to assess the presentation and document inconsistency in a non-pejorative way, not to pronounce on honesty or fraud.

DSM-5-TR POINTER	WHAT IT LOOKS LIKE
Medicolegal context	**Attorney referral, or self-referral while litigation or criminal charges are pending**
Discrepancy	**Claimed stress or disability far exceeds the objective findings and observed behaviour**
Non-cooperation	**Poor engagement with the evaluation and with the prescribed treatment regimen**
Antisocial personality disorder	**Present as an associated feature (a pointer, not proof, and not a prerequisite)**

The four DSM-5-TR features that should raise suspicion of malingering; any combination warrants suspicion, none alone confirms it (DSM-5-TR 2022).

PEARL

Certainty in malingering is *graded, not binary*. The literature codes it along a ladder, indeterminate, then strongly suspected, then provisional, then probable, then deemed likely present, according to how far the pointers, associated features and collateral corroboration line up (Kaplan and Sadock CTP 2024). Genuine illness, by contrast, is consistent across examiners, contexts and even when the person is distracted; feigned illness tends to be inconsistent and shifting.

COMMON TRAP

Treating antisocial personality disorder as proof of malingering. It is only one associated pointer. Studies do *not* show that people with antisocial personality disorder malinger more often, or more successfully, than the general population, and mild symptom exaggeration or ordinary impression management is not malingering. A firm attribution needs converging, documented evidence, not a personality label.

SUBTYPES AND DETECTION INSTRUMENTS

Resnick's classical trichotomy (Resnick) names three forms: *pure malingering*, complete fabrication of a non-existent disorder; *partial malingering*, exaggeration of genuinely present symptoms; and *false imputation*, consciously attributing real symptoms to a false, unrelated cause. Structured aids to detection, used mainly in forensic assessment, include the SIRS (Structured Interview of Reported Symptoms), the M-FAST (Miller Forensic Assessment of Symptoms Test), the SIMS (Structured Inventory of Malingered Symptomatology) and the TOMM (Test of Memory Malingering); they support, but never replace, the clinical judgement built on collateral data.

5.3 Factitious disorder in one frame: feigned, but no external incentive

Deliberate deception with an internal motive, to be a patient. Factitious disorder imposed on self (F68.10) is defined by the falsification of physical or psychological signs or symptoms, or the induction of injury or disease, associated with identified deception; the person presents themselves to others as ill, impaired or injured; and, the decisive clause, the deceptive behaviour is evident *even in the absence of obvious external rewards*, and is not better explained by another mental disorder such as a psychotic illness (DSM-5-TR 2022). The symptoms are produced intentionally and consciously, exactly as in malingering, but the aim is internal: to occupy the sick role and receive care, attention and medical procedures. This is the classical **primary gain** of the sick role, not the tangible **secondary gain** of the malingerer. The diagnosis rests on objectively identifying the falsification, not on proving the inner motive. Its severe, chronic, wandering form is Munchausen syndrome, named for the Baron of tall tales, estimated at roughly five to ten percent of factitious cases; when the illness is fabricated or induced in another person, usually a dependent, it is factitious disorder imposed on another, and the perpetrator, not the victim, receives the diagnosis.

4	5	5-10%
DSM-5-TR MALINGERING POINTERS	CODED DEGREES OF CERTAINTY	MUNCHAUSEN SHARE OF FACTITIOUS CASES

GOOD TO REMEMBER

- **Factitious disorder imposed on self (the diagnosis, F68.10):** falsification of signs or symptoms, or induction of injury or disease, with identified deception; presents self as ill; the deception is present *even without obvious external rewards*; not better explained by another disorder. The one discriminator to hold is Criterion C, the absence of an external reward, which is exactly the axis that separates it from malingering.

5.4 Voluntariness by incentive: the factitious versus malingering grid

Two feigned conditions, told apart on one axis. Both malingering and factitious disorder are, by definition, deliberately produced, so **voluntariness** of symptom production is *shared* and cannot separate them; what separates them is the incentive. This is the whole of the **factitious versus malingering** discrimination: an external incentive present points to malingering (secondary gain, a reward or an avoided obligation), an external incentive absent points to factitious disorder (primary gain, the sick role itself). The genuine somatic disorders sit off this axis entirely, because their symptoms are not intentionally produced at all. The accompanying figure lays this out as a

two-by-two of intentional-or-not production against external-or-internal incentive; the same content is tabulated below (New Oxford Textbook of Psychiatry; DSM-5-TR 2022).

CONDITION	SYMPTOM INTENTIONALLY PRODUCED?	INCENTIVE PRESENT
Genuine somatic disorder (somatic symptom disorder, conversion)	No, involuntary and unintentional	**Neither; symptoms are not made to a purpose (secondary gain may coexist but is not the cause)**
Factitious disorder	Yes, intentional and conscious	**Internal only, the sick role (primary gain); external reward absent**
Malingering	Yes, intentional and conscious	**External incentive present, a tangible reward or avoided obligation (secondary gain)**

The voluntariness-by-incentive grid; production separates the genuine disorders from the two feigned conditions, and the external incentive separates malingering from factitious disorder (New Oxford Textbook of Psychiatry).

RULE **Same production, different incentive.** Malingering and factitious disorder are both consciously feigned; the discriminator is the external incentive, present in malingering (secondary gain), absent in factitious disorder (the internal sick role, primary gain).

5.5 The fuller somatic-disorders discriminator

Five neighbours placed on production and motivation. The examiner's synthesis extends the grid across the whole chapter, so that **somatic versus factitious** versus malingering can be read off two columns at a glance. Somatic symptom disorder carries genuine, not feigned, distressing symptoms with an excessive psychological response and no external incentive; illness anxiety disorder is genuine health fear on a largely quiet body; conversion, or functional neurological symptom disorder, is a genuine deficit whose *internal inconsistency* is demonstrated by positive signs rather than being deliberately produced. Factitious disorder is feigned with no external incentive, and malingering is feigned with an external incentive. The full bedside recognition of conversion, its positive signs and its management, is developed in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes, and body dysmorphic disorder, which belongs on the obsessive-compulsive spectrum, in the Anxiety, OCD and trauma-related disorders notes. The figure alongside renders this as the five-row discriminator map.

CONDITION	SYMPTOMS GENUINE OR FEIGNED	EXTERNAL INCENTIVE	THE ONE-LINE DISCRIMINATOR
Somatic symptom disorder	Genuine, not feigned	Absent	**Real distressing symptom plus excessive thoughts, feelings and behaviours (positive psychological criterion)**
Illness anxiety disorder	Genuine, not feigned	Absent	**Fear of having or acquiring illness on a quiet body; symptoms absent or mild**
Conversion / functional neurological symptom disorder	Genuine, not intentionally produced	Absent	**Positive signs of internal inconsistency (for example Hoover's sign) rule it in**
Factitious disorder	Feigned, intentionally produced	Absent	**Deception to occupy the sick role; primary gain, no external reward**
Malingering	Feigned, intentionally produced	Present	**External incentive drives the feigning; secondary gain**

The five-way somatic-disorders discriminator, keyed on symptom production and on motivation; the two feigned conditions split only on the external incentive (Kaplan and Sadock CTP 2024; New Oxford Textbook of Psychiatry; DSM-5-TR 2022).

GOOD TO REMEMBER

- **The discriminator (the synthesis):** ask production first, then motivation. Genuine somatic disorders are not intentionally produced. Of the two feigned conditions, factitious disorder has no external incentive (sick role, primary gain) and malingering has an external incentive (secondary gain). That single external-incentive axis carries the **differentiation**.

TRAP **Confusing factitious disorder with malingering.** Both are consciously feigned, so they feel alike; the difference is the external incentive. Factitious disorder has none (the motive is the sick role); malingering has one. Reading a compensation or medicolegal claimant as factitious, or a sick-role patient as a malingerer, inverts the whole grid.

INDIA

Malingering surfaces most in Indian medicolegal work, where it is examined rather than treated: disability and compensation certification, fitness assessments, and above all the malingered insanity defence, where a defendant may feign mental illness to influence criminal responsibility (Jiloha, Forensic Psychiatry, An Indian Perspective). Assessment leans on structured aids such as the SIRS, M-FAST, SIMS and TOMM alongside collateral history, and Indian professional guidance recognises malingering as one of the grounds on which a doctor may decline to continue a therapeutic relationship. The register throughout is medical and non-accusatory: the task is to document inconsistency and weigh evidence, not to brand a person dishonest, which also guards against the opposite error of dismissing a genuinely ill or culturally distressed patient, such as one presenting somatically, as a fraud.

HOLD THIS

Malingering and factitious disorder are both consciously feigned, so voluntariness of production does not separate them; the discriminator is the external incentive, present in malingering (secondary gain, coded Z76.5 as a condition and not a mental disorder, strongly suspected on the four DSM-5-TR pointers) and absent in factitious disorder (Criterion C, the deception persists without obvious external reward, the motive being the sick role and primary gain), while the genuine somatic disorders, somatic symptom disorder, illness anxiety disorder and conversion, are not intentionally produced at all, so the whole map reduces to two questions asked in order, is the symptom produced, and if so, for an external incentive or for the sick role.

6 · Body dysmorphic disorder and bodily distress

Two disorders, one abbreviation, opposite chapters. This short topic sits at the somatic/BDD boundary and its whole job is to keep two look-alikes apart. **Body dysmorphic disorder** is a preoccupation with a perceived defect in appearance and lives on the **obsessive-compulsive and related disorders** spectrum; **bodily distress disorder** is the ICD-11 label for the symptom-led somatic presentation that replaced somatoform disorder. Both get shortened to "BDD" in the wards and both were once filed as somatoform, which is exactly why examiners like the trap. Name each, place each, and know the one mapping the examiner wants: how ICD-11 bodily distress disorder lines up against DSM-5 somatic symptom disorder.

This is a naming and cross-reference note, not the full teaching. The complete work-up of body dysmorphic disorder, its phenomenology, comorbidity, suicidality and management, belongs with its obsessive-compulsive kin and is developed in the Anxiety, OCD and trauma-related disorders notes. The two somatic siblings that bracket it, somatic symptom disorder and illness anxiety disorder, are their own topics in these notes, and the wider transcultural frame, including Dhat as a culturally patterned bodily-distress presentation, is in the Consultation-liaison, geriatric and transcultural psychiatry notes.

6.1 Body dysmorphic disorder on the OCRD spectrum

A perceived flaw, not a real one. Body dysmorphic disorder is a preoccupation with one or more perceived defects or flaws in physical appearance that are *not observable or appear only slight to others* (DSM-5-TR 2022, Criterion A). The person performs repetitive behaviours (mirror

checking, excessive grooming, skin picking, reassurance seeking) or mental acts (comparing their appearance with others) in response to the concern (Criterion B), which causes clinically significant distress or impairment (Criterion C) and is not better explained by an eating disorder's concern with fat or weight (Criterion D). There is no duration threshold. Two riders matter: the muscle dysmorphia specifier (preoccupation that the build is too small or insufficiently muscular, seen mostly in men), and an insight specifier graded good-or-fair, poor, or absent insight with delusional beliefs, the last absorbing what older systems called delusional dysmorphophobia.

It migrated out of the somatic chapter. Historically dysmorphophobia was treated as a variant of hypochondriasis and filed with the somatoform disorders (Companion to Psychiatric Studies), which is the root of the confusion. DSM-5 and ICD-11 both moved it firmly onto the obsessive-compulsive and related disorders grouping (ICD-11 code 6B21), alongside hypochondriasis, olfactory reference disorder and hoarding, on the strength of the shared engine of intrusive preoccupation plus repetitive checking and reassurance. That placement on the OCRD spectrum is the single fact this note exists to fix. The full teaching, the bdd cross-reference for phenomenology, comorbidity and treatment, is carried in the Anxiety, OCD and trauma-related disorders notes.

RULE **Appearance, not illness, and OCRD, not somatic.** A preoccupation with a perceived *appearance* defect that others cannot see, driven by checking and comparing, is body dysmorphic disorder and it belongs on the obsessive-compulsive and related disorders spectrum, not among the somatic disorders.

GOOD TO REMEMBER

- **Body dysmorphic disorder (the syndrome):** preoccupation with one or more perceived defects or flaws in appearance that are unobservable or slight to others, plus repetitive behaviours (mirror checking, grooming, reassurance seeking) or mental acts (comparing with others), causing distress or impairment and not explained by an eating disorder; the defining criterion is the unobservable-defect preoccupation, the classic specifier is muscle dysmorphia, and its home is the OCRD spectrum (6B21), not the somatic chapter (DSM-5-TR 2022; ICD-11 CDDR).

6.2 Bodily distress disorder and its map to somatic symptom disorder

The ICD-11 replacement for somatoform. Bodily distress disorder (ICD-11 code 6C20) is the major ICD-11 change that swept away ICD-10's somatoform disorders. It is characterised by the presence of bodily symptoms that are distressing to the person and by an *excessive* level of attention directed toward those symptoms, which typically leads to repeated contact with health services (ICD-11 CDDR; Kaplan and Sadock 2024). Like DSM-5's construct, it is defined by *positive* psychological features, the distress and the excessive thoughts and behaviours, rather than by the old requirement that symptoms be medically unexplained. It is graded on a continuum as mild, moderate or severe by the impact on functioning, and it sits in the ICD-11 grouping "disorders of bodily distress and bodily experience" alongside the rare body integrity dysphoria.

The mapping the examiner wants. ICD-11 bodily distress disorder maps onto DSM-5 somatic symptom disorder: both are symptom-led, both rest on the positive psychological criterion, and

both replaced the medically-unexplained model. The two are easily held side by side. They are close cousins rather than identical twins, ICD-11 foregrounds the excessive attention and repeated healthcare contact, DSM-5 foregrounds disproportionate thoughts, feelings and behaviours, but for a one-line answer they are the same idea in two manuals.

AXIS	ICD-11 BODILY DISTRESS DISORDER (6C20)	DSM-5 SOMATIC SYMPTOM DISORDER
Replaces	**ICD-10 somatoform disorders**	**DSM-IV somatoform disorders**
Core feature	Distressing bodily symptoms plus excessive attention to them	Distressing somatic symptoms plus disproportionate thoughts, feelings, behaviours
Basis of diagnosis	**Positive psychological features, not absence of medical explanation**	**Positive psychological criterion, not medically unexplained**
Severity grading	Mild, moderate, severe by functional impact	Mild, moderate, severe; specify persistent, predominant pain
Grouping	Disorders of bodily distress and bodily experience	Somatic symptom and related disorders

ICD-11 bodily distress disorder against DSM-5 somatic symptom disorder; the one line to hold is that both are the symptom-led, positive-criterion successors to somatoform disorder (ICD-11 CDDR; DSM-5-TR 2022; Kaplan and Sadock 2024).

TRAP **"BDD" is not one thing.** Body dysmorphic disorder is an appearance-preoccupation on the OCRD spectrum; bodily distress disorder is the ICD-11 somatic-symptom successor to somatoform disorder. Same abbreviation, different chapters, do not let the letters merge them.

GOOD TO REMEMBER

- **Bodily distress disorder (the syndrome):** ICD-11 6C20, distressing bodily symptoms with an excessive level of attention directed to them and repeated healthcare contact, defined by positive psychological features rather than absence of a medical explanation and graded mild to severe; it is the ICD-11 replacement for somatoform disorder and maps onto DSM-5 somatic symptom disorder (ICD-11 CDDR; Kaplan and Sadock 2024).

6.3 Where each is taught, and the one management anchor

Route the reader, then give the anchor. Keep the full detail where it belongs: body dysmorphic disorder with its obsessive-compulsive relatives in the Anxiety, OCD and trauma-related disorders notes, and the symptom-led somatic disorders in the somatic symptom disorder and illness anxiety disorder topics here. For the one management fact worth carrying: body dysmorphic disorder is treated first-line with CBT that centres on exposure and response prevention, and where symptoms are moderate to severe an SSRI is added, used at the higher doses and the longer trial familiar from obsessive-compulsive disorder rather than the lower antidepressant doses of depression (NICE CG31; Maudsley Prescribing Guidelines). Bodily distress disorder is managed as its somatic siblings are, physician-centred care and psychological therapy first, with drugs reserved for a genuine comorbid depression or anxiety, developed in the somatic symptom disorder topic.

GOOD TO REMEMBER

- **Management anchor (guideline):** body dysmorphic disorder is first-line CBT with exposure and response prevention, adding an SSRI for moderate-to-severe illness at OCD-range higher doses and a longer trial (NICE CG31, obsessive-compulsive disorder and body dysmorphic disorder); bodily distress disorder follows the somatic pathway, physician-centred care and psychological therapy first, medication only for a comorbid mood or anxiety disorder.

INDIA

Bodily distress disorder fits the Indian clinic closely: symptom-led somatic presentations dominate general practice, and the ICD-11 positive-criterion frame is a better match than the old "medically unexplained" label for patients who present the body first. The culturally patterned bodily-distress presentation of Dhat, and its transcultural handling, are developed in the Consultation-liaison, geriatric and transcultural psychiatry notes. Body dysmorphic disorder is easy to miss here because it hides behind repeated requests for cosmetic and dermatological procedures rather than a psychiatric complaint, so the referral often arrives from the skin or surgery clinic, not from the patient naming a fear.

2.4%	13 to 15%	16 to 17
BODY DYSMORPHIC DISORDER POINT PREVALENCE, ADULTS (DSM-5-TR)	AMONG GENERAL COSMETIC SURGERY PATIENTS (DSM-5-TR)	MEAN AGE AT ONSET, BODY DYSMORPHIC DISORDER (YEARS)

HOLD THIS

Body dysmorphic disorder is an appearance-defect preoccupation on the OCRD spectrum (ICD-11 6B21, DSM-5 obsessive-compulsive and related disorders), unobservable or slight defect plus checking and comparing, muscle dysmorphia and insight specifiers, full teaching in the Anxiety, OCD and trauma-related disorders notes; bodily distress disorder is the different beast, ICD-11 6C20, the positive-criterion successor to somatoform disorder that maps onto DSM-5 somatic symptom disorder; same three letters, opposite chapters.

7 · Management of medically unexplained symptoms

This topic takes the somatic patient from diagnosis to a plan. **Medically unexplained symptoms** (MUS) are bodily complaints that persist without a physical disease that fully accounts for them; over decades the label has drifted from "somatization" through "medically unexplained symptoms" to "functional somatic symptoms" (Kaplan and Sadock CTP 2024). The examinable point is that the **management of medically unexplained symptoms** is *not* a hunt for the missing disease and *not* a prescription. It is a physician-led, relationship-based, psychological-first package in which drugs are held back deliberately. Get the order of moves right and you have the marks: physician-centred care first, structured psychological work next, and medication only for a genuine comorbidity.

How to use this page. Fix the guideline spine first, that being the Indian Psychiatric Society somatoform CPG (IPS_CPG 2020) and the WHO mhGAP OTH module (WHO mhGAP 2016), because both put non-pharmacological, physician-centred strategies as the first line and both restrict medication for the somatic symptoms themselves. Then learn the **reattribution** model as

the signature technique, the psychological evidence, the one restrictive drug rule, and the follow-up posture. Close with the functional somatic syndromes, which are managed on exactly these principles. The one cross-chapter link to keep is that the positive signs and specific management of a conversion presentation belong to the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes.

Medically unexplained symptoms (MUS)
Distressing bodily symptoms not fully explained by a detectable physical disease; a description, not a single diagnosis.

Physician-centred care
A management stance in which one coordinating clinician builds an alliance, validates the distress, restrains testing, and shifts focus to functioning.

Reattribution
A brief structured technique that helps the patient re-link bodily symptoms to psychosocial stress, moving from cause to functioning.

7.1 Physician-centred care: the foundation of management

One coordinating clinician, one continuing relationship, and restraint. The guideline foundation is physician-centred care (IPS_CPG 2020; WHO mhGAP 2016): emphasise non-pharmacological strategies as the first line of treatment, and build management on a small number of durable moves. A single named clinician holds the case, so care stops fragmenting across specialists. That clinician establishes a **therapeutic relationship**, the alliance that carries every other step and is undermined by prescription-drug dependence, benzodiazepine dependence in particular, which the IPS CPG flags explicitly. The clinician validates that the symptoms and the distress they cause are real and not imaginary, restricts unnecessary diagnostic testing and specialist referrals to those with a clear medical indication such as abnormal vital signs, identifies and treats any concurrent anxiety or depression early, and shifts the clinical focus away from symptoms and toward functioning. WHO mhGAP adds two explicit "do not" moves that sit inside this foundation, that being not ordering further investigations without a clear indication and not giving vitamin injections or other ineffective treatments.

ELEMENT OF PHYSICIAN-CENTRED CARE	WHAT IT MEANS IN THE ROOM	WHY IT IS THE ACTIVE INGREDIENT
Single coordinating clinician	One named doctor holds and continues the case	**Stops fragmented care and repeated doctor-shopping**
Therapeutic relationship	A trusting, continuing alliance with the patient	**The vehicle every other step travels on**
Validate symptoms and distress	State plainly that the symptoms are real, not imagined	**Removes the felt need to keep proving illness**
Restrict testing and referrals	Investigate only on a clear medical indication	**Breaks the iatrogenic loop of chasing disease**
Treat comorbidity early	Screen and treat concurrent depression or anxiety	**Relieves a driver of the somatic burden itself**
Shift toward functioning	Target coping and function, not symptom eradication	**A realistic, sustainable goal that reduces distress**

The six elements of physician-centred care, the guideline-anchored first line for medically unexplained symptoms (IPS_CPG 2020, WHO mhGAP 2016), set out step by step here.

MNEMONIC

CARVES

The physician-centred foundation, the way it carves the plan out of the chaos. **C**oordinating clinician, one named doctor; **A**lliance, the therapeutic relationship; **R**estrict tests and referrals; **V**alidate the real symptoms and distress; treat **E**motional comorbidity early; **S**hift from symptoms to functioning. Six moves, no prescription among them.

RULE **Physician-centred care is the treatment, not the waiting room.** In MUS the coordinating clinician, the therapeutic alliance, validation and restraint on testing *are* the active first-line intervention (IPS_CPG 2020); the alliance is the first move, not the prescription.

GOOD TO REMEMBER

- **Physician-centred care (the management step):** the guideline first line (IPS_CPG 2020, WHO mhGAP 2016), being a single coordinating clinician, a therapeutic relationship, validation that the symptoms and distress are real, restriction of unnecessary tests and referrals, early treatment of comorbid mood or anxiety, and a shift toward functioning. The first move is the alliance, and non-pharmacological strategies lead.

7.2 The reattribution model

A brief four-stage technique that re-links body to mind. Reattribution is the classic structured method for MUS in primary and general-hospital care, originally taught by Goldberg and Gask, and it runs in four stages (AIIMS Consultation Liaison handbook 2020). The first stage is *feeling understood*: focused history, focused physical examination and supportive listening to build the alliance. The second stage *broadens the agenda*: give feedback, acknowledge the concerns, and pick up cues of underlying distress. The third stage is the *normalising link*: connect the bodily symptoms to psychosocial problems, negotiate that unnecessary tests and doctor-shopping will

be curtailed with the assurance that tests will be arranged if genuinely needed, and target improved functioning and reduced distress rather than complete symptom removal; scheduling regular follow-up here both strengthens the alliance and prevents the patient from generating a new symptom as a ticket to a new physician. The fourth stage *makes the change*: agree a management plan and negotiate the goals of treatment. The model is effective, feasible and acceptable in routine primary care.

REATTRIBUTION STAGE	TASK IN THE ROOM	THE MOVE TO HOLD
Feeling understood	Focused history and examination, supportive listening	**Build the alliance before anything else**
Broadening the agenda	Give feedback, acknowledge concerns, read distress cues	**Open the emotional agenda without confrontation**
Making the link	Normalise, connect symptoms to psychosocial stress	**Aim at functioning, curb needless testing**
Negotiating the plan	Agree a management plan and shared goals	**Convert insight into an actionable plan**

The four stages of the reattribution model, from feeling understood to negotiating a plan (AIIMS Consultation Liaison handbook 2020, after Goldberg and Gask), run in order here.

GOOD TO REMEMBER

- **Reattribution (the management step):** a brief four-stage technique, that being feeling understood (focused history and examination), broadening the agenda (feedback and distress cues), making the link (normalise symptoms to psychosocial stress, aim at functioning, curb needless testing), and negotiating the plan. The guiding principle is to move from proving or disproving disease to restoring function.

7.3 The psychological therapies: CBT, psychoeducation, relaxation, mindfulness

Cognitive behaviour therapy leads, with a brief package close behind. The structured psychological evidence is the substance of the plan once the alliance is set. Cognitive behaviour therapy for somatic presentations, the **cbt somatic** evidence base, is the first-line psychotherapy (IPS_CPG 2020): it targets the catastrophic misinterpretation of bodily sensations, adds relaxation and pleasurable, meaningful activities, and rebuilds functioning through behavioural activation and cognitive restructuring; sessions typically range from four to sixteen over roughly three months. Running alongside it as an alternative first-line approach is a brief intervention that bundles psychoeducation, relaxation therapy and reattribution training. Psychoeducation emphasises the *mechanism* of symptoms over their cause, acknowledges the symptoms as real, communicates normal clinical and test findings, elicits the patient's own explanatory model, and discusses the links between emotional stress and bodily sensation, optionally paired with a consultation letter to the referring physician. Relaxation therapy means progressive muscular relaxation and slow diaphragmatic breathing. For a hypochondriacal, illness-anxiety flavoured presentation the IPS CPG puts CBT with cognitive restructuring and exposure and response prevention first line, with mindfulness-based cognitive therapy or acceptance and commitment therapy as second line; for persistent somatoform pain it puts CBT plus biofeedback first line.

WORKED EXAMPLE

A woman in her thirties has had two years of shifting aches, fatigue and gut symptoms, three normal specialist work-ups and a growing conviction that "something is being missed". Rather than order a fourth panel, her one coordinating clinician validates the symptoms as real, restricts new testing to a clear indication, and starts reattribution: focused examination to feel understood, then a gentle link between symptom flares and a difficult caregiving load, then a shared plan of scheduled monthly reviews with a functioning goal of returning to part-time work. Referral for cognitive behaviour therapy targets her catastrophic reading of each new sensation. No antidepressant is started, because there is no comorbid depression and no dominant pain syndrome.

GOOD TO REMEMBER

- **Psychological therapy in MUS (the management step):** the guideline first line (IPS_CPG 2020) is cognitive behaviour therapy targeting catastrophic misinterpretation and rebuilding function, with an alternative brief package of psychoeducation, relaxation and reattribution; psychoeducation stresses mechanism over cause. For illness-anxiety presentations, CBT with cognitive restructuring and exposure and response prevention leads, mindfulness-based approaches second line.

7.4 The pharmacological rule: restraint first, drugs for the comorbidity

Do not medicate the somatic symptoms; medicate a genuine comorbidity. The single most testable management fact here is a "do not": under the WHO mhGAP OTH module, do NOT prescribe anti-anxiety or antidepressant medicines for medically unexplained somatic complaints unless advised by a specialist (WHO mhGAP 2016). Medication is not the first-line treatment of the somatic symptoms themselves. It becomes appropriate only when there is a genuinely co-existing depressive or anxiety disorder, or a dominant chronic-pain component. Where mood or anxiety comorbidity dominates, the **ssri somatic** question is answered by treating that disorder on its own merits, so an SSRI is reasonable. Where persistent pain dominates, the analgesic antidepressants are preferred: an SNRI such as duloxetine, typically **60 mg** once daily, or a low-dose tricyclic such as amitriptyline for its analgesic action. The evidence keeps drugs firmly in a supporting role: the Cochrane review of pharmacological interventions for somatoform disorders (Kleinstauber 2014) found only low to very-low-quality evidence that new-generation antidepressants reduce somatic symptoms, so drugs supplement, and never replace, physician-centred and psychological care. Polypharmacy is avoided, and side effects matter especially here because they can amplify symptom perception.

TRAP **Reflexively prescribing an antidepressant or anxiolytic for the somatic symptoms.** WHO mhGAP (2016) says do not, unless a specialist advises; reserve drugs for a genuine comorbid depression or anxiety disorder, or a persistent-pain component, and never as a substitute for physician-centred and psychological care.

GOOD TO REMEMBER

- **Medication in MUS (the drug principle):** the guideline rule (WHO mhGAP 2016) is not to prescribe anti-anxiety or antidepressant drugs for medically unexplained somatic symptoms unless a specialist advises. Reserve them for comorbid depression or anxiety, or for a dominant chronic-pain component, where an SNRI (duloxetine, typically 60 mg once daily) or a low-dose tricyclic (amitriptyline) is preferred; deep antidepressant pharmacology sits in the Mood disorders: pharmacotherapy notes.

7.5 Follow-up: shifting from symptoms to functioning

Scheduled, time-based reviews replace symptom-driven visits. The management does not end at the first plan; the follow-up posture is itself guideline content. WHO mhGAP asks the clinician to review the person in 2 to 4 weeks if symptoms do not improve or worsen, and to reconsider the protocol or consult a specialist if there is no improvement or repeated insistence on further investigation (WHO mhGAP 2016). The IPS CPG frames maintenance around encouraging functioning and coping, keeping stable patients on roughly one visit every 1 to 2 months, and educating patient and family that minor symptom variations are common and need not be attributed to new pathology (IPS_CPG 2020). The deep principle across both is that visits are scheduled by the calendar, not triggered by each new symptom, which is precisely what stops a fresh complaint becoming the only permit to be seen.

15	4	2-4
PHQ-15 HIGH-SEVERITY CUT-OFF (0 TO 30 SCALE)	STAGES OF THE REATTRIBUTION MODEL	MHGAP SYMPTOM-REVIEW WINDOW (WEEKS)

PEARL

Grade severity with a scale to pitch the intensity of care: the PHQ-15 rates fifteen common somatic symptoms, each scored 0 to 2, for a total out of a possible 30, with cut-points at 5, 10 and 15 marking low, medium and high burden. The reproduced PHQ-15 form sits with the somatic-symptom scales; here the high-severity cut-off, at 15, is the number to carry.

GOOD TO REMEMBER

- **Follow-up in MUS (the management step):** the guideline posture (WHO mhGAP 2016, IPS_CPG 2020) is calendar-scheduled review, reassess in 2 to 4 weeks if no improvement, keep stable patients to roughly one visit every 1 to 2 months, and educate that minor symptom variation is normal and not new disease. Time-based visits replace symptom-triggered ones.

7.6 The functional somatic syndromes: chronic fatigue syndrome, fibromyalgia, phantom limb pain

Named syndromes, same management principles. Several defined conditions sit close to MUS as functional somatic syndromes and are managed on the same physician-centred, psychological-first footing, with medication reserved for a pain or comorbidity component. Chronic fatigue syndrome is defined by severe, disabling mental and physical fatigue that is not relieved by rest and is worsened by exertion, persisting for at least several months and not explained by another condition; graded activity and cognitive behaviour therapy are the mainstay psychological

approaches (Shorter Oxford; New Oxford). Fibromyalgia is defined by chronic widespread pain with tenderness, fatigue, unrefreshing sleep and cognitive complaints; here an antidepressant with an analgesic action, an SNRI such as duloxetine, has an established role alongside exercise and CBT, illustrating the "pain component" exception to the drug rule (Stahl; Kaplan and Sadock CTP 2024). Phantom limb pain is pain experienced in a limb that has been amputated, distinct from localised stump pain, and it responds in part to the analgesic antidepressants and to graded, functionally oriented rehabilitation (Kaufman's Clinical Neurology for Psychiatrists; Kaplan and Sadock CTP 2024). Across all three, the trap is to keep re-investigating a settled functional diagnosis rather than treating it.

GOOD TO REMEMBER

- **Chronic fatigue syndrome (the syndrome):** severe, disabling fatigue not relieved by rest and worsened by exertion, for at least several months, unexplained by another condition; the discriminator is post-exertional worsening, and graded activity plus CBT lead.
- **Fibromyalgia (the syndrome):** chronic widespread pain with tenderness, fatigue and unrefreshing sleep; the discriminator is the widespread pain, and an analgesic antidepressant (an SNRI such as duloxetine) plus exercise and CBT is the standard approach.
- **Phantom limb pain (the syndrome):** pain felt in an amputated limb, distinct from stump pain confined to the injury; the discriminator is the referred location in the absent limb, managed with analgesic antidepressants and functionally oriented rehabilitation.

INDIA

Somatic presentation is the Indian norm rather than the exception: emotional distress is very commonly first brought to the clinician as bodily symptoms, so a large share of primary-care and general-hospital attenders sit in exactly this management space, and the Indian evidence base is anchored by the IPS_CPG 2020 guideline on psychotherapies for somatoform disorders and by indigenous tools such as the Scale for Assessment of Somatic Symptoms. The most examinable India-specific presentation is the culture-bound Dhat syndrome, a somatic and anxious preoccupation attributed to perceived loss of vital fluid; it is managed with the same physician-centred alliance, validation and psychoeducation rather than reflexive medication, and must be read through a cultural formulation rather than pathologised on its surface content. Its wider transcultural frame and the consultation-liaison service model that most often meets these patients are developed in the Consultation-liaison, geriatric and transcultural psychiatry notes.

HOLD THIS

The management of medically unexplained symptoms is physician-centred and psychological first: one coordinating clinician builds a therapeutic relationship, validates the real symptoms and distress, restricts unnecessary testing and referrals, treats comorbid depression or anxiety early, and shifts the focus from symptoms to functioning, with reattribution and CBT as the first-line psychological work; per WHO mhGAP, anti-anxiety or antidepressant drugs are NOT prescribed for the somatic symptoms themselves unless specialist-advised, and are reserved for a comorbid mood or anxiety disorder or a persistent-pain component (an SNRI such as duloxetine, or low-dose amitriptyline), while chronic fatigue syndrome, fibromyalgia and phantom limb pain are managed on the very same principles.

Sexual dysfunctions, paraphilias and gender

8 · The normal sexual response cycle

Learn the map before the malfunctions. The **sexual response cycle** is the physiological scaffold on which every sexual dysfunction is classified, so it is the first thing to fix before any of the disorders make sense. Both DSM-5-TR and ICD-11 name their sexual dysfunctions by the *phase of response that is disrupted*, which means the examiner who asks you to define hypoactive desire, erectile dysfunction, or premature ejaculation is really asking whether you can place each complaint on this cycle. The topic is small, high-yield, and almost entirely about three named models plus one piece of neuroanatomy.

This note owns the normal frame: the **masters and johnson** four-phase model with its refractory period, Helen Singer Kaplan's triphasic reformulation that added the **desire phase** and turned the cycle into a diagnostic map, Rosemary Basson's circular model of female response, and the autonomic wiring of erection and ejaculation. The dysfunctions themselves, their criteria, and their guideline-anchored management are developed in the sexual dysfunction topics of these notes; the culture-bound frame of Dhat and semen-loss anxiety sits in the Consultation-liaison, geriatric and transcultural psychiatry notes. Keep the whole discussion clinical and schematic, and lean on the accompanying figure of the response-cycle curves.

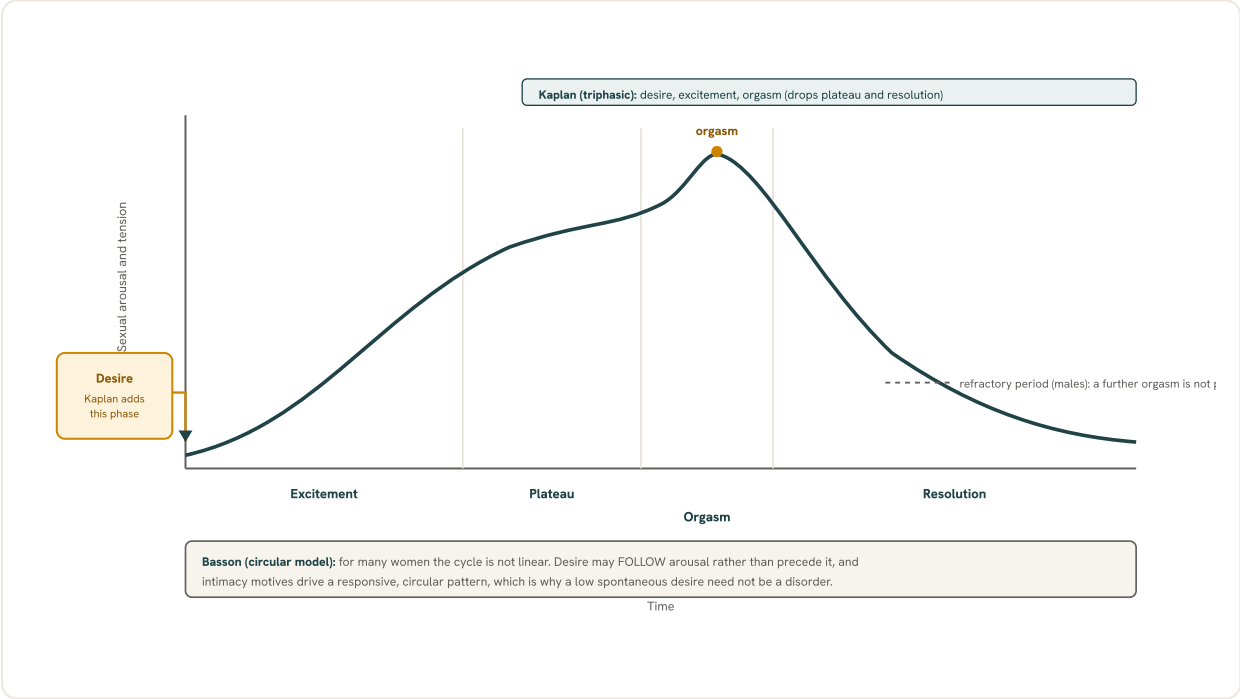

Fig 18.5 The normal sexual response cycle. Masters and Johnson described four sequential phases, drawn here as a single arousal curve: excitement, a sustained plateau, the orgasmic peak, and resolution, with a refractory period in males during which a further orgasm is not possible. Kaplan reframed the cycle as triphasic by adding an initial desire phase and collapsing plateau and resolution, which is the clinically useful version because it separates the desire, arousal and orgasm disorders. Basson's circular model captures a common female pattern in which desire is responsive, following arousal and intimacy rather than preceding them, so that a low level of spontaneous desire is not in itself a dysfunction. The figure is a neutral physiological schematic.

8.1 The Masters and Johnson four-phase model

Four sequential phases. From their laboratory observations, published as *Human Sexual Response* in 1966, Masters and Johnson described the human sexual response cycle as four sequential phases: **excitement**, **plateau**, **orgasm** and **resolution** (Kaplan and Sadock 2024). This is the **four-phase model**, and the two physiological engines running through it are vasocongestion (increased genital blood flow, giving erection in men and clitoral and vaginal engorgement with lubrication in women) and myotonia (rising skeletal and smooth-muscle tension). Excitement is the initial arousal and engorgement; plateau is the sustained high-arousal state just short of climax; orgasm is the brief peak that releases the accumulated tension; and resolution is the return to the unaroused baseline with a sense of well-being.

The refractory period is the sex difference to name. In men, resolution includes a refractory period, a recovery interval during which a further orgasm is physiologically not possible; it is short in youth and lengthens with age. Women characteristically have little or no true refractory period and can return to the orgasm phase with minimal delay, which is the physiological basis of multi-orgasmic capacity (Kaplan and Sadock 2024). That single asymmetry is the classic viva point and is exactly what the accompanying figure of the response-cycle curves is drawn to show.

PHASE (MASTERS AND JOHNSON)	CORE PHYSIOLOGICAL EVENTS
Excitement	**Onset of arousal: vasocongestion begins, penile erection, clitoral and vaginal engorgement with lubrication, breast and nipple changes, rising heart rate and blood pressure**
Plateau	Arousal sustained near its peak: testes elevate, the outer vaginal third forms the orgasmic platform, muscle tension and cardiorespiratory rate climb further
Orgasm	**Brief climax releasing sexual tension: rhythmic pelvic contractions, in men emission then ejaculation, in women rhythmic contraction of the orgasmic platform**
Resolution	**Return to baseline: detumescence and de-engorgement, well-being; men enter a refractory period, women may not**

The four-phase model of Masters and Johnson; the discriminating detail is the male refractory period in resolution, absent or minimal in women (Kaplan and Sadock 2024).

GOOD TO REMEMBER

- **Four-phase model (Masters and Johnson):** excitement, plateau, orgasm, resolution, driven by vasocongestion and myotonia; the defining sex difference is the refractory period in resolution, present in men and minimal or absent in women.

8.2 Kaplan's triphasic model and the desire phase

Kaplan added the mind before the body. Helen Singer Kaplan's 1979 reformulation, the **kaplan triphasic** model, collapsed the physiological cycle into three interdependent components and,

crucially, prefixed a psychological one: **desire**, **excitement** and **orgasm** (Kaplan and Sadock 2024). The addition of the desire phase mattered because Masters and Johnson had begun the cycle at excitement and so had no place for the many patients whose problem is an absence of sexual interest or drive before arousal is ever attempted. Desire is appetitive and centrally driven; excitement and orgasm are the peripheral, largely vascular and reflexive events that follow.

The triphasic model is a diagnostic map. Its enduring value is that each phase corresponds to a family of dysfunctions, which is why DSM-5-TR uses these phases as the basis for classifying the sexual dysfunctions (Kaplan and Sadock 2024). A desire-phase problem gives low libido (hypoactive sexual desire) or, at the other pole, hypersexuality; an excitement or arousal-phase problem gives erectile dysfunction in men and female arousal disorder; and an orgasm-phase problem gives anorgasmia or premature ejaculation. Localise the complaint to a phase and you have narrowed the differential before you have named a single criterion.

TRIPHASIC PHASE (KAPLAN)	NATURE OF THE PHASE	DYSFUNCTION WHEN DISRUPTED
Desire	**Central, appetitive drive and interest**	Hypoactive sexual desire; sexual aversion; hypersexuality
Excitement (arousal)	Peripheral vascular arousal (erection, engorgement, lubrication)	**Erectile dysfunction; female sexual arousal disorder**
Orgasm	Reflex peak and release of tension	**Anorgasmia (delayed or absent orgasm); premature ejaculation**

Kaplan's triphasic model as a map of the dysfunctions; the examiner's use of it is to place any presenting complaint on desire, excitement or orgasm (Kaplan and Sadock 2024; DSM-5-TR 2022).

RULE **The disrupted phase names the disorder.** Desire-phase failure means a desire disorder, excitement-phase failure means an arousal disorder such as erectile dysfunction, orgasm-phase failure means anorgasmia or premature ejaculation; classification follows the phase.

GOOD TO REMEMBER

- **Kaplan triphasic model:** desire, excitement, orgasm; the point of the model is that it adds the desire phase that Masters and Johnson omitted and maps each phase onto a family of dysfunctions, which is the framework DSM-5-TR adopts for classification.

8.3 Basson's circular model of female response

A circle, not a straight line. The linear models assume desire is spontaneous and always precedes arousal, an assumption that fits male experience better than female and risks over-diagnosing hypoactive desire in women (Kaplan and Sadock 2024). Rosemary Basson's circular model (2000), developed for female response, replaces the straight line with a loop that many women enter from a position of sexual neutrality, moved by a wish for emotional intimacy rather than by a spontaneous surge of drive. Willingness leads to sexual stimuli, arousal builds, and desire is then triggered by that arousal, so here desire can *follow* arousal rather than lead it. This is responsive desire, as opposed to the spontaneous desire the linear models presume.

Why it matters clinically. Basson's model reframes emotional satisfaction and relationship quality as drivers of the cycle, not incidental extras, and it normalises a pattern of desire that the linear models would misread as pathology. Although built around female response, its principles are now applied across sexes, and it is the reason a purely mechanistic, erection-and-orgasm account of a couple's difficulty so often misses the point.

PEARL

The single most testable idea from Basson is that arousal can precede desire. In her circular model a woman may begin from neutrality, choose to be receptive for intimacy, and only then, as arousal builds, experience desire. Reading that responsive pattern as a disorder of the desire phase is the commonest conceptual error at the bedside, and it is exactly what the model was written to correct.

8.4 The neurophysiology: point and shoot

Two autonomic divisions, two jobs. Erection is a parasympathetic event and ejaculation is a sympathetic one, and the whole of it is captured by the classic mnemonic point and shoot (Kaufman's Clinical Neurology for Psychiatrists). Excitatory descending impulses raise parasympathetic outflow from the sacral cord, which relaxes the smooth muscle of the genital arteries; the relaxed arteries dilate, blood flows in, and the result is penile erection in men and clitoral engorgement in women. At the tissue level the mediator is nitric oxide, released in the corpus cavernosum during arousal; it raises cyclic guanosine monophosphate, which produces the smooth-muscle relaxation and blood inflow that give the erection, and it is the enzyme that degrades that second messenger, phosphodiesterase-5, which the erectile-dysfunction drugs inhibit (Kaplan and Sadock 2024; Stahl).

Sympathetic outflow closes the sequence. With continued stimulation a predominantly sympathetic, thoracolumbar discharge produces emission (movement of seminal fluid into the posterior urethra) and then ejaculation, after which arterial tone returns and detumescence follows in resolution (Kaufman). The parasympathetic outflow for erection arises from the sacral cord at **S2 to S4** and the sympathetic outflow for emission and ejaculation from the thoracolumbar cord at **T11 to L2**; the somatic pudendal nerve (also S2 to S4) carries the erotic afferents and drives the rhythmic pelvic-floor contractions of the ejaculatory reflex. A failure of sympathetic bladder-neck closure diverts semen backwards, which is why sperm in a post-orgasm urine sample signals retrograde ejaculation.

MNEMONIC

POINT and SHOOT

Point is **P**arasympathetic and gives the erection (S2 to S4, acetylcholine, nitric oxide, arterial relaxation and inflow); **Shoot** is **S**ympathetic and gives emission and ejaculation (T11 to L2). The clinical payoff: sympathetic overdrive, as in anxiety, both softens erection and hurries ejaculation.

TRAP **Do not swap the autonomic assignments.** Erection is parasympathetic (point), not sympathetic; it is sympathetic overactivity, the physiology of anxiety, that abolishes erection and precipitates premature ejaculation, which is why performance anxiety is self-perpetuating.

GOOD TO REMEMBER

- **Autonomic control (point and shoot):** parasympathetic sacral outflow (S2 to S4) mediates erection via nitric oxide and cyclic guanosine monophosphate with smooth-muscle relaxation; sympathetic thoracolumbar outflow (T11 to L2) mediates emission and ejaculation; the pudendal nerve is the somatic afferent and ejaculatory-motor route.

8.5 Using the cycle in the clinic

The cycle is the sexual-history template. A good psychosexual history walks the phases in order and asks, for the patient and the partner, where the difficulty falls: is it a lack of interest before anything begins (desire), a failure to become or stay aroused (excitement), or a problem with climax and its timing (orgasm), with pain considered alongside (Kaplan and Sadock 2024). Because illnesses that cause pain, fatigue, vascular disease or hormonal deficiency and many medications each strike particular phases, phase-localisation immediately raises the organic differential as well as the psychological one. Erectile dysfunction, an excitement-phase vascular failure, is a recognised early marker of endothelial and cardiovascular disease, so the cycle is a screening prompt, not merely a taxonomy.

WORKED EXAMPLE

A man reports that his interest and arousal are intact and erections are firm, but climax arrives far sooner than he wishes. Walk the triphasic model: desire is normal, excitement is normal, and the problem is squarely in the orgasm phase, which localises the complaint to premature ejaculation rather than a desire or erectile disorder before a single management step is chosen. Reverse the pattern, so that interest is present but the erection will not form or hold, and you have moved one phase upstream to an excitement-phase disorder, erectile dysfunction, with its own vascular and organic differential. Localise first, name second.

INDIA

Indian teaching uses the same frame: it anchors the sexual dysfunctions to disturbance across the phases of the sexual response cycle, so the Masters and Johnson and Kaplan models are the standard Indian exam scaffold. The everyday clinical relevance is that orgasm-phase and desire-phase complaints, particularly premature ejaculation and low desire, dominate Indian psychosexual and andrology clinics, and they are frequently overlaid on the culture-bound anxiety of Dhat, in which normal physiology is misread through a fear of semen loss. Placing such a presentation on the cycle first, and reading the cultural meaning second, keeps the assessment grounded; the transcultural frame of Dhat is developed in the Consultation-liaison, geriatric and transcultural psychiatry notes.

4	3	S2 to S4	T11 to L2
MASTERS AND JOHNSON PHASES (EXCITEMENT, PLATEAU, ORGASM, RESOLUTION)	KAPLAN TRIPHASIC PHASES (DESIRE, EXCITEMENT, ORGASM)	PARASYMPATHETIC ERECTILE OUTFLOW (SACRAL)	SYMPATHETIC EMISSION AND EJACULATION OUTFLOW

HOLD THIS

Masters and Johnson gave four phases, excitement, plateau, orgasm and resolution, with a male-only refractory period; Kaplan's triphasic model added the desire phase to make desire, excitement and orgasm, and that phase map is how DSM-5-TR classifies the dysfunctions; Basson's circular model shows female desire can be responsive and follow arousal; and the wiring is point and shoot, parasympathetic sacral outflow (S2 to S4, nitric oxide) for erection and sympathetic thoracolumbar outflow (T11 to L2) for emission and ejaculation.

9 · Male sexual dysfunctions and their management

This topic sits at the *desire* and *arousal* hinge of the chapter, and it is examined as a hybrid: a short discrimination task that places a man's complaint in the right diagnostic box, then a longer, guideline-anchored management answer that most candidates under-prepare. The high-yield entity is **erectile dysfunction**, both because it is the commonest presentation the psychiatrist meets (as a side effect, a comorbidity of diabetes or vascular disease, or a primary complaint) and because its first-line drug treatment is one of the cleanest guideline positions in the syllabus. The consensus across the sexual-medicine bodies, the International Society for Sexual Medicine, the British Society for Sexual Medicine, and the operative urology guidelines, is uniform, so marks are lost only by getting a dose, a contraindication, or the testosterone principle wrong.

How to use this page. First fix the DSM-5-TR and ICD-11 map of the **male sexual dysfunctions** and the common criteria that gate every one of them; then learn erectile dysfunction properly, its definition, the organic against psychogenic split, and its meaning as a cardiovascular warning; then commit the stepwise management, first-line **PDE5 inhibitor** with its verified doses and the absolute nitrate rule, second-line injection or vacuum, third-line prosthesis, and the single testosterone principle that carries the classic trap. Finish with the **ejaculatory disorders** and hypoactive desire. The one error this page is built to prevent is starting long-term testosterone in a man whose testosterone is normal.

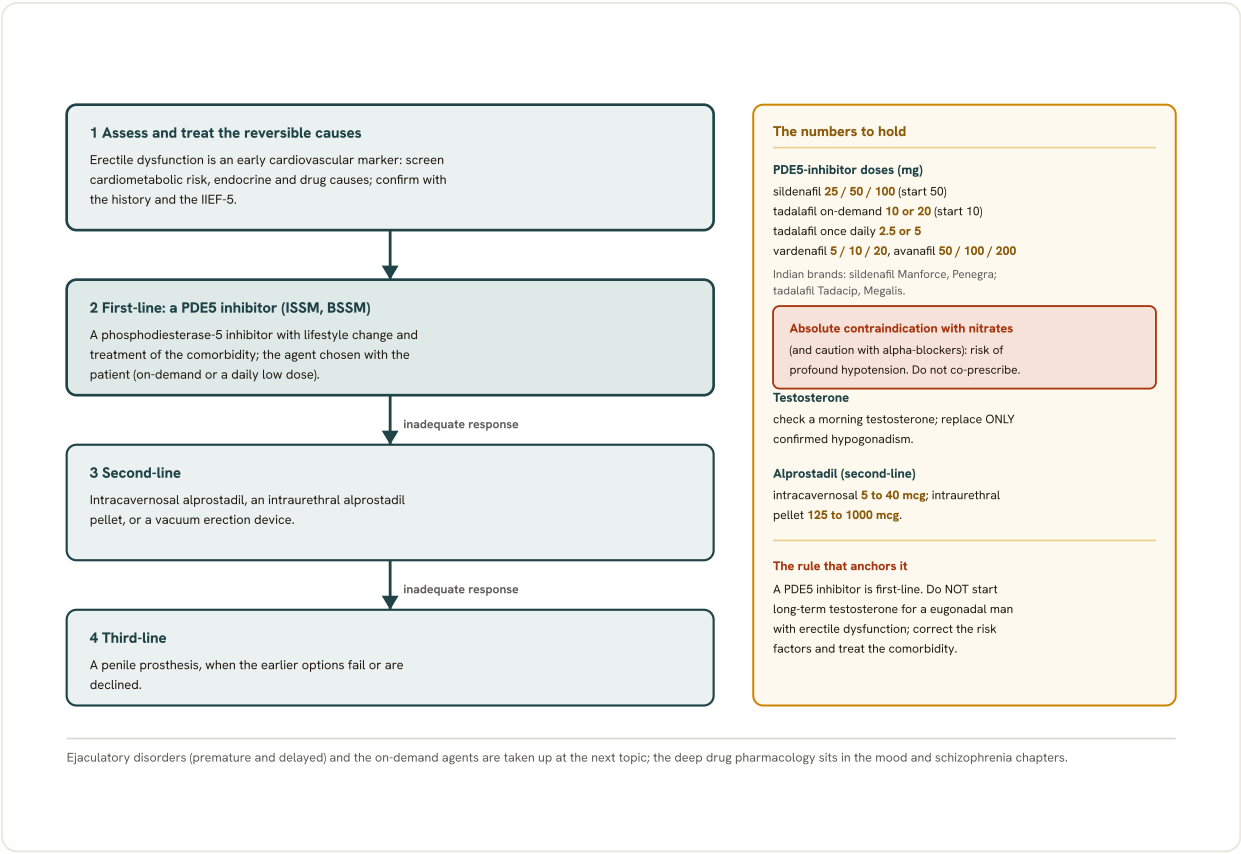

Fig 18.6 The erectile-dysfunction management algorithm. Because erectile dysfunction is often an early sign of vascular disease, the first step is to screen and treat the reversible cardiometabolic, endocrine and drug causes and to confirm the picture with the history and the IIEF-5. First-line treatment, anchored on the ISSM and BSSM guidance, is a phosphodiesterase-5 inhibitor alongside lifestyle change and treatment of the comorbidity, chosen with the patient as an on-demand agent or a daily low dose; the doses are held in the panel. A phosphodiesterase-5 inhibitor is absolutely contraindicated with nitrates. Where it fails or is unsuitable, second-line options are intracavernosal or intraurethral alprostadil or a vacuum device, and third-line is a penile prosthesis. Testosterone is checked and replaced only where hypogonadism is confirmed; empirical testosterone for a eugonadal man is a classic error.

IIEF-5 (SHIM), THE 5-ITEM SEXUAL HEALTH INVENTORY FOR MEN	SCORE BAND (OUT OF A POSSIBLE 25)
5 items on erectile function and satisfaction over the past 6 months, each scored 1 to 5; total out of a possible 25	22 to 25: no erectile dysfunction
higher total means better erectile function	17 to 21: mild
a brief screen and severity grade, not a diagnosis on its own	12 to 16: mild to moderate
tracks response to treatment over time	**8 to 11: moderate; 5 to 7: severe**

IIEF-5 / SHIM (Rosen et al, 1999); copyright the authors, permission required, so typeset here in summary with the verified severity bands rather than reproduced.

ASEX (ARIZONA SEXUAL EXPERIENCE SCALE)	INTERPRETATION
5 items: sex drive, arousal, erection or lubrication, ability to reach orgasm, and satisfaction from orgasm	each item scored 1 to 6; total out of a possible 30; higher means more sexual dysfunction
useful for tracking medication-induced sexual dysfunction across either sex	**clinically significant dysfunction if the total is 19 or more, OR any one item is 5 or more, OR any three items are 4 or more**

ASEX (McGahuey et al, 2000); University of Arizona, permission required, so typeset here in summary with the verified thresholds rather than reproduced.

Fig 18.7 The sexual-function rating scales. The IIEF-5, or Sexual Health Inventory for Men, is the brief erectile-function screen and severity grade: five items each scored 1 to 5 for a total out of a possible 25, banded from 22 to 25 (no dysfunction) down to 5 to 7 (severe). The Arizona Sexual Experience Scale is a short five-item measure usable across either sex and especially handy for tracking medication-induced sexual dysfunction, each item scored 1 to 6 for a total out of a possible 30, with clinically significant dysfunction flagged by a total of 19 or more, any single item of 5 or more, or any three items of 4 or more. Both are permission-required instruments and are summarised here from their verified structure and thresholds.

9.1 The DSM-5-TR and ICD-11 map of the male dysfunctions

Four male-relevant disorders, one shared set of gates. DSM-5-TR groups the male sexual dysfunctions into male hypoactive sexual desire disorder, erectile dysfunction (erectile disorder), premature ejaculation (premature or early ejaculation), and delayed ejaculation, alongside substance or medication-induced sexual dysfunction (Kaplan and Sadock CTP 2024; DSM-5-TR 2022). Whichever one you are diagnosing, the same qualifying criteria apply: the symptom must persist for at least six months, cause clinically significant distress, and not be better explained by a non-sexual mental disorder, relationship distress, another medical condition, or a substance. Erectile dysfunction, premature ejaculation and delayed ejaculation add a frequency gate on top of these, the symptom on roughly 75 to 100 percent of occasions; male hypoactive sexual desire disorder carries no frequency threshold in DSM-5-TR. Each is then coded as lifelong or acquired and as generalised or situational, and its severity is graded by the distress it causes. ICD-11 made the parallel move of lifting these out of the mental disorders chapter into a dedicated chapter, "Conditions related to sexual health", with gender-parallel dysfunctions (ICD-11).

DISORDER (DSM-5-TR)	CORE FEATURE	THE ONE THING TO ATTACH TO IT
Male hypoactive sexual desire disorder	Persistently deficient or absent sexual thoughts, fantasies and desire	**Measure morning testosterone; low desire correlates with low testosterone**
Erectile dysfunction (erectile disorder)	Difficulty obtaining or maintaining an erection, or reduced erectile rigidity	**Classify as organic, psychogenic or mixed; it is a cardiovascular marker**
Premature (early) ejaculation	Ejaculation within roughly one minute of penetration, sooner than wished	**On-demand dapoxetine is the only licensed first-line drug**
Delayed ejaculation	Marked delay, infrequency or absence of ejaculation	**Separate it from retrograde ejaculation, which is always organic**

The male sexual dysfunctions under DSM-5-TR, all gated by the shared six-month, distress and exclusion criteria, with the 75-to-100-percent frequency gate on every one except male hypoactive sexual desire disorder; ICD-11 places the parallel set in its sexual-health chapter (Kaplan and Sadock CTP 2024, DSM-5-TR 2022, ICD-11).

GOOD TO REMEMBER

- **The shared criteria (every male dysfunction):** at least six months, causing significant distress, not better explained by another disorder, relationship distress, a medical condition or a substance; then specify lifelong or acquired and generalised or situational. The symptom on about 75 to 100 percent of occasions is required for erectile dysfunction, premature ejaculation and delayed ejaculation, but not for male hypoactive sexual desire disorder. Missing the six-month and distress gates is the commonest under-diagnosis error.

9.2 Erectile dysfunction: definition and the organic, psychogenic, mixed split

One definition, three aetiological patterns. Erectile dysfunction is the persistent or recurrent inability to attain or maintain a penile erection sufficient for satisfactory sexual performance (Kaplan and Sadock CTP 2024). It is classified as psychogenic, organic, or mixed, and the split is drawn largely from the history. Psychogenic erectile dysfunction, driven by performance anxiety, relationship conflict or mood, tends to be situational and sudden in onset, and the man still has normal erections with masturbation, on waking, and during sleep. Organic erectile dysfunction is insidious, and erections are deficient in most contexts, nocturnal and masturbatory included. Organic causes are vascular (arterial insufficiency or venous leak), neurogenic, endocrine (hypogonadism, hyperprolactinaemia), medication-induced, or systemic; the vasculogenic type is the commonest organic aetiology, and antipsychotic and antidepressant contributions are a reversible cause the psychiatrist must actively exclude (developed in the Schizophrenia: management, antipsychotics and adverse effects notes and the Mood disorders: pharmacotherapy notes). Where doubt persists, nocturnal penile tumescence testing (erections during sleep) helps separate organic from psychogenic disease, since preserved sleep erections point away from an organic lesion.

RULE **The history sorts organic from psychogenic before any test.** Preserved morning, masturbatory and nocturnal erections with sudden, situational onset make organic disease negligible and the picture psychogenic; insidious onset with erections deficient in every context points organic and warrants a vascular and endocrine work-up.

GOOD TO REMEMBER

- **Erectile dysfunction (the diagnosis):** persistent inability to attain or maintain an erection sufficient for satisfactory performance, for at least six months, with distress; the defining discriminator is the organic, psychogenic or mixed classification, read first from preserved versus absent nocturnal and masturbatory erections.

9.3 Erectile dysfunction as a cardiovascular sentinel

The penis is a window on the arteries. Erectile dysfunction and cardiovascular disease share a pathophysiology of endothelial dysfunction, inflammation and low testosterone, and erectile dysfunction is independently associated with coronary heart disease, stroke and all-cause mortality (Kaplan and Sadock CTP 2024). The clinically load-bearing point is that vasculogenic erectile dysfunction in a younger man can be an early indicator of future cardiovascular disease, so these men should receive an appropriate cardiovascular risk assessment rather than a prescription alone. The epidemiology reinforces it: in the Massachusetts Male Aging Study the combined prevalence reached 52 percent, and after adjusting for age the strongest correlates were the classic vascular markers, hypertension, diabetes and heart disease. Among men with diabetes, roughly 50 to 75 percent develop erectile dysfunction, about three times the rate in non-diabetic men.

PEARL

Treat new-onset erectile dysfunction in a man under 50 as a reason to check blood pressure, fasting glucose or HbA1c, and lipids, not only to write for a tablet. The small penile arteries narrow before the larger coronaries, so the complaint can arrive years ahead of a cardiac event, which is exactly why the sexual-medicine guidelines fold cardiovascular risk stratification into the ED work-up.

9.4 Assessment: history, morning testosterone, the ISSM labs and the IIEF-5

Clinical diagnosis, targeted bloods, one screening scale. Diagnosis rests on the clinical presentation, not on laboratory values; the examination emphasises the nervous, vascular and genital systems, looking for signs of hypogonadism (small testes, gynaecomastia, sparse body and facial hair). The ISSM (International Society for Sexual Medicine) recommends a focused panel of fasting glucose, HbA1c, lipids and a morning total testosterone (Kaplan and Sadock CTP 2024). The morning sample matters because testosterone follows a diurnal peak; a low result must be confirmed on a repeat before it is called hypogonadism. The abridged International Index of Erectile Function, the five-item IIEF-5 or Sexual Health Inventory for Men, both screens for and grades erectile dysfunction; the reproduced instrument sits in the accompanying scale plate. A separate five-item scale, the ASEX (Arizona Sexual Experience Scale), is the tool of choice for tracking psychotropic-induced sexual dysfunction across a one-week window, scored so that a

higher total means more dysfunction (Clinical Methods in Psychiatry, AIIMS 2024; Maudsley Prescribing Guidelines).

IIEF-5 BAND (OUT OF A POSSIBLE 25)	INTERPRETATION
22 to 25	No erectile dysfunction
17 to 21	Mild
12 to 16	Mild to moderate
8 to 11	Moderate
5 to 7	Severe

The IIEF-5 (Sexual Health Inventory for Men) severity bands; the five items each score up to 5, so the total runs from 5 to 25, read against these cut-offs (see gaps). The typeset form is reproduced in the accompanying scale plate.

GOOD TO REMEMBER

- **The ED work-up (guideline-anchored, ISSM):** diagnosis is clinical; order fasting glucose, HbA1c, lipids and a morning total testosterone, confirm any low testosterone before acting on it, and use the IIEF-5 to grade severity, a total in the 22-to-25 band out of a possible 25 meaning no erectile dysfunction and 5 to 7 out of a possible 25 meaning severe.

9.5 First-line management: the PDE5 inhibitor

An oral PDE5 inhibitor plus risk-factor optimisation, for everyone without a contraindication. First-line treatment of erectile dysfunction is a phosphodiesterase-5 inhibitor combined with lifestyle and cardiovascular risk-factor management (ISSM; BSSM; EAU male sexual dysfunction; AUA 2018). All the oral agents are similarly effective, and choice turns on how the man wants to use them: sildenafil is the high-efficacy pick and tadalafil the tolerability pick with the longest window. Sildenafil is taken on-demand, roughly one hour before intercourse, with a recommended **50 mg** starting dose titrated between 25 and 100 mg; onset is 30 to 60 minutes and the window runs up to 12 hours, with absorption delayed by a high-fat meal. Tadalafil on-demand starts at **10 mg** (range 10 to 20 mg), acts from about 30 minutes, and gives the longest window of any agent, up to 36 hours, with the least food effect. Tadalafil also has a once-daily low-dose regimen, 2.5 or 5 mg with 5 mg preferred, which decouples dosing from sexual activity and is the only PDE5 inhibitor licensed for concurrent erectile dysfunction and lower-urinary-tract symptoms of benign prostatic hyperplasia at 5 mg once daily. Vardenafil (start 10 mg) and avanafil (start 100 mg, the fastest onset, taken 15 to 30 minutes before) complete the class.

AGENT	ON-DEMAND DOSE (MG)	ONSET AND WINDOW	DISTINGUISHING POINT
Sildenafil	25, 50, 100; start 50	30 to 60 min; up to 12 h	**High-efficacy pick; high-fat meal delays absorption**
Tadalafil (on-demand)	10, 20; start 10	from 30 min; up to 36 h	**Longest window; least affected by food**
Tadalafil (once-daily)	2.5 or 5 once daily; use 5	continuous, off the clock of sex	**Only PDE5 inhibitor licensed for ED with LUTS or BPH**
Vardenafil	5, 10, 20; start 10	from about 30 min	**Orally disintegrating form available**
Avanafil	50, 100, 200; start 100	fastest; take 15 to 30 min before	**Quickest onset of the class**

The oral PDE5 inhibitors, all first-line and similarly effective; the doses are the verified EAU and AUA 2018 figures (EAU male sexual dysfunction; AUA 2018).

25 IIEF-5 TOP SCORE, NO-ED BAND STARTS AT 22	**50** SILDENAFIL ON-DEMAND START (MG)	**5** TADALAFIL ONCE-DAILY DOSE (MG)	**30** DAPOXETINE ON-DEMAND START (MG)

INDIA

The generics are widely marketed in India: sildenafil (Manforce, Penegra), tadalafil (Tadacip, Megalis), and, for premature ejaculation, dapoxetine (Duralast, Sustinex, also sold in sildenafil and tadalafil fixed-dose combinations). Keep the generic primary and confirm the exact product and strength locally; the sexual-medicine advice is that dapoxetine should not routinely be combined with a PDE5 inhibitor because of syncope risk.

MNEMONIC

PILLS → POKES & PUMPS → PROSTHESIS

The ED treatment ladder in order: oral **Pills** (PDE5 inhibitor) first, then the injection and vacuum second line (**Pokes and Pumps**), then the surgical **Prosthesis** third line. You climb only when the rung below fails or is declined.

GOOD TO REMEMBER

- **First-line ED drug (guideline-anchored, ISSM, BSSM, EAU, AUA 2018):** an oral PDE5 inhibitor plus risk-factor optimisation; sildenafil starts at 50 mg on-demand about one hour before, tadalafil at 10 mg on-demand or 5 mg once daily, and tadalafil is the only agent licensed for concurrent ED and benign prostatic hyperplasia.

9.6 The absolute nitrate contraindication

One combination you never allow. The concurrent use of any organic nitrate, nitric oxide donor, nicorandil, or a recreational nitrite with any PDE5 inhibitor is an absolute contraindication (EAU male sexual dysfunction; AUA 2018). Both act on the nitric-oxide, cyclic-GMP pathway, so the combination causes cyclic-GMP to accumulate and can precipitate profound, unpredictable

hypotension. Before prescribing, ask specifically about nitrate use for angina and about recreational nitrites. Alpha-blockers are a caution rather than an absolute bar and may be co-prescribed carefully, watching for postural hypotension.

COMMON TRAP

Forgetting to ask about a nitrate. A man on a nitrate for angina, or using a recreational nitrite, must never receive a PDE5 inhibitor; screening for nitrate use is a mandatory step, not an optional one, and no dose is "safe" in this combination.

GOOD TO REMEMBER

- **PDE5 inhibitor caution (guideline-anchored, EAU and AUA 2018):** any organic nitrate, nitric oxide donor, nicorandil or recreational nitrite is an absolute contraindication with any PDE5 inhibitor because of cyclic-GMP accumulation and profound hypotension; alpha-blockers are a relative caution only.

9.7 Second-line and third-line therapy

When the tablet fails or is contraindicated, step down the ladder. Second-line options are for men in whom oral therapy fails or is contraindicated (EAU male sexual dysfunction; AUA 2018). Intracavernosal alprostadil is the most effective injectable monotherapy, dosed at 5 to 40 micrograms and producing an erection within 5 to 15 minutes, with over 70 percent efficacy but needing in-office injection training; its risks are penile pain, priapism and fibrosis. An intraurethral alprostadil pellet (MUSE, 125 to 1000 micrograms, usually initiated at 500 micrograms) and topical alprostadil cream (200 to 300 micrograms) are alternatives with more limited data. A vacuum erection device is a non-invasive, drug-free choice for well-informed men, with variable satisfaction. Third-line therapy is a surgically implanted penile prosthesis (inflatable or semi-rigid), reserved for when less-invasive options fail or the man wants a definitive solution; it has the highest satisfaction of any ED treatment, over 90 percent, its main risks being mechanical failure and infection in about 2 to 3 percent.

LINE	OPTION	DOSE OR FORM	KEY POINT
Second	Intracavernosal alprostadil	5 to 40 micrograms	**Most effective injectable; erection in 5 to 15 min; needs training**
Second	Intraurethral pellet (MUSE)	125 to 1000 micrograms, start 500	**Alternative for men declining injection; data more limited**
Second	Topical alprostadil cream	200 to 300 micrograms	**Non-injection local option**
Second	Vacuum erection device	mechanical device	**Non-invasive, drug-free; satisfaction variable**
Third	Penile prosthesis	inflatable or semi-rigid implant	**Highest satisfaction, over 90 percent; infection about 2 to 3 percent**

The second-line and third-line ED ladder, entered when oral PDE5 inhibitors fail or are contraindicated; the full stepwise route is drawn in the accompanying figure (EAU male sexual dysfunction; AUA 2018).

GOOD TO REMEMBER

- **Second and third-line ED therapy (guideline-anchored, EAU and AUA 2018):** after oral failure, intracavernosal alprostadil (5 to 40 micrograms, the most effective injectable) or a vacuum device is second line, and a penile prosthesis, with the highest satisfaction of any treatment, is third line.

9.8 Testosterone: test, then treat only confirmed hypogonadism

Measure it, but do not treat a normal level. Measure a morning total testosterone in any man with erectile dysfunction, low desire, or clinical signs of hypogonadism, and treat only biochemically confirmed hypogonadism (EAU male sexual dysfunction; AUA 2018). In a genuinely hypogonadal man who has not responded to a PDE5 inhibitor, adding testosterone can improve the response, so the two are complementary in that specific setting. What you must not do is give empirical, long-term testosterone for erectile dysfunction to a man whose testosterone is normal: it will not fix a eugonadal man's erectile dysfunction and it exposes him to the risks of unnecessary androgen therapy. This is the single commonest examined error in the topic.

TRAP **Starting long-term testosterone in a eugonadal man with erectile dysfunction.** Testosterone helps only when a low level is confirmed on a morning sample; in a man with normal testosterone it does not correct erectile dysfunction and adds needless risk. Test first, treat only proven hypogonadism.

GOOD TO REMEMBER

- **Testosterone principle (guideline-anchored, EAU and AUA 2018):** check a morning total testosterone, treat only confirmed hypogonadism, use it to rescue a hypogonadal PDE5-inhibitor non-responder, and never give empirical long-term testosterone for erectile dysfunction in a eugonadal man.

9.9 The ejaculatory disorders and hypoactive sexual desire

Three more entities, each with a clean discriminator. **Premature ejaculation** is ejaculation, before or within roughly one minute of penetration, that occurs sooner than the man wishes and causes distress; DSM-5-TR grades it as mild (about 30 seconds to 1 minute), moderate (about 15 to 30 seconds), or severe (at the start of, or within about 15 seconds of, penetration). Its pharmacological management, on-demand dapoxetine as the only licensed first-line agent (started at 30 mg, maximum 60 mg, not more than once every 24 hours), off-label daily SSRIs, topical anaesthetics, and behavioural methods, is developed in full in the dedicated premature ejaculation and dapoxetine topic. **Delayed ejaculation** is a marked delay in, infrequency of, or absence of ejaculation despite adequate stimulation; the crucial separation is from retrograde ejaculation, in which semen passes backward into the bladder, which always has an organic cause (genitourinary surgery, autonomic neuropathy, alpha-blocker or anticholinergic drugs). Male hypoactive sexual desire disorder is persistently deficient or absent sexual thoughts and desire for at least six months with distress, and it prompts a check of testosterone, mood, relationship factors and medication.

GOOD TO REMEMBER

- **Premature ejaculation (the diagnosis):** ejaculation within about one minute of penetration, sooner than wished, with distress and control lost; graded mild, moderate or severe by latency, with on-demand dapoxetine the only licensed first-line drug.
- **Delayed and retrograde ejaculation (the discriminator):** delayed ejaculation is a marked delay or absence of ejaculation; retrograde ejaculation, semen passing into the bladder, is a separate, always-organic entity tied to genitourinary surgery, autonomic neuropathy, or alpha-blocker and anticholinergic drugs.
- **Male hypoactive sexual desire disorder (the diagnosis):** persistently deficient or absent sexual thoughts and desire for at least six months with distress; the first step is to measure a morning testosterone and screen mood, medication and relationship factors.

HOLD THIS

Erectile dysfunction is first-line an oral PDE5 inhibitor (sildenafil start 50 mg on-demand, tadalafil start 10 mg on-demand or 5 mg once daily); a nitrate in any form is an absolute contraindication, and you never start long-term testosterone in a man whose testosterone is normal.

10 · Female sexual dysfunctions and their management

This topic mirrors the male-dysfunction page but flips the emphasis: the diagnostic map is a little denser (a merger and a subsuming category the examiner loves), and the management answer is deliberately un-showy, because the pharmacology is thin and the marks live in the **biopsychosocial** and couple-based first line. The high-yield facts are the DSM-5-TR trio and their shared gates, the single biggest reclassification (desire and arousal merged into one female disorder), and the one management principle that the whole chapter turns on: in women, psychological, relational and physiotherapeutic approaches lead, and drugs are a narrow, licence-limited adjunct.

How to use this page. First fix the DSM-5-TR and ICD-11 map of the female sexual dysfunctions; then take each of the three disorders in turn, learning the defining criterion and the one discriminator that keeps you out of trouble; then commit the multimodal management, biopsychosocial assessment, psychoeducation and sex therapy, the pelvic-floor and graded-dilator work that is specific to the pain-penetration disorder, local oestrogen for the menopausal cause of painful sex, and the two licensed drugs, which are covered fully in the dedicated flibanserin topic. The error this page is built to prevent is labelling painful sex psychogenic before an organic cause has been excluded.

10.1 The DSM-5-TR trio and the ICD-11 framing

Three female disorders, one shared set of gates, one big merger. DSM-5-TR recognises three **female sexual dysfunctions**: female sexual interest/arousal disorder, female orgasmic disorder, and genito-pelvic pain/penetration disorder (alongside substance or medication-induced sexual dysfunction, which is gender-neutral) (Kaplan and Sadock CTP 2024; DSM-5-TR 2022). The defining structural change from DSM-IV is that the old hypoactive sexual desire disorder and female sexual arousal disorder were combined into a single interest-and-arousal category, on the evidence that low desire and low arousal co-occur so heavily in women that separating them is artificial. Whichever of the three you are diagnosing, the same qualifying gates apply: the symptom must persist for at least approximately six months, cause clinically significant distress in the woman herself, and not be better explained by a non-sexual mental disorder, severe relationship distress or partner violence, another medical condition, or a substance. Each is then specified as lifelong or acquired and graded mild, moderate or severe by distress. ICD-11 lifts the equivalent conditions out of the mental disorders chapter into a new chapter, "Conditions related to sexual health"; notably, and unlike DSM-5, ICD-11 does *not* merge desire and arousal, keeping hypoactive sexual desire dysfunction and female sexual arousal dysfunction as separate categories, with anorgasmia and a sexual pain-penetration disorder completing the female set (ICD-11).

DISORDER (DSM-5-TR)	CORE FEATURE	THE DISCRIMINATOR TO ATTACH
Female sexual interest/arousal disorder	Merged low desire and low arousal; at least three of six indicators	**A mere "desire discrepancy" with a partner is not the disorder**
Female orgasmic disorder	Marked delay, infrequency, absence, or reduced intensity of orgasm	**Clitoral-not-coital orgasm, and inadequate stimulation, are excluded**
Genito-pelvic pain/penetration disorder	Penetration difficulty, pain, fear of pain, or pelvic-floor tensing (any one of four)	**Subsumes vaginismus and dyspareunia; the only one with no generalized/situational specifier**

The three DSM-5-TR female sexual dysfunctions, each gated by the shared roughly-six-month, distress and exclusion criteria and specified lifelong or acquired; ICD-11 places the parallel set in its "Conditions related to sexual health" chapter but keeps desire and arousal apart (Kaplan and Sadock CTP 2024, DSM-5-TR 2022, ICD-11).

GOOD TO REMEMBER

- **The shared gates (every female dysfunction):** the symptom for at least approximately six months, causing clinically significant distress in the woman, not better explained by another mental disorder, severe relationship distress, a medical condition or a substance; then specify lifelong or acquired and grade by distress. The single biggest DSM-5 change is the merger of desire and arousal into one female disorder.

10.2 Female sexual interest arousal disorder, the merged desire and arousal disorder

At least three of six indicators, for at least approximately six months, with distress. Female sexual interest/arousal disorder requires a lack of, or significantly reduced, sexual interest or arousal shown by at least three of six indicators: reduced interest in sexual activity; reduced erotic thoughts or fantasies; no or reduced initiation and typical unreceptiveness to a partner's initiation; reduced excitement or pleasure during almost all (roughly 75 to 100 percent) encounters; reduced arousal to any internal or external erotic cues; and reduced genital or non-genital sensations during almost all encounters (DSM-5-TR 2022). Two diagnostic subtleties carry marks. First, a *desire discrepancy*, where a woman simply wants sex less often than her partner, is explicitly not sufficient for the diagnosis. Second, a lifelong lack of desire that is better understood as an asexual identity is excluded. The concept of *responsive desire* matters clinically: many women do not begin an encounter with spontaneous desire but develop it once arousal is under way, so an assessment of the adequacy of sexual stimulation is part of the diagnosis, not an afterthought. Around 30 percent of women report chronic low desire, but only a fraction of those experience the personal distress the criteria demand.

RULE **Distress in the woman is the gate, not the frequency of sex.** Low desire, low arousal, or a mismatch with a partner is a disorder only when it persists for about six months and causes clinically significant distress in the woman herself; a desire discrepancy alone, or an asexual identity, is not female sexual interest/arousal disorder.

PEARL

Ask about **responsive desire** before you label a woman as having low desire. If desire reliably arrives once she is aroused with adequate, wanted stimulation, the problem may be the context, the stimulation or the relationship rather than a dysfunction, and the treatment is couple-based, not pharmacological.

GOOD TO REMEMBER

- **Female sexual interest arousal disorder (the merged desire and arousal disorder):** at least three of six Criterion A indicators of reduced sexual interest or arousal, for at least approximately six months, causing distress in the woman; a desire discrepancy with a partner and an asexual identity are both excluded, and the disorder combines the old hypoactive desire and female arousal diagnoses.

10.3 Female orgasmic disorder

Delay, infrequency, absence, or reduced intensity of orgasm on almost all occasions. Female orgasmic disorder, historically called inhibited female orgasm or anorgasmia, is a marked delay in,

marked infrequency of, or absence of orgasm, or a markedly reduced intensity of orgasmic sensations, experienced on almost all (roughly 75 to 100 percent) occasions of sexual activity for at least approximately six months and causing distress (DSM-5-TR 2022; Kaplan and Sadock CTP 2024). Two exclusions do the discriminating. A woman who reaches orgasm with clitoral stimulation but not during penile-vaginal intercourse does *not* meet the criteria, because clitoral-dependent orgasm is a normal variant, not a disorder. And where orgasm difficulty is the result of demonstrably inadequate sexual stimulation, the diagnosis is not made even though the woman may still need help. The lifelong subtype, present since the woman first became sexually active, can be specified further as "never experienced an orgasm under any situation," which is the classic indication for the directed-masturbation programme. Internationally, roughly 10 percent of women report never experiencing orgasm across their lifetime.

GOOD TO REMEMBER

- **Female orgasmic disorder (the diagnosis):** marked delay, infrequency, absence or reduced intensity of orgasm on about 75 to 100 percent of occasions, for at least approximately six months, with distress; a clitoral-only (non-coital) orgasm and orgasm difficulty from inadequate stimulation are both excluded, and the lifelong "never orgasmed" subtype is the target for directed masturbation.

10.4 Genito-pelvic pain penetration disorder, subsuming vaginismus and dyspareunia

One category built from four complaints, any one of which qualifies. Genito-pelvic pain/penetration disorder gathers the two older diagnoses of vaginismus and dyspareunia into a single category defined by persistent difficulty with one or more of: vaginal penetration during intercourse; marked vulvovaginal or pelvic pain during penetration attempts; marked fear or anxiety about that pain; and marked tensing or tightening of the pelvic-floor muscles on attempted penetration, for at least approximately six months with distress (DSM-5-TR 2022). Because these four so often drive one another (fear leads to guarding, guarding to pain, pain to more fear), DSM-5-TR reasoned it was cleaner to combine them, though the split remains useful clinically. The single most important step is not psychiatric at all: somatic causes of painful sex must be actively excluded, because a substantial minority of women presenting to sex-therapy clinics have identifiable pelvic pathology.

Vaginismus (historical term)

Involuntary spasm or tensing of the pelvic-floor and perivaginal muscles that prevents or makes painful any vaginal penetration; now the "penetration/tensing/fear" pole of genito-pelvic pain penetration disorder.

Dyspareunia (historical term)

Recurrent or persistent genital pain before, during or after intercourse; DSM-5-TR notes roughly 15 percent of North American women report recurrent painful intercourse. Somatic causes must be ruled out first.

Genitourinary syndrome of menopause

Oestrogen-deficiency vulvovaginal atrophy causing dryness and dyspareunia in peri- and post-menopausal women; the leading organic driver of acquired painful sex, and the one with a specific,

effective treatment.

TRAP **Calling painful sex psychogenic before excluding an organic cause.** Dyspareunia and the pain-penetration disorder demand a genital and pelvic examination first: genitourinary syndrome of menopause, infections, vulvodynia, endometriosis, episiotomy scarring and dermatoses all cause it, and up to a third of sex-clinic attenders with the complaint have pelvic pathology. Rule out the organ before you formulate the mind.

INDIA

In Indian sexual-medicine and psychiatry practice, genito-pelvic pain/penetration disorder frequently presents not as "painful sex" but as **non-consummation of marriage**, often as a couple, sometimes with the man's erectile difficulty layered on, and typically laden with cultural expectation and shame. Neither licensed female-desire drug (flibanserin, bremelanotide) is marketed in India, so Indian management leans even harder on psychoeducation, couple therapy, graded dilators and pelvic-floor work; local oestrogen preparations for menopausal atrophy are, however, readily available generically. Frame the assessment for the couple, non-judgementally, and rule out organic pathology early.

GOOD TO REMEMBER

- **Genito-pelvic pain penetration disorder (subsuming vaginismus and dyspareunia):** persistent difficulty with any one of four (penetration, vulvovaginal or pelvic pain, fear of that pain, pelvic-floor tensing), for at least approximately six months with distress; it is the only female dysfunction with no generalized/situational specifier, and the first step is always to exclude an organic cause of the pain.

10.5 Biopsychosocial assessment and the multimodal management

Assess and treat biopsychosocially first; psychological and couple-based approaches lead. The ISSM (International Society for Sexual Medicine) and the general consensus frame female sexual dysfunction as biopsychosocial, so assessment and management run in parallel across relationship factors, mood and anxiety, medication effects, hormonal and genital contributors, and the woman's beliefs and knowledge about sex (ISSM; Kaplan and Sadock CTP 2024). Assessment is clinical, supported where useful by a rating instrument: the FSFI (Female Sexual Function Index), a nineteen-item scale across six domains, is the standard measure, with a validated cut-off at or below **26.55** flagging a woman at risk of sexual dysfunction (Rosen et al 2000; Wiegel et al 2005); the validated cut-off is carried in the text. Management is multimodal and stepwise: first, *psychoeducation* that corrects myths and reframes the response cycle; second, *treat the contributors*, which means optimising mood and anxiety, reviewing and where possible switching a culprit drug (antidepressant-induced dysfunction is developed in the Mood disorders: pharmacotherapy notes, and antipsychotic hyperprolactinaemia in the Schizophrenia: management, antipsychotics and adverse effects notes), and treating hormonal or genital pathology; third, *sex therapy*. The core behavioural method is sensate focus (Masters and Johnson), a graded programme of non-demand mutual pleasuring with intercourse initially interdicted, which lifts performance pressure and interrupts **spectatoring** (the anxious self-observation that sabotages arousal). Disorder-specific techniques layer on top: lifelong female

orgasmic disorder is treated with directed masturbation, sometimes with a vibrator, plus Kegel exercises to build pelvic-floor awareness.

MODALITY	BEST SUITED TO	WHAT IT DOES
Psychoeducation	All three disorders	**Corrects myths, reframes the response cycle and responsive desire**
Treat the contributors	Acquired dysfunction of any kind	**Mood, anxiety, culprit drugs, hormonal and genital pathology**
Sensate focus (sex therapy)	Interest/arousal and orgasmic disorder, as a couple	**Non-demand pleasuring, intercourse banned early, ends spectatoring**
Directed masturbation and Kegels	Lifelong female orgasmic disorder	**Self-exploration to first orgasm; pelvic-floor awareness**
Pelvic-floor physiotherapy and graded dilators	Genito-pelvic pain/penetration disorder	**Desensitises penetration, retrains guarding; see 10.6**

The multimodal management of the female sexual dysfunctions, biopsychosocial and psychological-first; the full stepwise route is biopsychosocial-first (ISSM; Kaplan and Sadock CTP 2024).

3 FSIAD CRITERION A INDICATORS NEEDED, FROM A SET OF SIX	**6** MONTHS MINIMUM DURATION FOR EVERY DYSFUNCTION	**26.55** FSFI CUT-OFF FLAGGING FEMALE SEXUAL DYSFUNCTION	**100** FLIBANSERIN BEDTIME DOSE (MG PER DAY)

MNEMONIC

PLISSIT

The escalating sex-therapy framework (Annon): **P**ermission (normalise and permit the sexual concern), **LI** Limited Information (targeted education), **SS** Specific Suggestions (concrete behavioural tasks such as sensate focus, dilators or directed masturbation), and **IT** Intensive Therapy (referral for in-depth psychotherapy). Most female sexual dysfunction is resolved at the first three, lighter levels, which is exactly why the drugs sit so far down the list.

GOOD TO REMEMBER

- **Multimodal management (guideline-anchored, ISSM, biopsychosocial):** assess and treat biopsychosocially, lead with psychoeducation, treat the contributors (mood, anxiety, culprit drugs, hormones), and use sensate focus as the core sex-therapy method, with directed masturbation for lifelong female orgasmic disorder; the FSFI (cut-off at or below 26.55) grades severity, and pharmacology is a narrow adjunct, not the first move.

10.6 The pain-penetration physiotherapy, local oestrogen, and the limited drug options

For pain and penetration, physiotherapy and graded dilators; for menopausal atrophy, local oestrogen; drugs are last and licence-limited. Genito-pelvic pain/penetration disorder has the most concrete physical management of the three: pelvic-floor physiotherapy and progressive vaginal trainers, in which the woman uses her own fingers or size-graduated dilators to

desensitise penetration and unlearn the protective muscle guarding, ideally with a pelvic-floor physiotherapist, and combined with the psychological and sex-therapy work described above (Kaplan and Sadock CTP 2024). Where the driver is genitourinary syndrome of menopause, low-dose local vaginal oestrogen (for example estriol or estradiol cream or pessary) is effective for the dryness and dyspareunia and is preferred over systemic hormone therapy for isolated urogenital symptoms (NICE NG23 menopause 2015; ISSM). Pharmacotherapy for low desire is genuinely limited: the two agents licensed anywhere, flibanserin (a 5-HT1A agonist and 5-HT2A antagonist, 100 mg once daily at bedtime) and bremelanotide (a melanocortin agonist, 1.75 mg subcutaneously on demand), are approved only for acquired, generalised hypoactive sexual desire disorder in *premenopausal* women, and both are covered in full, with their boxed warnings and cautions, in the dedicated flibanserin topic (Addyi FDA label 2015; Vyleesi FDA label 2019). PDE5 inhibitors are not established for female sexual dysfunction (results are inconsistent), and systemic testosterone for female low desire is off-label and unapproved in most jurisdictions; the overall pharmacological evidence base is weak, which is precisely why couple-based and psychological approaches remain the lead.

GOOD TO REMEMBER

- **Pain-penetration and oestrogen management (guideline-anchored):** genito-pelvic pain penetration disorder is treated with pelvic-floor physiotherapy and graded dilators plus the psychological work; menopausal dyspareunia (genitourinary syndrome of menopause) is treated with low-dose local vaginal oestrogen, preferred over systemic therapy for isolated urogenital symptoms (NICE NG23).
- **The limited drug options (guideline-anchored):** flibanserin (100 mg once daily at bedtime) and bremelanotide (1.75 mg subcutaneous, on demand) are licensed only for acquired, generalised hypoactive sexual desire disorder in premenopausal women; PDE5 inhibitors are not established and systemic testosterone is off-label. Full pharmacology sits in the dedicated flibanserin topic.

HOLD THIS

The female sexual dysfunctions are the DSM-5-TR trio, female sexual interest/arousal disorder (desire and arousal merged), female orgasmic disorder, and genito-pelvic pain/penetration disorder (vaginismus plus dyspareunia), each gated by about six months and distress; management is biopsychosocial-first (psychoeducation, sensate focus, dilators and pelvic-floor work, local oestrogen for menopausal painful sex), and the only licensed drugs treat premenopausal acquired generalised low desire alone.

11 · Dapoxetine and flibanserin

These two agents pair naturally as a compact pharmacology question: both are *repurposed serotonergic drugs licensed for a single sexual dysfunction*, one for the man and one for the woman, and each carries a small cluster of verified facts, a dose, a timing, a label restriction, and one signature caution, that the examiner can pull straight out. **Dapoxetine** is the short-acting, on-demand pill taken before sex for **premature ejaculation**; **flibanserin** is the daily bedtime pill taken for months for **hypoactive sexual desire disorder** in premenopausal women. Confuse their dosing rhythms and you lose the mark; hold the rhythm and the rest follows.

How to use this page. Learn each drug as a triad: what it is (mechanism and class), how it is dosed (the verified on-demand versus daily rhythm), and its one examinable caution. Then read the pair against each other, because the discriminator most often tested is the licensing restriction, on-demand for **premature ejaculation** against acquired, generalised, **premenopausal** HSDD, and the single interaction that gets each drug into trouble.

11.1 Dapoxetine: the on-demand short-acting SSRI for premature ejaculation

The only agent licensed for on-demand treatment of premature ejaculation. Dapoxetine is a **short-acting SSRI** developed specifically for premature ejaculation, and it is the first and only drug approved for its **on-demand** treatment (Kaplan and Sadock CTP 2024; New Oxford Textbook of Psychiatry). The sexual-medicine and regulatory positions are uniform: an **on-demand SSRI** is the first-line pharmacological option for lifelong premature ejaculation (ISSM 2014; EAU male sexual dysfunction; AUA 2018), and the drug is licensed for this indication under the label recommendation of a **30 mg** starting dose taken **1 to 3 hours** before sexual activity (NICE ESNM40; Priligy SPC). It is titrated upward only if the starting dose is insufficient and tolerated, to a maximum **60 mg**, and it is never taken more than once every 24 hours. Benefit and risk are reappraised after the first 4 weeks or at least 6 doses.

Why the on-demand rhythm works. Unlike the long-acting SSRIs (paroxetine, sertraline, fluoxetine, citalopram), which delay ejaculation only when taken daily for a fortnight or more and are unsuitable as an as-needed drug, dapoxetine is absorbed and cleared rapidly, so a single pre-coital dose raises synaptic serotonin over the window that matters and then washes out. Its common adverse effects are the expected serotonergic ones, nausea, dizziness, headache and diarrhoea, together with an orthostatic, syncope-prone effect that drives its cardinal interaction rule (Priligy SPC). It is contraindicated in significant cardiac disease and in a history of mania or severe depression.

FEATURE	DAPOXETINE (VERIFIED)
Class	Short-acting, on-demand SSRI
Indication	Premature ejaculation (the only licensed on-demand agent)
Start dose (mg)	30, taken 1 to 3 hours before activity
Ceiling dose (mg)	60, only if 30 is insufficient and tolerated
Frequency	Not more than once every 24 hours
Review	Reappraise after 4 weeks or at least 6 doses
Signature caution	Do not combine with a PDE5 inhibitor (syncope); avoid in cardiac disease and in a mania or severe-depression history

Dapoxetine at a glance: the on-demand short-acting SSRI for premature ejaculation, with its verified label doses and the PDE5-inhibitor caution (ISSM 2014; NICE ESNM40; Priligy SPC).

PEARL

The pharmacokinetics are the point. A chronic SSRI blunts ejaculation through gradual receptor adaptation that takes days to build, which is why paroxetine and its peers must be taken every day off-label. Dapoxetine's rapid absorption and short half-life invert that logic: it acts within about an hour and clears quickly, so it can be swallowed only when needed, roughly 1 to 3 hours ahead. That single kinetic difference, not any special receptor trick, is what makes it the on-demand agent.

INDIA

Dapoxetine is heavily marketed in India: lead the generic, with Duralast and Sustinex the common brands, and it is also sold in sildenafil-dapoxetine and tadalafil-dapoxetine fixed-dose combinations. The verified caution runs against exactly those combinations, since the Priligy SPC advises against pairing dapoxetine with a PDE5 inhibitor because of syncope risk; keep the generic primary and confirm the exact product and strength locally before any prescribing use. Flibanserin has no established routine Indian presence, so it is examined as a name to recognise rather than a drug to prescribe here.

GOOD TO REMEMBER

- **Dapoxetine (guideline-anchored, ISSM 2014, NICE ESNM40, Priligy SPC):** the only agent licensed for on-demand premature ejaculation; a short-acting SSRI started at 30 mg taken 1 to 3 hours before activity, raised to a maximum 60 mg only if needed and tolerated, not more than once every 24 hours, and never combined with a PDE5 inhibitor because of syncope risk.

11.2 Flibanserin: the daily bedtime pill for premenopausal HSDD

A daily serotonergic agent for acquired, generalised HSDD in premenopausal women. Flibanserin (brand Addyi) is a 5-HT1A agonist and 5-HT2A antagonist that, in the prefrontal cortex, lowers serotonin while raising noradrenaline and dopamine activity; it was originally trialled as an antidepressant, failed, and was repurposed for desire (Kaplan and Sadock CTP 2024; Goodman and Gilman). It is one of the two FDA-approved agents (with bremelanotide) for acquired, generalised hypoactive sexual desire disorder (HSDD) in **premenopausal** women only, and its licensed dose is **100 mg** once daily at bedtime (Addyi FDA label 2015). Unlike dapoxetine it is a maintenance, not an as-needed, drug: it is taken every night for weeks, and it is discontinued after 8 weeks if there is no improvement.

Modest effect, three named cautions. The efficacy is real but modest, a small rise in satisfying sexual events and desire with reduced desire-related distress (Tasman; New Oxford Textbook of Psychiatry). Its adverse-effect and interaction profile is where marks sit: hypotension, syncope, somnolence, dizziness, nausea and dry mouth (Maudsley Prescribing Guidelines). The bedtime timing is deliberate, limiting daytime hypotension and sedation. It carries a boxed warning for severe hypotension and syncope with alcohol and with strong CYP3A4 inhibitors, so alcohol co-use and CYP3A4 interactions are the two cautions to state, alongside its CNS-depressant, sedating profile. Reassuringly, it appears safe in women already taking antidepressants (Maudsley Prescribing Guidelines).

FEATURE	FLIBANSERIN (VERIFIED)
Mechanism	5-HT1A agonist, 5-HT2A antagonist (lowers cortical serotonin, raises noradrenaline and dopamine)
Indication	Acquired, generalised HSDD in premenopausal women only
Dose (mg)	100 once daily at bedtime
Rhythm	Daily maintenance; stop at 8 weeks if no benefit
Effect size	Modest
Signature cautions	Alcohol and strong CYP3A4 inhibitors (hypotension, syncope); CNS depression and sedation

Flibanserin at a glance: the daily bedtime pill for premenopausal, acquired, generalised HSDD, with its modest effect and the alcohol, CYP3A4 and CNS-depression cautions (Addyi FDA label 2015; Maudsley Prescribing Guidelines).

GOOD TO REMEMBER

- **Flibanserin (guideline-anchored, Addyi FDA label 2015):** a 5-HT1A agonist and 5-HT2A antagonist for acquired, generalised HSDD in premenopausal women, dosed 100 mg once daily at bedtime, of modest effect, stopped at 8 weeks if no benefit; the examinable cautions are alcohol and strong CYP3A4 inhibitors (hypotension and syncope) plus its sedating, CNS-depressant profile.

11.3 Reading the pair: on-demand versus daily, and the label restrictions

Two rhythms, two label boxes, two cautions. The pair is designed to be discriminated. Dapoxetine is *on-demand and short-acting*, swallowed before a single act of sex for premature ejaculation, its trap being combination with a PDE5 inhibitor. Flibanserin is *daily and maintenance*, taken every bedtime for premenopausal HSDD, its trap being alcohol. The restriction most often tested on flibanserin is the population box: it is licensed only for *acquired* (not lifelong), *generalised* (not situational), *premenopausal* HSDD, so offering it to a postmenopausal woman or for a situational, relationship-bound loss of desire is off-label and off-target.

AXIS	DAPOXETINE	FLIBANSERIN
Target	Premature ejaculation (men)	**Acquired, generalised HSDD (premenopausal women)**
Class	Short-acting SSRI	**5-HT1A agonist, 5-HT2A antagonist**
Rhythm	On-demand, 1 to 3 hours before	**Daily, at bedtime**
Dose (mg)	30 start, up to 60 max	**100 once daily**
Signature caution	Not with a PDE5 inhibitor (syncope)	**Not with alcohol or strong CYP3A4 inhibitors (hypotension, syncope)**
Review point	After 4 weeks or 6 doses	**Stop at 8 weeks if no benefit**

The discrimination table for the pair; the contrast in dosing rhythm and licensed population is the highest-yield line, and the two kinetic profiles could hardly differ more (ISSM 2014; NICE ESNM40; Addyi FDA label 2015).

30	60	100	8
DAPOXETINE START DOSE (MG)	DAPOXETINE CEILING DOSE (MG)	FLIBANSERIN NIGHTLY DOSE (MG)	FLIBANSERIN REVIEW POINT (WEEKS)

RULE **The rhythm names the drug and the caution.** On-demand short-acting SSRI before sex means dapoxetine for premature ejaculation, caution the PDE5 inhibitor; daily bedtime 5-HT1A agonist for premenopausal, acquired, generalised HSDD means flibanserin, caution the alcohol.

TRAP **Prescribing flibanserin as an on-demand libido pill, or ignoring its label box.** It is a daily maintenance drug, not a pre-sex tablet, and it is licensed only for acquired, generalised HSDD in premenopausal women; offering it to a postmenopausal woman, for situational loss of desire, or without the alcohol and CYP3A4 warning is the classic error.

COMMON TRAP

Pairing dapoxetine with a PDE5 inhibitor. It is tempting when a man reports both premature ejaculation and erectile difficulty, and Indian fixed-dose combinations make it easy, but the Priligy SPC advises against the combination because of syncope risk. Treat the dominant complaint first and keep the two apart unless a specialist judges otherwise.

HOLD THIS

Dapoxetine is the on-demand short-acting SSRI for premature ejaculation, 30 mg (up to 60) taken 1 to 3 hours before sex and never with a PDE5 inhibitor; flibanserin is the daily bedtime 5-HT1A agonist / 5-HT2A antagonist for acquired, generalised, premenopausal HSDD, 100 mg nightly, of modest effect and never with alcohol.

12 · Paraphilic disorders

This topic is examined less on the exotic list of behaviours and more on one clean conceptual line: the difference between a **paraphilia**, an atypical sexual interest that is not in itself a disorder, and a **paraphilic disorder**, which the current nosology reserves for a paraphilia that

either causes the person *distress* or impairment or entails *harm*, or the risk of it, to another, usually non-consenting, person. Get that threshold right and most of the marks follow; miss it and you pathologise a private, harmless preference or, worse, treat a criminal charge as if the label alone made a diagnosis. The class also carries a genuine forensic and risk-assessment dimension the psychiatrist is asked to comment on, and a graded, guideline-anchored management answer that candidates rarely rehearse.

How to use this page. First fix the paraphilia-against-disorder distinction and the shared criteria that gate every entity; then learn the eight named DSM-5-TR types and the sharp place where ICD-11 parts company; then the forensic frame, where the cardinal error is diagnosing from the offence rather than the sexual preference; and finally the **management of paraphilia**, psychological relapse-prevention first, with SSRIs and, for severe or high-risk cases, testosterone-lowering antiandrogen or GnRH-agonist treatment reserved for the top of the ladder. The combined map of the paraphilic and impulse-control classes is drawn in the accompanying figure at the impulse-control block; refer to it there. The tone throughout is clinical, nosological, and non-graphic.

12.1 Paraphilia against paraphilic disorder: the consent, distress and harm threshold

An interest is not a disorder until it crosses a threshold. DSM-5 and DSM-5-TR define a paraphilia as "any intense and persistent sexual interest other than sexual interest in genital stimulation or preparatory fondling with phenotypically normal, physically mature, consenting human partners" (Kaplan and Sadock CTP 2024; DSM-5-TR 2022). The pivotal 2013 move was to separate that interest from the paraphilic disorder, defined as a paraphilia that is *currently causing distress or impairment to the individual*, or *a paraphilia whose satisfaction has entailed personal harm, or the risk of harm, to others*. In other words a paraphilia is a disorder only when criterion B is met: the person is distressed or impaired, or the interest is acted on with, or poses a risk to, a non-consenting person. This preserves the line between non-normative and pathological sexual behaviour without branding every unusual preference a mental illness. Across every named entity the shared gates are the same: the pattern is present for at least six months, and either causes clinically significant distress or impairment, or the urges have been acted on with a non-consenting person.

RULE **Distress, impairment, or harm to a non-consenting other is what makes a paraphilia a disorder.** The atypical interest alone is not diagnosable; you need criterion B, distress or impairment to the person, or personal harm or the risk of it to another who has not consented. Consent, distress and harm are the three words that carry the diagnosis.

GOOD TO REMEMBER

- **Paraphilic disorder (the defining criterion):** an intense, persistent atypical sexual interest present for at least six months that *either* causes the individual clinically significant distress or impairment *or* is acted on with, or risks harm to, a non-consenting person; without that criterion B threshold it is a paraphilia, not a disorder.

12.2 The named types, and where ICD-11 parts from DSM-5-TR

Eight named disorders in DSM-5-TR, grouped three ways. DSM-5-TR names eight specific paraphilic disorders, organised into anomalous *activity* preferences (voyeuristic disorder, exhibitionistic disorder, frotteuristic disorder), *algolagnic* disorders of pain and suffering (sexual masochism disorder and sexual sadism disorder), and anomalous *target* preferences (paedophilic disorder, fetishistic disorder, transvestic disorder), with residual "other specified" and "unspecified" categories replacing the old NOS label (Kaplan and Sadock CTP 2024; DSM-5-TR 2022). The activity-preference and sadism disorders require that the urges be acted on with a non-consenting person, or that they cause marked distress or impairment; the target-preference disorders can qualify on distress or impairment alone. Two age anchors are examined: for paedophilic disorder the individual must be at least 16 years of age and at least five years older than the prepubescent child, generally 13 years of age or younger; for voyeuristic disorder the individual must be at least 18 years of age. Course specifiers, "in a controlled environment" and "in full remission", apply to all except paedophilic disorder.

ICD-11 draws the line by consent, and drops three DSM entities. The ICD-11 "Paraphilic disorders" grouping is built explicitly around whether the arousal focus is on non-consenting others or on solitary or consenting behaviour. It lists exhibitionistic (6D30), voyeuristic (6D31), paedophilic (6D32), coercive sexual sadism (6D33) and frotteuristic (6D34) disorders, plus a catch-all for other paraphilic disorder involving non-consenting individuals (6D35), and a separate residual for paraphilic disorder involving solitary behaviour or consenting individuals (6D36), diagnosed only where there is marked distress and not merely because the behaviour is socially disapproved (ICD-11 CDDR). Crucially, ICD-11 *removed* fetishistic disorder, fetishistic transvestism and sadomasochism as named disorders, on the reasoning that private, consensual variants are not, by themselves, mental disorders. That DSM-keeps / ICD-drops split for fetishism and transvestism is a favourite discriminator.

DISORDER	CORE AROUSAL FOCUS	AGE OR DURATION ANCHOR	THE ONE THING TO ATTACH
Exhibitionistic disorder	Exposing one's genitals to an unsuspecting person	At least six months	**One of the three commonest offences in police records (with voyeurism and paedophilia)**
Voyeuristic disorder	Observing an unsuspecting person naked or in sexual activity	Individual at least 18 years of age	**The only paraphilia with a stated minimum age for the diagnosis**
Frotteuristic disorder	Touching or rubbing against a non-consenting person	At least six months	**Typically in crowded settings; from French "frotter", to rub**
Paedophilic disorder	Prepubescent child, generally 13 years of age or younger	Individual at least 16 years and at least five years older	**No course specifiers; the forensic core of the class**

DISORDER	CORE AROUSAL FOCUS	AGE OR DURATION ANCHOR	THE ONE THING TO ATTACH
Sexual sadism disorder	Physical or psychological suffering of another	At least six months	**ICD-11 names only the coercive, non-consenting form (6D33)**
Sexual masochism disorder	Being humiliated, beaten, bound or made to suffer	At least six months	**Often benign; asphyxiophilia is the dangerous specifier**
Fetishistic disorder	Non-living objects or a specific non-genital body part	At least six months	**Kept in DSM-5-TR; removed from ICD-11**
Transvestic disorder	Sexual arousal from cross-dressing	At least six months	**Kept in DSM-5-TR; removed from ICD-11. Not the same as gender incongruence**

The eight DSM-5-TR paraphilic disorders and their discriminators; ICD-11 retains only the five non-consenting-focused disorders plus two residual codes and drops fetishistic, transvestic and sadomasochism as named entities (Kaplan and Sadock CTP 2024; DSM-5-TR 2022; ICD-11 CDDR).

PEARL

Read the "in a controlled environment" and "in full remission" specifiers carefully. The absence of paraphilic behaviour in a prison or secure unit, where opportunity is removed, is *not* remission; it is simply a controlled environment. Full remission requires no acting on the urges with a non-consenting person and no distress or impairment for at least five years while in an *un*controlled setting, which is why a quiet custodial record cannot, on its own, be read as recovery.

GOOD TO REMEMBER

- **Exhibitionistic disorder (the criterion):** recurrent, intense arousal from exposing the genitals to an unsuspecting person for at least six months, acted on with a non-consenting person or causing distress or impairment; exhibitionism is one of the three commonest offences in police records.
- **Voyeuristic disorder (the discriminator):** arousal from observing an unsuspecting naked or sexually active person; the individual must be at least 18 years of age, the only paraphilia with a stated minimum diagnostic age, distinguishing it from adolescent curiosity.
- **Frotteuristic disorder (the criterion):** arousal from touching or rubbing against a non-consenting person, usually in a crowd, acted on or causing distress or impairment.
- **Paedophilic disorder (the criterion):** recurrent, intense arousal to a prepubescent child, generally 13 years of age or younger, with the individual at least 16 years of age and at least five years older than the child; it uniquely carries no course specifiers.
- **Fetishistic and transvestic disorder (the divergence):** both remain named DSM-5-TR disorders (objects or non-genital body parts; cross-dressing), but ICD-11 has removed them, treating private, consensual variants as non-disordered.

12.3 The forensic and risk-assessment dimension

A paraphilic disorder is a diagnosis; a sex offender is a legal category, and the two are not interchangeable. The term "sex offender" describes a person legally convicted of a sexual offence and says nothing about the presence or absence of a paraphilia; conversely a person with a paraphilia may never offend, obtaining gratification through fantasy alone (Kaplan and Sadock

CTP 2024). The diagnosis rests on the individual's *sexual preference*, established through a comprehensive psychosexual evaluation, collateral history and, where used, objective measures, penile plethysmography (phallometry), visual reaction time and, in mandated settings, the sexual-history polygraph, since self-report is unreliable and there is no definitive test. Crossover between paraphilias is common, so a single presentation should prompt a review for the others. Risk is best estimated with structured actuarial instruments such as the Static-99R, which the evidence shows predict relative recidivism risk more accurately than unstructured clinical judgement, and clinicians must also weight the dynamic risk factors, deviant sexual interest, sexual preoccupation, poor self-regulation, intimacy deficits and offence-supportive attitudes.

TRAP **Diagnosing the paraphilia from the offence, or equating "sex offender" with a paraphilic disorder.** A man who has molested a child may have paedophilic disorder, but the same act can arise from intellectual disability, a manic episode, dementia or intoxication; you diagnose from the sustained pattern of sexual *preference*, not from the behaviour or the conviction alone.

WORKED EXAMPLE

A man is referred after exposing himself; he is distressed, describes a decade of recurrent urges to expose to unsuspecting strangers, and has acted on them repeatedly. That sustained pattern, with acting on the urges toward non-consenting others, supports exhibitionistic disorder. Contrast a first-ever episode of public undressing during a florid manic episode, with no prior deviant arousal: here the behaviour is a symptom of the mood disorder, and a paraphilic diagnosis would be wrong. Screen him for the other paraphilias, because crossover is the rule, and estimate risk with a structured actuarial tool rather than a gut impression.

12.4 Management principles: the graded, psychological-first ladder

The management of paraphilia is graded to severity and risk, and begins with psychological work. There is no evidence any paraphilic disorder can be "cured"; the realistic goal is control of behaviour and reduction of risk (New Oxford Textbook of Psychiatry). The World Federation of Societies of Biological Psychiatry (WFSBP) algorithm assigns treatment by severity: the less severe are managed with psychotherapy alone, while more severe or higher-risk presentations add pharmacotherapy on top (Kaplan and Sadock CTP 2024). The empirically supported psychological approach is structured cognitive-behavioural treatment, historically framed as relapse prevention, targeting the cognitive distortions (minimisations, justifications) and the offence cycle, and increasingly delivered through strength-based rehabilitation frameworks, the Good Lives Model and the Risk-Need-Responsivity model. Meta-analysis finds cognitive-behavioural treatment reduces reoffending more than didactic or purely psychodynamic work, and is comparable to antiandrogen treatment in effect; a non-judgemental, empathic therapist improves outcomes regardless of the programme's orientation.

MNEMONIC

PSYCHOTHERAPY → SSRI → ANTIANDROGEN → GnRH AGONIST

The WFSBP severity ladder for the management of paraphilia: cognitive-behavioural relapse-prevention for the less severe, an SSRI added as the first drug step, then a testosterone-lowering antiandrogen, and a long-acting GnRH agonist at the top for severe, treatment-refractory or high-risk cases. You climb only as severity and risk demand.

GOOD TO REMEMBER

- **Psychological management (guideline-anchored, WFSBP):** structured cognitive-behavioural relapse-prevention is first-line for less severe paraphilic disorders and the base of every plan; it targets cognitive distortions and the offence cycle, is comparable to antiandrogens in effect, and is now embedded in strength-based frameworks such as the Good Lives Model.

12.5 Pharmacological options: SSRIs, antiandrogens and GnRH agonists

Drugs work by dampening serotonergically driven urges or by lowering testosterone, and they escalate with severity. When medication is indicated, the first step is an SSRI, on the OCD analogy that enhanced central serotonergic tone reduces paraphilic arousal; it suits milder cases and those with prominent obsessive, compulsive urges, though the controlled evidence is limited (Kaplan and Sadock CTP 2024). For more severe or higher-risk cases the strategy is to lower testosterone. The antiandrogens cyproterone acetate and medroxyprogesterone acetate are synthetic progestogens that reduce serum testosterone and thereby libido, erections and ejaculation; cyproterone acetate is used at **50 to 100 mg** per day and is licensed for male hypersexuality in some countries at 50 mg twice daily (Maudsley Prescribing Guidelines; Goodman and Gilman). At the top of the ladder, the long-acting GnRH agonists leuprolide and triptorelin produce a reversible chemical castration by exhausting the hypothalamic-pituitary axis; leuprolide is given as **7.5 mg** intramuscularly every month and triptorelin as 11.25 mg intramuscularly every 12 weeks, and they are generally better tolerated than the older antiandrogens (Maudsley Prescribing Guidelines; Kaplan and Sadock CTP 2024). Surgical castration is now nearly obsolete. All testosterone-lowering agents demand baseline and follow-up monitoring, testosterone, liver function, glucose and bone mineral density, given risks of osteopenia, weight gain, hyperglycaemia, gynaecomastia and, for cyproterone, meningioma.

STEP	AGENT	TYPICAL DOSE AND ROUTE	KEY POINT AND MONITORING
First drug	SSRI (e.g. sertraline, fluoxetine)	Standard antidepressant range, oral daily	**For milder or obsessive-type urges; evidence limited but low-risk**
Antiandrogen	Cyproterone acetate	50 to 100 mg oral, daily	**Lowers testosterone; watch LFTs, meningioma risk, feminisation**
Antiandrogen	Medroxyprogesterone acetate	Oral or intramuscular depot	**Progestogenic androgen-lowering; weight gain, glucose, thromboembolism**
GnRH agonist	Leuprolide	7.5 mg IM, monthly	**Chemical castration for severe or refractory cases; monitor bone density**
GnRH agonist	Triptorelin	11.25 mg IM, every 12 weeks	**Depot GnRH agonist; better tolerated than antiandrogens**

The pharmacological ladder for paraphilic disorders, escalated by severity and risk; doses are the verified Maudsley and Goodman and Gilman figures, and every testosterone-lowering agent needs testosterone, LFT, glucose and bone-density monitoring (Maudsley Prescribing Guidelines; Kaplan and Sadock CTP 2024; see gaps for paraphilia-specific dosing).

INDIA

The forensic frame is the genuine Indian anchor. Child sexual offences fall under the POCSO Act (Protection of Children from Sexual Offences Act, 2012), which also imposes a mandatory-reporting duty on the clinician, the practical face of the paedophilic-disorder risk assessment. Indian criminal law separately makes voyeurism a distinct statutory offence (introduced by the Criminal Law Amendment Act, 2013, and carried into the Bharatiya Nyaya Sanhita, 2023). India has *no* chemical-castration sentencing statute, and the Justice Verma Committee (2013) advised against castration as punishment, so testosterone-lowering treatment stays a consented clinical decision, not a court penalty. On availability, leuprolide is marketed in India as Lupride Depot and triptorelin as Decapeptyl; cyproterone acetate is available as an antiandrogen (confirm the exact standalone product and strength locally, see gaps). Keep the generic primary.

GOOD TO REMEMBER

- **Pharmacological management (guideline-anchored, WFSBP and Maudsley):** an SSRI is the first drug step for milder or obsessive-type urges; for severe or high-risk cases lower testosterone with an antiandrogen (cyproterone acetate at 50 to 100 mg per day) and, at the top, a long-acting GnRH agonist (leuprolide 7.5 mg IM monthly, triptorelin every 12 weeks), all with testosterone, LFT, glucose and bone-density monitoring; surgical castration is obsolete.

8	6	18	50 to 100
DSM-5-TR NAMED PARAPHILIC DISORDERS	MONTHS MINIMUM SYMPTOM DURATION	VOYEURISTIC DISORDER MINIMUM AGE (YEARS)	CYPROTERONE ACETATE DOSE (MG PER DAY)

HOLD THIS

A paraphilia becomes a paraphilic *disorder* only when it causes the person distress or impairment, or entails harm or the risk of harm to a non-consenting other, the consent-distress-harm threshold; you diagnose from the sexual preference, not the offence, and the management of paraphilia climbs from cognitive-behavioural relapse-prevention to SSRIs to testosterone-lowering antiandrogen or GnRH-agonist treatment for the severe and high-risk.

13 · Gender incongruence and gender-affirmative care

This is the topic where nosology and human rights meet, and the examiner wants to see that you can hold both. The single biggest fact is a reclassification: the eleventh revision of the international classification moved **gender incongruence** out of the mental disorders chapter altogether, so the modern, guideline-anchored position is that being transgender is *not* a mental illness. The management answer is not a drug at all but a stance, the gender-affirmative approach codified in the WPATH Standards of Care, and a clear sense of what the psychiatrist does and does not do within it. High-yield marks cluster on four things: the terminology (and the incongruence-versus-dysphoria distinction), the ICD-11 **depathologisation** contrasted with the DSM retention of a diagnosis, the affirmative-care pathway, and the Indian legal frame.

How to use this page. First fix the vocabulary, because half of the errors on this topic are vocabulary errors; then learn the two competing constructs, gender incongruence (the mismatch, no distress required) and gender dysphoria (the mismatch plus clinically significant distress); then hold the two classifications side by side, ICD-11 having depathologised where DSM-5-TR retains a diagnosis; then anchor the affirmative-care pathway and the mental-health professional's bounded role; and finally the Indian statute. The one error this page is built to prevent is treating the identity itself as the pathology, when the guideline-anchored target of care is the distress, the comorbidity and the stigma, never the person's gender.

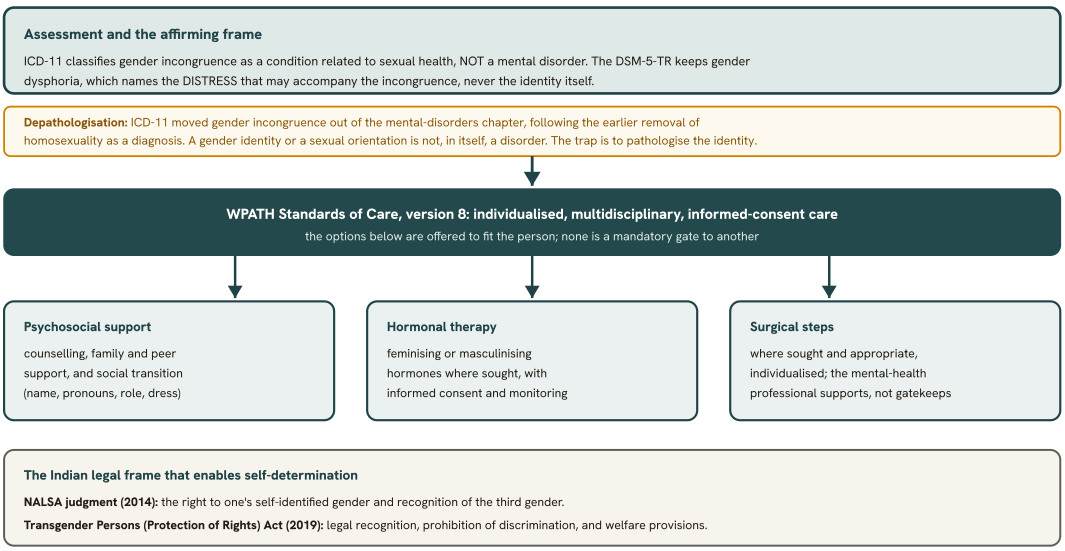

Fig 18.8 The gender-affirmative-care pathway. Care begins from an affirming frame: ICD-11 treats gender incongruence as a condition related to sexual health and not a mental disorder, having moved it out of the mental-disorders chapter just as homosexuality was removed as a diagnosis decades earlier, while the DSM-5-TR retains gender dysphoria to name the distress that may accompany incongruence rather than the identity itself. The WPATH Standards of Care, version 8, then set out an individualised, multidisciplinary, informed-consent model in which psychosocial support and social transition, hormonal therapy, and surgical steps are offered to fit the person, none a mandatory gate to another, and the mental-health professional supports rather than gatekeeps. In India this is underpinned by the NALSA judgment of 2014 and the Transgender Persons (Protection of Rights) Act of 2019.

13.1 The terminology: sex, gender identity and the umbrella terms

Separate sex, gender identity, gender expression and sexual orientation before anything else. The words are examined precisely, and conflating any two of them is the classic slip (Kaplan and Sadock CTP 2024). *Sex* refers to biological variables (genes, chromosomes, gonads, internal and external genital structures) that are male-typical or female-typical; sex assigned at birth is the sex recorded for an individual in infancy. *Gender* is the social category and role. Gender identity, a term coined by Robert Stoller in the mid-twentieth century, is a person's persistent inner sense of their own gender, which today is recognised as possibly male, female, both, in between or neither. Critically, gender identity says nothing about sexual orientation: whether a person experiences their gender as man or woman does not predict whom they are attracted to. Keep these four axes independent and most of the traps disappear.

Sex assigned at birth

The sex (male or female) recorded for a person at birth on the basis of observed anatomy; the reference point against which incongruence is later described.

Gender identity

A person's persistent internal sense of their own gender (Stoller); may be male, female, non-binary or another category, and is independent of sexual orientation.

Gender expression / gender role

The outward presentation of gender (dress, mannerisms, social role); the visible counterpart of the inner identity.

Transgender

Umbrella term (coined by Virginia Prince, early 1970s) for a person whose gender identity or expression differs from the sex assigned at birth; it is an identity, not a diagnosis.

Cisgender

A person whose gender identity aligns with the sex assigned at birth.

RULE **Identity is not orientation, and neither is a diagnosis.** Gender identity (who I am), gender expression (how I present), sex assigned at birth, and sexual orientation (whom I am drawn to) are four independent axes; being transgender or cisgender tells you nothing about a person's sexual orientation, and neither is, in itself, a disorder.

13.2 Gender incongruence versus gender dysphoria: where distress enters

Incongruence is the mismatch; dysphoria is the mismatch plus clinically significant distress. This one distinction earns marks repeatedly. **Gender incongruence** is a marked and persistent mismatch between a person's experienced or expressed gender and the sex assigned at birth. It is a description, not a pathology, and by itself it requires no distress. **Gender dysphoria** is the term the DSM uses for the aversive distress that *some* people with that incongruence experience; it is defined by the presence of clinically significant distress or impairment, and that distress, not the identity, is what a clinician treats (Kaplan and Sadock CTP 2024; DSM-5-TR 2022; ICD-11). The move from "gender identity disorder" to "gender dysphoria" in DSM-5 was deliberately made to strip the word "disorder" from the label and to shift clinical focus from identity to distress. Much of that distress is now understood through *gender minority stress*, the chronic strain of stigma, misgendering and discrimination, which is linked to elevated rates of depression, anxiety and suicidality; affirmation and the reduction of stigma are correspondingly protective. The population-level and public-health dimension of that suicide risk is developed in the Community psychiatry and public health notes; here the point is simply that distress is contingent and modifiable, not intrinsic to being transgender.

PEARL

A memorable way to hold the pair: **incongruence** is a noun of fact (the two do not match), **dysphoria** is a noun of feeling (and it hurts). You can have the first without the second, and a large proportion of transgender people do. The clinician's business is the second.

13.3 The ICD-11 depathologisation: out of the mental disorders chapter

ICD-11 renamed transsexualism as gender incongruence and moved it out of mental disorders entirely. This is the headline reform. In the eleventh revision (ICD-11), the WHO replaced "transsexualism" with gender incongruence and, for the first time, removed the diagnosis from the Mental and Behavioural Disorders chapter, relocating it to a new chapter, "Conditions related to sexual health" (ICD-11; New Oxford Textbook of Psychiatry). This is the formal **depathologisation** of gender diversity: the reclassification keeps a diagnostic code, so that people can still access publicly funded and insured gender-affirming care, while explicitly stating

that gender incongruence is *not* a mental disorder. The adolescent-and-adult category (gender incongruence of adolescence and adulthood, code HA60) requires a marked and persistent incongruence between experienced gender and assigned sex, shown by at least two of the four indicators (discomfort with one's sex characteristics, a desire to be rid of them, a desire for those of the experienced gender, and a desire to be treated as the experienced gender), continuously present for at least several months, and it cannot be assigned before the onset of puberty. Notably, unlike the DSM, ICD-11 requires *no* distress or dysfunction criterion at all.

GOOD TO REMEMBER

- **Gender incongruence, ICD-11 (the criterion anchor):** a marked, persistent mismatch between experienced gender and sex assigned at birth, at least two of the four indicators, continuously present for at least several months, not assignable before puberty; there is NO distress criterion. It sits in "Conditions related to sexual health," not the mental disorders chapter, so it is explicitly not a mental disorder.

13.4 The DSM-5-TR retention of gender dysphoria, and the contrast

DSM-5-TR keeps a diagnosis, gender dysphoria, gated by distress; ICD-11 does not. DSM-5-TR (DSM-5-TR) renamed gender identity disorder as gender dysphoria but, unlike ICD-11, retains it as a diagnosis within the manual. The adolescent-and-adult diagnosis requires at least two of the six criteria (a marked incongruence between experienced gender and one's sex characteristics, a strong desire to be rid of those characteristics, a desire for those of the other gender, a desire to be the other or an alternative gender, a desire to be treated as such, and a conviction of having the typical feelings of that gender), present for at least approximately six months, plus a mandatory Criterion B of clinically significant distress or impairment (DSM-5-TR 2022). There are four DSM diagnoses in the family (gender dysphoria in adolescents and adults, in children, other specified, and unspecified), with a "with a disorder of sex development" specifier and a "post-transition" specifier, the latter letting a person retain the diagnosis needed to access ongoing hormones after transition. The examiner's favourite contrast is captured below.

FEATURE	ICD-11 GENDER INCONGRUENCE	DSM-5-TR GENDER DYSPHORIA
Chapter placement	Conditions related to sexual health (moved OUT of mental disorders)	**Retained as a diagnosis within the manual**
Former name	Transsexualism	**Gender identity disorder**
Distress required?	No distress or dysfunction criterion	**Yes, Criterion B distress or impairment is mandatory**
Adult criteria threshold	At least two of the four indicators, several months	**At least two of the six, at least approximately six months**
Core stance	Explicit depathologisation of gender diversity	**Destigmatised label, but still a diagnosis**

The single highest-yield table on this topic: ICD-11 depathologises by relocation and needs no distress; DSM-5-TR destigmatises the label but keeps a distress-gated diagnosis (ICD-11; DSM-5-TR 2022; Kaplan and Sadock CTP 2024).

GOOD TO REMEMBER

- **Gender dysphoria, DSM-5-TR (the criterion anchor):** at least two of the six criteria for at least approximately six months, PLUS Criterion B clinically significant distress or impairment; formerly gender identity disorder, retained as a diagnosis (four sub-types) with "with a DSD" and "post-transition" specifiers. The distress is the gate; the identity is not.

13.5 The historical precedent: the depathologisation of homosexuality

Gender incongruence is following the road homosexuality already travelled: out of the manuals. The reclassification of gender incongruence is best understood as the second act of a story whose first act was the removal of homosexuality from psychiatric classification. The American Psychiatric Association removed homosexuality as a disorder from its diagnostic manual in **1973** (a residual "ego-dystonic homosexuality" category persisted for a time and was later dropped), and the WHO's ICD followed, with ICD-11 finally clearing all such orientation categories out (Kaplan and Sadock CTP 2024). The enduring principle is the one to quote in a viva: a sexual *orientation* is a normal human variation, not a disorder. Historically, gender-diverse presentations were conflated with homosexuality and both were pathologised; the modern separation of gender identity from sexual orientation, and the depathologisation of each in turn, is the same corrective applied twice. Framing gender incongruence this way signals to the examiner that you grasp the trajectory of the whole field, not just one diagnostic row.

MNEMONIC

ORIENTATION IS NOT A DISORDER

The line to carry from the homosexuality precedent into the gender discussion: what a person *is* (their orientation, their gender identity) is not the illness. Distress, comorbidity and the effects of stigma may need care; the identity does not. The two depathologisations, homosexuality first and gender incongruence second, rest on this single principle.

13.6 The WPATH SOC-8 gender-affirmative care pathway

The affirmative-care standard is individualised, multidisciplinary and informed-consent based, with a flexible staged transition. The reference framework for management is the World Professional Association for Transgender Health Standards of Care, eighth version (WPATH SOC-8 2022), which adopts the ICD-11 gender incongruence diagnosis and frames gender-affirmative care as individualised, multidisciplinary and grounded in informed consent, deliberately moving away from mental-health assessment as a universal gatekeeping hurdle while still recommending mental-health support where it is needed (WPATH SOC-8 2022; Kaplan and Sadock CTP 2024). The pathway is staged and flexible, not a rigid ladder: fully reversible steps (living as, and being socially recognised as, one's experienced gender, a *social transition*) usually precede partially reversible steps (gender-affirming hormone therapy) which precede irreversible surgical steps. Importantly, version 8 removed the older requirement for a minimum period of living in the experienced gender role before hormones, and it individualises timing rather than fixing minimum ages. For genital surgery, SOC-8 suggests at least **12 months** of continuous gender-affirming hormone therapy (unless contraindicated) and **two** referral letters documenting

persistent gender incongruence and the capacity to give fully informed consent; for adolescents seeking puberty suppression, one criterion is that the young person has reached Tanner stage 2. The full staged route, from social transition through the hormonal and surgical options with the mental-health professional sitting alongside rather than as a gate, is drawn in the accompanying figure. Not every person wants or reaches every step, and that is by design.

TRAP **Pathologising gender incongruence instead of affirming it.** The examiner's cardinal error here is to treat the gender identity as the disorder and reach for something that tries to change it (so-called conversion or reparative efforts, which are ineffective, harmful and legislatively banned in many places). The affirmative stance is the standard of care: affirm the identity, and treat only the distress, the comorbidity and the effects of stigma.

8	12	2	2014
THE CURRENT WPATH STANDARDS OF CARE VERSION (SOC-8, 2022)	MONTHS OF CONTINUOUS GENDER-AFFIRMING HORMONE THERAPY WPATH SUGGESTS BEFORE GENITAL SURGERY	REFERRAL LETTERS WPATH SOC-8 ASKS FOR BEFORE GENITAL SURGERY	THE NALSA JUDGMENT YEAR, AFFIRMING SELF-IDENTIFIED GENDER IN INDIA

GOOD TO REMEMBER

- **WPATH SOC-8 pathway (the guideline anchor):** individualised, multidisciplinary, informed-consent affirmative care; a flexible staged transition running fully reversible (social) then partially reversible (hormones) then irreversible (surgery); version 8 dropped the minimum "real-life" duration before hormones; genital surgery is preceded by about 12 months of continuous gender-affirming hormones and two referral letters documenting persistent incongruence and capacity to consent.

13.7 The role of the mental-health professional (and the endocrine overview)

The psychiatrist assesses readiness and capacity, treats comorbidity and supports; the psychiatrist does not initiate hormones. Within affirmative care, the mental-health professional's role is bounded and specific: assessment of the person's readiness and capacity to give informed consent, accurate diagnosis and treatment of any co-occurring mental illness (which does not by itself preclude transition but should be stabilised so it will not compromise a safe transition plan), psychoeducation, and psychosocial support (WPATH SOC-8 2022; Kaplan and Sadock CTP 2024). Hormone therapy can be initiated on a referral from a qualified mental-health professional or another suitably trained health professional able to assess incongruence, eligibility and readiness, but the prescribing itself is endocrinology-led. On the endocrine overview only (prescribing detail is out of scope and belongs to the endocrinologist): feminising therapy combines an oestrogen with an anti-androgen, and masculinising therapy uses testosterone, with the Endocrine Society Clinical Practice Guideline (2017) as the standard specialist, monitored reference (Endocrine Society 2017). The psychiatrist may co-monitor mood and adherence during hormone use, but does not start or titrate the regimen.

GOOD TO REMEMBER

- **Mental-health professional's role (the guideline anchor):** assess readiness and capacity for informed consent, diagnose and treat comorbid mental illness (stabilise, do not exclude), give psychoeducation and psychosocial support, and refer; the psychiatrist does NOT initiate or titrate gender-affirming hormones, which are endocrinology-led (feminising = oestrogen plus anti-androgen; masculinising = testosterone; Endocrine Society 2017).

13.8 The Indian legal frame: NALSA and the Transgender Persons Act

India recognises the self-identified gender and a third gender in law. The Indian frame is genuinely examinable and must be verified rather than paraphrased loosely. In the NALSA judgment (National Legal Services Authority v Union of India, Supreme Court, 15 April 2014), the court recognised transgender persons as a "third gender" and affirmed the constitutional right to self-identified gender. This was followed by statute: the Transgender Persons (Protection of Rights) Act, 2019 (the Transgender Persons Act) provides the framework for legal recognition of a transgender identity, prohibits discrimination in areas such as employment, education and healthcare, and sets out welfare provisions (NALSA 2014; Transgender Persons (Protection of Rights) Act 2019). India also has long-standing traditional third-gender communities, the hijra being the most widely known, which the two-spirit and hijra examples in the standard texts cite as evidence that a strict male/female binary is not culturally universal. For an Indian candidate, pairing the affirmative-care clinical stance with the NALSA judgment and the 2019 Act is the complete, high-scoring answer.

INDIA

Weave the legal frame into any Indian answer on this topic. The clinical stance (affirm, do not pathologise) and the legal stance now point the same way: the 2014 **NALSA judgment** gave transgender persons the right to self-identified gender and third-gender recognition, and the 2019 Transgender Persons (Protection of Rights) Act turned that into anti-discrimination and welfare law. Culturally, the hijra community is India's own long-standing third-gender tradition. The psychiatrist works within this frame, offering affirmative, non-judgemental care and appropriate referral, not gatekeeping and never conversion approaches.

GOOD TO REMEMBER

- **Indian legal frame (the statute anchor):** the NALSA judgment (Supreme Court, 15 April 2014) recognised a third gender and the right to self-identified gender; the Transgender Persons (Protection of Rights) Act, 2019 gives legal recognition, prohibits discrimination and provides welfare measures. Both are verified; quote them by name and year.

HOLD THIS

Gender incongruence is the mismatch between experienced gender and sex assigned at birth and is NOT a mental disorder (ICD-11 moved it to "Conditions related to sexual health," needing no distress); gender dysphoria is the DSM-5-TR term for the distress that only some experience; management is the WPATH SOC-8 individualised, informed-consent, gender-affirmative pathway (social, then hormonal, then surgical steps, hormones endocrinology-led) with the psychiatrist treating distress and comorbidity, not the identity, all within the Indian frame of the NALSA judgment (2014) and the Transgender Persons (Protection of Rights) Act, 2019.

14 · Dhat syndrome and culture-bound sexual presentations

This topic is the Indian examiner's favourite in the sexual-and-somatic band, and it is tested as a discrimination-and-management task rather than a memory dump. **dhat syndrome** is the presentation in which a young man attributes fatigue, weakness, anxiety and a range of somatic and sexual symptoms to the loss of semen, and the marks turn on recognising it as a **culture-bound** presentation, placing it correctly in the nosology, reading it through a cultural formulation, and managing the semen-loss belief without confronting it. It is the most examinable member of a small family of culturally shaped sexual presentations that examiners like to pair.

How to use this page. Fix the cardinal feature and the semen-loss attribution first; then the Ayurvedic cultural roots and the prevalence and comorbidity in the Indian and South Asian setting; then the nosological status as a cultural concept of distress and the cultural formulation the whole topic turns on; then the related **culture-bound sexual presentations**, koro chief among them; and finally the non-confrontational psychoeducational management. The complaint usually crystallises around **semen-loss anxiety** tied to masturbation and nocturnal emission, and the single error this page is built to prevent is reading a Dhat presentation without the cultural formulation.

14.1 Dhat syndrome: the semen-loss attribution

A cluster of symptoms pinned on the loss of semen. dhat syndrome is a term coined in South Asia about half a century ago to account for young men who attribute diverse symptoms to the loss of semen (DSM-5-TR 2022). Despite the name it is not, in the modern reading, a discrete disease but a culturally shaped presentation: the patient reports anxiety, fatigue, weakness, weight loss, poor concentration, multiple somatic complaints, low mood and sexual difficulty such as erectile dysfunction or premature ejaculation, and attributes them all to the passage of *dhat*, a whitish discharge he notices in urine or after defecation and believes to be semen. The cardinal feature is the anxiety and distress about the perceived loss of semen in the absence of any identifiable physiological dysfunction. The belief commonly carries marked guilt and dysphoria, and it typically fastens onto masturbation and nocturnal emission as the feared sources of loss (Companion to Psychiatric Studies).

RULE **The attribution is the disorder, not the discharge.** What defines Dhat syndrome is the distressing belief that semen is being lost and is causing the symptoms; the whitish urinary discharge is usually normal, so the target of assessment and treatment is the semen-loss belief, not a genitourinary lesion.

GOOD TO REMEMBER

- **Dhat syndrome (the presentation):** diverse somatic, anxious, depressive and sexual symptoms that the patient attributes to the loss of semen (dhat), a whitish discharge in urine or after defecation that he believes to be semen; the defining feature is the distressing preoccupation with semen loss in the absence of an identifiable physiological cause, not any single somatic symptom.

14.2 The cultural roots and the Indian and South Asian context

An Ayurvedic belief that semen is a precious, finite essence. The attribution rests on an old and widely held sociocultural belief: semen is regarded as an extremely precious bodily substance, distilled from many drops of blood, so its loss is felt to drain vitality and to cause weakness and sexual failure. The idea connects to dhatu, the word for semen in Ayurveda, counted as one of seven essential bodily fluids whose balance is held to maintain health (DSM-5-TR 2022). Because the belief is culturally normative rather than idiosyncratic, Dhat presentations are common wherever it is shared. The figures from the **indian context** and the wider South Asian setting are striking: **64 percent** of men attending psychiatric clinics in India for sexual complaints, and **30 percent** of men attending general medical clinics in Pakistan, have been described with the concept (DSM-5-TR 2022). It is most often seen in young men from lower socioeconomic backgrounds, though middle-aged men are also affected, and a parallel concern about white vaginal discharge (the leukorrhea variant) forms the female counterpart. Depression and anxiety are frequent companions, and the classic teaching is that a Dhat presentation should prompt an active search for a comorbid depressive or anxiety disorder rather than being taken as a stand-alone complaint.

Dhat
the whitish urinary or post-defecation discharge the patient believes to be lost semen

Dhatu
semen in Ayurveda, counted among the seven essential bodily fluids whose balance is held to maintain health

Leukorrhea variant
the parallel female concept that attributes symptoms to the loss of white vaginal discharge

64%	30%	7
DHAT CONCEPT IN INDIAN PSYCHIATRIC SEXUAL-COMPLAINT ATTENDERS	DHAT CONCEPT IN PAKISTANI GENERAL-MEDICAL ATTENDERS	DHATU, THE ESSENTIAL BODILY FLUIDS OF AYURVEDA

INDIA

Dhat is one of the commonest ways sexual and general distress reaches the Indian clinic, and it sits at the meeting point of the somatic symptom disorders, the sexual dysfunctions and depression. The examiner's expectation is that you name it as a culture-bound presentation, read it through the cultural formulation, search for and treat the comorbid depression, anxiety or sexual dysfunction, and address the semen-loss belief with psychoeducation rather than dismissal. Its wider transcultural frame, alongside the other culture-bound presentations, is developed in the Consultation-liaison, geriatric and transcultural psychiatry notes.

14.3 A cultural concept of distress, not a discrete disorder: the cultural formulation

A cultural explanation of distress, held in the DSM-5-TR glossary rather than the disorder list. DSM-5-TR does not classify Dhat as a discrete disorder; it lists it among the cultural concepts of distress in the Section III chapter on culture and psychiatric diagnosis, and specifically as a cultural causal explanation, the semen loss being the culturally recognised cause to which a range of somatic and psychological symptoms are attributed (DSM-5-TR 2022; Clinical Methods in Psychiatry, AIIMS 2024). ICD-10 placed it, with koro and the other geographically localised presentations, among the other specified neurotic disorders as a culture-specific or culture-bound syndrome (ICD-10). The practical consequence is the same in both systems: the presenting symptoms map onto ordinary diagnoses, a somatic symptom disorder, an illness anxiety disorder, a depressive or anxiety disorder, or a sexual dysfunction, while Dhat names the cultural idiom through which they are expressed. This is why the assessment must run through a cultural formulation. The Cultural Formulation Interview, the person-centred protocol in the same DSM-5 chapter, elicits the patient's own definition of the problem, its perceived cause and context, and his coping and help-seeking, so that the semen-loss explanatory model is understood rather than overwritten (DSM-5-TR 2022).

TRAP **Reading a Dhat presentation without the cultural formulation.** Taken at surface value the semen-loss belief can be mislabelled as a delusion, or the discharge chased as an organic genitourinary disease; the cultural formulation reframes it as a shared explanatory model and redirects assessment to the comorbid depression, anxiety or sexual dysfunction beneath it.

GOOD TO REMEMBER

- **Dhat as a cultural concept of distress (the nosological status):** DSM-5-TR holds Dhat in the Section III glossary of cultural concepts of distress as a cultural causal explanation (semen loss as the perceived cause), not as a codeable disorder; ICD-10 placed it among the other specified neurotic disorders as a culture-bound syndrome. The first step is a cultural formulation, after which the presenting symptoms are coded as the ordinary depressive, anxiety, somatic-symptom or sexual diagnosis they represent.

14.4 Related culture-bound sexual presentations: koro

Genital retraction and the fear of death. The related **culture-bound sexual presentations** are worth naming because examiners pair them with dhat syndrome, and koro is the classic partner (DSM-5-TR 2022). Koro is an episode of sudden, intense anxiety that the penis, or in the female form the nipples, breasts or vulva, is shrinking or retracting into the body, accompanied by a

belief that this will prove fatal. It is reported across Asia including India, and notably in Southeast Asia, where it can spread rapidly through a community in epidemic form, often built on the same culturally elaborated fears about nocturnal emission and masturbation. The Chinese counterpart, shen-k'uei or kidney deficiency, attributes weakness and sexual symptoms to semen loss much as Dhat does. The discriminator to hold is the object of the fear: koro fixes on genital retraction and imminent death, Dhat on the draining loss of a vital fluid, and both differ from body dysmorphic disorder, whose preoccupation is with perceived ugliness rather than shrinkage or depletion (body dysmorphic disorder is developed on the obsessive-compulsive spectrum in the Anxiety, OCD and trauma-related disorders notes).

PRESENTATION	CORE ATTRIBUTION OR FEAR	THE DISCRIMINATOR
Dhat syndrome	Symptoms attributed to loss of semen (dhat) in urine	**Fear of depletion of a vital fluid; South Asian; comorbid depression and anxiety common**
Koro	Penis, or nipples, breasts and vulva, retracting into the body	**Fear of genital retraction and death; can occur in epidemics**
Shen-k'uei (kidney deficiency)	Weakness and sexual symptoms attributed to semen loss	**Chinese counterpart of the semen-loss concept**
Body dysmorphic disorder	Preoccupation with a perceived defect in appearance	**Fear of ugliness, not depletion or retraction; obsessive-compulsive spectrum**

The culture-bound sexual presentations separated by the object of the fear; body dysmorphic disorder is set beside them as the appearance-based contrast (DSM-5-TR 2022).

PEARL

Separate the three by the *object of the fear*: **Dhat** fears the *loss* of semen (depletion of a vital fluid), **koro** fears the *retraction* of the genitals into the body and death, and **body dysmorphic disorder** fears the *appearance* of the body (perceived ugliness). Loss, retraction, ugliness.

GOOD TO REMEMBER

- **Koro (the presentation):** sudden intense anxiety that the genitals (the penis, or the nipples, breasts or vulva in women) are retracting into the body, with a belief that death will follow; it can occur in epidemics. The discriminator is the fear of genital retraction and death, which separates it from body dysmorphic disorder (perceived ugliness) and from Dhat syndrome (perceived semen loss).

14.5 Non-confrontational psychoeducational management

Correct the myth, treat the comorbidity, never confront. Management of Dhat syndrome is non-pharmacological and non-confrontational first. The core is counselling and psychoeducation directed at gently correcting the misconception about semen loss: explain, in the patient's own explanatory language, that the whitish discharge is not a dangerous drain of vitality, that occasional masturbation and nocturnal emission are normal and harmless, and pair this with straightforward sex education to allay the specific fears. Cognitive behavioural techniques fold easily into this model, addressing the catastrophic beliefs about depletion and the checking and

reassurance-seeking they drive. The second, equally important step is to identify and treat any comorbidity by the usual means, a depressive or anxiety disorder, a sexual dysfunction, or a hypochondriacal concern, reserving an antidepressant for a genuine comorbid depression or anxiety rather than for the semen-loss belief itself. Throughout, the stance stays respectful of the cultural model: the aim is to reframe the belief collaboratively, not to dismiss or ridicule it, since confrontation ruptures the alliance and drives the patient away.

GOOD TO REMEMBER

- **Management of Dhat syndrome (the first step):** non-confrontational counselling and psychoeducation that corrects the semen-loss myth and normalises masturbation and nocturnal emission, combined with sex education and, where useful, cognitive behavioural techniques; then treat any comorbid depression, anxiety or sexual dysfunction by the usual means, reserving antidepressants for a genuine comorbidity, not for the belief itself. The first move is psychoeducation within the patient's cultural model, never confrontation.

HOLD THIS

Dhat syndrome is a culture-bound presentation, not a disease: the patient attributes anxiety, fatigue, weakness and sexual symptoms to the loss of semen (dhat, a whitish urinary discharge he believes to be semen), so DSM-5-TR holds it as a cultural concept of distress rather than a codeable disorder; read it through the cultural formulation, search for and treat the comorbid depression, anxiety or sexual dysfunction, and manage the semen-loss belief with non-confrontational psychoeducation, never by confronting or pathologising it.

15 · Medication-induced sexual dysfunction

Sexual dysfunction is one of the most common, most under-reported and most adherence-wrecking adverse effects in all of psychiatric prescribing. Patients rarely volunteer it, quietly stop the drug, and relapse; the examiner therefore wants you to *ask about it, attribute it correctly, and manage it by a guideline ladder rather than by simply stopping treatment*. Two drug classes dominate the answer. **Antidepressant-induced sexual dysfunction** is a **serotonergic** problem of desire, arousal and orgasm; **antipsychotic-induced sexual dysfunction** is largely a **prolactin** problem driven by dopamine blockade. The mechanism you name decides the management you offer.

How to use this page. Hold each class as a triad: the clinical picture (which functions fail, how prevalent, whether reversible), the mechanism (serotonin versus hyperprolactinaemia), and the verified management ladder (watchful waiting, **dose reduction**, **switch** to a lower-liability agent, then an **adjunct**). The deep receptor pharmacology behind each antidepressant sits in the Mood disorders: pharmacotherapy notes, and the full antipsychotic-by-antipsychotic prolactin and receptor profile in the Schizophrenia: management, antipsychotics and adverse effects notes; this page is the clinical syndrome and its management.

15.1 Antidepressant-induced sexual dysfunction: the clinical picture, prevalence and PSSD

A serotonergic triad of reduced desire, delayed or absent orgasm, and erectile difficulty. Selective serotonin reuptake inhibitor (SSRI) treatment lowers sexual function so reliably that the SSRI sexual side effects carry their own teaching profile, clustering into three failures: reduced

libido, delayed or absent orgasm (anorgasmia and delayed ejaculation), and erectile difficulty in men with reduced lubrication in women (Kaplan and Sadock CTP 2024; Maudsley Prescribing Guidelines). It is a class effect of serotonergic antidepressants and it is *largely dose dependent* and *generally fully reversible* on stopping (Maudsley Prescribing Guidelines). Prevalence is high but agent-dependent: treatment-emergent sexual dysfunction reaches 70 to 80 per cent with venlafaxine, citalopram, paroxetine and fluoxetine, and is lower, 25 to 45 per cent, with imipramine, phenelzine, duloxetine, escitalopram and fluvoxamine (Kaplan and Sadock CTP 2024).

Post-SSRI sexual dysfunction is the one to flag. In a minority the sexual symptoms persist despite stopping the drug, a state termed post-SSRI sexual dysfunction (PSSD): decreased libido, erectile dysfunction, delayed ejaculation, anorgasmia, vaginal dryness and, in men, genital numbness and pleasureless orgasm (Maudsley Prescribing Guidelines). Its prevalence and pathophysiology remain uncertain, one estimate putting the risk at **1 in 216** patients treated; diagnosis rests on the medication history, the onset and profile of symptoms and the exclusion of other causes, and no definitive treatment exists (Maudsley Prescribing Guidelines). Examine it as a name to recognise and a caution to counsel, not a syndrome to over-diagnose.

FUNCTION AFFECTED	ANTIDEPRESSANT-INDUCED PATTERN (VERIFIED)
Desire	**Reduced libido (serotonergic; also driven by the depression itself)**
Arousal	**Erectile difficulty in men; reduced lubrication in women**
Orgasm	**Delayed or absent orgasm, delayed ejaculation, anorgasmia (the most drug-specific)**
Course	**Largely dose dependent; generally fully reversible on stopping**
Persistent variant	**Post-SSRI sexual dysfunction (PSSD): symptoms continue despite discontinuation**

The antidepressant-induced sexual dysfunction picture: a serotonergic desire, arousal and orgasm triad, dose dependent and usually reversible, with PSSD as the persistent exception (Kaplan and Sadock CTP 2024; Maudsley Prescribing Guidelines).

GOOD TO REMEMBER

- **Antidepressant-induced sexual dysfunction (criterion-anchored, Maudsley Prescribing Guidelines):** a serotonergic triad of reduced desire, delayed or absent orgasm and erectile or lubrication difficulty; highest rates (70 to 80 per cent) with venlafaxine, citalopram, paroxetine and fluoxetine, largely dose dependent and generally fully reversible.
- **Post-SSRI sexual dysfunction / PSSD (criterion-anchored):** the defining feature is persistence of the sexual symptoms despite the antidepressant being discontinued; prevalence and mechanism uncertain, risk estimated near 1 in 216 treated, no definitive treatment, diagnosis by history and exclusion.

15.2 Managing antidepressant-induced sexual dysfunction: the strategy ladder

Confirm the cause, then climb the ladder. The Maudsley ladder is a sequence, not a menu (Maudsley Prescribing Guidelines Table on sexual adverse effects; Kaplan and Sadock CTP 2024). First, *rule out other causes*: compare sexual functioning on the antidepressant against functioning *before the antidepressant*, not before the depressive illness, since untreated depression itself lowers desire, and screen for alcohol or substance use, diabetes, vascular disease and other medications such as diuretics and beta-blockers. Then, where the depression is stable, work upward: **watchful waiting** for spontaneous tolerance, then dose reduction, then a **switch**, then an **adjunct**.

Watchful waiting is weak; switching is the well-supported move. Spontaneous remission occurs in only a small minority (roughly 5 to 10 per cent) and can take 4 to 6 months, so it is the least effective option and suits milder cases. Dose reduction to the lowest effective dose is reasonable once the patient is in full remission. A short planned *drug holiday* (missing a dose or two before planned intimacy) may help shorter-half-life agents but is useless for fluoxetine given its long half-life, and it risks discontinuation symptoms, reduced adherence and relapse, so it is used cautiously. The best-supported strategy, when mood is stable, is to switch to a lower-liability antidepressant: bupropion, mirtazapine, vortioxetine, agomelatine or moclobemide, with agomelatine, bupropion and vortioxetine carrying the best evidence (Maudsley Prescribing Guidelines).

Add-ons, where a switch is not wanted. In men with antidepressant-related erectile difficulty, adding a PDE5 inhibitor (sildenafil has the strongest evidence, tadalafil also works) improves function on randomised-trial evidence (Nurnberg, JAMA 2003). Add-on bupropion is used, chiefly in women at higher doses (300 mg per day), but its evidence is more modest and mixed (see gaps). Transdermal testosterone has randomised support in women with serotonergic-antidepressant-emergent low libido and in men on serotonergic antidepressants with low or low-normal testosterone.

STEP	WHAT TO DO	VERIFIED DETAIL
1. Confirm cause	Compare with function before the drug, not before the illness; exclude other causes	**Depression itself lowers desire; screen alcohol, diabetes, vascular disease, diuretics, beta-blockers**
2. Watchful waiting	Await spontaneous tolerance in milder cases	**Only 5 to 10 per cent remit; can take 4 to 6 months; least effective**
3. Dose reduction	Lower to the lowest effective dose	**Reserve for patients already in full remission**
4. Switch	Move to a lower-liability agent (mood stable)	**Bupropion, mirtazapine, vortioxetine, agomelatine, moclobemide; best evidence agomelatine, bupropion, vortioxetine**
5. Adjunct	Add an agent without switching	**PDE5 inhibitor for male erectile difficulty (sildenafil best evidence); add-on bupropion or testosterone in selected cases**

The verified management ladder for antidepressant-induced sexual dysfunction; the ordering, and the switch targets, are the highest-yield lines, ordered as a clear algorithm (Maudsley Prescribing Guidelines).

GOOD TO REMEMBER

- **Switch strategy (guideline-anchored, Maudsley Prescribing Guidelines):** where mood is stable, the well-supported move for antidepressant-induced sexual dysfunction is to switch to bupropion, mirtazapine, vortioxetine or agomelatine (best evidence: agomelatine, bupropion, vortioxetine), after first excluding depression itself as the cause and trying watchful waiting or dose reduction.
- **Add-on PDE5 inhibitor (guideline-anchored):** for antidepressant-related erectile difficulty in men, adding sildenafil (the best-supported PDE5 inhibitor) improves function on randomised-trial evidence; keep the antidepressant and add the PDE5 inhibitor rather than sacrificing a working mood response.

15.3 Antipsychotic-induced sexual dysfunction: hyperprolactinaemia and the prolactin ladder

Dopamine blockade, then raised prolactin. Sexual dysfunction is an adverse effect of most antipsychotics; individual susceptibility varies and the effect is usually reversible (Maudsley Prescribing Guidelines). Two linked mechanisms operate: antipsychotics decrease dopaminergic transmission, which itself lowers libido, and the same dopamine blockade raises prolactin by removing tonic inhibition of its release, producing **hyperprolactinaemia**. Prolactin elevation suppresses the hypothalamic-pituitary-gonadal axis, giving sexual dysfunction, menstrual disturbance, infertility, breast enlargement and galactorrhoea in both sexes, and rarely delusions of pregnancy; long-term risks are reduced bone mineral density and a possible increase in breast-cancer risk. Note the nuance: hyperprolactinaemia is *a key factor but may explain only about 40 per cent* of antipsychotic-related sexual dysfunction, the rest coming from anticholinergic effects (impaired arousal), peripheral alpha-1 blockade (erection and ejaculation problems in men) and sedation or weight gain (reduced desire).

The prolactin ladder ranks the culprits. The propensity of an antipsychotic to cause sexual dysfunction tracks its facility to raise prolactin: risperidone is the worst, then haloperidol, then olanzapine, then quetiapine, then aripiprazole (Maudsley Prescribing Guidelines). Aripiprazole, a partial dopamine agonist, is relatively free of sexual adverse effects as monotherapy and even improves function when patients are switched to it. Group the agents by prolactin: prolactin-raising (risperidone, paliperidone, amisulpride, sulpiride and the typicals) versus prolactin-sparing (aripiprazole first, then quetiapine, olanzapine, clozapine and the newer partial agonists). Before you blame the drug, exclude other causes of a raised prolactin: pregnancy, stress at venepuncture, hypothyroidism, renal impairment and prolactinoma, sampling early in the morning.

PROLACTIN LIABILITY	ANTIPSYCHOTIC (VERIFIED LADDER)
Highest (worst for dysfunction)	**Risperidone, paliperidone, amisulpride, sulpiride, first-generation antipsychotics such as haloperidol**
Intermediate to low	**Olanzapine, then quetiapine**
Prolactin-sparing (least)	**Aripiprazole (partial agonist), also clozapine, brexpiprazole, cariprazine, asenapine**

The prolactin ladder underlying antipsychotic-induced sexual dysfunction, risperidone worst and aripiprazole least (Maudsley Prescribing Guidelines).

GOOD TO REMEMBER

- **Antipsychotic-induced sexual dysfunction (criterion-anchored, Maudsley Prescribing Guidelines):** largely driven by hyperprolactinaemia from dopamine blockade, worst with risperidone and the typicals and least with aripiprazole; but prolactin explains only about 40 per cent, so also consider anticholinergic, alpha-1-blockade and sedative contributions, and exclude pregnancy, hypothyroidism and prolactinoma before attributing a raised prolactin to the drug.

15.4 Managing antipsychotic-induced sexual dysfunction: switch or aripiprazole adjunct

Two verified routes: switch down the ladder, or add low-dose aripiprazole. Where a prolactin-raising antipsychotic is driving the problem through hyperprolactinaemia, the first choice is to **switch** to a prolactin-sparing agent, aripiprazole first, or to a lesser extent quetiapine or olanzapine, remembering that any switch carries a risk of destabilising the illness (Maudsley Prescribing Guidelines). Where switching is not feasible, add low-dose aripiprazole to the existing treatment: it lowers prolactin in a dose-dependent way, 3 mg per day is effective and 6 mg per day more so, with higher doses unnecessary, and a 2022 network meta-analysis confirmed aripiprazole near 5 mg per day (and vitamin B6 at 600 mg per day) as effective. This aripiprazole adjunct is the standard way to normalise prolactin in patients kept on a raising agent such as haloperidol or risperidone long-acting injection. Dose reduction of the offending antipsychotic, and treating modifiable bone-health risks, sit alongside these.

ROUTE	INDICATION	VERIFIED DETAIL
Switch	Prolactin-raising agent is the driver and switching is feasible	**Move to a prolactin-sparing agent (aripiprazole first; quetiapine or olanzapine to a lesser extent); watch for relapse**
Aripiprazole adjunct	Must stay on the current antipsychotic	**Add aripiprazole 3 to 6 mg per day; lowers prolactin dose-dependently; 5 mg per day confirmed by 2022 network meta-analysis**
Exclude first	Before either route	**Rule out pregnancy, hypothyroidism, renal impairment and prolactinoma; sample prolactin in the early morning**

The two verified management routes for antipsychotic-induced hyperprolactinaemic sexual dysfunction, switch or aripiprazole adjunct, with the dose that matters (Maudsley Prescribing Guidelines).

GOOD TO REMEMBER

- **Aripiprazole adjunct (guideline-anchored, Maudsley Prescribing Guidelines):** where a prolactin-raising antipsychotic must be continued, adding aripiprazole 3 to 6 mg per day normalises prolactin dose-dependently (5 mg per day confirmed by 2022 network meta-analysis); the alternative first-line move is to switch to a prolactin-sparing agent, aripiprazole itself being the least offending.

15.5 Assessing it: baseline, the ASEX scale, and the common traps

Ask directly, and measure at baseline. Sexual dysfunction is far commoner than spontaneous reports suggest, so direct questioning or a rating scale beats waiting for the patient to volunteer it (Maudsley Prescribing Guidelines). The most examinable instrument is the ASEX (Arizona Sexual Experience Scale, McGahuey and colleagues), a brief five-item scale covering sex drive, arousal, penile erection or vaginal lubrication, ability to reach orgasm, and satisfaction from orgasm over one week; each item scores 1 to 6 so the total runs from 5 to 30 (out of a possible 30), with higher scores meaning worse dysfunction (Clinical Methods in Psychiatry, AIIMS 2024). Its practical value is a *baseline*: a score before the drug lets you attribute a later change to the medication rather than to the illness. The ASEX form sits on the sexual-function scale plate with the male sexual dysfunctions.

1 in 216	40%	3 to 6	5 to 30
PSSD RISK ESTIMATE (MAUDSLEY)	ANTIPSYCHOTIC SEXUAL DYSFUNCTION VIA RAISED PROLACTIN	ARIPIPRAZOLE ADJUNCT DOSE (MG PER DAY)	ASEX TOTAL RANGE (HIGHER IS WORSE)

INDIA

The add-on PDE5 inhibitor for antidepressant-related erectile difficulty in men is everyday practice here: lead the generic sildenafil, with Manforce and Penegra the common Indian brands, and tadalafil (Tadacip, Megalis) as the alternative; keep the generic primary and confirm the product and strength locally before prescribing. Risperidone and the typicals, the worst prolactin raisers, are among the most-used antipsychotics in Indian public practice, so hyperprolactinaemic sexual dysfunction and galactorrhoea are common bedside findings, and low-dose aripiprazole augmentation is a familiar fix. The ASEX itself is taught in the AIIMS Clinical Methods text as the standard bedside scale for drug-induced sexual dysfunction.

RULE **Switch or spare, do not just stop.** For medication-induced sexual dysfunction the guideline move is to confirm the cause, try watchful waiting or dose reduction, then switch to a lower-liability agent (bupropion, mirtazapine, vortioxetine or agomelatine for antidepressants; a prolactin-sparing antipsychotic or an aripiprazole adjunct for antipsychotics), never abrupt cessation that risks relapse.

TRAP **Blaming the drug without a baseline or a prolactin work-up.** Reduced libido may be the untreated depression, not the SSRI, unless you compare with function before the drug; and galactorrhoea on an antipsychotic must not be pinned on the drug before pregnancy, hypothyroidism and prolactinoma are excluded. A parallel error is trying a drug holiday with fluoxetine, whose long half-life makes it useless.

COMMON TRAP

Reflexively stopping the antidepressant or antipsychotic when a patient reports a sexual side effect. Abrupt cessation trades a manageable adverse effect for a relapse of depression or psychosis. The verified sequence is to confirm the drug is the cause, optimise the dose, and switch or augment within the guideline ladder while keeping the psychiatric illness treated.

HOLD THIS

Medication-induced sexual dysfunction splits by mechanism: for the antidepressant, rule out depression as the cause, try watchful waiting or dose reduction, then switch to bupropion, mirtazapine, vortioxetine or agomelatine (or add a PDE5 inhibitor in men); for the antipsychotic it is largely hyperprolactinaemia, worst with risperidone and the typicals and least with aripiprazole, so switch to a prolactin-sparing agent or add low-dose aripiprazole 3 to 6 mg per day.

Psychosomatic medicine and consultation-liaison

16 · The psychosomatic concept

This topic teaches the idea that the whole somatic band rests on, the **psychosomatic concept**: the position that mind and body are one system, so that emotional life can shape the onset, course and outcome of frank physical disease and, conversely, that physical illness reshapes the mind. It is the conceptual hinge of the chapter, sitting between the disorders where the body *complains* without disease (somatic symptom disorder) and the consultation-liaison service that

meets the medically ill patient at the bedside. Examiners test it in three predictable ways: define **psychosomatic medicine** and its modern diagnostic label, state the organising model (Engel's biopsychosocial frame), and list the classical psychosomatic illnesses with the theorist who named them.

How to use this page. Fix the terminology first, then learn the one formal diagnosis this concept carries in DSM-5-TR, that being **psychological factors affecting other medical conditions** (F54), and hold its cardinal discriminator from somatic symptom disorder: PFAOMC requires a *genuine* medical condition that psychological or behavioural factors are worsening. Then internalise the **biopsychosocial model** as the frame, sketch the stress-illness pathways at concept level (the neuroendocrine, autonomic and immune routes), and finish with the seven classical conditions. The service model and the disease-specific psychiatric aspects, the psycho-oncology, HIV, endocrine, cardiac, transplant and surgical or intensive-care work, are developed in the Consultation-liaison, geriatric and transcultural psychiatry notes; here we teach only the concept.

16.1 What "psychosomatic" means, and the field of psychosomatic medicine

One word, from two Greek roots, badly misused by the lay ear. The term derives from *psyche*, soul or mind, and *soma*, body, and refers literally to how the mind affects the body (Kaplan and Sadock CTP 2024). The popular misreading, that a psychosomatic complaint is imaginary or "all in the head", is precisely the error the concept exists to correct: the suffering and the disease are real, and the psychological contribution is to their course, not to their reality. Psychosomatic medicine is the subspecialty that studies and treats this mind-body interface in the medically ill. Its nomenclature has moved twice, and both moves are examinable. First, the American classification retired the older label psychophysiological disorders (also called psychosomatic disorders) in 1980 and replaced it with the descriptive diagnosis psychological factors affecting other medical conditions. Second, in 2017 the subspecialty itself was formally renamed consultation-liaison psychiatry to align the field's name with its actual bedside practice, though the historic term survives in the journals and societies (the American Psychosomatic Society, the journal *Psychosomatic Medicine*).

> **THE THREE LABELS, AND WHAT EACH ONE IS**
>
> **Psychosomatic medicine** is the *field* (now practised as consultation-liaison psychiatry). **Psychophysiological / psychosomatic disorders** was the old *nosological* term, deleted from DSM in 1980. **Psychological factors affecting other medical conditions (F54)** is the current *diagnosis* that replaced it. Do not conflate the field, the obsolete category and the live diagnostic code; a viva can ask for all three in one breath.

16.2 Psychological factors affecting other medical conditions (F54): the DSM-5-TR diagnosis

The concept's one formal home in the manual. Psychological factors affecting other medical conditions is the diagnosis given when psychological or behavioural factors adversely affect a real, diagnosed medical condition (DSM-5-TR 2022). It rests on three criteria. **Criterion A** requires that a medical symptom or condition, other than a mental disorder, is actually present. **Criterion B** requires that psychological or behavioural factors adversely affect that condition in at least one

of four ways (below). **Criterion C** requires that those factors are not better explained by another mental disorder, such as panic disorder, major depressive disorder or post-traumatic stress disorder. Severity is then graded in **four** steps by how much harm the psychological factor does, from mild through to extreme. The whole diagnosis turns on Criterion A: there must be an underlying general medical condition for the psychological factor to act upon, which is exactly what separates it from the somatic disorders where no such disease is required.

CRITERION B ROUTE	WORKED ILLUSTRATION
The factor influences the course of the condition (a close temporal link to its onset, exacerbation or delayed recovery)	**A stressful period immediately preceding an asthma exacerbation**
The factor interferes with the treatment of the condition	**Poor adherence to antihypertensive medication**
The factor constitutes an additional well-established health risk	**Continued smoking despite established chronic lung disease**
The factor influences the underlying pathophysiology, precipitating or exacerbating symptoms or necessitating care	**Anxiety aggravating angina or an arrhythmia**

The four routes by which psychological or behavioural factors adversely affect a medical condition under Criterion B; any one route suffices (DSM-5-TR 2022).

Mild

Increases medical risk (for example, inconsistent adherence with antihypertensive treatment)

Moderate

Aggravates the underlying medical condition (for example, anxiety aggravating asthma)

Severe

Results in medical hospitalisation or an emergency-department visit

Extreme

Results in a severe, life-threatening risk (for example, ignoring the symptoms of a heart attack)

GOOD TO REMEMBER

- **Psychological factors affecting other medical conditions / F54 (the diagnosis):** Criterion A, a genuine non-psychiatric medical condition is present; Criterion B, psychological or behavioural factors adversely affect it by one of four routes (course, treatment, health risk, or pathophysiology); Criterion C, not better explained by another mental disorder; severity graded mild, moderate, severe or extreme. The single discriminator to hold is Criterion A, the requirement for a real underlying medical disease that the psychological factor worsens.

RULE **PFAOMC needs a real disease being worsened; somatic symptom disorder needs an excessive response to symptoms.** Diagnose psychological factors affecting other medical conditions only when a genuine medical condition (Criterion A) is present and psychological or behavioural factors are demonstrably harming its course, treatment, risk profile or pathophysiology.

TRAP **Reading PFAOMC as "the same as somatic symptom disorder".** They are mirror images: PFAOMC starts from a real medical condition made worse by the mind, whereas somatic symptom disorder starts from distressing bodily symptoms plus a disproportionate psychological response, with no medical disease required. The presence or absence of a genuine underlying condition sorts them.

16.3 The biopsychosocial model: the organising frame

Engel's answer to biomedical reductionism. The biopsychosocial model was developed and named by George Engel in **1977** as a deliberate alternative to the purely biomedical model of disease (developmental and general-medicine texts). Its fundamental purpose is anti-reductionist: it insists that biological, psychological and social domains all be accounted for in every case, regardless of how obviously "physical" a lesion appears, so that psychological and social contributors are never excluded by default. This is not a neutral mediator between three domains; it is a positive stance against biological, psychological and social reductionism alike, and it has since become the standard formulation frame across medicine, not psychiatry alone. For the psychosomatic concept it is the load-bearing frame: it is what lets a genuine coronary lesion, a depressive state and chronic occupational stress be held in one explanatory picture rather than forced into a single cause.

Biological domain

Genetic predisposition, neuroendocrine and physiological processes, the organ pathology itself and the drugs acting on it

Psychological domain

Mood, personality, coping style, health beliefs and behaviours, illness cognitions and the meaning of the illness to the person

Social domain

Family, work and financial context, social support, culture and access to care

PEARL

The biopsychosocial model is easy to *recite* and hard to *use*. In an exam formulation, do not list the three domains and stop; show the *interaction*, for example how a social stressor (job loss) drives a psychological state (depression) that changes a biological pathway (adherence and inflammation) to worsen a real disease. The interaction is the mark-earning part.

16.4 The stress-illness pathways, at concept level

Three biological roads from an emotion to an organ. The mechanism that gives the concept its credibility is that psychological stress reaches the body through well-mapped physiological systems, so the mind-body link is not mystical but neuroendocrine, autonomic and immune (Kaplan and Sadock CTP 2024; Tasman). The neuroendocrine road is the hypothalamic-pituitary-adrenal axis: perceived stress drives cortisol and other counter-regulatory hormones, which over time impair glucose handling, immune surveillance and healing. The autonomic road is sympathetic and adrenomedullary activation, releasing catecholamines that raise heart rate and blood pressure and can, in vulnerable coronary vessels, provoke ischaemia. The immune road is

captured by psychoneuroimmunology, the study of how psychological states, particularly depression and chronic stress, shift immune function and raise pro-inflammatory cytokines that are themselves implicated in cardiovascular and autoimmune disease. When these systems are activated repeatedly rather than briefly, the cumulative physiological cost is described as allostatic load, the wear-and-tear price of chronic adaptation. Hold the three roads by system, not by disease; the disease-specific detail belongs to the consultation-liaison chapter.

PATHWAY	PRINCIPAL MEDIATORS	ILLUSTRATIVE ORGAN CONSEQUENCE
Neuroendocrine (HPA axis)	Cortisol and counter-regulatory hormones	**Impaired glucose control, immunosuppression, delayed healing**
Autonomic (sympatho-adrenal)	Adrenaline and noradrenaline (catecholamines)	**Raised heart rate and blood pressure, coronary ischaemia in vulnerable vessels**
Immune (psychoneuroimmunology)	Pro-inflammatory cytokines, altered lymphocyte function	**Contribution to cardiovascular and autoimmune disease activity**

The three concept-level stress-illness pathways linking a psychological state to physical disease, each linking a psychological state to physical disease (Kaplan and Sadock CTP 2024; Tasman).

16.5 The classical psychosomatic conditions

Alexander's seven, and how the list outgrew them. Franz Alexander, styled the Father of Psychosomatic Medicine, originally described **seven** classical psychosomatic illnesses. His specificity hypothesis proposed that a specific unconscious emotional conflict, occurring in a person with a genetically predetermined vulnerable organ, produces a specific physical disease. The seven he named are the classic viva list, easiest to hold as the mnemonic below. The specificity hypothesis has not survived as a literal theory, but it seeded the field, and as biopsychosocial causation became clearer the roster of illnesses in which psychological factors matter grew until the list of psychosomatic illnesses is virtually endless. That expansion is exactly why the modern manual abandoned a fixed list of "psychosomatic diseases" for the general, mechanism-neutral diagnosis of psychological factors affecting other medical conditions.

ALEXANDER'S CLASSICAL PSYCHOSOMATIC ILLNESSES	SYSTEM
Bronchial asthma	**Respiratory**
Ulcerative colitis	**Gastrointestinal**
Peptic ulcer	**Gastrointestinal**
Neurodermatitis (atopic dermatitis)	**Dermatological**
Thyrotoxicosis	**Endocrine**
Rheumatoid arthritis	**Musculoskeletal or autoimmune**
Essential hypertension	**Cardiovascular**

The seven classical psychosomatic illnesses described by Franz Alexander, the anchor list for the specificity hypothesis.

MNEMONIC

PURE ANT

A study aid for Alexander's seven: **P**eptic ulcer, **U**lcerative colitis, **R**heumatoid arthritis, **E**ssential hypertension, **A**sthma (bronchial), **N**eurodermatitis, **T**hyrotoxicosis. Seven letters, seven organs; recite the mnemonic and name the theorist (Alexander) and his mechanism (the specificity hypothesis) alongside it.

GOOD TO REMEMBER

- **Alexander's classical psychosomatic illnesses (the anchor list):** bronchial asthma, ulcerative colitis, peptic ulcer, neurodermatitis, thyrotoxicosis, rheumatoid arthritis and essential hypertension (PURE ANT); the discriminator to attach is the specificity hypothesis, that a specific emotional conflict in a genetically vulnerable organ yields a specific disease, a theory now historical but foundational.
- **George Engel, 1977 (the model):** the biopsychosocial model, requiring the biological, psychological and social domains all be accounted for, is the organising frame of the psychosomatic concept and the standard formulation frame across medicine.

7	1977	4
ALEXANDER'S CLASSICAL PSYCHOSOMATIC ILLNESSES	ENGEL'S BIOPSYCHOSOCIAL MODEL	PFAOMC SEVERITY GRADES (DSM-5-TR)

INDIA

The psychosomatic concept is not a Western import for the Indian resident; the canonical Indian textbook is itself the source that names Franz Alexander the Father of Psychosomatic Medicine, sets out the specificity hypothesis and Engel's 1977 model, and lists the seven classical illnesses, so this is examined from the Indian syllabus directly. It also lands with unusual force in Indian practice, where psychological distress is very commonly first voiced as bodily symptoms and where most of these patients are met not in a psychiatry clinic but on a general-hospital ward through the consultation-liaison service; the wider transcultural frame of somatic idioms of distress, including the culture-bound presentations, is developed in the Consultation-liaison, geriatric and transcultural psychiatry notes.

HOLD THIS

The psychosomatic concept holds mind and body as one system: its live diagnosis is psychological factors affecting other medical conditions (F54), which requires a REAL medical condition (Criterion A) that psychological or behavioural factors adversely affect, and is therefore the mirror image of somatic symptom disorder, not a synonym; Engel's 1977 biopsychosocial model is its organising frame, the HPA, autonomic and immune systems are its mechanisms, and Franz Alexander's specificity hypothesis gave the seven classical illnesses (PURE ANT).

17 · Consultation-liaison psychiatry and the disease-specific aspects

The interface discipline, mapped here and taught elsewhere. **Consultation-liaison psychiatry** is the branch of clinical psychiatry that works at the border between psychiatry and the rest of medicine, seeing the psychological problems of patients who are physically ill and

advising the teams that look after them. It matters to the examiner as a named model of care and as a pattern: the same short list of psychiatric problems (delirium, depression, anxiety, self-harm-related presentations and substance withdrawal) recurs across every medical and surgical setting, and each physical illness carries its own psychiatric signature. This note is a compact map. It names the field, its service model and its disease-specific reach, and hands the full teaching to the canonical home.

This is a naming and cross-reference note, not the full teaching. The principles of consultation-liaison psychiatry and the working service model, psycho-oncology and the psychiatric aspects of chronic medical illness, the psychiatric aspects of HIV, endocrine disease and cardiac disease, and the psychiatric aspects of organ transplantation and the surgical and critical-care settings are all developed in *the Consultation-liaison, geriatric and transcultural psychiatry notes*; the acute triage of an actively suicidal patient is in *the emergency psychiatry chapter*. What follows is only where each piece belongs and the one or two facts worth carrying across.

17.1 The principles of consultation-liaison psychiatry and the C-L service model

Consultation versus liaison. Lipowski, in 1971, defined the field as the clinical, teaching and research work of psychiatrists in the non-psychiatric parts of a general hospital. The two words name two activities. *Consultation* is patient-focused: a referral arrives, the psychiatrist answers a specific question and writes back. *Liaison* is team-focused: the psychiatrist teaches and works alongside the medical or surgical team so that psychological care is built into the ward, achieving **integration of care** rather than a single integrated clinician. Six service models are described worldwide, and which one a service runs is largely a question of resources. The commonest reasons a consult is requested are delirium, self-harm and suicide-related presentations, substance withdrawal, disturbed behaviour on the ward, an uncertain diagnosis and the psychological reaction to physical illness; the acute management of the actively suicidal patient is handled compassionately in *the emergency psychiatry chapter*.

C-L SERVICE MODEL	WHO OR WHAT IS THE FOCUS
Consultation model	**The patient is the focus; the consultant answers a specific referral question and hands back a note (the default model in India)**
Liaison model	The consultee and the clinical team are the focus; ongoing teaching and joint care integrate psychiatry into the ward
Bridge model	The C-L psychiatrist is embedded at a primary-care teaching site and trains primary-care physicians
Hybrid model	The psychiatrist sits as a member of a multidisciplinary team
Autonomous psychiatric model	The C-L psychiatrist is unaffiliated to any department and is hired directly by primary-health-care services
Postgraduate training model	A physician is trained in a mental-health setting for one to two years

The six C-L service models (AIIMS Handbook of Consultation-Liaison Psychiatry, 2020); India defaults to the consultation model, with the hybrid model emerging at tertiary centres.

RULE **Delirium until proven otherwise.** New behavioural or cognitive disturbance in a medical or surgical inpatient is delirium, the single commonest reason for a general-hospital psychiatric referral, until an organic cause is found and corrected; screen and treat the physical cause before reaching for a psychiatric label.

GOOD TO REMEMBER

- **The C-L service model (the organising principle):** consultation-liaison psychiatry (Lipowski, 1971) provides psychiatric consultation to the medically and surgically ill and liaises with the treating team, integrating care rather than taking it over; six service models run worldwide (consultation, liaison, bridge, hybrid, autonomous and postgraduate-training), and the first discipline on any referral is to answer the referral question and exclude delirium before attributing a new disturbance to psychiatric illness.

6 C-L SERVICE MODELS WORLDWIDE	**~20%** DELIRIUM AMONG GENERAL-HOSPITAL INPATIENTS	**0.15-3.6%** INDIAN GENERAL-HOSPITAL REFERRAL RATE	**79%** UPPER PSYCHIATRIC-MORBIDITY RATE IN INPATIENT SAMPLES

INDIA

In India, consultation-liaison psychiatry is still practised largely as part of general adult psychiatry rather than a formal subspecialty, and general-hospital psychiatric referral rates are strikingly low, spanning roughly **0.15 to 3.6%** against the Western figure near **10%**. Formal C-L units exist at only a few centres such as AIIMS New Delhi, PGIMER Chandigarh and NIMHANS Bengaluru; most settings run the consultation model, with informal liaison delivered through the National Mental Health Programme and its District Mental Health Programme outreach. The examinable point is that scaling C-L care in a low-resource system starts with the consultation model, layering hybrid and collaborative-care models on as capacity grows.

17.2 Psycho-oncology, chronic medical illness, and the psychiatric aspects of HIV, endocrine disease and cardiac disease

Each illness has a psychiatric signature. **Psycho-oncology** covers the mental health of cancer patients and families: adjustment reactions, depression and demoralisation, anxiety around procedures and prognosis, delirium in advanced disease, and the communication and palliative-care skills that carry it. The psychiatric aspects of **chronic medical illness** are bidirectional, physical illness raising the risk of depression and anxiety while those disorders worsen adherence and outcome. The psychiatric aspects of HIV run from HIV-associated neurocognitive disorder through depression, mania and the neuropsychiatric effects of the infection and its treatment. Endocrine disease is the classic organic mimic: hyperthyroidism presents with anxiety, depression or mania and hypothyroidism with apathy, cognitive slowing, depression and the historical "myxoedema madness". In cardiac disease the link is prognostic, depression is both a risk factor for and a poor prognostic marker after coronary artery disease and myocardial infarction. Every one of these is developed in *the Consultation-liaison, geriatric and transcultural psychiatry notes*; the point to carry is the pattern, not the detail.

TRAP **Do not normalise depression in the physically ill.** Depression in the medically ill is common, more than twice general-population rates, and a poor prognostic marker in cardiac disease, so it is screened and treated, not written off as an understandable reaction to being unwell.

17.3 Organ transplantation, the surgical setting and the ICU

The high-stakes settings. Organ transplantation brings the psychiatrist in twice: before, for a structured evaluation of the candidate's capacity, adherence, substance use and psychosocial support, and after, for delirium, steroid-induced mood change and immunosuppressant neurotoxicity. The surgical setting turns on preoperative anxiety, postoperative delirium and questions of capacity and consent. The ICU is dominated by delirium, which is very common in critically ill and ventilated patients, alongside sedation-related states and the longer post-intensive-care syndrome of cognitive, mood and stress symptoms after discharge. These settings, and the disease-specific detail above, are taught in full in *the Consultation-liaison, geriatric and transcultural psychiatry notes*; this note only fixes their address and the recurring theme that delirium is the thread running through all of them.

HOLD THIS

Consultation-liaison psychiatry (Lipowski) works at the medicine-psychiatry interface, providing consultation to the medically and surgically ill and liaising to integrate care through six service models (India mostly runs the low-referral consultation model); its disease-specific reach, psycho-oncology and chronic medical illness, the psychiatric aspects of HIV, endocrine disease and cardiac disease, and the transplantation, surgical and ICU settings, is developed in the Consultation-liaison, geriatric and transcultural psychiatry notes, and the working rule at the bedside is delirium-first, treat the depression, and hand back a clear, actionable note.

Impulse-control disorders

18 · The impulse-control disorders

This is a small, self-contained class that examiners love precisely because it is compact and rule-bound. An **impulse-control disorder** is defined by a single repeating engine: a failure to resist an impulse, drive or temptation to perform an act that is harmful to the person or to others, a rising sense of *tension or affective arousal* before the act, and *pleasure, gratification or relief* at the moment of doing it. Learn that tension-relief cycle once and it carries most of the marks across all four entities. The modern nosology regroups these behaviours: ICD-11 collects four named disorders under an ICD-11 impulse-control grouping, while DSM-5-TR scatters them, placing intermittent explosive disorder, pyromania and kleptomania in "Disruptive, impulse-control and conduct disorders" and handling compulsive sexual behaviour differently again.

How to use this page. Take each entity as a keyword with one defining criterion and one management line. Fix the shared tension-relief template first; then the four disorders, intermittent explosive disorder (impulsive aggression), kleptomania (impulsive stealing), pyromania (deliberate fire-setting) and compulsive sexual behaviour disorder; then the sharp discriminators that separate each from its "ordinary" mimic, and the graded, psychological-first management. The combined map of the paraphilic and impulse-control classes is drawn in the accompanying figure, which sits with this block; refer to it for how these entities relate to the paraphilias. The tone throughout is clinical and non-graphic.

MNEMONIC

TENSION → ACT → RELIEF

The shared engine of every impulse-control disorder: mounting *tension* or affective arousal, an impulsive *act* the person cannot resist, then *relief*, pleasure or gratification. Present this cycle and you have named what unifies intermittent explosive disorder, kleptomania, pyromania and compulsive sexual behaviour disorder, and what separates them from goal-directed, premeditated behaviour.

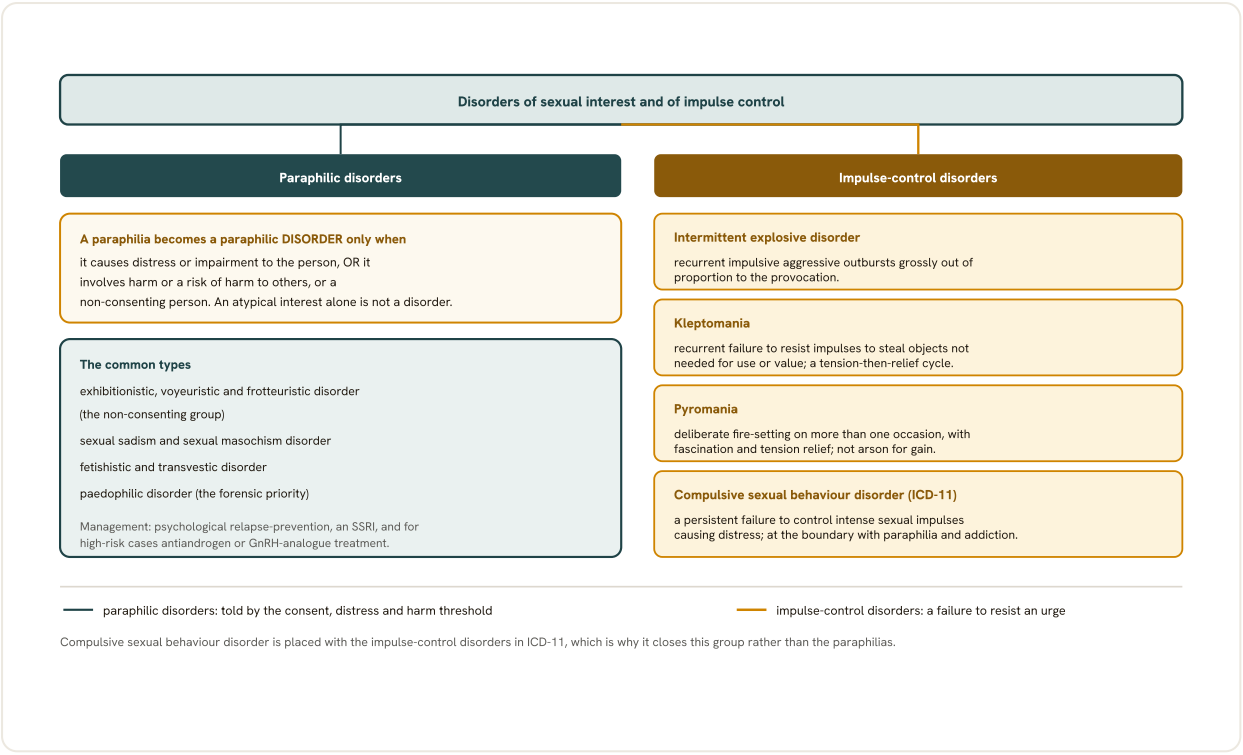

Fig 18.9 The paraphilic and impulse-control disorders. The two groups share a theme of behaviour driven by an urge, but they are recognised differently. A paraphilia becomes a paraphilic disorder only when it causes the person distress or impairment or entails harm or risk of harm to others or a non-consenting person, so an atypical interest by itself is not a disorder; the common types run from the non-consenting exhibitionistic, voyeuristic and frotteuristic disorders through sexual sadism and masochism, fetishistic and transvestic disorder, to paedophilic disorder as the forensic priority. The impulse-control disorders, intermittent explosive disorder, kleptomania and pyromania, are each a recurrent failure to resist an urge with a tension-then-relief cycle, and ICD-11 adds compulsive sexual behaviour disorder here, at the boundary with the paraphilias and with the addiction model.

18.1 Intermittent explosive disorder: impulsive aggression out of proportion to provocation

Recurrent, impulsive aggressive outbursts grossly out of proportion to the trigger. The core of intermittent explosive disorder is recurrent impulsive aggression, that is, aggressive outbursts that are impulsive or anger-based and grossly out of proportion, in intensity or duration, to the provocation (DSM-5-TR 2022; Kaplan and Sadock CTP 2024). DSM-5-TR operationalises the frequency two ways: verbal aggression (temper tantrums, tirades, verbal arguments) or non-damaging, non-injurious physical aggression occurring **twice weekly** on average over a 3-month period (Criterion A1), *or* three behavioural outbursts causing damage, destruction or physical injury within a 12-month period (Criterion A2). The magnitude is grossly out of proportion to the provocation (Criterion B); the outbursts are *not premeditated* and are not committed to achieve a tangible objective such as money, power or intimidation (Criterion C); and the chronological age is at least **6 years** (Criterion E). Outbursts have a rapid onset, little prodrome, and typically last under 30 minutes.

CRITERION	AGGRESSION TYPE	FREQUENCY THRESHOLD
A1	Verbal, or physical without damage or injury	Twice weekly on average, over a 3-month period
A2	Physical, causing damage, destruction or injury	Three outbursts within a 12-month period

The two frequency routes to the intermittent explosive disorder diagnosis; either the frequent low-grade route (A1) or the infrequent destructive route (A2) satisfies Criterion A (DSM-5-TR 2022).

RULE **The aggression is impulsive, not instrumental.** Intermittent explosive disorder is diagnosed only when the outbursts are impulsive or anger-based and are not premeditated and not aimed at a tangible objective; that is what separates it from the proactive, predatory aggression of conduct disorder and antisocial personality disorder.

GOOD TO REMEMBER

- **Intermittent explosive disorder (the criterion):** recurrent impulsive aggressive outbursts grossly out of proportion to provocation, either verbal or non-injurious aggression twice weekly over a 3-month period, or three destructive or injurious outbursts within a 12-month period; not premeditated, not for tangible gain, age at least 6 years.

Management: serotonergic pharmacotherapy plus cognitive-behavioural work. Because low central serotonergic function is linked to impulsive aggression, the drug evidence centres on SSRIs, and fluoxetine carries the single positive double-blind, placebo-controlled trial in intermittent explosive disorder (Coccaro and colleagues), dosed into the standard antidepressant range up to **60 mg** per day; across five controlled trials SSRIs reduce impulsive aggression, though under a third of patients reach full remission (Kaplan and Sadock CTP 2024). Where impulsivity is high, mood-stabilising anticonvulsants, divalproex sodium and valproate, reduce aggressive acts, with the largest effect in the most impulsive patients; the deeper pharmacology sits in the Mood disorders: pharmacotherapy notes. On the psychological side, cognitive-behavioural therapy targeting anger, hostile appraisals and the outburst cycle has a positive randomised trial (McCloskey and colleagues, twelve weekly sessions, group and individual formats), with gains sustained at follow-up.

GOOD TO REMEMBER

- **Intermittent explosive disorder management (guideline and evidence-anchored):** an SSRI is the drug of choice, fluoxetine having the one positive controlled trial (up to 60 mg per day); anticonvulsant mood stabilisers such as divalproex suit high trait impulsivity; and manualised cognitive-behavioural therapy for anger and impulse control is the evidence-based psychotherapy.

18.2 Kleptomania: the impulse to steal what is not needed

Recurrent failure to resist stealing objects not needed for use or value. Kleptomania is the recurrent failure to resist impulses to steal objects that are *not needed for personal use or for their monetary value* (Criterion A); the individual feels a rising subjective tension before the theft (Criterion B) and pleasure, gratification or relief at the moment of committing it (Criterion C); and the stealing is not done to express anger or vengeance and is not a response to a delusion or hallucination (Criterion D) (DSM-5-TR 2022). This is **impulsive stealing** driven by the tension-relief cycle, not by want: the objects are typically of little value to a person who could have paid for them, and are often given away, discarded, hoarded or secretly returned. It is distinct from ordinary theft, which is goal-directed, planned, and motivated by the value or usefulness of the item.

TRAP **Reading everyday shoplifting as kleptomania.** Most theft is ordinary, planned and motivated by the object's value; kleptomania is rare and defined by stealing things the person does not need or want, driven by mounting tension and relief. Equally, do not miss it in reverse, a patient who steals trinkets they discard, feels shame and cannot stop, is not simply "a thief".

GOOD TO REMEMBER

- **Kleptomania (the criterion):** recurrent failure to resist impulses to steal objects not needed for personal use or monetary value, with rising tension before and gratification or relief after the theft, not for anger, vengeance or in response to a delusion; the tension-relief cycle and the pointlessness of the object separate it from ordinary, goal-directed theft.

Management: an opioid antagonist and cognitive-behavioural therapy. There is no licensed drug, and the controlled evidence is thin, but the standout pharmacological signal is the opioid antagonist naltrexone, which reduces the urge to steal and stealing behaviour in trial data, plausibly by dampening the reward and relief that reinforce the act; SSRIs give mixed results and are used on the obsessive-compulsive-spectrum rationale (Kaplan and Sadock CTP 2024). The evidence-based psychotherapy is cognitive-behavioural therapy, classically covert sensitisation and imaginal desensitisation to the urge.

GOOD TO REMEMBER

- **Kleptomania management (evidence-anchored):** the opioid antagonist naltrexone has the best pharmacological support for reducing stealing urges; SSRIs are second-line with mixed evidence; and cognitive-behavioural therapy, particularly covert sensitisation and imaginal desensitisation, is the psychotherapy of choice. No agent is formally licensed.

18.3 Pyromania: deliberate fire-setting with fascination and relief

Purposeful, repeated fire-setting driven by fascination, not gain. Pyromania is **deliberate fire-setting** on more than one occasion (Criterion A), preceded by tension or affective arousal (Criterion B), with a *fascination with, interest in, curiosity about or attraction to* fire and its situational contexts, its paraphernalia, uses and consequences (Criterion C), and pleasure, gratification or relief when setting fires or witnessing their aftermath (Criterion D) (DSM-5-TR 2022). The diagnosis is exclusionary: the fire-setting must *not* be done for monetary gain, as an expression of sociopolitical ideology, to conceal criminal activity, to express anger or vengeance, to improve one's circumstances, in response to a delusion or hallucination, or as a result of impaired judgement; and it is not diagnosed if better explained by conduct disorder, a manic episode or antisocial personality disorder (Criteria E and F). This is what separates pyromania from arson, which is fire-setting for an external purpose such as profit, revenge or concealment.

GOOD TO REMEMBER

- **Pyromania (the criterion):** deliberate, purposeful fire-setting on more than one occasion, with pre-act tension, a fascination with fire, and relief on setting or watching it; not for monetary gain, ideology, concealment, anger or in response to a delusion; the intrinsic fascination and tension-relief, and the absence of external motive, separate it from arson.

Management: psychological, with no established drug. There are no controlled trials and no approved medication for pyromania; scattered case reports of SSRIs, naltrexone, topiramate and mood stabilisers are inconclusive (Kaplan and Sadock CTP 2024). The mainstay is cognitive-behavioural therapy combined with fire-safety education, an approach with the best support, especially in younger fire-setters where structured cognitive-behavioural programmes outperform education alone.

GOOD TO REMEMBER

- **Pyromania management (evidence-anchored):** no drug is licensed and pharmacotherapy evidence is only anecdotal; the first-line and best-supported treatment is cognitive-behavioural therapy paired with structured fire-safety education, targeting the antecedents of fire-setting and the fascination that reinforces it.

18.4 Compulsive sexual behaviour disorder: the ICD-11 impulse-control entity

A persistent failure to control intense, repetitive sexual impulses, classed with the impulse-control disorders. Compulsive sexual behaviour disorder (ICD-11 code 6C72) is a persistent pattern of failure to control intense, repetitive sexual impulses or urges resulting in repetitive sexual behaviour, over an extended period, at least six months (ICD-11 CDDR). The behaviour becomes a central life focus to the neglect of health, care and responsibilities; the person makes repeated unsuccessful efforts to reduce it; continues despite adverse consequences (relationship, financial, legal or health) and even when deriving little satisfaction; and the pattern causes marked distress or significant impairment. It is the ICD-11 successor to ICD-10 excessive sexual drive, the older label for **hypersexuality**, and it is deliberately placed among the impulse-control disorders. DSM-5-TR, by contrast, does *not* recognise it; a proposed "hypersexual disorder" was rejected for DSM-5 for want of evidence.

PEARL

Two boundaries are the whole exam answer. Against *paraphilia*: in compulsive sexual behaviour disorder the sexual interest itself is typically normative and it is *control* that fails, whereas a paraphilia is defined by an *atypical arousal focus*. Against *addiction*: ICD-11 declined to call this a behavioural addiction, reserving that grouping for gambling and gaming alone, and located it with the impulse-control disorders instead, so "sex addiction" is not an ICD-11 diagnosis. Crucially, distress arising *only* from moral or religious disapproval of one's own sexual behaviour does not qualify.

GOOD TO REMEMBER

- **Compulsive sexual behaviour disorder (the criterion):** a persistent, at-least-six-month failure to control intense, repetitive sexual impulses producing repetitive sexual behaviour that becomes a central focus, resists control and continues despite adverse consequences or little satisfaction, causing marked distress or impairment; classed as an ICD-11 impulse-control disorder, not a paraphilia and not a behavioural addiction, and not diagnosed on moral or religious distress alone.

DISORDER	THE IMPULSE RESISTED AND FAILED	TENSION THEN RELIEF	THE DISCRIMINATOR FROM ITS "ORDINARY" MIMIC
Intermittent explosive disorder	Impulsive aggressive outburst	Yes; anger or arousal, then release	**Impulsive, not pre-meditated and not for tangible gain (versus conduct disorder or antisocial aggression)**
Kleptomania	Stealing an unneeded, low-value object	Yes; tension, then grati-fication or relief	**Object not needed for use or value (versus ordinary, goal-direct-ed theft)**
Pyromania	Deliberate fire-setting	Yes; arousal, then relief on watching	**Fascination with fire, no external motive (versus arson for gain, revenge or concealment)**
Compulsive sexual be-haviour disorder	Repetitive sexual behaviour	Often to modulate neg-ative affect	**Control fails over a normative interest (versus a paraphilia's atypical arousal focus; not an addiction in ICD-11)**

The four ICD-11 impulse-control disorders share one engine, the tension-relief cycle, and each is separated from a common mimic by a single discriminator (DSM-5-TR 2022; ICD-11 CDDR; Kaplan and Sadock CTP 2024).

INDIA

The genuine Indian anchor is the medico-legal interface. Kleptomania, pyromania and impulsive aggression frequently surface in forensic settings, but the disorder is not, in itself, a legal defence: the insanity defence under Indian law, Indian Penal Code Section 84 (the M'Naughten rules, re-enacted in the Bharatiya Nyaya Sanhita 2023), acquits only where unsoundness of mind rendered the person incapable of knowing the nature of the act or that it was wrong or contrary to law. Most people with an impulse-control disorder retain that knowledge, so they are usually held criminally responsible for the theft, the fire-setting or the assault; the diagnosis bears on mitigation and sentencing, not automatic exculpation. The clinician's task is to distinguish the disorder from the crime (kleptomania from theft, pyromania from arson) and to say so honestly. Drug choices here, SSRIs, naltrexone and mood stabilisers, are the same generics used across the mood and obsessive-compulsive chapters; entity-specific Indian brand equivalents were not confirmable in the available sources, so the generic name stays primary.

4
ICD-11 NAMED IMPULSE-CONTROL DISORDERS

6
INTERMITTENT EXPLOSIVE DISORDER MINIMUM AGE (YEARS)

3
DESTRUCTIVE OUTBURSTS (PER 12-MONTH PERIOD, CRITERION A2)

HOLD THIS

Every impulse-control disorder is a failure to resist an impulse harmful to self or others, with **tension before and relief after**; ICD-11 names four, intermittent explosive disorder, kleptomania, pyromania and compulsive sexual behaviour disorder, and you diagnose each by the tension-relief cycle and the *absence* of an external, goal-directed motive, managing them psychologically first (cognitive-behavioural therapy) with an SSRI, fluoxetine or naltrexone as the drug adjunct.

Suicide and self-harm

19 · Suicide risk assessment

This is the topic on which the whole chapter closes, the *Defence of life* that the master mnemonic seeded at the outset, and it is examined as a skill rather than as a fact-list. The task is a **suicide risk assessment**: a compassionate, structured clinical interview that gathers the patient's suicidal thinking and behaviour, weighs the **risk factors** against the **protective factors** and the current mental state and social context, and then translates that synthesis into a plan for care and **safety**. It is emphatically *not* a numerical prediction of who will die, and the single most important idea on the page is that no instrument, and no clinician, can predict individual suicide reliably; the assessment exists to guide management, not to forecast an outcome.

How to use this page. Fix the vocabulary first (ideation, plan, intent, attempt, self-harm), then the two halves of the balance, the static and dynamic risk factors and the protective factors, then the framing of chronic against acute risk. After that, learn the three named aides in the way examiners want them, the Columbia Suicide Severity Rating Scale as the gold-standard structured interview, SAD PERSONS as a teaching aide with a stated limitation, and PHQ-9 item nine as a screen, and finally the two principles that bind the whole topic together: the limits of individual prediction, and the Indian statute that has recriminalised nothing and decriminalised the attempt. Throughout, hold the cardinal error the topic is built to prevent, treating a denied ideation as a cleared risk when the structured evaluation and the risk-and-protective balance say otherwise.

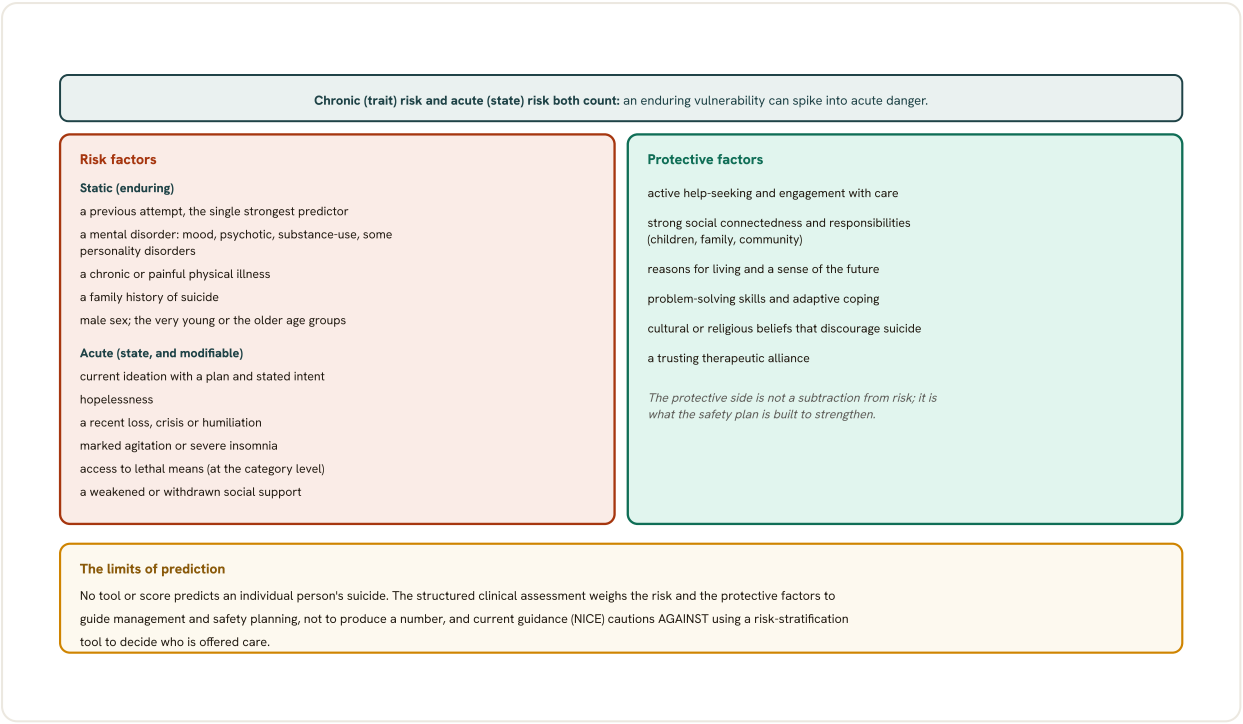

Fig 18.10 The balance of suicide risk and protective factors. A structured assessment weighs the two sides. The risk factors divide into static, enduring markers, of which a previous attempt is the single strongest, and acute, modifiable state factors such as current ideation with a plan and intent, hopelessness and a recent crisis, which is where intervention acts. The protective factors, from help-seeking and social connectedness to reasons for living and a therapeutic alliance, are not simply subtracted from risk; they are what a safety plan is built to strengthen. The decisive teaching point is the limit of prediction: no instrument forecasts an individual suicide, so the assessment guides care rather than generating a number, and current guidance discourages using a risk-stratification score to allocate treatment.

The Columbia Suicide Severity Rating Scale (C-SSRS): the screener structure

THE SCREENER ASKS, IN ESCALATING ORDER	WHAT IT MARKS
1. A wish to be dead, or to go to sleep and not wake up	ideation, lower
2. Non-specific active thoughts of ending one's life	ideation
3. Active thoughts with a general sense of acting, but no plan	ideation, rising
4. Active thoughts with some intention of acting	**high concern**
5. Active thoughts with a specific plan and intent	**highest ideation**
6. Behaviour: any preparatory act or attempt (asked of everyone)	**behaviour, highest**

Items 1 to 5 form an escalating ideation ladder and item 6 captures behaviour. The screener's own published triage points are: yes to item 1 or 2, low risk (behavioural-health referral at discharge); yes to item 3, moderate risk (behavioural-health consult); yes to item 4 or 5, high risk (patient safety precautions); yes to the behaviour item, high risk only if the behaviour fell within the past three months, otherwise moderate. The behaviour item is stated here in general terms, without method detail.

Fig 18.11 The Columbia Suicide Severity Rating Scale, shown as its structure. The screener walks up an ideation ladder, from a passive wish to be dead, through non-specific and then active thoughts, to active thoughts with intent and finally with a specific plan, and closes with a single behaviour question asked of everyone. It is used not to generate a number but to place where a person sits on that ladder: a positive answer at the second step already signals concern, and a positive answer higher up, or to the behaviour item, signals high risk needing immediate safety measures. It is presented here typeset and safe-messaged, with the behaviour examples abstracted so that no method detail appears.

SAD PERSONS: a teaching aide-memoire (out of a possible 10)

LETTER	ITEM (ONE POINT EACH)
S / A / D	male Sex; extremes of Age; Depression
P / E / R	Previous attempt; Ethanol or substance use; Rational thinking loss (psychosis)
S / O / N / S	Social supports lacking; Organised plan; No spouse or partner; Sickness (chronic illness)
Banding	**0 to 4 lower, 5 to 6 medium (consider admission), 7 to 10 high concern**

SAD PERSONS (Patterson et al, 1983). A teaching mnemonic, NOT a validated predictor of individual risk (Bolton 2012); use it to structure recall, never to clear a patient.

Over the last 2 weeks, how often have you been bothered by any of the following problems? *(Use "✔" to indicate your answer)*	Not at all	Several days	More than half the days	Nearly every day
9. Thoughts that you would be better off dead or of hurting yourself in some way	0	1	2	3

Developed by Drs. Robert L. Spitzer, Janet B.W. Williams, Kurt Kroenke and colleagues, with an educational grant from Pfizer Inc. No permission required to reproduce, translate, display or distribute.

PHQ-9 item 9 is the single suicidality-screen item, scored 0 to 3 over two weeks; any score of 1 or more is a positive screen that prompts a full risk assessment. It is a screen, not a risk assessment.

PHQ-9 (Kroenke, Spitzer and Williams); PHQ family, no permission required to reproduce; free non-commercial educational use.

BECK SUICIDE INTENT SCALE (SIS): INTENT BEHIND AN ACTUAL ATTEMPT	STRUCTURE AND BAND
Part 1, objective circumstances (8 items): isolation, timing, precautions against discovery, acting to gain help, final acts, active preparation, a note, and communication of intent	each item scored 0 to 2
Part 2, self-report (7 items): expectation of fatality, conception of lethality, seriousness, attitude to living or dying, premeditation, reaction to survival, stated purpose	each item scored 0 to 2; total out of a possible 30
rated for a person who HAS made an attempt (distinct from the ideation scales)	**a commonly used categorisation (Pierce 1977): 0 to 3 low, 4 to 10 medium, 11 or more high intent; Beck did not fix a validated cut-off**

Beck Suicide Intent Scale (Beck, Schuyler and Herman, 1974); copyright the authors, permission required, so typeset here in summary with the verified structure rather than reproduced.

Fig 18.12 The suicide-risk aides. SAD PERSONS is a ten-point mnemonic that helps a trainee remember the domains to ask about, banded 0 to 4 lower, 5 to 6 medium and 7 to 10 high concern; it is explicitly a teaching aide and not a validated predictor of an individual's risk, so it structures recall but never clears a patient. PHQ-9 item 9 is the single screening question for suicidality, positive at a score of 1 or more, which prompts, rather than replaces, a full assessment. The Beck Suicide Intent Scale is the instrument for a person who has already made an attempt, rating the objective circumstances and the self-reported intent across fifteen items for a total out of a possible 30, with the widely quoted Pierce bands recorded as a common categorisation rather than a Beck-validated cut-off. All are presented safe-messaged, without method detail.

19.1 The vocabulary of suicidality

Precise terms, arranged along a continuum from thought to act. A defensible assessment begins with clean definitions, because the examiner marks the discrimination between them and because muddled terms produce muddled clinical decisions (Kaplan and Sadock CTP 2024; Clinical Methods in Psychiatry, AIIMS 2024). **Suicidal ideation** is thinking about, considering or planning ending one's life, and it ranges from a passive wish to be dead to active, specific thoughts. A **plan** is a formulated method and set of circumstances for acting. **Intent** is the subjective determination to act on those thoughts, the will to die, and it is separable from both ideation and plan. A **suicide attempt** is a non-fatal, self-directed, potentially injurious act carried out with at least some intent to die. **Self-harm**, in the sense used by the self-harm guideline, is any act of self-injury or self-poisoning irrespective of intent or motive, so it is the broadest term and deliberately does not assume a wish to die; non-suicidal self-injury is the subset undertaken with no suicidal intent. The clinician's job is to place the patient on the continuum, ideation, then plan, then intent, then behaviour, because escalation along it is one of the things that raises acute concern.

GOOD TO REMEMBER

- **The suicidality continuum (the terminology):** ideation (passive wish to active thought) → plan (a formulated method) → intent (the will to act) → attempt (a self-directed potentially injurious act with some intent to die); self-harm is the umbrella term for any self-injury or self-poisoning regardless of intent, so it does not by itself imply a wish to die. The discriminator to hold is that intent is separable from ideation and plan and must be asked about in its own right.

19.2 What a structured evaluation is, and is not

A weighing interview, not a checklist score. The guideline-anchored method is a **structured evaluation** through a clinical interview that explores the patient's suicidal thinking and its history, the current mental state, the psychosocial context and the balance of risk and protective factors, and that arrives at a clinical judgement about care and safety (NICE-aligned, NG225 2022). It is structured in that it systematically covers the same domains every time so that nothing high-yield is missed, but it is not a tally of ticked boxes producing a risk number. Asking directly and calmly about suicidal thoughts is a core skill and, contrary to a persistent myth, does not plant the idea or increase risk; a graded set of questions, moving from low mood to thoughts that life is not worth living, to active thoughts, plan and intent, is the safe and validated way in. The cardinal trap sits precisely here: a patient may deny ideation, yet when the static and dynamic risk load is high and protective factors are thin, a bare verbal denial cannot be treated as a cleared risk. Denial is one data point among many, not the end of the assessment.

WORKED EXAMPLE

A man in his forties is seen after a recent attempt was medically managed. He now says, flatly, "no, I am not thinking of anything like that, I just want to go home." Against that single denial sits a strong static load (a prior attempt, current severe depression) and a heavy dynamic load (marked hopelessness, agitation, a job loss the week before, living alone), with few protective factors and poor engagement. Taking the denial at face value and discharging him would be the cardinal error. The structured evaluation, weighing the whole balance rather than the last sentence spoken, keeps him in active care with a collaborative safety plan; the denial is noted, not trusted as an all-clear.

TRAP **Treating a denied ideation as a cleared risk.** A verbal "no" does not neutralise a high risk-and-protective balance. When the structured evaluation shows heavy static and dynamic loading with thin protection, the denial is one data point; the assessment, not the last answer, governs the safety plan.

19.3 Static risk factors: the fixed background

Historical, slow-changing markers that set the baseline. Static (distal, largely unchangeable) risk factors describe the population a patient belongs to and raise the baseline against which the current state is read (Kaplan and Sadock CTP 2024). The single strongest of them is a *previous suicide attempt*; a prior attempt and a current major depressive episode are together described as the two strongest predictors of future suicide, and the first attempt materially raises the risk of a later one. Mental illness is the next great pillar: mood disorders (unipolar and, with the highest risk, bipolar), schizophrenia and other psychoses, and substance use disorders including alcohol all carry substantially elevated risk, and psychological-autopsy data find a diagnosable mental disorder in the large majority of those who die. Chronic, disabling or painful physical illness adds independently, especially when depression is also present. A family history of suicide or of psychiatric disorder raises risk, partly heritable and partly environmental. Demographic markers (for example male sex for completed suicide, older age, social isolation, single or bereaved status) are statistically real but weak in the individual case, which is exactly why they must never be used alone.

STATIC (DISTAL) RISK FACTOR	WHY IT RAISES BASELINE RISK	THE POINT TO HOLD
Previous suicide attempt	Strongest single historical predictor	**Prior attempt plus current depression are the two strongest predictors**
Mental illness	Mood disorder, psychosis, substance use disorder all elevate risk	**Bipolar and severe depression carry the highest mood-disorder risk**
Chronic pain or physical illness	Disabling, painful or fatal disease, amplified by depression	**Independent contributor, greatest when depression co-occurs**
Family history	Family history of suicide or psychiatric disorder	**Part heritable, part environmental transmission**
Demographics	Male sex (completion), older age, isolation, single or bereaved	**Statistically real but weak alone; never a stand-alone basis**

The static background factors that set baseline risk, mapped in the accompanying figure against the dynamic and protective factors (Kaplan and Sadock CTP 2024).

RULE **Prior attempt is the strongest static factor.** A previous suicide attempt, with current major depression, is among the two strongest predictors of future suicide; every assessment asks explicitly about past attempts and never discounts them because the method was medically minor.

19.4 Dynamic risk factors: the modifiable foreground

Current, changeable states that drive acute concern and are the levers of intervention. Dynamic (proximal, modifiable) factors are what shift a patient from their chronic baseline into a period of heightened risk, and because they can change they are where treatment acts (Kaplan and Sadock CTP 2024; NICE NG225 2022). *Hopelessness* is the pre-eminent dynamic factor, more tightly linked to eventual suicide than depressed mood itself, which is why it is asked about and rated directly. *Current suicidal ideation with a plan and stated intent* is the most immediate marker, and escalation along the ideation continuum is itself a warning. *Agitation, severe anxiety and insomnia* raise near-term risk, particularly in a depressive or mixed mood state. A *recent loss or interpersonal crisis* (bereavement, separation, humiliation, job or financial loss, legal trouble) commonly precedes an act. *Access to lethal means* is a dynamic factor named only at the category level, because reducing that access is one of the most effective things a clinician and family can do. *Social isolation and disconnection*, and recent discharge from inpatient or emergency care, complete the picture, the transition period after any care contact being a recognised window of elevated risk.

GOOD TO REMEMBER

- **Dynamic risk factors (the modifiable foreground):** hopelessness (the pre-eminent one), current ideation with plan and intent, agitation or severe anxiety and insomnia, a recent loss or crisis, access to means (named at category level only), isolation, and the high-risk period just after any care contact. The point to hold is that these are the treatable levers; the assessment targets them, and reducing access to means is framed positively as putting time and distance between a person and a crisis.

19.5 Protective factors: the other half of the balance

Reasons and connections that buffer risk and must be actively sought. A one-sided hunt for risk is a poor assessment; the protective factors are weighed alongside and are themselves things to strengthen (Kaplan and Sadock CTP 2024; Clinical Methods in Psychiatry, AIIMS 2024). The best-evidenced are *help-seeking* and a willingness to engage with care; *social connectedness and support*, a partner, family, friends or community; concrete *reasons for living*, which can be elicited directly and are the object of the Reasons for Living Inventory; *responsibilities to others*, notably dependent children; *religious or cultural beliefs* that frame suicide as unacceptable; and *active engagement with treatment* with a therapeutic alliance the patient values. Protective factors are not fixed either; they are dynamic, they can erode in a crisis, and part of management is deliberately building and mobilising them. Crucially, protection does not cancel risk arithmetically; a patient with strong reasons for living and a high acute risk is still at high acute risk, so protective factors qualify the plan rather than override the concern.

PEARL

Asking directly about suicide does *not* plant the idea or raise risk, a myth worth retiring on sight. A graded approach works best: start with mood, move to whether life feels worth living, then to active thoughts, then plan, then intent, and, just as deliberately, ask what has kept the person going, their reasons for living, so the interview surfaces the protective half of the balance rather than only the risk half.

19.6 Chronic and acute risk

Two time-frames, held separately, because they call for different responses. Risk is not a single dial; the useful clinical framing separates **chronic and acute risk** (Kaplan and Sadock CTP 2024). *Chronic (baseline) risk* is the long-standing, trait-like elevation carried by the static factors, a history of attempts, an enduring mental illness, a personality vulnerability; it changes slowly and is managed over the long term through treatment of the underlying disorder and durable supports. *Acute (state) risk* is the near-term elevation driven by the dynamic factors, a surge of hopelessness, an intensifying ideation, a fresh loss, agitation, intoxication; it changes fast and demands an immediate response. The two combine: a patient may be chronically high-risk yet acutely settled, or chronically moderate yet acutely dangerous because of a proximal crisis, and it is the person at chronic *and* acute elevation who is of greatest immediate concern. Documenting both, and stating what specifically has changed to raise the acute component, is what makes an assessment defensible and actionable.

DIMENSION	CHRONIC (BASELINE) RISK	ACUTE (STATE) RISK
Driven by	Static, trait-like factors	**Dynamic, modifiable factors**
Time-course	Slow-changing, long-standing	**Fast-changing, near-term**
Typical content	Past attempts, chronic illness, vulnerability	**Rising hopelessness, ideation with intent, recent crisis, agitation**
Response	Long-term treatment and durable support	**Immediate safety plan, closer care, mobilise supports**

Chronic and acute risk are documented as two separate axes; the person elevated on both warrants the most immediate response (Kaplan and Sadock CTP 2024).

19.7 The Columbia Suicide Severity Rating Scale (C-SSRS)

The gold-standard structured interview, an ideation-severity ladder plus a behaviour subscale. The Columbia Suicide Severity Rating Scale (C-SSRS; Posner and colleagues, 2011) is a clinician-administered interview widely regarded as the gold-standard instrument for assessing and monitoring suicidality, free for clinical use and available in many languages and versions (a lifetime-recent baseline version, a since-last-visit version for follow-up, and a brief screener for non-specialists) (Clinical Methods in Psychiatry, AIIMS 2024). Its power for the exam is its structure. The *ideation-severity subscale* is an ordered ladder of five levels of increasing severity: a wish to be dead; non-specific active suicidal thoughts; active thoughts with some general consideration of how one might act but no worked-out plan and no intent; active ideation with some intent to act but without a specific plan; and active ideation with both a specific plan and intent. A separate *behaviour subscale* records the spectrum of suicidal behaviour, from preparatory acts through interrupted and aborted attempts to an actual attempt, together with a general rating of its severity, and one further category captures self-injurious behaviour without suicidal intent. It structures the interview and tracks change over time; it does not, and is not meant to, output a probability that a given person will act.

C-SSRS IDEATION LEVEL	WHAT IT CAPTURES (NO METHOD SPECIFICS)	THE ESCALATION IT MARKS
Level one	Wish to be dead	**Passive; a wish not to be alive, without active thoughts**
Level two	Non-specific active suicidal thoughts	**Active thoughts of ending one's life, no method, plan or intent**
Level three	Active ideation with general thought of acting	**Some consideration of how one might act, no worked-out plan, no intent**
Level four	Active ideation with some intent	**Some intent to act, still without a specific plan**
Level five	Active ideation with plan and intent	**Both a specific plan and intent present; the top of the ladder**

The C-SSRS ideation-severity ladder, described without method detail; the full instrument is reproduced in the accompanying scale plate (Posner 2011; AIIMS Clinical Methods 2024).

GOOD TO REMEMBER

- **C-SSRS (the instrument):** the gold-standard clinician-administered interview for assessing and monitoring suicidality, comprising eleven categories in all, five ordered ideation-severity levels (from a wish to be dead up to active ideation with a specific plan and intent), five behaviour categories, and one for self-injurious behaviour without suicidal intent. The discriminator to hold is that it structures and tracks the assessment but is explicitly not a predictor of who will act.

19.8 SAD PERSONS and PHQ-9 item nine: two teaching aides

A mnemonic best used to teach, and a single screening item, each with clear limits. Two further aides appear in every viva, and each must be quoted with its caveat (Patterson and colleagues, 1983; Kroenke and Spitzer, 2001). The SAD PERSONS scale is a ten-item mnemonic of classic risk factors, scored out of a possible ten, devised as a memory aid and teaching tool. Its examinable weakness is decisive: it is *not* a validated predictor of individual suicide, it performs poorly at discriminating who will act, and it must never be used to decide who receives care or admission; it earns its place as a way to remember factors, not to grade risk. The PHQ-9 depression questionnaire carries item nine, which asks about "thoughts that you would be better off dead, or of hurting yourself in some way," scored 0 to 3 over the past two weeks. It is a widely used single-item screen: any endorsement is a positive flag that must trigger a full structured suicide risk assessment, but on its own it screens rather than assesses, and it neither confirms risk nor grades it. Both aides feed the interview; neither replaces it, and the reproduced forms sit in the accompanying scale plate.

MNEMONIC

SAD PERSONS

Sex (male), **A**ge (older), **D**epression; **P**revious attempt, **E**thanol or substance use, **R**ational-thinking loss (psychosis), **S**ocial supports lacking, **O**rganised plan, **N**o spouse (isolation), **S**ickness (chronic illness). Ten items, one point each, scored out of a possible ten. Hold it for what it is: a teaching aide that lists risk factors, explicitly not a validated instrument for predicting an individual's risk or for rationing care.

GOOD TO REMEMBER

- **SAD PERSONS (the aide):** a ten-item risk-factor mnemonic scored out of a possible ten; its defining limitation is that it is not validated to predict individual suicide and must not be used to allocate care, so it teaches factors rather than grades risk.
- **PHQ-9 item nine (the screen):** a single item on death and self-harm thoughts, scored 0 to 3 over two weeks; any positive answer mandates a full structured suicide risk assessment. It screens, it does not assess or grade risk.

19.9 The limits of individual-risk prediction

No instrument predicts individual suicide; the assessment guides care, it does not forecast. This is the intellectual centre of the topic and its most heavily tested principle. Suicide is, at the

level of the individual, a rare and fluctuating event, and the evidence is consistent that no scale, algorithm or clinical judgement reliably identifies which person will act; this is the **limits of prediction** (Kaplan and Sadock CTP 2024; NICE NG225 2022). Two practical consequences follow. First, risk-assessment and stratification *tools* must not be used to predict future suicide or self-harm, nor to decide who is offered treatment or admission; a global "low, medium, high" label must not drive clinical decisions. Second, the field has moved from prediction toward a prevention-oriented *suicide risk formulation*, which summarises the gathered data into four judgements that inform action rather than forecast an event: the patient's *risk status* (relative to a comparison group), their *risk state* (relative to their own baseline or other time-points), the *resources* they can draw on in crisis, and the *foreseeable changes* that could raise risk (Pisani, Murrie and Silverman 2016). The purpose of the assessment is to identify modifiable, treatable factors and to shape a safety-focused plan, not to generate a number.

FROM PREDICTION TO PREVENTION-ORIENTED FORMULATION

The traditional model sorted patients into low, medium and high risk and stopped there, which both over-labels and mis-directs care. The prevention-oriented formulation instead asks four action-linked questions, risk status, risk state, available resources and foreseeable changes, and uses the answers to build management and a collaborative safety plan. Stratifying into fixed categories to allocate care is discouraged precisely because it borrows a predictive authority the data do not support, while diverting attention from the modifiable factors that treatment can actually change.

GOOD TO REMEMBER

- **Limits of prediction (the governing principle):** no tool or clinician reliably predicts individual suicide; risk-stratification scores must not be used to predict or to ration care. The assessment identifies modifiable factors and drives a safety plan, and the field favours a prevention-oriented formulation (risk status, risk state, resources, foreseeable changes) over a low-medium-high label.

19.10 From assessment to safety: the management link

The assessment earns its keep only when it produces a safety plan and the right level of care. A risk assessment is a means to safety, not an end; the guideline-anchored next steps are a collaborative safety plan, means-restriction counselling framed positively, a disposition matched to need, and treatment of the driving disorder (NICE NG225 2022; WHO LIVE LIFE 2021). The Stanley-Brown Safety Planning Intervention is the standard collaborative tool, built *with* the person: recognise personal warning signs, use internal coping strategies, use social contacts and settings that provide distraction, identify people who can help, list professionals and crisis or helpline contacts, and make the environment safer by reducing access to means. Means restriction, counselled as putting time and distance between a person and a crisis and involving family where appropriate, is the single most robustly evidenced lever and is always framed positively and never punitively. The level of care, community follow-up, crisis-team involvement or admission, is matched to assessed need and safety, with rapid follow-up after any emergency contact because the transition period is high-risk. Where a driving disorder is present, its

treatment is itself anti-suicidal, and two agents carry specific evidence: lithium in mood disorders and clozapine in schizophrenia; their pharmacology, dosing and monitoring sit in the Mood disorders: pharmacotherapy notes and the Schizophrenia: management, antipsychotics and adverse effects notes, and the acute triage of the actively suicidal patient in crisis is developed in the emergency psychiatry chapter.

GOOD TO REMEMBER

- **Safety planning and means restriction (the management step):** the guideline first move after assessment (NICE NG225 2022) is a collaborative safety plan (the Stanley-Brown components) plus positively framed means-restriction counselling, then a level of care matched to need with rapid follow-up. Means restriction is the highest-yield lever; drugs treat the driving disorder (lithium in mood disorders, clozapine in schizophrenia, both with specific anti-suicidal evidence) and supplement, never replace, the plan.

19.11 India: the Mental Healthcare Act 2017 and the decriminalised attempt

The statute every Indian candidate must quote precisely. The pivotal Indian provision is Section 115 of the Mental Healthcare Act 2017 (VERIFIED against the Act and Indian Kanoon). It provides that any person who attempts to commit suicide is presumed, unless proved otherwise, to have severe stress, and shall not be tried or punished for it, effectively overriding the old Section 309 of the Indian Penal Code that had criminalised the attempt. The Act goes further and places a duty on the appropriate government to provide care, treatment and rehabilitation to a person under such severe stress, so as to reduce the risk of recurrence. The clinical translation is humane and examinable: a person who attempts suicide is presumed to be suffering, not offending, and is owed care rather than a charge. This reframes the clinician's role at the bedside toward compassionate assessment and safety planning, in keeping with the safeguarding, non-judgemental tone the whole topic demands.

INDIA

India carries a large share of the world's suicide deaths, and suicide is among the leading causes of death in young Indian adults, a burden that makes competent assessment a public-health priority; it is stated here without any reference to means. The legal frame is the anchor: **Section 115** of the Mental Healthcare Act 2017 decriminalised the attempt through a presumption of severe stress and a duty of care, superseding Section 309 of the Indian Penal Code, and this is reinforced by national helpline infrastructure such as Tele MANAS. The Indian Psychiatric Society endorses the shift from punishment to care. The population-level and public-health suicide-prevention deep dive, including the WHO LIVE LIFE strategy and gatekeeper programmes, is developed in the Community psychiatry and public health notes.

RULE **The attempt is decriminalised, not the person blamed.** Under Section 115 of the Mental Healthcare Act 2017 a person who attempts suicide is presumed to be under severe stress and shall not be punished, and the state owes them care to reduce recurrence.

5	10	4
C-SSRS IDEATION-SEVERITY LEVELS	SAD PERSONS ITEMS (SCORE OUT OF A POSSIBLE TEN)	PREVENTION-ORIENTED FORMULATION JUDGEMENTS

HOLD THIS

A suicide risk assessment is a compassionate **structured evaluation** that weighs static and dynamic risk factors against protective factors and separates **chronic and acute risk**, using the C-SSRS ideation ladder to structure the interview and PHQ-9 item nine to screen, while remembering that SAD PERSONS is only a teaching aide and that **no instrument predicts individual suicide**, so stratifying into low-medium-high to ration care is discouraged; the assessment exists to drive a collaborative safety plan and means restriction, a denied ideation is never a cleared risk, and in India the attempt is decriminalised by Section 115 of the Mental Healthcare Act 2017.

20 · Epidemiology and aetiology of suicide

This is a CORE topic in the suicide band, and it is examined as two linked demands: state the **epidemiology of suicide** responsibly, and explain the **aetiological models** that make sense of it. The marks turn on framing the burden without sensationalising it, knowing the demographic and clinical risk patterns, placing the Indian picture with its student and agrarian dimensions accurately, and holding the three complementary explanatory frames: the individual-level **stress-diathesis model** that integrates the field, Durkheim's sociological typology, and Joiner's interpersonal theory. Throughout, the register stays compassionate, non-judgemental and means-free.

How to use this page. Fix the vocabulary and the spectrum first; then the global **suicide burden** and who is most at risk; then the Indian context and the legal frame set by the Mental Healthcare Act; and finally the aetiology, working from the integrating stress-diathesis model out to the sociological and interpersonal theories. The one idea to carry off the page is that suicide is best read as an enduring vulnerability meeting an acute stressor, never as a simple reaction to circumstances.

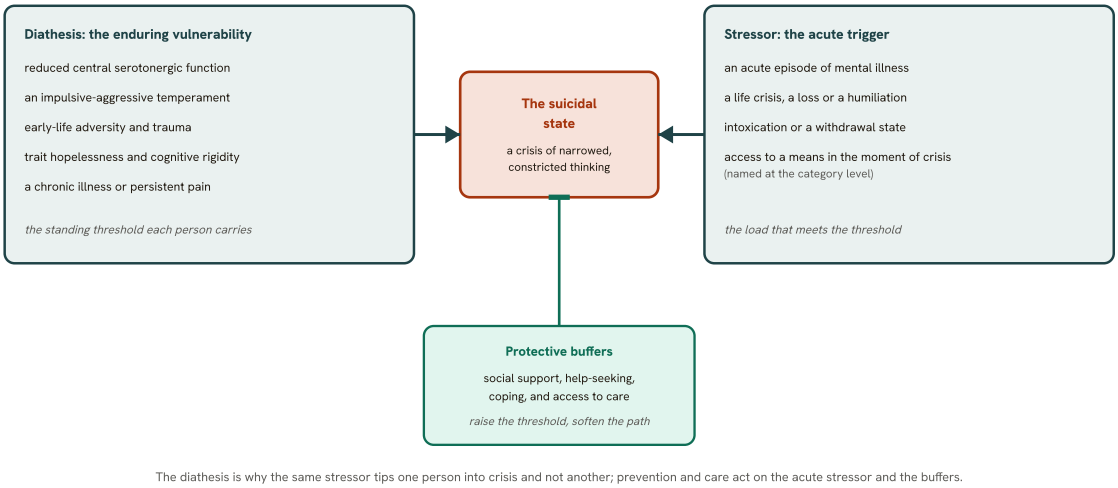

Fig 18.13 The stress-diathesis model of suicidal behaviour. Suicidal crises are best understood not as a simple product of a stressor but as the meeting of two things. Each person carries a standing diathesis, an enduring vulnerability made of reduced serotonergic function, impulsive-aggressive temperament, early adversity, trait hopelessness and chronic illness, which sets the threshold at which a crisis can occur. An acute stressor, an episode of illness, a loss or humiliation, or intoxication, then supplies the load that may meet that threshold, producing the suicidal state of narrowed, constricted thinking. Protective buffers, social support, help-seeking, coping and access to care, raise the threshold and soften the path. The model explains why an identical stressor tips one person and not another, and it shows where prevention and care can act, on the acute stressor and on the buffers.

20.1 The vocabulary and the spectrum of suicidal behaviour

Precise terms, because the examiner marks them. Suicidal phenomena form a spectrum, and the words are not interchangeable (Shorter Oxford Textbook of Psychiatry; Companion to Psychiatric Studies). Suicide is death caused by a self-directed act undertaken with at least some intent to die. Suicidal ideation ranges from a passive wish to be dead to active, planned thinking. A suicide attempt is a non-fatal self-directed act with some intent to die. Older literature used deliberate self-harm and parasuicide for intentional self-injury that need not carry a wish to die, and the acute triage of these presentations belongs to the emergency psychiatry chapter. The clinical point is that intent, not the medical severity of the act, defines where a presentation sits, and that the assessment stays needs-based and safety-focused rather than a graded checklist.

Suicidal ideation

thoughts of ending one's life, from a passive wish to be dead through to active planning

Suicide attempt

a non-fatal self-directed act carried out with some degree of intent to die

Suicide

death from a self-directed act undertaken with at least some intent to die

Deliberate self-harm

intentional self-injury that may or may not carry a wish to die; assessed for needs and safety, not by severity alone

20.2 The global suicide burden

A common, largely preventable cause of death. The **epidemiology of suicide** is examined first as scale. The World Health Organization reports that more than one in every hundred deaths worldwide is a suicide, and that suicide is the fourth leading cause of death among people aged 15 to 29 (WHO). More than three-quarters of suicides occur in low- and middle-income countries, so the **suicide burden** falls disproportionately on the settings with the least specialist provision (WHO; New Oxford Textbook of Psychiatry). The responsible way to state this in an exam is as a public-health problem that is common, unevenly distributed and amenable to prevention, keeping to counts and rates and never to method or graphic detail. The population and public-health approach to lowering these figures, including the highest-yield lever of reducing access to lethal means, is developed in the Community psychiatry and public health notes.

12	>1 in 100	4th	3
INDIA SUICIDE RATE PER 100,000 POPULATION (NCRB, 2021)	OF ALL DEATHS WORLDWIDE ARE SUICIDE (WHO)	LEADING CAUSE OF DEATH WORLDWIDE AT AGES 15 TO 29 (WHO)	COMPONENTS IN JOINER'S INTERPERSONAL THEORY

20.3 Who is most at risk: demographic and clinical patterns

Distal markers and the one proximal driver. Risk is patterned. Internationally more men die by suicide, while non-fatal self-harm is reported more often among women, a divergence usually called the gender paradox; part of the difference in case-fatality between groups reflects differences in the methods used and in help-seeking, which is one reason reducing access to lethal means is protective (Shorter Oxford; New Oxford). Risk rises at the extremes of adult age in many settings, and is higher among people who are unmarried, separated, widowed, socially isolated, unemployed or bereaved, and among those with chronic painful illness (Kaplan and Sadock CTP 2024). These are distal, largely fixed markers and they set the background rate. The single strongest proximal, and crucially modifiable, factor is active psychiatric illness: a mood disorder above all, and also alcohol and substance use, psychosis and personality disorder. Naming and treating that illness is where the epidemiology becomes clinical.

RULE **Treat the illness behind the statistic.** Active psychiatric illness, a mood disorder chief among them, is the strongest and most modifiable proximal risk factor for suicide; sociodemographic markers set the background rate, but the treatable target is the current disorder.

20.4 The Indian context

A rising rate, a young profile and a decriminalised act. India carries a large share of the global burden. Its national source is the National Crime Records Bureau (NCRB), whose annual Accidental Deaths and Suicides in India report is the primary dataset. The recorded suicide rate reached 12 per 100,000 (per lakh) population in 2021, up from about 10 per lakh in 2017, a steady year-on-year rise (NCRB, 2021). The majority of recorded deaths are among men, and the age profile skews young. Two dimensions are examined repeatedly and must be stated without sensationalising. The student dimension: NCRB records a marked and rising trend in suicides among students, widely linked to academic and examination stress. The agrarian dimension: the

farming sector, captured in NCRB's farmers and agricultural labourers category, is a recognised at-risk group tied to agrarian distress such as indebtedness and crop failure. The legal frame is a favourite: under Section 115 of the Mental Healthcare Act, 2017, a person who attempts suicide is presumed, unless proved otherwise, to be under severe stress and is not to be tried or punished, effectively overriding the old offence at Section 309 of the Indian Penal Code, and the appropriate government is placed under a duty to provide care and rehabilitation (Mental Healthcare Act 2017, Section 115).

INDIA

The Indian examiner expects three moves: the burden stated soberly from the NCRB source (a rate near 12 per lakh in 2021, up from about 10 per lakh in 2017), the two salient at-risk dimensions named (students, driven by examination and academic stress; and the farming sector, tied to agrarian distress), and the statute quoted precisely. Section 115 of the Mental Healthcare Act, 2017 decriminalises attempted suicide by presuming severe stress and directs the state toward care rather than punishment; the Indian Psychiatric Society has supported this reframing of the attempt as a signal of distress and a call for help.

GOOD TO REMEMBER

- **Section 115, Mental Healthcare Act, 2017 (the statute):** any person who attempts suicide is presumed, unless proved otherwise, to have severe stress and shall not be tried or punished, which in practice overrides Section 309 of the Indian Penal Code; the state carries a duty to provide care, treatment and rehabilitation. The first response to an attempt is compassionate assessment and care, not sanction.

20.5 Aetiological models: the stress-diathesis model as the integrating frame

An enduring vulnerability meeting an acute stressor. The **aetiological models** operate at three levels, and the individual-level frame that ties them together is the stress-diathesis model (associated with Mann and colleagues). It holds that suicidal behaviour arises when an acute state stressor acts on an enduring trait diathesis, and only when their combined load crosses a threshold (Kaplan and Sadock CTP 2024). The diathesis is the stable, trait-like vulnerability: a disposition to hopelessness and pessimism, impulsive-aggressive traits, a heritable component and the imprint of early-life adversity, associated at the biological level with reduced central serotonergic function. The stressor is the acute, state-dependent trigger: an episode of psychiatric illness, intoxication, or a humiliating interpersonal, financial or occupational crisis. The model earns its place because it explains what a stressor alone cannot: why the overwhelming majority of people exposed to severe adversity do not act, while the same stressor precipitates an act in a vulnerable individual. The accompanying figure sets the enduring diathesis and the acute stressor as two converging axes crossing a threshold, and it is the mental model to carry into any risk formulation.

TRAP **Reading a suicide as a simple reaction to circumstances.** Explaining an act by the precipitating stressor alone (a job loss, an exam failure, a relationship breakdown) misses the enduring diathesis and, with it, the treatable psychiatric illness that amplifies it; the stress-diathesis frame keeps both the vulnerability and the modifiable disorder in view.

GOOD TO REMEMBER

- **Stress-diathesis model (the defining structure):** suicidal behaviour emerges when an acute state stressor (a psychiatric episode, intoxication, a life crisis) acts on an enduring trait diathesis (hopelessness, impulsive-aggressive traits, reduced central serotonergic function, early adversity) and their combined load crosses a threshold. The discriminator from a purely social account is the enduring, trait-level vulnerability, which is why most people under severe stress do not act.

20.6 Sociological and interpersonal models: Durkheim and Joiner

The social frame: Durkheim's two axes. The sociological level was set by Durkheim, whose 1897 monograph argued that suicide rates track two social forces: the degree of social integration and the degree of social regulation (Shorter Oxford; Companion to Psychiatric Studies). Too little or too much of each yields a type. Low integration produces egoistic suicide, in a person cut off from the group and its shared meanings; excessive integration produces altruistic suicide, in which the self is sacrificed for the group or a cause. Low regulation, when the normative bonds that guide behaviour break down in times of upheaval, produces anomic suicide; excessive, oppressive regulation produces fatalistic suicide. The enduring value of the model is that it explains suicide as a social fact whose rate rises and falls with social conditions, not only as an individual event.

DURKHEIM TYPE	SOCIAL FORCE	THE DEFINING CONDITION
Egoistic	Integration	**Too little integration: the person is detached from the group and its shared meanings**
Altruistic	Integration	**Too much integration: the self is sacrificed for the group or a greater cause**
Anomic	Regulation	**Too little regulation: normative bonds break down amid social or economic upheaval**
Fatalistic	Regulation	**Too much regulation: life is over-controlled by oppressive rules or blocked futures**

Durkheim's four types arranged on the two axes of social integration and social regulation, each as a deficiency or an excess (Shorter Oxford; Companion).

PEARL

Hold Durkheim as a two-by-two: the *integration* axis gives **egoistic** (too little) and **altruistic** (too much); the *regulation* axis gives **anomic** (too little) and **fatalistic** (too much). Every type is a deficiency or an excess of one of the two forces.

The interpersonal frame: Joiner's desire and capability. At the psychological level, Joiner's interpersonal theory, the Interpersonal-Psychological Theory of Suicide, proposes that the desire to die arises from two interpersonal states: thwarted belongingness, the sense of being disconnected, excluded or alienated, and perceived burdensomeness, the belief that one is a burden on others (Kaplan and Sadock CTP 2024; DSM-5-TR 2022). Desire alone, however, is not enough for a serious attempt; the theory adds acquired capability, a fearlessness about pain, injury and death that is built up through repeated exposure to painful or provocative experiences. A near-lethal attempt requires all three: the desire (thwarted belongingness plus perceived burdensomeness) and the capability. The framework is clinically useful because it separates the wish from the ability to act, and because belongingness and burdensomeness are addressable in therapy.

MNEMONIC

DESIRE + CAPABILITY

Joiner's interpersonal theory needs both to converge on a serious attempt. DESIRE = thwarted **B**elongingness + perceived **B**urdensomeness (the two B's); CAPABILITY = the **acquired capability** to enact lethal self-harm, built by habituation to pain and fear. Wish plus ability, not wish alone.

GOOD TO REMEMBER

- **Durkheim's typology (the two axes):** suicide rates track social integration (egoistic = too little, altruistic = too much) and social regulation (anomic = too little, fatalistic = too much); each of the four types is a deficiency or excess of one force.
- **Joiner's interpersonal theory (the three components):** the desire to die comes from thwarted belongingness plus perceived burdensomeness, and a serious attempt additionally requires acquired capability (fearlessness about pain and death); all three must converge, which is why desire without capability, or capability without desire, does not produce a lethal act.

HOLD THIS

Suicide is an enduring diathesis (trait vulnerability: hopelessness, impulsive-aggressive traits, reduced serotonergic function, early adversity) meeting an acute stressor (a psychiatric episode above all, intoxication, or a life crisis) and crossing a threshold; epidemiology tells you who is at risk (men, the socially isolated, the young in India, students and those in agrarian distress, and anyone with active psychiatric illness), Durkheim explains the social rate through integration and regulation, and Joiner explains the individual act as desire (thwarted belongingness plus perceived burdensomeness) meeting acquired capability.

21 · Management of the suicidal patient and safety planning

This is the CORE management topic of the chapter, and it is examined as a sequence of clinical moves rather than a single fact. The **management of the suicidal patient** begins the moment the person is in the room: an engaging, non-judgemental relationship, a structured assessment that weighs risk against protective factors rather than a predictive score, a collaborative **safety plan**, a disposition matched to need, treatment of the disorder that is driving the risk, and active follow-up through the vulnerable weeks that come after. Get the order and the principles right

and the marks follow; the guideline spine here is NICE (NICE NG225 2022), which frames every step around compassion, needs and safety.

How to use this page. Fix the NICE position first, because it is the lead guideline and it sets two rules that reorganise older teaching: assess needs and safety through a person-centred interview, and do *not* use risk-stratification scores to predict future suicide or to decide who is offered care (NICE NG225 2022). Then learn the collaborative Safety Planning Intervention as the signature intervention, means restriction counselling framed positively, the level-of-care decision, the disorder-directed and anti-suicidal pharmacology, and the follow-up posture. The India anchor to carry is Section 115 of the Mental Healthcare Act, 2017. The acute triage of the person who is actively suicidal in the emergency room is developed in the emergency psychiatry chapter, and the population-level prevention deep dive sits in the Community psychiatry and public health notes. Throughout, the tone is safeguarding and the burden is stated without any reference to method.

Therapeutic stance

The engaging, warm, non-judgemental clinical posture that makes honest disclosure and collaboration possible; the vehicle every other step travels on.

Safety planning

A collaborative, prioritised written plan the person can use in a crisis, co-produced with the clinician; the Stanley-Brown Safety Planning Intervention.

Means restriction counselling

Working with the person and family to reduce access to lethal means, framed positively as putting time and distance between the person and a crisis.

Disposition

The level-of-care decision that matches the setting (community follow-up, crisis team, or admission) to assessed need and safety.

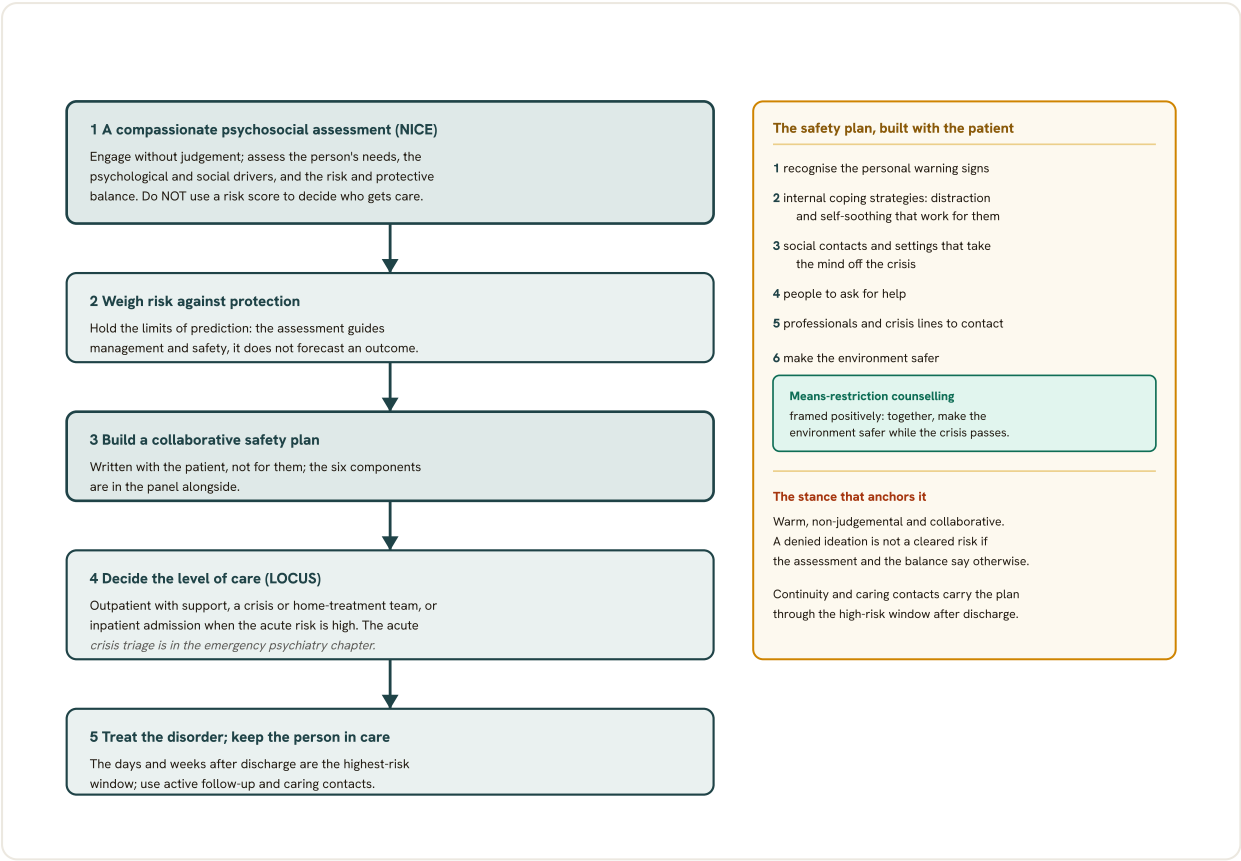

Fig 18.14 The management of the suicidal patient and safety planning. The pathway is anchored on a compassionate, non-judgemental psychosocial assessment, in the NICE tradition, that maps the person's needs and the drivers of their distress rather than reducing them to a risk score; guidance is explicit that a stratification score should not decide who receives care. The clinician then weighs the risk and protective balance, holding the limits of prediction, and builds a safety plan with the patient across its six components, from recognising warning signs to internal coping, social and professional contacts, and making the environment safer through means-restriction counselling framed positively. The level of care follows, from outpatient support through a crisis team to admission when acute risk is high, with the acute crisis triage taken up in the emergency psychiatry chapter. Because the period after discharge is the highest-risk window, active follow-up and caring contacts carry the plan forward.

21.1 The therapeutic stance: engage before you assess

A compassionate, non-judgemental relationship is the first intervention, not the preamble. The guideline foundation is that every person presenting after self-harm or with suicidal thoughts receives a compassionate, non-judgemental, needs-based assessment, regardless of the apparent medical seriousness of the act (NICE NG225 2022). The **therapeutic stance** means sitting with distress without recoiling from it, asking about suicidal thoughts directly and calmly (asking does not plant the idea), validating the pain that drives the thoughts, and conveying that help is possible. This stance is itself protective: it reduces the shame that keeps people from disclosing, and it is the precondition for an honest account of ideation, intent and access to means. NICE is explicit that the assessment is an intervention in its own right, an opportunity for engagement rather than a bureaucratic risk-scoring exercise. The clinician who leads with warmth gets the information the assessment depends on; the clinician who leads with a checklist often does not.

RULE **Engagement is the intervention, not the waiting room.** The compassionate, needs-based assessment is itself therapeutic (NICE NG225 2022); asking directly about suicidal thoughts does not increase risk, and the honest disclosure it earns is what every later step depends on.

GOOD TO REMEMBER

- **Therapeutic stance (the management step):** the guideline first move (NICE NG225 2022) is a compassionate, non-judgemental, needs-based assessment offered to everyone, whatever the medical severity of the act; ask about suicidal thoughts directly and calmly, treat the assessment as an engagement opportunity, and build the alliance before anything else.

21.2 Structured assessment and risk formulation, not a predictive score

Weigh risk against protective factors and current state; do not stratify to ration care. The examinable pivot of modern practice is that individual-level prediction of suicide is poor, risk fluctuates, and no tool reliably identifies who will act (NICE NG225 2022). The clinician therefore builds a **risk formulation** through a structured interview that weighs static and dynamic risk factors against protective factors, current mental state, intent and access to means, and the social context, and that arrives at a clinical judgement of current risk rather than a fixed low, medium or high label. NICE is unambiguous: do not use risk-assessment or stratification scales to predict future self-harm or suicide, and do not use their scores to determine who is offered treatment or admission. Structured instruments still have a role as aids to a thorough interview: the C-SSRS (Columbia Suicide Severity Rating Scale) is regarded as the gold-standard structured tool for assessing and monitoring suicidal ideation and behaviour, with five ideation levels of increasing severity (Clinical Methods in Psychiatry, AIIMS 2024); the older SAD PERSONS acronym scale and the ninth item of the PHQ-9 are screening aids; and SAFE-T (the Suicide Assessment Five-Step Evaluation and Triage) offers a five-step framework for structuring the interview and the disposition. All support, and none replace, the clinical formulation.

INSTRUMENT	WHAT IT IS	VERIFIED USE AND CAUTION
C-SSRS	Structured scale with five ideation levels of increasing severity, plus behaviour items	**Gold-standard aid (AIIMS 2024); supports, never replaces, the interview**
SAD PERSONS	Ten-item acronym-based screen, historically used in the emergency room	**A prompt only, not a substitute for clinical evaluation (AIIMS CL 2020)**
PHQ-9, the ninth item	Single screening item for thoughts of being better off dead or of self-harm	**A positive item triggers fuller assessment, never a decision on its own**
SAFE-T	Suicide Assessment Five-Step Evaluation and Triage framework	**Structures interview and disposition; a scaffold, not a predictive score**

Structured instruments as aids to the interview, with the NICE caution that scores must not predict risk or ration care (NICE NG225 2022); the reproduced forms sit with the suicide-risk assessment scales.

TRAP **Reading a denied ideation as a cleared risk.** A flat denial does not clear risk when the risk-and-protective balance, the collateral history and the mental state point the other way; formulate from the whole picture, and never let a global scale score or a single answer make the disposition (NICE NG225 2022).

GOOD TO REMEMBER

- **Risk formulation (the management step):** the guideline principle (NICE NG225 2022) is a structured, person-centred interview that weighs risk against protective factors, current state, intent and access to means, producing a clinical formulation rather than a predictive category; do not use stratification scores to predict or to ration care. The C-SSRS is the gold-standard structured aid but never a substitute for the interview.

21.3 Collaborative safety planning: the Safety Planning Intervention

Six prioritised steps the person can use in a crisis, co-produced not prescribed. The signature intervention is the Safety Planning Intervention (the Stanley-Brown Safety Plan), developed collaboratively for anyone at moderate to high risk when hospitalisation is not clinically indicated (Kaplan and Sadock CTP 2024; NICE NG225 2022). It is a written, personalised, prioritised ladder that the person moves down as distress rises: recognise personal warning signs; use internal coping strategies alone; draw on social contacts and settings that provide distraction; reach out to people who can be asked for help; contact professionals and crisis or helpline services; and make the environment safer by reducing access to lethal means. The plan is co-produced, kept where the person can find it, and reviewed and updated over time. Crucially it is a clinical instrument, not a promise: a signed no-suicide or no-harm contract has no evidence base and can falsely reassure the clinician, whereas the safety plan gives the person concrete, ranked actions and gives the clinician a structured way to involve family and services.

COMPONENT	WHAT IT LOOKS LIKE IN THE PLAN	THE POINT OF THIS RUNG
Recognise warning signs	The person's own thoughts, images, moods and situations that precede a crisis	**Catch the crisis early, at the person's own first signal**
Internal coping strategies	Things the person can do alone to take their mind off the crisis	**Self-soothing tried before anyone else is contacted**
Social contacts and settings	People and places that provide healthy distraction	**Distraction, not disclosure, at this rung**
People to ask for help	Family or friends the person can tell they are in crisis	**The first human help, when self-management is not enough**
Professionals and crisis contacts	Clinicians, crisis lines, helplines and emergency services	**The professional safety net, with numbers to hand**
Making the environment safer	Means restriction counselling, framed positively, with the person and family	**Time and distance between the person and a crisis**

The six components of the Safety Planning Intervention, a ranked ladder from internal coping to professional help ending in a safer environment (Stanley-Brown; NICE NG225 2022), laid out in the accompanying figure.

MNEMONIC

WISE-PS

The Safety Planning Intervention as a ranked ladder. **W**arning signs the person recognises; **I**nternal coping strategies used alone; **S**ocial contacts and settings for distraction; **E**nlist people to ask for help; **P**rofessional and crisis contacts; make the environment **S**afer through means restriction counselling. Six rungs, climbed only as far as each crisis needs.

RULE **A safety plan is a collaborative clinical act, not a contract.** The six-step Safety Planning Intervention is co-produced and works because the person descends a ranked ladder that ends in a safer environment; a signed no-suicide contract is not evidence-based and is not a substitute.

GOOD TO REMEMBER

- **Safety Planning Intervention (the management step):** the guideline-anchored intervention (Stanley-Brown; NICE NG225 2022; Kaplan and Sadock CTP 2024) is a collaborative, prioritised, written plan with six components, that being warning signs, internal coping, social contacts and settings for distraction, people to ask for help, professional and crisis contacts, and making the environment safer through means restriction. It is co-produced for moderate to high risk when admission is not indicated, and it replaces the discredited no-suicide contract.

21.4 Means restriction counselling: framed positively

Reduce access to lethal means, and frame it as time and distance, never as blame. The sixth rung of the safety plan is its own high-yield step. **Means restriction counselling** is a collaborative conversation about reducing access to lethal means, framed positively as putting time and distance between the person and a crisis so that an intense, often short-lived suicidal impulse can pass safely (NICE NG225 2022). The clinician works with the person, and where appropriate with family or carers, to make the immediate environment safer, at the category level of reducing access to lethal means rather than dwelling on any specific method. This is deliberately non-judgemental and non-alarming; it is offered as a shared act of care, not an accusation or a restriction imposed from outside. At the population level the same principle is the single highest-yield, most robustly evidenced suicide-prevention lever (WHO LIVE LIFE 2021), which is why the individual conversation matters so much; the population and public-health treatment of means restriction is developed in the Community psychiatry and public health notes.

GOOD TO REMEMBER

- **Means restriction counselling (the management step):** the guideline principle (NICE NG225 2022; WHO LIVE LIFE 2021) is to reduce access to lethal means, framed positively as putting time and distance between the person and a crisis, worked out collaboratively with the person and family and kept at the category level. It is the sixth safety-plan component and, at the population level, the highest-yield prevention lever.

21.5 Disposition: matching the level of care to need

Community, crisis team, or admission, decided by need and safety, not by a score. The **disposition** is the level-of-care decision, and it is matched to assessed need and safety rather than to a stratification label (NICE NG225 2022). The lowest-restriction option that keeps the person safe is preferred: outpatient management with a safety plan and social support where risk can be contained; a crisis resolution and home treatment team for intensive community support when risk is higher but hospitalisation can be avoided; and inpatient admission, voluntary wherever possible, when acute risk is high and cannot be managed safely in the community. Every step in the pathway includes rapid follow-up after any emergency contact and an active, documented handover, because the transition between services is itself a period of elevated risk. Where a person at imminent risk lacks capacity or declines care, admission may proceed under the relevant provisions of the Mental Healthcare Act, 2017. The acute triage of the person who is actively suicidal in crisis, the immediate **crisis intervention** in the emergency room, belongs to the emergency psychiatry chapter.

LEVEL OF CARE	WHEN IT FITS	THE SAFEGUARD THAT MUST TRAVEL WITH IT
Outpatient with support	Risk containable in the community, safety plan in place, supports available	**Rapid review and a documented follow-up appointment**
Crisis resolution / home treatment team	Higher risk needing intensive daily support, admission avoidable	**Frequent contact and a clear escalation route**
Inpatient admission	Acute risk high, cannot be kept safe in the community	**Voluntary where possible; least restriction that keeps the person safe**

The disposition ladder, from outpatient support to admission, chosen by assessed need and safety and not by a stratification score (NICE NG225 2022); sketched in the figure alongside.

WORKED EXAMPLE

A man in his forties is brought in by his wife after an episode of self-harm following job loss. Rather than reach for a scale score, the clinician spends the first minutes building rapport, then formulates: prominent hopelessness and recent loss on one side, a supportive spouse, help-seeking and no current intent on the other. Together they build a safety plan, moving through the man's own warning signs, a few internal coping steps, his brother as a first contact, and a crisis line; the last rung is a calm, positive conversation with the couple about making the home environment safer. Because risk is judged containable and support is strong, the disposition is outpatient management with a review within a few days and a crisis number in hand, rather than admission.

GOOD TO REMEMBER

- **Disposition (the management step):** the guideline principle (NICE NG225 2022) is to match the level of care, that being outpatient with support, a crisis or home-treatment team, or inpatient admission, to assessed need and safety at the least restriction that keeps the person safe; ensure rapid follow-up and an active handover at every transition, and take the immediate emergency-room crisis intervention to the emergency psychiatry chapter.

21.6 Treat the underlying disorder, and the anti-suicidal agents

The most durable risk reduction is treating the disorder that drives the risk. Suicidal risk usually sits on a treatable psychiatric disorder, most often a mood disorder, a psychotic illness, or a substance use disorder, and vigorous treatment of that disorder is central to management. Two agents carry a specific anti-suicidal effect that goes beyond simply treating the episode. Lithium reduces the risk of suicide and of death from any cause in mood disorders; the meta-analysis by Cipriani and colleagues, pooling 48 randomised controlled trials, established this signal, which is at least partly distinct from control of the mood episode itself (Cipriani 2013 BMJ), making lithium a specific reason to favour it in mood disorders carrying suicide risk. Clozapine is the only antipsychotic with a specific anti-suicidal indication; the International Suicide Prevention Trial showed it superior to olanzapine in reducing suicidal behaviour in schizophrenia and schizoaffective disorder at high risk (InterSePT, Meltzer 2003). Ketamine and intranasal esketamine produce a rapid, short-term fall in suicidal ideation as an adjunct within comprehensive care (the ASPIRE programme), but an effect on completed suicide is not established. The deep pharmacology of these agents lives elsewhere: lithium in the Mood disorders: pharmacotherapy notes, and clozapine in the Schizophrenia: management, antipsychotics and adverse effects notes.

AGENT	POPULATION AND EVIDENCE	WHAT TO HOLD
Lithium	Mood disorders; Cipriani 2013 BMJ, pooling 48 randomised trials	**Cuts suicide and all-cause death; effect partly independent of mood control**
Clozapine	Schizophrenia and schizoaffective disorder at high risk; InterSePT, Meltzer 2003	**Only antipsychotic with a specific anti-suicidal indication; beat olanzapine**
Ketamine / esketamine	Depression with acute suicidal ideation; the ASPIRE programme	**Rapid, short-term fall in ideation only; no proven effect on completed suicide**

The three anti-suicidal agents and the strength of their evidence; doses and full pharmacology sit in the mood and schizophrenia chapters, cross-referenced above.

GOOD TO REMEMBER

- **Lithium (the anti-suicidal drug):** reduces suicide and all-cause mortality in mood disorders, an effect partly distinct from mood control (Cipriani 2013 BMJ, 48 trials); favour it where a mood disorder carries suicide risk. Deep pharmacology in the Mood disorders: pharmacotherapy notes.
- **Clozapine (the anti-suicidal drug):** the only antipsychotic with a specific anti-suicidal indication, superior to olanzapine in high-risk schizophrenia and schizoaffective disorder (InterSePT, Meltzer 2003). Deep pharmacology in the Schizophrenia: management, antipsychotics and adverse effects notes.
- **Esketamine (the anti-suicidal drug):** rapid, short-term reduction in ideation as an adjunct (the ASPIRE programme); a reduction in completed suicide is not established, so it supplements rather than replaces comprehensive care.

21.7 Follow-up and continuity: the high-risk period after discharge

The weeks after an emergency contact or discharge are the most dangerous, so follow-up must be active. Management does not end at the plan or the discharge letter. Transitions between services and the period soon after discharge from inpatient care carry markedly elevated risk, so continuity of care with a prompt, actively arranged follow-up is essential rather than optional (NICE NG225 2022). The clinician ensures a named follow-up is booked before the person leaves, that the safety plan travels with them, and that the handover to community services is explicit and closed-loop. A low-cost, well-recognised adjunct in this window is the use of **caring contacts**, brief, non-demanding follow-up messages or letters that simply convey that the service is thinking of the person and remains available; they are offered as a supportive supplement to, not a replacement for, active clinical follow-up. The aim across the whole period is to keep the person connected to care exactly when disengagement is most dangerous.

6	5	4	115
SAFETY PLANNING INTERVENTION COMPONENTS	C-SSRS IDEATION LEVELS	WHO LIVE LIFE INTERVENTIONS	MENTAL HEALTHCARE ACT 2017 SECTION DECRIMINALISING THE ATTEMPT

PEARL

The post-discharge window is where a good inpatient stay is either consolidated or lost. Book the follow-up before discharge, send the safety plan out with the person, and treat any missed appointment in this window as a signal to reach out actively rather than to wait; caring contacts are the cheap, humane way to hold the thread.

GOOD TO REMEMBER

- **Follow-up and continuity (the management step):** the guideline posture (NICE NG225 2022) is that the period after an emergency contact or discharge is high-risk, so arrange prompt, active follow-up with an explicit closed-loop handover, send the safety plan with the person, and use caring contacts as a supportive adjunct. Disengagement is most dangerous exactly here, so continuity is a safety intervention.

21.8 India: Section 115 of the Mental Healthcare Act, 2017

The attempt is decriminalised, and severe stress is presumed. The India anchor for this topic is a statute, not a scale. Under Section 115 of the Mental Healthcare Act, 2017, any person who attempts suicide is presumed, unless proved otherwise, to have severe stress, and shall not be tried and punished for the attempt; this provision effectively overrides the older Section 309 of the Indian Penal Code (Mental Healthcare Act, 2017, Section 115). The same section places a duty on the appropriate government to provide care, treatment and rehabilitation to a person who has attempted suicide, to reduce the risk of a recurrence. For the clinician this reframes the encounter decisively: the person who has attempted suicide is a patient in severe distress owed care, not an offender, and the entire management sequence in this topic follows from that stance. The wider burden of suicide in India is substantial and is a public-health priority; it is stated here without any reference to method, and the population-level response is taken up in the Community psychiatry and public health notes.

INDIA

Section 115 of the Mental Healthcare Act, 2017 is the single most examinable India point on this topic: a person who attempts suicide is presumed, unless proved otherwise, to be under severe stress and shall not be tried or punished, effectively overriding Section 309 of the Indian Penal Code, and the government carries a duty to provide care, treatment and rehabilitation. The Indian Psychiatric Society has supported this decriminalisation. Read every suicidal presentation through this lens, that being distress owed care rather than conduct to be judged.

GOOD TO REMEMBER

- **Section 115, Mental Healthcare Act, 2017 (the statute):** a person who attempts suicide is presumed, unless proved otherwise, to have severe stress and shall not be tried or punished, effectively overriding Section 309 of the Indian Penal Code; the government has a duty to provide care, treatment and rehabilitation. The defining point is decriminalisation with a presumption of severe stress.

HOLD THIS

The management of the suicidal patient runs as a sequence: an engaging, non-judgemental therapeutic stance; a structured assessment that weighs risk against protective factors and current state, a formulation rather than a predictive score (and never a stratification score to ration care, NICE NG225 2022); a collaborative six-step Safety Planning Intervention ending in positively framed means restriction counselling; a disposition matched to need across outpatient support, crisis team, or admission; vigorous treatment of the underlying disorder, with lithium reducing suicide in mood disorders (Cipriani 2013 BMJ) and clozapine the only antipsychotic with a specific anti-suicidal indication (InterSePT, Meltzer 2003); and active follow-up through the high-risk post-discharge weeks with caring contacts. In India, Section 115 of the Mental Healthcare Act, 2017 presumes severe stress and decriminalises the attempt.

22 · Deliberate self-harm

This is the topic where a compassionate stance and a guideline become the same thing. Deliberate self-harm is any intentional self-poisoning or self-injury, carried out irrespective of motive and

irrespective of the degree of suicidal intent, so the term deliberately spans the person who wished to die, the person who did not, and every ambivalent state between. The examiner tests two moves: the clean *discrimination* between self-harm, non-suicidal self-injury and a suicide attempt, and the *management* that flows from a single landmark guideline. That guideline is NICE NG225 (Self-harm: assessment, management and preventing recurrence, 2022), and it reframed the field so decisively that the whole of **self-harm management** can be reconstructed from its two central positions: every episode earns a full, needs-based psychosocial assessment, and risk-stratification scores must never be used to predict future self-harm or to decide who receives care.

How to use this page. Fix the vocabulary first, then the relationship between self-harm and suicide (any self-harm raises future suicide risk, whatever the apparent medical seriousness), then the NICE NG225 stance as the lead: a **compassionate non-judgemental**, needs-based psychosocial assessment for every presentation, no risk score used to ration treatment, a collaborative safety plan, the carefully stated harm-minimisation debate, and continuity of care. Throughout, safe-messaging is absolute: the burden and the risk are stated without any reference to method, and access to means is named only at the category level. The accompanying management-and-safety-planning algorithm, drawn at the management topic, also maps the self-harm pathway, so this page refers to it generically rather than redrawing it.

22.1 What deliberate self-harm is, and what it is not

An umbrella term that spans intent, not a synonym for a suicide attempt. The defining feature of self-harm is that it is intentional self-poisoning or self-injury *regardless* of the person's motive or the degree of suicidal intent (NICE NG225 2022; Kaplan and Sadock CTP 2024). This is why the guideline prefers the bare term "self-harm" to the older "deliberate self-harm (DSH)": it refuses to assume a wish to die and refuses to grade the act by its apparent medical seriousness. Sitting inside that umbrella are two narrower ideas held apart by intent. Non-suicidal self-injury (NSSI) is intentional self-inflicted injury to the body undertaken with the expectation that it will cause only minor physical harm and with no intent to die, a distinct entity coded in the ICD-11 (MB23.E); it is frequently a means of regulating unbearable affect rather than an attempt to end life. A *suicide attempt*, by contrast, is a self-directed, potentially injurious act carried out with at least some intent to die, however ambivalent. The clinician's first task is therefore not to label the method but to establish where on this intent-spanning field the person stands, because that is what the assessment, never the score, then addresses.

TERM	DEFINING FEATURE	THE DISCRIMINATOR TO HOLD
Deliberate self-harm (self-harm)	Intentional self-poisoning or self-injury, irrespective of motive or suicidal intent	**The umbrella term; does not, by itself, imply a wish to die**
Non-suicidal self-injury (NSSI)	Self-inflicted bodily injury with the expectation of only minor harm and no intent to die (ICD-11 MB23.E)	**The subset explicitly without suicidal intent, often to regulate affect**
Suicide attempt	A self-directed, potentially injurious act with at least some intent to die	**Intent to die is present, however ambivalent or fleeting**

The terminology of self-harm, arranged by the single axis of suicidal intent; the umbrella and its two subsets separate cleanly on that axis (NICE NG225 2022; ICD-11 CDDR).

GOOD TO REMEMBER

- **Deliberate self-harm (the definition):** intentional self-poisoning or self-injury irrespective of motive or degree of suicidal intent, so it is the broadest term and spans intent rather than assuming it. The discriminator to hold is that NSSI is the intent-free subset (ICD-11 MB23.E, expectation of only minor harm, no wish to die) while a suicide attempt carries at least some intent to die; the act is never graded by its apparent medical seriousness.

22.2 The relationship to suicide

Self-harm and suicide are neither identical nor safely separable: any self-harm raises future suicide risk. The two are distinct in the moment, because self-harm can occur with or without suicidal intent, yet they are bound together over time (NICE NG225 2022; New Oxford Textbook of Psychiatry). The examinable principle is that any episode of self-harm, including an episode with no suicidal intent at the time, is among the strongest markers of later suicide, and the risk of suicide is substantially elevated in the period immediately after a presentation. This has an immediate clinical consequence that the guideline insists upon: the apparent medical triviality of an act carries no reassurance about future risk, so a superficial injury or an apparently minor act is not a lesser presentation and must not be triaged as one. A history of prior self-harm is likewise one of the strongest static risk factors weighed in any suicide risk assessment, a link developed in the assessment topic. The safeguarding reading is the humane one: a person who has harmed themselves has signalled distress and elevated risk, and is owed a full assessment and active care rather than a rapid discharge.

RULE **Any self-harm raises future suicide risk, whatever the method or medical seriousness.** An episode with no suicidal intent still marks elevated future risk; the triviality of the act never justifies a lesser response, and every presentation earns a full assessment.

GOOD TO REMEMBER

- **Self-harm and suicide (the relationship):** self-harm occurs with or without suicidal intent and is distinct from a completed attempt in the moment, yet any self-harm, including non-suicidal episodes, is among the strongest predictors of subsequent suicide and the risk is highest soon after a presentation. The point to hold is that apparent medical seriousness does not grade future risk.

22.3 The NICE NG225 lead: a needs-based psychosocial assessment for every episode

The single first move, for every presentation, is a compassionate needs-based psychosocial assessment. The leading position of NICE NG225 (2022) is that every person presenting after an episode of self-harm should receive a full **psychosocial assessment**, carried out in a compassionate non-judgemental manner, regardless of the apparent medical seriousness of the act and regardless of whether they are seen in an emergency department, in primary care or on a ward. The assessment is *needs-based*, not risk-scored: it explores the person's own needs, the psychological and social factors that drove and surround the episode (the precipitants, the mental state, the relationships, the losses, the supports), and the risks to their safety, and it does so as an engagement, not an interrogation. NICE is explicit that this assessment is itself therapeutic, an intervention in its own right rather than a gate to one, because being heard without judgement is what re-establishes safety and the willingness to accept help. This is the anchor of self-harm management, and every downstream step (the safety plan, the level of care, the aftercare) is built on it.

RULE **Every episode gets a full compassionate psychosocial assessment (NICE NG225).** Whatever the apparent medical seriousness and wherever the person is seen, the needs-based assessment of needs, psychosocial factors and safety is the guideline first move, and it is itself therapeutic.

PEARL

Treat the psychosocial assessment as an intervention, not a triage form. NICE NG225 frames the compassionate, unhurried, non-judgemental conversation as therapeutic in itself: it validates distress, rebuilds engagement, and is often the moment a person first accepts help. Asking directly and calmly about self-harm and suicidal thoughts does not plant the idea or raise risk, a myth worth retiring on sight.

GOOD TO REMEMBER

- **The psychosocial assessment (the management step, NICE NG225 2022):** a compassionate non-judgemental, needs-based assessment for every episode covering the person's needs, the psychological and social factors, and the risks, given regardless of the medical seriousness of the act. The guideline principle to hold is that it is itself an intervention, not a gateway that screens people out of care.

22.4 The critical position: risk-stratification tools must not gate care

Do not use a risk score to predict future self-harm, and never to decide who is offered treatment. This is the position on which NICE NG225 (2022) most sharply broke with earlier practice, and it is the single most heavily tested point in the topic. Risk-assessment and stratification tools and scales must not be used to predict future self-harm or suicide, and must not be used to determine who is offered treatment, admission or discharge. A global label of "low", "medium" or "high" risk must not drive any clinical decision, because such instruments perform poorly at identifying who will act at the individual level and, used as a gate, they systematically deny care to people who need it. What replaces the score is the person-centred assessment of the preceding subtopic: the individualised weighing of needs, psychosocial factors and safety. The

instruments may still have narrow uses, for structuring a conversation or prompting domains, but the moment a number is used to ration care the guideline has been broken.

APPROACH	WHAT IT DOES	NICE NG225 VERDICT
Needs-based psychosocial assessment	Weighs the person's needs, psychosocial context and safety, individually, every episode	**Endorsed as the standard for every presentation**
Risk-stratification tool or global score	Sorts the person into low, medium or high to predict future self-harm	**Must not be used to predict, nor to decide who is offered care or admission**

The reframe at the centre of NICE NG225: a needs-based assessment governs care, a stratification score never does (NICE NG225 2022).

TRAP **Using a risk-stratification score to withhold care.** Reading a "low risk" label off a scale and discharging on the strength of it is the cardinal error NICE NG225 forbids; the score does not predict the individual, and it must never decide who is offered treatment.

COMMON TRAP

A young person is seen after a first episode of self-harm, appears calm, and a stratification tool returns a "low" global score. Discharging on that number, without the compassionate needs-based assessment, is exactly the practice NICE NG225 was written to end: the calm presentation and the low score neither predict future risk nor address the psychosocial drivers of the episode. The person is instead offered a full psychosocial assessment, a collaborative safety plan and active follow-up. The number never governs the disposition.

GOOD TO REMEMBER

- **Risk-stratification tools (the critical NICE NG225 position):** do not use risk-assessment or stratification scores to predict future self-harm or suicide, and do not use them to decide who is offered treatment or admission. A low-medium-high label must not drive care; a person-centred assessment of needs and safety does. The trap to hold is treating a "low risk" score as a licence to discharge.

22.5 Safety planning and access to help

A collaborative safety plan, built with the person, plus positively framed means-restriction counselling. After the assessment, the guideline first move is a collaborative safety plan developed *with* the person, together with clear, concrete information on how to get help during a crisis (NICE NG225 2022). The standard structure is the Stanley-Brown Safety Planning Intervention, whose components map directly onto self-harm: recognise personal warning signs; use internal coping strategies; use social contacts and settings that provide distraction; identify people who can be asked for help; list professionals and crisis or helpline contacts; and make the environment safer by reducing access to means. Means restriction is named only at the category level and is always framed positively and collaboratively, as putting time and distance between the person and a crisis rather than as a punitive removal, and family or carers are involved where

appropriate. The plan is a living document, revisited and revised, not a form signed once, and giving the person a real route to help between contacts is part of what makes it work.

GOOD TO REMEMBER

- **Safety planning (the management step, NICE NG225 2022):** build a collaborative safety plan with the person (the Stanley-Brown components) and give clear crisis-access information; counsel on reducing access to means, framed positively as time and distance from a crisis, involving carers where appropriate. The guideline principle to hold is that the plan is collaborative and revisited, and means restriction is named only at the category level, never punitively.

22.6 The harm-minimisation debate, stated carefully

Harm minimisation is an adjunct considered only within a wider plan, for people who continue to self-harm, and its evidence and ethics remain contested. For a person who continues to self-harm despite treatment, an individualised harm-minimisation approach, aimed at reducing the frequency, severity and impact of episodes, may be *considered* as one part of a wider, individualised plan (NICE NG225 2022). It is never a first-line or stand-alone intervention, and it is offered only after, and alongside, a proper assessment and the attempt to treat the underlying difficulties. The guideline is careful, and so must the candidate be: the overarching aim always remains reducing and ultimately stopping self-harm, and the evidence base and the ethics of harm minimisation are genuinely contested, so it is stated as a considered, bounded option rather than an endorsed strategy. In an exam answer the safe formulation is to name it, place it firmly as an adjunct for continuing self-harm within a broader plan, and flag the contested evidence, without specifying techniques.

GOOD TO REMEMBER

- **Harm minimisation (the contested adjunct, NICE NG225 2022):** consider it only as part of a wider individualised plan for people who continue to self-harm, never as first-line or stand-alone; the aim stays reducing and stopping self-harm, and the evidence and ethics are contested. The principle to hold is that it is a bounded adjunct, not an endorsed strategy, and it is stated without specifying techniques.

22.7 Continuity of care, aftercare and the self-harm-focused intervention

The period after an episode is high risk; continuity of care and active handover are protective. Self-harm management does not end at the safety plan. NICE NG225 (2022) emphasises continuity of care and follow-up after every episode, because transitions between services, the move from an emergency department to community care, from a ward to home, from one team to another, are recognised periods of elevated risk that demand an active, named handover rather than a passive referral. Rapid follow-up after any emergency contact and a clear, agreed plan for who is responsible are the protective moves. Beyond immediate aftercare, the guideline supports offering a psychological intervention specifically tailored to self-harm, and, for adults with recurrent self-harm and emotional dysregulation, dialectical behaviour therapy has the strongest evidence; the specific modality and session structure are taken from the guideline itself and are flagged here rather than fixed. Where a driving disorder underlies the self-harm, treating it is part of care, and two agents carry specific anti-suicidal evidence, lithium in mood

disorders and clozapine in schizophrenia, whose pharmacology, dosing and monitoring sit in the Mood disorders: pharmacotherapy notes and the Schizophrenia: management, antipsychotics and adverse effects notes; the acute triage of the actively suicidal patient in crisis is developed in the emergency psychiatry chapter, and the population and public-health suicide-prevention deep dive in the Community psychiatry and public health notes.

GOOD TO REMEMBER

- **Continuity and aftercare (the management step, NICE NG225 2022):** ensure continuity of care and rapid follow-up after every episode, and treat every service transition as a high-risk period needing an active named handover. Offer a psychological intervention tailored to self-harm, with dialectical behaviour therapy best evidenced for adults with recurrent self-harm and emotional dysregulation; treat any driving disorder, using the anti-suicidal evidence for lithium and clozapine held in the mood and psychosis chapters.

22.8 India: the decriminalised attempt and the duty of care

A person who self-harms is presumed to be under severe stress and is owed care, not a charge. The Indian frame for any self-harm or attempt is Section 115 of the Mental Healthcare Act 2017 (verified against the Act and Indian Kanoon). It provides that any person who attempts to commit suicide is presumed, unless proved otherwise, to have severe stress, and shall not be tried or punished for the attempt, effectively overriding the old Section 309 of the Indian Penal Code that had criminalised it. The Act goes further, placing a duty on the appropriate government to provide care, treatment and rehabilitation to reduce the risk of recurrence. The clinical translation is exactly the safeguarding stance the whole topic demands: a person who has harmed themselves is suffering, not offending, and the bedside response is compassionate assessment and safety planning, not judgement. India carries a large share of the world's suicide burden, and self-harm is a common emergency presentation, a burden stated here without any reference to means; the Indian Psychiatric Society endorses the shift from punishment to care.

INDIA

The anchor is **Section 115** of the Mental Healthcare Act 2017: the attempt is decriminalised through a presumption of severe stress and a duty of care, superseding Section 309 of the Indian Penal Code. The clinical stance follows directly, compassionate assessment and a collaborative safety plan rather than a charge, reinforced by national helpline infrastructure such as Tele MANAS. The burden is real and large but is stated without method, in keeping with safe-messaging, and the population-level prevention strategy sits in the Community psychiatry and public health notes.

GOOD TO REMEMBER

- **Section 115, Mental Healthcare Act 2017 (the statute, India):** a person who attempts suicide is presumed to be under severe stress and shall not be punished, overriding Section 309 of the Indian Penal Code, and the state owes them care to reduce recurrence. The principle to hold is that self-harm is met with care, not criminal liability.

2022	0	2017
NICE NG225, THE LEADING SELF-HARM GUIDELINE	RISK-STRATIFICATION SCORES THAT MAY PREDICT SELF-HARM OR RATION CARE	MENTAL HEALTHCARE ACT, SECTION 115 (ATTEMPT DECRIMINALISED)

HOLD THIS

Deliberate self-harm is intentional self-poisoning or self-injury irrespective of intent, so it spans NSSI (no wish to die) and the suicide attempt alike, and any self-harm raises future suicide risk whatever its medical seriousness; **NICE NG225** makes the management a single stance, a compassionate non-judgemental needs-based **psychosocial assessment** for every episode, no risk-stratification score ever used to predict self-harm or to decide who is offered care, a collaborative safety plan with positively framed means restriction, harm minimisation only as a contested adjunct, and active continuity of care, while in India Section 115 of the Mental Healthcare Act 2017 presumes severe stress and owes the person care rather than a charge.

23 · Suicide prevention

This topic asks you to lift the gaze from the single patient to the population: not *how do I assess this person*, but *what actually lowers the suicide rate of a community*. The marks turn on holding one organising frame, the World Health Organization's **WHO LIVE LIFE** guide, and its four evidence-based interventions; on placing each intervention on the right tier of a three-level public-health model; on knowing which single lever carries the strongest evidence; and on stating the Indian frame, the **National Suicide Prevention Strategy** and the decriminalising statute, accurately. Throughout, the register stays compassionate, non-judgemental and free of any method detail, and **means restriction** is framed positively as putting time and distance between a person and a crisis.

How to use this page. Fix the public-health scaffold first (the three tiers and the universal, selective and indicated classification); then load the WHO LIVE LIFE four interventions as the spine; then the highest-yield population lever, means restriction; then the community tier (gatekeeper training, crisis lines and responsible media reporting) and the individual and pharmacological tiers; and finally the Indian national frame. The population and public-health deep dive, including the epidemiology behind these levers, is developed in the Community psychiatry and public health notes; this page is the prevention framework itself. The one idea to carry off the page is that the biggest gains in **suicide prevention** come not from better individual prediction but from population-level action that reduces access to lethal means.

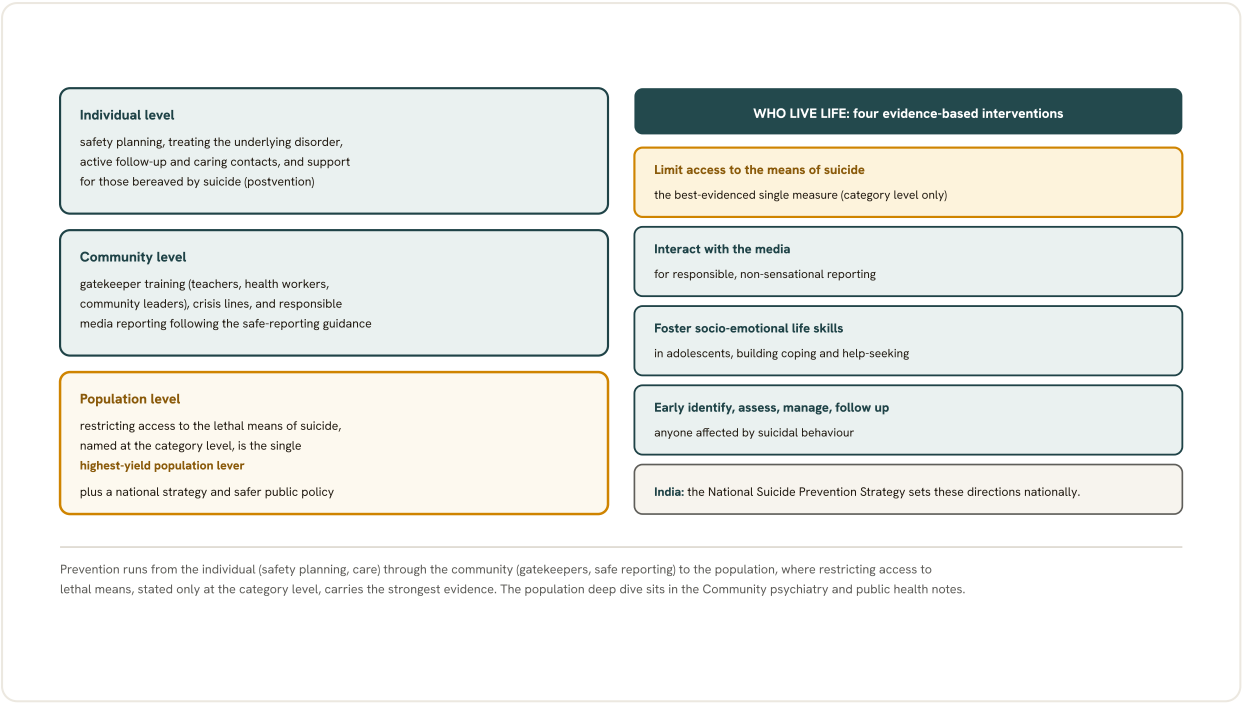

Fig 18.15 Suicide prevention. Prevention works across three levels. At the individual level it is safety planning, treatment of the disorder, active follow-up and caring contacts, and support for those bereaved. At the community level it is gatekeeper training, crisis lines and responsible, non-sensational media reporting. At the population level, restricting access to the lethal means of suicide, named only at the category level, is the single highest-yield lever, alongside a national strategy. The WHO LIVE LIFE framework gathers the four best-evidenced interventions, limiting access to means, engaging the media, fostering socio-emotional life skills in adolescents, and early identification and follow-up, and India expresses these through its National Suicide Prevention Strategy. The wider population and public-health treatment sits in the Community psychiatry and public health notes.

23.1 Prevention as a public-health task: the three tiers

From the clinic to the population. Because individual-level prediction of who will act is poor, prevention is best organised as a public-health task working across three tiers at once (New Oxford Textbook of Psychiatry; Kaplan and Sadock CTP 2024). The **individual tier** works with people already at risk or already affected: structured assessment, collaborative safety planning, follow-up and, above all, treating the underlying illness. The **community tier** works with at-risk groups and settings: gatekeeper training, crisis helplines, school-based life-skills programmes and responsible media reporting. The **population tier** works with everyone: reducing access to lethal means, reducing stigma, and the surveillance and strategy that underpin them. A complementary classification, borrowed from Gordon and used throughout the field, is universal, selective and indicated prevention: universal reaches a whole population regardless of risk (a canonical example is regulating access to hazardous means); selective targets a higher-risk subgroup (adolescents, farming communities); indicated targets identified individuals already showing warning signs. The two schemes map onto each other, and the examiner rewards the candidate who can place any given intervention correctly on the grid.

TIER	WHO IT TARGETS	EXEMPLAR INTERVENTIONS
Individual (indicated)	People already at risk or affected	**Structured assessment, collaborative safety planning, follow-up, treating the underlying disorder**
Community (selective)	At-risk groups and settings	**Gatekeeper training, crisis helplines, school life-skills, responsible media reporting**
Population (universal)	The whole population	**Means restriction (the highest-yield lever), stigma reduction, national strategy and surveillance**

The three-tier public-health model of prevention, aligned with the universal, selective and indicated classification (New Oxford; Kaplan and Sadock CTP 2024).

23.2 The WHO LIVE LIFE frame: four evidence-based interventions

The spine of every prevention answer. The World Health Organization LIVE LIFE implementation guide sets out four evidence-based interventions, and naming all four in order is the reliable way to structure an essay (WHO LIVE LIFE, 2021). They are: **L**imit access to the means of suicide; **I**nteract with the media for responsible reporting of suicide; foster socio-emotional **L**ife skills in adolescents; and **E**arly identify, assess, manage and follow up anyone affected by suicidal thoughts and behaviours. These four are supported by four foundational pillars (situation analysis, multisectoral collaboration, awareness-raising and capacity-building), underpinned in turn by adequate financing, surveillance, and monitoring and evaluation. The frame is deliberately cross-sectoral: it reaches beyond the health system into agriculture, media, education and regulation, which is precisely why it can move a population rate that clinical services alone cannot.

LIVE LIFE INTERVENTION	WHAT IT MEANS	TIER AND EVIDENCE
Limit access to means	Regulation reducing population access to lethal means	**Population; highest-yield, most robustly evidenced**
Interact with the media	Promote responsible, safe reporting; counter contagion	**Community; addresses the Werther effect**
Foster life skills	School-based socio-emotional and coping-skills programmes	**Universal, adolescent-directed**
Early identification and follow-up	Detect, assess, manage and actively follow up those affected	**Individual and clinical; gatekeeper-supported**

The four WHO LIVE LIFE interventions, each mapped to its prevention tier (WHO LIVE LIFE, 2021).

MNEMONIC

LIVE LIFE = LIMIT · INTERACT · LIFE-skills · EARLY

The four WHO interventions in order: **LIMIT** access to the means; **INTERACT** with the media for responsible reporting; foster **LIFE-skills** in adolescents; and **EARLY** identify, assess, manage and follow up. Four interventions on four pillars.

4	10%	1/3	48
WHO LIVE LIFE EVIDENCE-BASED INTERVENTIONS	NATIONAL SUICIDE PREVENTION STRATEGY TARGET REDUCTION IN SUICIDE MORTALITY BY 2030 (INDIA)	WHO GLOBAL TARGET REDUCTION IN THE SUICIDE RATE BY 2030	RANDOMISED TRIALS POOLED IN THE LITHIUM ANTI-SUICIDAL META-ANALYSIS (CIPRIANI, 2013)

GOOD TO REMEMBER

- **WHO LIVE LIFE (the four interventions):** Limit access to the means of suicide; Interact with the media for responsible reporting; foster socio-emotional Life skills in adolescents; Early identify, assess, manage and follow up. The guideline principle is that these four population and community levers, not better individual prediction, produce the largest fall in the suicide rate.

23.3 Means restriction: the single highest-yield lever

The one intervention with the strongest evidence. Of the four, restricting access to the means of suicide is the most robustly evidenced and highest-yield population-level intervention (WHO LIVE LIFE, 2021; New Oxford). It works because many suicidal crises are acute and ambivalent: putting time and distance between a person and a readily available lethal means allows the crisis to pass, and substitution to an equally lethal alternative is often incomplete. Canonical, method-free examples taught at the policy level are the regulation of highly hazardous pesticides in agriculture, limiting the pack sizes of commonly available over-the-counter medicines, and securing recognised structural hotspots with barriers. The accompanying figure sets the three prevention tiers in three levels with means restriction at the population base carrying the greatest population impact, and it is the mental model to reproduce. The full population and public-health case for this lever, with its epidemiological evidence, is developed in the Community psychiatry and public health notes.

RULE **Reducing access to lethal means is the highest-yield lever.** Across the whole toolkit, population-level means restriction has the strongest and most consistent evidence for lowering the suicide rate; it is the intervention to name first when asked what works.

TRAP **The substitution myth.** Assuming that a person prevented from one means will simply and inevitably find another is not borne out by the evidence; because crises are often transient and ambivalent, restricting access reduces deaths rather than merely displacing them.

GOOD TO REMEMBER

- **Means restriction (the guideline principle):** reducing population access to lethal means is the single highest-yield, most robustly evidenced suicide-prevention intervention (WHO LIVE LIFE, 2021). It is framed positively, as putting time and distance between a person and a crisis, and it acts at the population tier where clinical services cannot reach.

23.4 The community tier: gatekeeper training, crisis lines and responsible media

Widening the net beyond the clinic. The community tier equips non-specialists and shapes the information environment. Gatekeeper training equips frontline non-specialists (teachers, primary-care staff, community and religious leaders, helpline volunteers) to recognise warning signs and refer people at risk; it reliably improves skills and is highly acceptable in schools and workplaces, though a direct effect on completed suicide is harder to demonstrate and it works best as one component of a layered programme (Savita Malhotra; Dulcan). Crisis helplines and telephone or text lines provide accessible, immediate support at the point of distress. Responsible media reporting follows established safe-reporting guidance: avoid prominent or repetitive coverage, omit method and location detail, avoid framing suicide as a solution or romanticising it, and instead signpost help. This matters because sensational reporting can drive imitation, the Werther effect of copycat suicide, whereas coverage that models coping and recovery can be protective, the Papageno effect (New Oxford; Shorter Oxford). The acute triage of a person in active crisis who reaches these services belongs to the emergency psychiatry chapter.

PEARL

Hold the two named media effects as a mirror pair: the **Werther effect** is harm from irresponsible, sensational reporting (imitation and contagion); the **Papageno effect** is protection from responsible reporting that models coping and help-seeking. Responsible media reporting is the deliberate move from Werther toward Papageno.

GOOD TO REMEMBER

- **Gatekeeper training (the guideline role):** train non-specialists to identify and refer people at risk; it improves recognition and is highly acceptable, and it supports the early-identification intervention of WHO LIVE LIFE, but is delivered as part of a layered programme rather than as a stand-alone guarantee of reduced deaths.
- **Responsible media reporting (the guideline principle):** follow safe-reporting guidance (no method or location detail, no sensationalising or romanticising, signpost help); it shifts the population effect of coverage from the harmful Werther effect toward the protective Papageno effect.

23.5 The individual tier and the pharmacological prevention levers

Care, safety planning and the disorder-specific drugs that lower risk. At the individual tier, prevention means the early identification, assessment, management and active follow-up of people affected, with a collaboratively built safety plan (the Stanley-Brown Safety Planning Intervention components: warning signs, internal coping strategies, social contacts, people and professionals to call, and making the environment safer by restricting access to means) and rapid follow-up after any crisis contact, since transitions between services are a period of raised risk.

Two drugs carry a specific, evidence-based anti-suicidal effect and so act as pharmacological prevention levers. Lithium reduces the risk of suicide in people with mood disorders, a signal at least partly distinct from control of the mood episode itself, shown in the meta-analysis by Cipriani and colleagues pooling **48** randomised trials (BMJ, 2013); its full pharmacology is in the Mood disorders: pharmacotherapy notes. Clozapine is the only antipsychotic with a specific anti-suicidal indication, established by the International Suicide Prevention Trial in schizophrenia and schizoaffective disorder at high risk (InterSePT; Meltzer, 2003); its full pharmacology is in the Schizophrenia: management, antipsychotics and adverse effects notes. Treating the underlying disorder, and choosing an agent with an anti-suicidal signal where one exists, is itself prevention.

GOOD TO REMEMBER

- **Lithium (the guideline evidence):** favour lithium in a mood disorder carrying suicide risk; it reduces suicide and all-cause mortality versus placebo across 48 pooled randomised trials (Cipriani, BMJ 2013), an anti-suicidal effect at least partly independent of mood control.
- **Clozapine (the guideline indication):** clozapine is the only antipsychotic with a specific indication for reducing recurrent suicidal behaviour, in schizophrenia and schizoaffective disorder, established by the InterSePT trial (Meltzer, 2003).

23.6 The Indian national frame: strategy and statute

A first national strategy and a decriminalised act. India's prevention frame has two pillars the examiner expects. The first is the National Suicide Prevention Strategy, announced by the Ministry of Health and Family Welfare in 2022 as the country's first such strategy, with the stated goal of reducing suicide mortality by **10%** by 2030 against a 2019 baseline (a modest target beside the WHO Comprehensive Mental Health Action Plan aim of a **one-third** reduction in the suicide rate by 2030). Its actions are staged as immediate (within 1 to 3 years: advocacy for responsible media reporting, reducing access to a commonly used agrarian means through pesticide regulation, and strengthening surveillance and the mental-health workforce), intermediate (within 4 to 7 years: integrating mental health into general health care, training helpline workers, and reducing workplace stressors) and long term (within 8 to 10 years: destigmatisation, safer agricultural alternatives, and embedding well-being in education). The second pillar is the statute: under Section 115 of the Mental Healthcare Act, 2017, a person who attempts suicide is presumed, unless proved otherwise, to be under severe stress, and shall not be tried or punished, which effectively overrides the old offence at Section 309 of the Indian Penal Code, and the government carries a duty to provide care and rehabilitation. The Indian Psychiatric Society has supported this reframing of an attempt as a signal of distress and a call for help rather than a crime.

INDIA

Three moves win the Indian answer. Name the National Suicide Prevention Strategy (Ministry of Health and Family Welfare, 2022; the first national strategy) with its headline goal, a 10% reduction in suicide mortality by 2030. Quote Section 115 of the Mental Healthcare Act, 2017 precisely: attempted suicide is presumed to arise from severe stress and is not punished, overriding Section 309 of the Indian Penal Code, with a state duty of care. And weave in the two Indian dimensions the strategy targets, the agrarian sector (means regulation and safer farming) and students and adolescents (well-being curricula and life skills), consistent with the WHO LIVE LIFE emphasis on cross-sectoral action.

GOOD TO REMEMBER

- **National Suicide Prevention Strategy (the policy anchor):** India's first national strategy (Ministry of Health and Family Welfare, 2022); goal is a 10% reduction in suicide mortality by 2030 against a 2019 baseline, staged across immediate, intermediate and long-term actions spanning health, agriculture, media and education.
- **Section 115, Mental Healthcare Act, 2017 (the statute):** any person who attempts suicide is presumed, unless proved otherwise, to have severe stress and shall not be tried or punished (overriding Section 309 of the Indian Penal Code); the state must provide care, treatment and rehabilitation. The first response to an attempt is compassionate care, not sanction.

HOLD THIS

Prevention works in tiers (individual, community, population); the WHO LIVE LIFE guide gives the four evidence-based interventions (Limit access to means, Interact with the media for responsible reporting, foster Life skills, Early identify and follow up); of these, means restriction is the single highest-yield population lever; and the Indian frame is the National Suicide Prevention Strategy (2022, a 10% reduction goal by 2030) sitting on Section 115 of the Mental Healthcare Act, 2017, which decriminalises the attempt and mandates care.

24 · The neurobiology of suicidal behaviour

This is a short, high-yield topic that supplies the biological half of the picture built elsewhere in the chapter: it names the substrate of the enduring vulnerability, the **diathesis**, on which an acute stressor acts. Kaplan frames the **neurobiology of suicide** around a small number of recurring findings, and the examinable ones are three: a reduced central **serotonergic** function (indexed classically by low cerebrospinal-fluid 5-HIAA and tied to impulsive-aggressive and suicidal behaviour), a dysregulated stress axis (the **HPA axis**), and a set of emerging polyamine and inflammatory signals. The thread that binds them is the stress-diathesis (stress-vulnerability) model: these are trait-level, largely cross-diagnostic vulnerability markers, not tests that predict who will act (Kaplan and Sadock CTP 2024; New Oxford Textbook of Psychiatry).

How to use this page. Learn the serotonergic finding first, because it is the single most consistent and most examined; then the HPA-axis stress-response finding and what post-mortem study adds; then the newer polyamine and inflammatory signals and how the whole set constitutes the biological diathesis. The one idea to carry off the page is that these markers describe an enduring vulnerability and have limited value for predicting an individual, so the clinical task remains the

compassionate, structured assessment, and the tone throughout stays safeguarding and non-sensational.

24.1 Reduced central serotonergic function

The most consistent biological finding in the field. A decreased, dysregulated central serotonergic system in people who die by suicide and in those who attempt it, seen regardless of the psychiatric diagnosis, is a very strong correlate of suicidal behaviour and is described as one of the most consistent neurobiologic findings in psychiatry (Kaplan and Sadock CTP 2024). The classic in-vivo marker is a low concentration of the main serotonin metabolite, 5-HIAA (5-hydroxyindoleacetic acid), in the cerebrospinal fluid; central serotonergic function is also probed by the neuroendocrine fenfluramine challenge test and by PET of prefrontal metabolism (Tasman Psychiatry). Crucially, decreased central serotonin metabolism maps more tightly onto trait impulsive-aggression than onto any specific disorder: low CSF 5-HIAA is associated with impulsive, aggressive behaviour and with more medically serious, determined attempts, so it behaves as a trait vulnerability that raises the likelihood of an act when an acute episode and adverse circumstances coincide (Kaplan and Sadock CTP 2024).

MARKER OR METHOD	WHAT IT INDEXES	THE POINT TO HOLD
CSF 5-HIAA	The principal serotonin metabolite in cerebrospinal fluid	**Low CSF 5-HIAA marks reduced central serotonergic function and tracks impulsive-aggression**
Fenfluramine challenge	Prolactin response to a serotonin-releasing probe	**A blunted response signals reduced serotonergic responsiveness**
PET of prefrontal metabolism	In-vivo prefrontal serotonergic and metabolic activity	**Reduced prefrontal serotonergic and metabolic response**
Post-mortem 5-HT2A binding	Receptor-level change in the prefrontal cortex	**Altered 5-HT2A binding in the prefrontal cortex of those who died by suicide**

The measures of central serotonergic function used to demonstrate the core neurobiological finding; these findings form the diathesis axis of the stress-diathesis model (Tasman Psychiatry; Kaplan and Sadock CTP 2024).

RULE **Serotonergic hypofunction is the trait signal, not a diagnostic one.** Reduced central serotonergic function, indexed by low CSF 5-HIAA, is the most consistent biological correlate of suicidal behaviour and is cross-diagnostic, binding to impulsive-aggressive traits rather than to any single disorder.

GOOD TO REMEMBER

- **Reduced central serotonergic function (the core finding):** a decreased, dysregulated serotonergic system, marked by low CSF 5-HIAA and probed by the fenfluramine challenge and prefrontal PET, is one of the most consistent neurobiologic findings and correlates with suicidal behaviour irrespective of diagnosis. The discriminator to hold is that it is a trait-level marker tied to impulsive-aggression, more so than to any specific psychiatric disorder.

24.2 HPA-axis dysregulation: the stress response

The stress-response pathway that links life adversity to biology. The most studied pathway after the serotonergic one is the stress-response pathway mediated by the HPA axis (hypothalamic-pituitary-adrenal axis); disruption of this axis with altered glucocorticoid activity may increase the risk for suicidal behaviour (Kaplan and Sadock CTP 2024). The robust, exam-ready form of the finding is that HPA-axis hyperactivity, reflected in an abnormal dexamethasone suppression test (that is, non-suppression of cortisol), is a robust predictor of future completed suicide, primarily among patients in a major depressive episode. Post-mortem brain study of people who died by suicide adds the anatomy and the downstream chemistry: significant alterations in the prefrontal cortex and hippocampus (regions of emotion regulation and executive function), altered 5-HT2A binding, decreased BDNF (brain-derived neurotrophic factor, a neuroprotective signal), and dysregulated glutamatergic and GABA signalling. Taken together, the HPA-axis and neuroplasticity findings show how an enduring stress-response vulnerability is written into the brain.

TRAP **Reading a biomarker as a clinical predictor for the individual.** An abnormal dexamethasone suppression test or a low CSF 5-HIAA describes group-level risk and enduring vulnerability; these findings have limited practical value in the individual case, so the clinical decision still rests on the structured risk-and-protective assessment, never on a biological test.

GOOD TO REMEMBER

- **HPA-axis dysregulation (the stress-axis finding):** hyperactivity of the HPA axis, shown as cortisol non-suppression on the dexamethasone suppression test, is a robust predictor of future completed suicide, primarily in mood disorder; post-mortem study adds prefrontal and hippocampal change, altered 5-HT2A binding and reduced BDNF. The discriminator is that it is a stress-response vulnerability marker, robust at the group level yet of limited use for predicting an individual.

24.3 Polyamine and inflammatory signals, and the diathesis synthesis

The newer signals, and how the biology assembles into a diathesis. Beyond serotonin and cortisol, two emerging areas complete the compact picture. Changes in the polyamine system are reported alongside the serotonergic and HPA-axis changes as candidate substrates that make some individuals more vulnerable to suicidal behaviour (New Oxford Textbook of Psychiatry). Separately, a growing literature links inflammation, depression and suicide, drawing attention to neuroinflammation and the microbiome, and GABAergic signalling is a further active area (Kaplan and Sadock CTP 2024). Overlaying all of these, epigenetic mechanisms are the bridge from experience to biology: childhood adversity can alter, through DNA methylation and histone modification, the genes governing neurotrophic signalling and neurotransmission, and this is how early environment loads the diathesis. Assembled, the serotonergic hypofunction, the dysregulated HPA axis and these newer signals are precisely what the stress-diathesis (stress-vulnerability) model calls the diathesis: an enduring, partly heritable, partly experience-shaped trait vulnerability on which an acute stressor acts to produce an act (Kaplan and Sadock CTP 2024). Where a driving disorder is present, its treatment is itself anti-suicidal, and two agents

carry specific evidence whose pharmacology sits elsewhere: lithium in mood disorders, in the Mood disorders: pharmacotherapy notes, and clozapine in schizophrenia, in the Schizophrenia: management, antipsychotics and adverse effects notes.

INDIA

The biology gives a compassionate reading of the Indian statute. Because a suicidal act needs an enduring diathesis for an acute stressor to act upon, "severe stress" is a signal of suffering rather than of choice, which is exactly the presumption written into Section 115 of the Mental Healthcare Act 2017: a person who attempts suicide is presumed, unless proved otherwise, to be under severe stress and is not to be punished, and the state owes them care. The neurobiology and the law point the same way, toward assessment and care, not blame.

2	3	5-HT2A
STRESS-DIATHESIS COMPONENTS: AN ACUTE STRESSOR ACTING ON AN ENDURING DIATHESIS	CORE SYSTEMS STUDIED: SEROTONERGIC, HPA AXIS, NEUROPLASTICITY	THE SEROTONIN RECEPTOR ALTERED IN THE PREFRONTAL CORTEX POST-MORTEM

HOLD THIS

The neurobiology of suicidal behaviour is the biological **diathesis** of the stress-diathesis model: a reduced central **serotonergic** function (low CSF **5-HIAA**, tied to impulsive-aggression and cross-diagnostic, the single most consistent finding) plus a dysregulated **HPA axis** (cortisol non-suppression on the dexamethasone suppression test predicting completed suicide in mood disorder), with newer polyamine, inflammatory and epigenetic signals, all describing an enduring trait vulnerability that has limited value for predicting the individual, so care rests on the structured assessment, not a biological test.

25 · Anti-suicidal pharmacology

Why this topic sits apart. Almost every effective psychiatric drug lowers suicide risk indirectly, by lifting the depression, settling the mania or quietening the psychosis that drove the risk. The examinable heart of **anti-suicidal pharmacology** is narrower and sharper: two agents, and only two, carry a specific, trial-level anti-suicidal effect that is at least partly *distinct* from their control of the mood episode or the psychosis, and each maps to a single disorder group. Lithium reduces suicide and self-harm in mood disorders (Cipriani 2013 BMJ), and clozapine reduces recurrent suicidal behaviour in schizophrenia and schizoaffective disorder (InterSePT, Meltzer 2003, and an FDA indication). A third strand, ketamine and esketamine, is the emerging rapid option for suicidal ideation. The topic then closes on post-attempt management, the ordered clinical response after an act.

How to use this page. Hold the set as two "specific-signal" drugs plus one "emerging" drug, then the ordered post-attempt sequence. For each drug learn three things: the disorder it applies to, the trial or evidence that anchors it, and the one line that names its status (a class effect for lithium, the only antipsychotic so indicated for clozapine). The deep pharmacology, the doses, serum levels and monitoring, lives in the mood-disorder and schizophrenia chapters and is cross-

referenced here; this page is about the anti-suicidal *effect* and its evidence. Safe-messaging holds throughout: the discussion stays at the level of risk, evidence and care, never method.

25.1 Two specific signals against a disorder-directed background

Treating the disorder is itself anti-suicidal; two drugs add more. The default and most important principle is that reducing suicide risk pharmacologically means treating the underlying disorder effectively, because an untreated mood episode or active psychosis is itself the main driver of risk (Kaplan and Sadock CTP 2024). The examinable exception is that two agents carry a dedicated anti-suicidal effect that sits over and above symptom control and is tied to one disorder group each: lithium in mood disorders and clozapine in schizophrenia and schizoaffective disorder. A third, newer strand, ketamine and esketamine, gives a rapid but short-term effect on suicidal ideation rather than on completed suicide. These two specific signals stand against the disorder-directed background so the exception is not mistaken for the rule.

AGENT	DISORDER GROUP	ANCHORING EVIDENCE	STATUS TO STATE
Lithium	Mood disorders (unipolar and bipolar)	Cipriani 2013 BMJ meta-analysis, 48 RCTs	**A class effect specific to lithium, not shared by other mood stabilisers; reduces suicide and death from any cause**
Clozapine	Schizophrenia and schizoaffective disorder	InterSePT (Meltzer 2003), 2-year RCT versus olanzapine	**The only antipsychotic with an anti-suicidal FDA indication; reduces recurrent suicidal behaviour**
Ketamine and esketamine	Major depression with acute suicidal ideation	ASPIRE programme (esketamine)	**Emerging adjunct; rapid, short-term fall in ideation, no proven effect on completed suicide**

The examinable core of anti-suicidal pharmacology: two specific signals plus one emerging option, each read as disorder, evidence, and status (Cipriani 2013 BMJ; InterSePT, Meltzer 2003; ASPIRE).

RULE **Only two agents carry a specific anti-suicidal signal.** Beyond treating the disorder, only lithium (in mood disorders) and clozapine (in schizophrenia and schizoaffective disorder) have a dedicated, trial-level anti-suicidal effect; for each, name the trial and the status.

TRAP **Assuming the benefit is class-wide.** Lithium's anti-suicidal effect is not reproduced by valproate, lamotrigine or carbamazepine, and clozapine's is not reproduced by other antipsychotics; InterSePT's comparator olanzapine is an effective drug yet was inferior, so do not assume any mood stabiliser or any antipsychotic delivers the same protection.

25.2 Lithium: the anti-suicidal effect in mood disorders

Lithium lowers suicide in mood disorders, and it behaves as a class-specific property. The lithium anti-suicidal effect is the best-established in the field. The meta-analysis by Cipriani and colleagues (BMJ 2013;346:f3646) pooled 48 randomised controlled trials and found that lithium

reduced the risk of suicide and the risk of death from any cause compared with placebo, across unipolar and bipolar mood disorders (Cipriani 2013 BMJ). Two points make it examinable. First, the anti-suicidal signal is at least partly *distinct* from control of the mood episode itself, so it is a reason to favour lithium specifically when a mood disorder carries suicide risk, not merely a by-product of better mood control. Second, it looks like a **class effect of lithium**, seen with lithium and not clearly shared by the other mood stabilisers, which is why the choice of the mood stabiliser can matter for risk. The proposed substrate is a dampening of impulsive aggression, consistent with the serotonergic diathesis of suicidal behaviour, but the effect is claimed on the outcome evidence, not on a mechanism. The deep pharmacology, the therapeutic range, monitoring and toxicity, belongs to the Mood disorders: pharmacotherapy notes; here the point is the effect and the trial that anchors it.

PEARL

The word to hold is *distinct*. If lithium only prevented suicide by preventing relapse, any equally good mood stabiliser would match it, but they do not. The anti-suicidal effect tracks lithium specifically and is partly separable from episode control, which is exactly why it changes the prescribing calculus in a mood disorder with suicide risk rather than being a footnote to relapse prevention.

GOOD TO REMEMBER

- **Lithium anti-suicidal effect (evidence-anchored, Cipriani 2013 BMJ):** lithium reduces the risk of suicide and of death from any cause in mood disorders, pooled across 48 randomised controlled trials; the effect is at least partly distinct from mood-episode control and is a class effect specific to lithium, not shared by the other mood stabilisers, so it favours lithium where a mood disorder carries suicide risk. Deep pharmacology in the Mood disorders: pharmacotherapy notes.

25.3 Clozapine: recurrent suicidal behaviour in schizophrenia

Clozapine reduces recurrent suicidal behaviour in schizophrenia, and it is the only antipsychotic licensed for it. The clozapine persistent suicidality data come from the International Suicide Prevention Trial (InterSePT; Meltzer et al, Archives of General Psychiatry 2003;60:82-91). It was a double-blind randomised comparison that enrolled roughly 980 patients with schizophrenia or schizoaffective disorder judged to be at high suicide risk, assigned them to clozapine or to olanzapine, and followed them for up to 2 years. Clozapine was superior in delaying recurrent suicidal behaviour, with fewer clozapine-treated patients requiring hospitalisation or rescue intervention to prevent suicide (InterSePT, Meltzer 2003). This underpins the FDA-approved indication for reducing the risk of recurrent suicidal behaviour in schizophrenia and schizoaffective disorder, the only antipsychotic to hold such an indication. As with lithium, the effect appears partly distinct from control of the positive symptoms, so a persistent suicide risk in schizophrenia is itself a recognised reason to move to clozapine. The deep pharmacology, the titration and the mandatory haematological monitoring, belongs to the Schizophrenia: management, antipsychotics and adverse effects notes.

GOOD TO REMEMBER

- **Clozapine persistent suicidality (guideline-anchored, InterSePT, Meltzer 2003, and the FDA indication):** clozapine reduces recurrent suicidal behaviour in schizophrenia and schizoaffective disorder, shown in the InterSePT 2-year randomised trial where it outperformed olanzapine, and it is the only antipsychotic FDA-indicated for reducing suicide risk. Persistent suicidality in schizophrenia is a recognised reason to move to clozapine; deep pharmacology and monitoring in the Schizophrenia: management, antipsychotics and adverse effects notes.

25.4 Ketamine and esketamine: the emerging rapid option

A rapid, short-term effect on suicidal ideation, not a proven effect on completed suicide. Ketamine and intranasal esketamine (Spravato) produce rapid, short-lived reductions in suicidal ideation, often within hours, which is what makes them of interest when ideation is acute (Kaplan and Sadock CTP 2024; Maudsley Prescribing Guidelines). Esketamine has been studied specifically in major depressive disorder with acute suicidal ideation or behaviour (the ASPIRE programme). The honest reading of the evidence is that it supports a rapid effect on *ideation*, as an adjunct within comprehensive care, whereas a reduction in completed suicide has not been established. So it functions as a bridge, buying time while definitive treatment and safety planning take hold, and never as a stand-alone answer to risk.

GOOD TO REMEMBER

- **Ketamine and esketamine (guideline-anchored, ASPIRE programme):** a rapid, short-term reduction in suicidal ideation, studied for esketamine in major depression with acute suicidal ideation or behaviour; it is an adjunct within comprehensive care that buys time, not a proven reducer of completed suicide and not a stand-alone treatment.

25.5 Post-attempt management: the ordered response

An ordered sequence, not a single drug. Post-attempt management is the structured response after an act, and it is examined as a sequence. First comes medical stabilisation of the physical consequences to medical fitness; only then, once the person is medically fit, a compassionate, non-judgemental psychosocial assessment of needs, context and safety, which every episode warrants regardless of the apparent medical seriousness of the act (NICE NG225 2022). This feeds a collaborative safety plan with means-restriction counselling framed positively, and, running underneath all of it, treatment of the underlying disorder, which is the main pharmacological lever against future risk and where the two specific agents above may enter. A recurring pitfall the guideline names is using a risk-stratification score to decide who is offered care; risk is assessed to guide a needs-based, collaborative response, not to ration it. The acute triage of the actively suicidal patient in crisis is covered in the emergency psychiatry chapter, and the population-level, public-health side of prevention in the Community psychiatry and public health notes.

STEP	THE ACTION	THE PRINCIPLE
Medical stabilisation	Treat the physical consequences of the act to medical fitness first	**Life and physical safety precede any psychiatric disposition**
Psychosocial assessment	Compassionate, non-judgemental assessment of needs, context and safety, every episode	**Needs-based, never a risk score (NICE NG225 2022)**
Safety plan and means counselling	Build a collaborative safety plan; counsel on reducing access to lethal means	**Means restriction is the highest-yield step, framed positively**
Treat the underlying disorder	Optimise treatment of the driving disorder; consider lithium or clozapine where indicated	**Effective disorder treatment is itself anti-suicidal**

The post-attempt management sequence, medical stabilisation then needs-based psychosocial assessment, safety planning and disorder treatment, with those post-attempt steps set in order (NICE NG225 2022).

INDIA

The statute frames post-attempt management compassionately. Under Section 115 of the Mental Healthcare Act, 2017, a person who attempts suicide is presumed, unless proved otherwise, to be under severe stress, and is not to be tried or punished, with a duty on the government to provide care, treatment and rehabilitation to reduce the risk of recurrence (Mental Healthcare Act 2017, Section 115). The clinical sequence and the law point the same way, toward assessment and care rather than blame. Both specific agents are available and used in India: lithium is inexpensive and widely prescribed, and clozapine is available with the mandatory haematological monitoring, so the anti-suicidal evidence is directly actionable here. The Indian Psychiatric Society position aligns with this care-first, means-restriction framing.

GOOD TO REMEMBER

- **Post-attempt management (guideline-anchored, NICE NG225 2022):** an ordered response, medical stabilisation of the physical consequences first, then a compassionate, non-judgemental psychosocial assessment of needs and safety in every episode, a collaborative safety plan with positively framed means-restriction counselling, and treatment of the underlying disorder, which is itself the main anti-suicidal lever. A risk-stratification score must never decide who is offered care. Acute crisis triage is in the emergency psychiatry chapter.

48	2	1
RANDOMISED TRIALS POOLED IN THE CIPRIANI LITHIUM META-ANALYSIS	INTERSEPT FOLLOW-UP (YEARS)	ANTIPSYCHOTIC WITH AN ANTI-SUICIDAL INDICATION (CLOZAPINE)

HOLD THIS

Beyond treating the disorder, only two drugs carry a specific anti-suicidal effect: **lithium** reduces suicide and all-cause death in mood disorders (Cipriani 2013 BMJ, 48 RCTs; a class effect specific to lithium) and **clozapine** reduces recurrent suicidal behaviour in schizophrenia and schizoaffective disorder (InterSePT, Meltzer 2003; the only antipsychotic so FDA-indicated); ketamine and esketamine cut ideation rapidly but not completed suicide; and post-attempt management runs medical stabilisation, then a needs-based psychosocial assessment and positively framed safety plan, then treatment of the underlying disorder.

Apply and consolidate

26 · Worked cases

This is the topic on which the whole chapter is *closed*, the point where the four rooms of the master mnemonic, the body that complains, the body that desires, the urge that acts and the life that must be protected, are walked through as reasoning rather than recited as fact. Everything before this page taught entities in isolation; here five **worked examples** put them back into the clinic, because the examiner rarely asks "define" and almost always asks "here is a patient, place them and defend the next step". A vignette is marked on two moves, the discrimination that names the frame and the guideline-anchored step that follows from it, so each case below is reasoned through both.

How to use this page. Read each case as a two-part act: first the diagnostic or discrimination reasoning that fixes the frame, then the management or safety step that the frame demands, with the cardinal error the case is built to catch called out in the open. The order tracks the arc, a somatic-symptom work-up, then the produced-symptom discrimination, then an erectile-dysfunction management case, then a gender-incongruence affirmative-care case, and finally a suicide-risk-assessment-and-safety-planning case, the last written under strict safe-messaging, with no method detail anywhere and a compassionate, safeguarding register throughout.

26.1 A somatic-symptom-disorder work-up

case: a woman in her late thirties has attended gastroenterology, cardiology and general medicine repeatedly over roughly eight months with abdominal pain, palpitations and fatigue; the investigations have been normal each time, yet she spends hours reading about serious illness, checks her pulse many times a day, has cut back her work, and cannot be settled by a normal review. She arrives at psychiatry surprised to be referred, insisting the trouble is physical.

The reasoning. Place her against the three DSM-5-TR criteria for somatic symptom disorder (DSM-5-TR 2022). Criterion A is met, there are distressing, disrupting somatic symptoms. Criterion C is met, the symptomatic state has persisted well beyond six months. The load-bearing move is Criterion B, the **positive psychological criterion**: her disproportionate and persistent thoughts about seriousness, her high health anxiety, and the excessive time and behaviour devoted to symptoms are all present, and any one of the three would suffice. The diagnosis is therefore secured by what she does and feels about the symptoms, not by the symptoms being

unexplained; even were an explanatory condition later found, the excessive response would still qualify her. Grade the burden with the PHQ-15 (fifteen items, each scored 0 to 2, a total out of a possible 30) and read Criterion B directly with the SSD-12 (a total out of a possible 48); the totals run out of a possible 48.

RULE **Diagnose on the positive criterion, not on a normal work-up.** Somatic symptom disorder is established by the excessive thoughts, feelings and behaviours of Criterion B alongside a distressing somatic symptom; whether the symptom is medically explained does not decide it.

WORKED EXAMPLE

The wrong turn is a fresh battery of tests to "reassure" her, which teaches the body to keep speaking. The right first move is physician-centred: a single named clinician, brief scheduled visits at a fixed interval rather than symptom-triggered ones, explicit validation that the symptoms and the distress are real, restriction of unnecessary investigation and referral, early treatment of any comorbid depression or anxiety, and a steady shift of the conversation from symptoms toward functioning. Cognitive behaviour therapy is the first-line psychotherapy, targeting the catastrophic reading of bodily sensations (IPS_CPG 2020). Drugs are not started for the somatic symptoms themselves.

GOOD TO REMEMBER

- **Somatic symptom disorder (the diagnosis):** Criterion A, distressing somatic symptoms; Criterion B, excessive thoughts, feelings or behaviours about them (the positive psychological criterion); Criterion C, a persistent symptomatic state beyond six months. The single discriminator is that Criterion B, not the absence of a medical explanation, defines it.
- **Physician-centred care (the management step):** the guideline first-line (IPS_CPG 2020), being a therapeutic alliance, validation of real symptoms, restriction of unnecessary tests and referrals, early treatment of comorbid mood or anxiety, and a shift toward functioning; the first move is the alliance, and antidepressants are not prescribed for medically unexplained somatic symptoms unless a specialist advises (WHO mhGAP 2016), reserved instead for a comorbid depressive or anxiety disorder or a dominant chronic-pain component.

If a conversion element sat within her presentation its bedside positive signs and management would be worked in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes; the consultation-liaison service model that most often meets such patients, and the transcultural reading of a Dhat-type presentation through a cultural formulation, are developed in the Consultation-liaison, geriatric and transcultural psychiatry notes.

26.2 A somatic-versus-factitious-versus-malingering discrimination

case: a man is referred with recurrent, dramatic and shifting symptoms that never quite match the objective findings, and the team is split on whether he is genuinely unwell, making himself unwell, or putting it on for a reason. The whole discrimination reduces to two questions asked in order: is the symptom *produced*, and if so, *why*.

The reasoning. The first question separates the genuine somatic disorders, whose symptoms are not intentionally produced, from the two feigned conditions, where they are. The second

question then splits the feigned pair on the incentive. If this man carries a pending compensation or medicolegal claim, shows disability far exceeding the objective findings, and is poorly cooperative with assessment and treatment, an **external incentive** is present and the frame is malingering, coded Z76.5 as a condition for clinical attention and expressly not a mental disorder; DSM-5-TR says it is *strongly suspected*, never confirmed, on any combination of its four pointers (DSM-5-TR 2022). If instead he consciously falsifies or induces signs, seeks procedures and admissions, and no external reward is in view, so that the motive is simply to occupy the sick role, the frame is factitious disorder imposed on self (F68.10), whose Criterion C is precisely that the deception persists in the absence of obvious external rewards. Voluntariness of production is shared by both feigned conditions and so cannot tell them apart; only the external incentive can.

THE PRESENTATION	SYMPTOM INTENTIONALLY PRODUCED?	EXTERNAL INCENTIVE
Genuine somatic symptom disorder	No, not intentionally produced	**Absent; the distress is real and the response excessive**
Factitious disorder (F68.10)	Yes, consciously feigned	**Absent; the motive is the sick role (primary gain)**
Malingering (Z76.5)	Yes, consciously feigned	**Present; a tangible reward or an avoided obligation (secondary gain)**

The voluntariness-by-incentive grid applied to the case; production separates the genuine disorder from the two feigned conditions, and the external incentive separates malingering from factitious disorder (New Oxford Textbook of Psychiatry; DSM-5-TR 2022).

TRAP **Confusing factitious disorder with malingering.** Both are consciously feigned, so they feel alike; the difference is the external incentive, present in malingering, absent in factitious disorder. Reading a compensation claimant as factitious, or a sick-role patient as a malingerer, inverts the whole grid.

GOOD TO REMEMBER

- **The discriminator (the criterion anchor):** ask production first, then motivation. Genuine somatic disorders are not intentionally produced; of the two feigned conditions, factitious disorder has no external incentive (the sick role, primary gain, Criterion C) and malingering has one (secondary gain, coded Z76.5, strongly suspected on the four DSM-5-TR pointers, not a disorder). The single external-incentive axis carries the differentiation, and the register stays medical and non-accusatory, documenting inconsistency rather than branding dishonesty.

26.3 An erectile-dysfunction management case

case: a man in his early fifties with type 2 diabetes and hypertension reports gradually worsening difficulty attaining and maintaining an erection across all contexts, including reduced early-morning erections, for the past several months, and is distressed by it. He asks for "the tablet".

The reasoning. The history sorts aetiology before any test: an insidious onset with erections deficient in every context, nocturnal and masturbatory included, points to organic, most likely vasculogenic, erectile dysfunction rather than a psychogenic pattern (Kaplan and Sadock CTP 2024). Two consequences follow. First, in a man with vascular risk factors the complaint is a

cardiovascular sentinel, the small penile arteries narrowing before the coronaries, so he needs a proper cardiovascular risk assessment, blood pressure, fasting glucose or HbA1c and lipids, and not a prescription alone. Second, measure a morning total testosterone and confirm any low result on a repeat before calling it hypogonadism. Grade severity with the five-item IIEF-5: the total runs from 5 to 25, read as no erectile dysfunction (22 to 25), mild (17 to 21), mild to moderate (12 to 16), moderate (8 to 11) and severe (5 to 7), out of a possible 25; the bands run out of a possible 25.

The management. First-line for a man without a contraindication is an oral PDE5 inhibitor plus lifestyle and risk-factor optimisation (EAU male sexual dysfunction; AUA 2018). All the oral agents are similarly effective, so choice turns on how he wants to use them: sildenafil (Manforce, Penegra) is the high-efficacy pick, taken on-demand about one hour before, starting at **50 mg**; tadalafil (Tadacip, Megalis) is the tolerability pick with the longest window, on-demand from **10 mg** or a once-daily **5 mg** that decouples dosing from sex. Before any prescription, screen specifically for nitrate use, because any organic nitrate, nitric oxide donor, nicorandil or recreational nitrite with any PDE5 inhibitor is an absolute contraindication (cyclic-GMP accumulation and profound hypotension); alpha-blockers are a caution only. If oral therapy fails or is contraindicated, second-line is intracavernosal alprostadil or a vacuum device, and third-line a penile prosthesis. Where an antidepressant or antipsychotic is contributing, that reversible cause is addressed as in the Mood disorders: pharmacotherapy notes and the Schizophrenia: management, antipsychotics and adverse effects notes.

ORAL PDE5 INHIBITOR	ON-DEMAND START (MG)	THE POINT TO HOLD
Sildenafil (Manforce, Penegra)	50	**High-efficacy pick; onset 30 to 60 min, window up to 12 h; a high-fat meal delays absorption**
Tadalafil (Tadacip, Megalis)	10 on-demand, or 5 once daily	**Longest window, up to 36 h; the only agent also licensed for erectile dysfunction with benign prostatic hyperplasia, at 5 mg once daily**
Vardenafil	10	**Orally disintegrating form available**
Avanafil	100	**Fastest onset; taken 15 to 30 min before**

The first-line oral PDE5 inhibitors, all similarly effective, with the verified EAU and AUA 2018 starting doses; units are carried in the header (EAU male sexual dysfunction; AUA 2018).

The trap to close. Because he has diabetes and low-ish energy, the tempting error is empirical, long-term testosterone. Measure it, but treat only biochemically confirmed hypogonadism; testosterone can rescue a genuinely hypogonadal PDE5-inhibitor non-responder, but it will not fix a eugonadal man's erectile dysfunction and only adds androgen risk.

GOOD TO REMEMBER

- **Erectile dysfunction (the diagnosis):** persistent inability to attain or maintain an erection sufficient for satisfactory performance; the defining discriminator is the organic, psychogenic or mixed classification, read first from preserved versus absent nocturnal and masturbatory erections, with vasculogenic disease a cardiovascular warning.
- **First-line erectile-dysfunction drug (guideline-anchored, EAU and AUA 2018):** an oral PDE5 inhibitor plus risk-factor optimisation; sildenafil starts at **50 mg** on-demand about one hour before, tadalafil at **10 mg** on-demand or **5 mg** once daily; any nitrate in any form is an absolute contraindication, and empirical long-term testosterone is never started in a man whose testosterone is normal.

26.4 A gender-incongruence affirmative-care case

case: a young adult, assigned male at birth, has experienced herself as female persistently since adolescence and comes seeking recognition, support and onward referral; her low mood and anxiety are driven largely by family rejection, misgendering and fear of discrimination, not by the gender identity itself.

The reasoning. The affirming frame is the whole answer. Gender incongruence, the marked and persistent mismatch between experienced gender and sex assigned at birth, is not a mental disorder; ICD-11 moved it out of the mental disorders chapter into "Conditions related to sexual health", and it carries no distress criterion at all (ICD-11). Gender dysphoria is the DSM-5-TR term for the clinically significant distress that only *some* people with that incongruence experience, and it is that distress, understood largely as gender minority stress, that is the target of care, never the identity. So the clinician affirms who she is and treats the distress, the comorbid depression and the effects of stigma.

The pathway. Management is a stance codified in the eighth version of the Standards of Care, WPATH SOC-8 (2022): individualised, multidisciplinary and informed-consent based, with a flexible staged transition running fully reversible (social) then partially reversible (hormonal) then irreversible (surgical) steps, and mental-health support offered where needed rather than as a universal gate. The psychiatrist's role is bounded and specific: assess readiness and capacity to give informed consent, diagnose and treat comorbid mental illness (stabilise it, do not use it to exclude her), give psychoeducation and psychosocial support, and refer. The psychiatrist does not initiate or titrate gender-affirming hormones, which are endocrinology-led (feminising therapy combines an oestrogen with an anti-androgen; Endocrine Society 2017).

RULE **Affirm the identity; treat only the distress.** Gender incongruence is not the pathology. The guideline-anchored target of care is the dysphoria, the comorbidity and the effects of stigma, and so-called conversion or reparative efforts are ineffective, harmful and outside the standard of care.

GOOD TO REMEMBER

- **Gender incongruence (the criterion anchor, ICD-11):** a marked, persistent mismatch between experienced gender and sex assigned at birth, at least two of the four indicators, present for at least several months, not assignable before puberty, and with NO distress criterion; it sits in "Conditions related to sexual health", so it is explicitly not a mental disorder. Gender dysphoria is the DSM-5-TR distress-gated term for the suffering only some experience.
- **Affirmative-care pathway (the guideline anchor, WPATH SOC-8 2022):** individualised, multidisciplinary, informed-consent care with a flexible social-then-hormonal-then-surgical transition; the psychiatrist assesses readiness and capacity, treats comorbidity, supports and refers, and does not initiate hormones (endocrinology-led).

INDIA

The clinical and legal stances now point the same way. In the NALSA judgment (National Legal Services Authority v Union of India, Supreme Court, 15 April 2014) the court recognised transgender persons as a third gender and affirmed the right to self-identified gender; the Transgender Persons (Protection of Rights) Act, 2019 then gave statutory recognition, prohibited discrimination in employment, education and healthcare, and set out welfare provisions. India also has its own long-standing third-gender tradition in the hijra community. The psychiatrist works within this frame, offering affirmative, non-judgemental care and appropriate referral, and never conversion approaches.

26.5 A suicide-risk-assessment-and-safety-planning case

case: a person is seen in acute distress after a recent crisis, a job loss and a relationship breakdown in the same fortnight, against a background of a current depressive episode and a past attempt in the history. This case is written under strict safe-messaging: no method is named anywhere, access to means is spoken of only at the category level, and the register stays compassionate and safeguarding throughout.

The reasoning. The task is a structured evaluation, a compassionate clinical interview that systematically covers the same domains every time, not a checklist score. Move gently along the continuum, from low mood to whether life feels worth living, to active thoughts, plan and intent, and, just as deliberately, ask what has kept the person going. Asking directly does not plant the idea. Then weigh the two halves of the balance. The static load is heavy here, a previous attempt with a current major depressive episode being together among the two strongest predictors of future suicide. The dynamic load is what has surged, hopelessness (the pre-eminent dynamic factor), a fresh interpersonal and financial crisis, agitation and isolation, with access to lethal means considered only at the category level. Against these sit the protective factors to seek out and strengthen, help-seeking, family support, concrete reasons for living and responsibilities to others. Hold chronic and acute risk as two separate axes; the person elevated on both warrants the most immediate response. Structure the interview with the Columbia Suicide Severity Rating Scale (C-SSRS), whose ideation subscale is an ordered ladder of five levels of increasing severity, and screen with PHQ-9 item nine; remember that SAD PERSONS is only a teaching aide, and that no instrument or clinician reliably predicts individual suicide, so a low-medium-high label must never be used to ration care.

TRAP **Treating a denied ideation as a cleared risk.** A bare verbal "no" does not neutralise a high risk-and-protective balance. When the structured evaluation shows heavy static and dynamic loading with thin protection, the denial is one data point, not an all-clear; the assessment governs the plan, not the last sentence spoken.

The management link. The assessment earns its keep only when it produces a plan. Build a collaborative Stanley-Brown Safety Planning Intervention *with* the person: recognise personal warning signs, use internal coping strategies, use social contacts and settings that provide distraction, identify people who can help, list professionals and crisis or helpline contacts, and make the environment safer by reducing access to lethal means, the last framed positively as putting time and distance between a person and a crisis, involving family where appropriate. Match the level of care, community follow-up, crisis team or admission, to assessed need, and arrange rapid follow-up, because the transition after any care contact is a high-risk window. Treating the driving disorder is itself anti-suicidal, and two agents carry specific evidence, lithium in mood disorders and clozapine in schizophrenia, developed in the Mood disorders: pharmacotherapy notes and the Schizophrenia: management, antipsychotics and adverse effects notes; the acute triage of the patient in crisis is developed in the emergency psychiatry chapter, and the population-level prevention deep dive in the Community psychiatry and public health notes.

WORKED EXAMPLE

Taking this person's eventual "I just want to go home" at face value and discharging would be the cardinal error, because the whole balance, not the last answer, is heavily loaded. The defensible course keeps them in active care, documents chronic and acute risk separately and states what has changed acutely, mobilises the protective factors, and produces a written collaborative safety plan with positively framed means-restriction counselling and a rapid follow-up appointment. The denial is noted, not trusted as an all-clear.

GOOD TO REMEMBER

- **Structured suicide risk assessment (the principle anchor):** a compassionate structured interview that weighs static against dynamic risk factors and protective factors, separates chronic from acute risk, structures with the C-SSRS ideation ladder (five levels) and screens with PHQ-9 item nine; no tool predicts individual suicide, so stratifying into low-medium-high to ration care is discouraged, and a denied ideation is never a cleared risk.
- **Safety planning and means restriction (the management step, NICE-aligned NG225 2022):** the first move after assessment is a collaborative Stanley-Brown safety plan plus positively framed means-restriction counselling, then a level of care matched to need with rapid follow-up; means restriction is the highest-yield lever, and drugs treat the driving disorder (lithium, clozapine) and supplement, never replace, the plan.

INDIA

India carries a large share of the world's suicide deaths, and suicide is among the leading causes of death in young Indian adults, a burden stated here without any reference to means and one that makes competent, compassionate assessment a public-health priority. The legal anchor is Section 115 of the Mental Healthcare Act 2017: a person who attempts suicide is presumed, unless proved otherwise, to be under severe stress, and shall not be tried or punished, effectively overriding Section 309 of the Indian Penal Code, and the state owes them care and rehabilitation to reduce recurrence. National helpline infrastructure such as Tele MANAS and the Indian Psychiatric Society position reinforce the shift from punishment to care.

25	50	30	5
IIEF-5 TOP SCORE (NO ED FROM 22 UP)	SILDENAFIL ON-DEMAND START (MG)	DAPOXETINE ON-DEMAND START (MG)	C-SSRS IDEATION-SEVERITY LEVELS

HOLD THIS

Read every vignette in two moves, the discrimination that names the frame and the guideline step that follows: somatic symptom disorder turns on the POSITIVE psychological criterion and is managed physician-centred; the produced-symptom pair splits only on the external incentive (present in malingering, absent in factitious disorder); erectile dysfunction is first-line an oral PDE5 inhibitor (sildenafil **50 mg** on-demand, tadalafil **10 mg** on-demand or **5 mg** once daily) with nitrates absolutely contraindicated and testosterone only for confirmed hypogonadism; gender incongruence is affirmed, not pathologised, along the WPATH SOC-8 pathway within the NALSA judgment and the Transgender Persons Act 2019; and the suicidal patient gets a structured assessment weighing risk against protection, never a predictive score, with a collaborative safety plan and positively framed means restriction, remembering that a denied ideation is not a cleared risk and that Section 115 of the Mental Healthcare Act 2017 decriminalised the attempt.

27 · Consolidation

The whole chapter now stands 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 somatic, sexual, impulse and suicide block; it rewards the one who can, cold and under pressure, name the positive criterion that defines a somatic symptom disorder, split factitious from malingering on a single axis, place a sexual complaint on the response cycle and reach for the first-line drug, state that being transgender is not an illness, and run a compassionate structured suicide assessment. 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 master **mnemonic** that ties the chapter together, paid off from where it was seeded at the somatic-symptom-disorder opener; a set of answer-free prompt lists for **active recall** across the somatic nosology and its positive criterion, the factitious-versus-malingering discriminator, the sexual response

cycle and the erectile-dysfunction first-line, the gender-incongruence depathologisation, and the suicide risk-assessment principles and safety planning; and a whole-chapter **synthesis map** you are meant to **redraw from memory**. It re-derives no criterion, dose or cut-off of its own; every figure quoted here is a pointer back to the topic that verified it. The deep antidepressant and lithium anti-suicidal pharmacology lives in the Mood disorders: pharmacotherapy notes, the clozapine anti-suicidal pharmacology in the Schizophrenia: management, antipsychotics and adverse effects notes, and the transcultural frame of Dhat in the Consultation-liaison, geriatric and transcultural psychiatry 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.

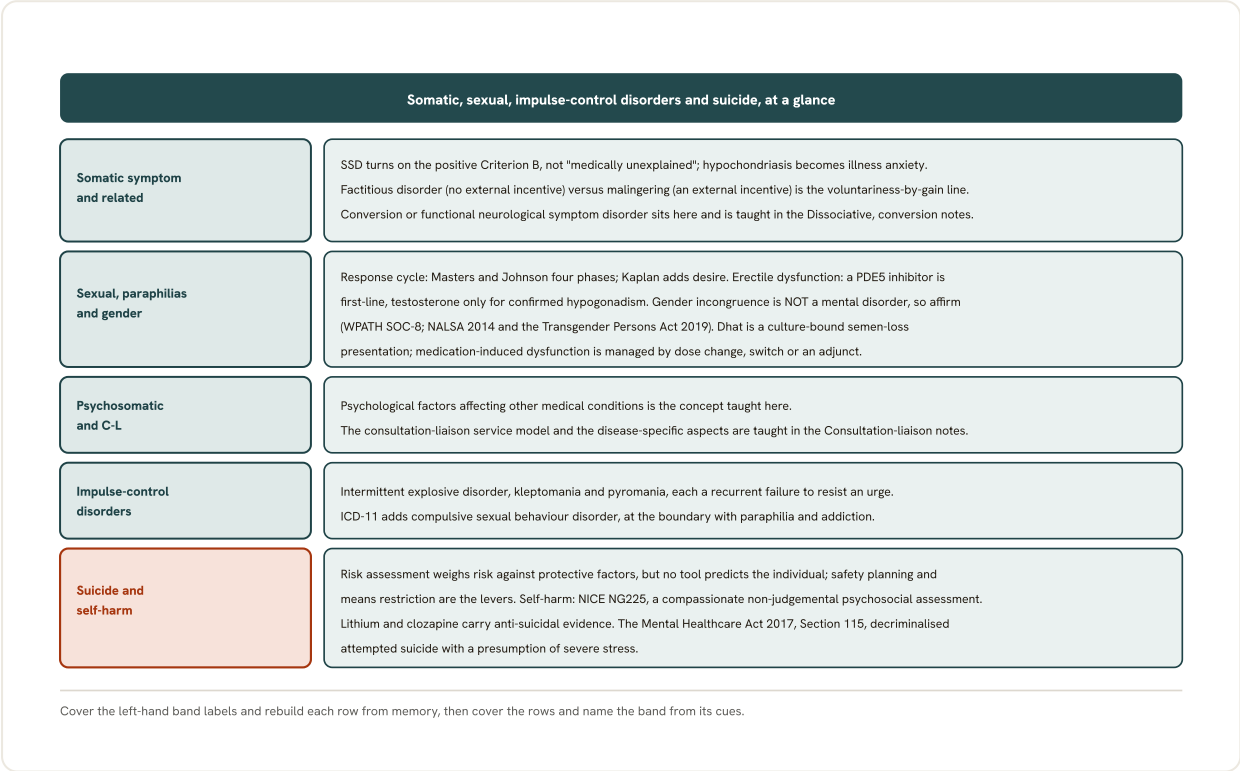

Fig 18.16 The chapter at a glance. Read across each band once, then use the map for retrieval: cover the left-hand labels and rebuild each row from its cues, then cover the rows and name the band. The somatic band turns on the positive Criterion B and the voluntariness-by-incentive line that separates factitious disorder from malingering. The sexual and gender band holds the response cycle, the phosphodiesterase-5 inhibitor as first-line for erectile dysfunction, the affirming frame for gender incongruence with its Indian legal basis, and Dhat. The psychosomatic and consultation-liaison band is the concept taught here with the service model cross-referenced. The impulse-control band gathers the disorders of urge. The suicide and self-harm band is the core: assessment that guides rather than predicts, safety planning and means restriction, the compassionate NICE stance to self-harm, the anti-suicidal evidence for lithium and clozapine, and the decriminalisation of attempted suicide under the Mental Healthcare Act.

27.1 The master mnemonic, paid off: SEED

Seeded at the opener, closed here. The chapter opened by seeding the master mnemonic **"SEED"** at somatic symptom disorder, and this is where it is reaped, because its four letters are the four rooms of a single house, arranged as the body moves from complaint to desire to urge to the protection of life. **S** is the *Somatic* room, where the body *complains*: somatic symptom disorder and the ICD-11 bodily distress disorder, with factitious disorder and malingering standing at its far door. **E** is *Eros*, where the body *desires* and identity is *affirmed*: the sexual dysfunctions and gender incongruence. **E** is the *Explosive urges*, where the urge *acts*: the impulse-

control disorders, each running the mounting-tension-then-relief arc. **D** is the *Defence of life*, the protective, compassionate stance the chapter closes on: suicide risk assessment, safety planning and prevention. If you can name the four rooms and walk their contents without looking, you hold the spine of the whole chapter.

MNEMONIC

SEED

The chapter master mnemonic, paid off. **S** = the *Somatic* room (the body complains): somatic symptom disorder defined by the positive psychological criterion, ICD-11 bodily distress disorder, and the feigned pair, factitious disorder and malingering, at the door. **E** = *Eros* (the body desires, identity is affirmed): the sexual dysfunctions mapped on the response cycle, and gender incongruence, which is not a mental illness. **E** = *Explosive urges* (the urge acts): the impulse-control disorders and their tension-then-relief arc. **D** = the *Defence of life*: compassionate, structured suicide risk assessment and collaborative safety planning. Four rooms, one house: S-E-E-D.

27.2 Active-recall prompt list: the somatic room, the positive criterion and the feigned pair

Cover the answers and pull each one. This is a **retrieval practice** list, not a summary. For each prompt below, say aloud (or write) the discriminator before you check it against the topic that verified it. The point is the effort of retrieval: the harder the pull, the stronger the trace.

- **The positive criterion.** Prompt: what now defines somatic symptom disorder, and what was abolished? (Answers in the somatic-symptom-disorder topic: Criterion B, the excessive *Thoughts, Anxiety and Behaviour* about the symptoms, is the defining **positive psychological criterion**; the old "medically unexplained" requirement is gone, so a medically explained symptom with an excessive response still qualifies, and severity is counted from Criterion B, not from the number of symptoms.)
- **The forward collapse.** Prompt: where did the DSM-IV somatoform family go? (Somatisation, undifferentiated and pain disorder folded into somatic symptom disorder; hypochondriasis split into somatic symptom disorder and illness anxiety disorder; conversion retained as functional neurological symptom disorder; body dysmorphic disorder migrated to the obsessive-compulsive spectrum. The positive signs of conversion are in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes, and body dysmorphic disorder in the Anxiety, OCD and trauma-related disorders notes.)
- **The ICD-11 parallel.** Prompt: the ICD-11 name and its logic? (Bodily distress disorder, code 6C20, distressing symptoms plus excessive attention, positively defined and not unexplained-based, present on most days for at least several months; ICD-11 sends its hypochondriasis to the obsessive-compulsive and related disorders.)
- **Factitious versus malingering.** Prompt: both are feigned, so what tells them apart? (Both are consciously produced, so voluntariness is shared and cannot separate them; the discriminator is the *external incentive*. Malingering has one, secondary gain, coded Z76.5 as a condition and not a mental disorder, strongly suspected on the four DSM-5-TR pointers; factitious disorder

has none, the motive being the sick role and primary gain, its severe wandering form being Munchausen syndrome. The genuine somatic disorders are not intentionally produced at all.)

RULE **Read production, then incentive.** The whole somatic room reduces to two questions asked in order: is the symptom intentionally produced, and if so, for an external incentive (malingering, secondary gain) or for the sick role (factitious disorder, primary gain); the genuine somatic disorders answer the first question with no.

GOOD TO REMEMBER

- **The somatic room (the criterion anchors):** somatic symptom disorder is defined by the positive Criterion B (excessive thoughts, feelings and behaviours), not by being medically unexplained; ICD-11 makes the same move as bodily distress disorder (6C20). The feigned pair separate only on the external incentive, present in malingering (Z76.5, not a disorder) and absent in factitious disorder (the sick role); a denied external motive does not turn a compensation claimant into a factitious patient.

27.3 Active-recall prompt list: the Eros room, the response cycle, the ED first-line and the depathologisation

Localise first, then treat, then affirm. This room holds a schematic model, one clean first-line drug position, and one reclassification. Retrieve each before checking it against its topic.

- **The response cycle.** Prompt: the three named models and the wiring? (Masters and Johnson gave four phases, excitement, plateau, orgasm and resolution, with a male-only refractory period; Kaplan's triphasic model added the desire phase to make desire, excitement and orgasm, and that phase map is how DSM-5-TR classifies the dysfunctions; Basson's circular model shows female desire can be responsive and follow arousal; the wiring is point and shoot, parasympathetic sacral outflow at **S2 to S4** for erection and sympathetic thoracolumbar outflow at **T11 to L2** for emission and ejaculation.)
- **The disrupted phase names the disorder.** Prompt: which phase gives which complaint? (A desire-phase failure gives hypoactive desire; an excitement-phase failure gives erectile dysfunction; an orgasm-phase failure gives anorgasmia or premature ejaculation. Localise to a phase and the differential is narrowed before a criterion is named.)
- **The erectile-dysfunction first-line.** Prompt: the first-line drug, the two doses, the one absolute contraindication, the testosterone rule? (An oral PDE5 inhibitor plus risk-factor optimisation; sildenafil starts at **50 mg** on-demand about one hour before, tadalafil at **10 mg** on-demand or **5 mg** once daily; any nitrate in any form is an absolute contraindication; test a morning testosterone and treat only confirmed hypogonadism, never long-term testosterone in a eugonadal man. The ladder is Pills, then Pokes and Pumps, then Prosthesis.)
- **Premature ejaculation.** Prompt: the only licensed first-line drug and its dosing rule? (On-demand dapoxetine, started at **30 mg**, taken one to three hours before, maximum 60 mg, not more than once every 24 hours; it should not routinely be combined with a PDE5 inhibitor because of syncope risk.)

- **The gender depathologisation.** Prompt: the headline reclassification, the DSM contrast, and the management stance? (ICD-11 renamed transsexualism as gender incongruence and moved it out of the mental disorders chapter to "Conditions related to sexual health," needing no distress criterion; DSM-5-TR keeps gender dysphoria, gated by Criterion B distress; management is not a drug but the affirmative-care pathway, individualised and informed-consent based, with the psychiatrist treating distress and comorbidity, never the identity.)

GOOD TO REMEMBER

- **Erectile dysfunction first-line (guideline-anchored, ISSM, BSSM, EAU, AUA 2018):** an oral PDE5 inhibitor plus risk-factor optimisation is first-line for every man without a contraindication; sildenafil (Manforce, Penegra) starts at 50 mg on-demand, tadalafil (Tadacip, Megalis) at 10 mg on-demand or 5 mg once daily; a nitrate is an absolute contraindication, and long-term testosterone is never started for a eugonadal man. Premature ejaculation is first-line on-demand dapoxetine (Duralast, Sustinex), started at 30 mg.
- **Gender incongruence (the criterion anchor and stance):** a marked, persistent mismatch between experienced gender and sex assigned at birth is *not* a mental disorder; ICD-11 relocated it out of the mental disorders chapter with no distress criterion, DSM-5-TR retains gender dysphoria gated by distress, and management is the WPATH SOC-8 individualised, informed-consent, gender-affirmative pathway. The discriminator to hold is that the target of care is the distress, the comorbidity and the stigma, never the person's gender.

27.4 Active-recall prompt list: the defence of life, risk assessment and safety planning

A weighing skill, held with a safeguarding tone. This is the room the chapter closes on, and it is examined as a skill, not a fact-list. Retrieve each principle before checking it against the suicide risk-assessment topic, and keep the register compassionate throughout: the assessment exists to guide care and safety, never to forecast an outcome or to allocate blame.

- **The structured evaluation.** Prompt: what is it, and what is it not? (A compassionate **structured evaluation** that weighs static and dynamic risk factors against protective factors and the current mental state, and separates chronic from acute risk; it is not a checklist score or a numerical prediction. Asking directly and calmly about suicidal thoughts is a core skill and does not plant the idea.)
- **The three named aides.** Prompt: the gold standard, the teaching aide, and the single screening item? (The C-SSRS is the gold-standard clinician-administered interview, its ideation subscale an ordered ladder of five levels; SAD PERSONS is only a teaching aide scored out of a possible ten, explicitly not validated to predict individual risk or to ration care; PHQ-9 item nine is a single screening item that, if endorsed, mandates a full structured assessment.)
- **The limits of prediction.** Prompt: the governing principle? (No tool or clinician reliably predicts individual suicide, so risk-stratification scores must not be used to predict or to ration care; the field favours a prevention-oriented formulation of four judgements, risk status, risk state, resources and foreseeable changes, over a low-medium-high label.)
- **Safety planning and means restriction.** Prompt: the guideline first move after assessment? (A collaborative Stanley-Brown Safety Planning Intervention built with the person, plus

means-restriction counselling framed positively as putting time and distance between a person and a crisis, then a level of care matched to need with rapid follow-up. Reducing access to lethal means, named only at the category level, is the single most robustly evidenced lever, and where a driving disorder is present its treatment is itself anti-suicidal, lithium in mood disorders and clozapine in schizophrenia.)

TRAP **Treating a denied ideation as a cleared risk.** A verbal "no" does not neutralise a high risk-and-protective balance; when the structured evaluation shows heavy static and dynamic loading with thin protection, the denial is one data point among many, and the assessment, not the last sentence spoken, governs the safety plan.

GOOD TO REMEMBER

- **Suicide risk assessment and safety planning (guideline-anchored, NICE NG225 2022, WHO LIVE LIFE 2021):** weigh static and dynamic risk factors against protective factors and separate chronic from acute risk; use the C-SSRS ideation ladder to structure and PHQ-9 item nine to screen, and remember no instrument predicts individual suicide, so stratifying to ration care is discouraged. The first move after assessment is a collaborative Stanley-Brown safety plan plus positively framed means restriction, the highest-yield lever; drugs (lithium, clozapine) treat the driving disorder and supplement, never replace, the plan.

INDIA

The Indian thread runs through every room and is worth carrying as one line at consolidation. In the Somatic room, distress is very commonly first brought to the clinician as bodily symptoms, so the culture-bound Dhat presentation must be read through a cultural formulation rather than pathologised on its surface, its transcultural frame sitting in the Consultation-liaison, geriatric and transcultural psychiatry notes. In the Eros room, the generics are familiar Indian brands, sildenafil (Manforce, Penegra), tadalafil (Tadacip, Megalis) and dapoxetine (Duralast, Sustinex), and gender-affirmative care is reinforced in law by the 2014 NALSA judgment, which recognised a third gender and the right to self-identified gender, and the Transgender Persons (Protection of Rights) Act, 2019. In the Defence-of-life room, India carries a large share of the world's suicide deaths, a burden stated here without any reference to means, and Section 115 of the Mental Healthcare Act 2017 decriminalised the attempt through a presumption of severe stress and a duty of care, superseding Section 309 of the Indian Penal Code; the Indian Psychiatric Society endorses the shift from punishment to care.

27.5 The whole-chapter synthesis map, framed cover-the-branches

Build the house, then redraw it blind. The strongest single revision act for this block is to **redraw from memory** a **synthesis map** that hangs every entity off its SEED room. The map has four rooms: the *Somatic* (the body complains), *Eros* (the body desires and identity is affirmed), the *Explosive urges* (the urge acts), and the *Defence of life* (the life protected). From each room hang its disorders, and from each disorder hang the one anchor to pull cold, its criterion, its discriminator or its first-line. 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.

SEED ROOM	WHAT HANGS FROM IT	THE ONE ANCHOR TO PULL COLD
S · Somatic (the body complains)	Somatic symptom disorder, illness anxiety, bodily distress disorder, conversion; factitious disorder and malingering at the door	**The positive psychological criterion (Criterion B), then production-before-incentive for the feigned pair**
E · Eros (the body desires, identity affirmed)	The response-cycle phases and their dysfunctions, erectile dysfunction, premature ejaculation, gender incongruence	**The disrupted phase names the disorder; ED first-line is a PDE5 inhibitor; gender incongruence is not a mental illness, so affirm**
E · Explosive urges (the urge acts)	The impulse-control disorders, each running the tension-then-relief arc	**Mounting tension relieved by the act, the ego-syntonic urge distinguishing them**
D · Defence of life (the life protected)	Suicide risk assessment, safety planning, prevention	**Weigh the whole balance not the last sentence; means restriction framed positively; the attempt is decriminalised**

The four-room SEED synthesis map, framed cover-the-branches; each room's single anchor is the fact to regenerate before uncovering the row (verified against the individual chapter topics, drawn in the accompanying figure).

How to use it. First pass: cover everything but the four room-names and list the disorders under each. Second pass: for each disorder, cover the anchor and pull it. Third pass: recite the one first-line or discriminator per room. 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.

5	15	50
C-SSRS IDEATION-SEVERITY LEVELS	PHQ-15 HIGH-SEVERITY CUT-OFF (0 TO 30 SCALE)	SILDENAFIL ON-DEMAND START DOSE (MG)

PEARL

The whole chapter collapses to four rooms and four anchors. The *Somatic* room turns on the positive criterion and the production-before-incentive grid; *Eros* turns on the response-cycle phase and the clean PDE5-inhibitor first-line, with gender incongruence affirmed rather than treated; the *Explosive urges* turn on the tension-then-relief arc; and the *Defence of life* turns on the weighed balance, the safety plan and the positively framed means restriction. Redraw the four rooms, name their anchors, and you own the block.

HOLD THIS

Redraw the four-room SEED synthesis map from memory: Somatic (defined by the positive Criterion B, not by being medically unexplained, with factitious and malingering split on the external incentive), Eros (the disrupted phase names the sexual disorder, erectile dysfunction is first-line a PDE5 inhibitor, and gender incongruence is not a mental illness so it is affirmed), Explosive urges (the mounting-tension-then-relief arc of the impulse-control disorders), and the Defence of life (a compassionate structured assessment that weighs the balance rather than trusting a denial, driving a collaborative safety plan and positively framed means restriction, with the attempt decriminalised by Section 115 of the Mental Healthcare Act 2017).

Previously asked

This is a broad territory that is examined at two levels, the syndrome (its clinical features, its differential diagnosis and its course) and the management, with one band, suicide and self-harm, carrying much of the essay weight. The reliable pattern is that the somatic-symptom and conversion disorders recur as full essays, that suicide is the dominant management essay and is repeatedly asked with an Indian and a medico-legal slant, and that the sexual dysfunctions, Dhat, the paraphilias, and the consultation-liaison and psychosomatic topics come as standalone notes. The impulse-control disorders are drawn together in an enumerate-and-describe essay. Every long essay ends in a management plan, so the first-line and the reason for it must be exam-ready, and the suicide answers must be written with the safe, structured, compassionate stance the block rewards. The genuine questions on record are grouped by band below.

HOW THIS TERRITORY IS ACTUALLY TESTED

Q **The somatic-symptom and conversion disorders, as essays.** "Describe the clinical features, differential diagnosis and management of Somatoform Disorder" has come as a 25-mark essay across more than one sitting, alongside "Discuss the etiology, clinical features and management of Conversion Disorder", "Clinical features and differential diagnosis of Conversion Disorder" and "Differential diagnosis of somatoform pain disorder and its prognosis"; "Pseudoseizures" and "Malingering" recur as 10-mark notes. Prepare the somatoform-to-somatic-symptom-disorder nosology shift and the positive psychological criterion, the somatic-versus-factitious-versus-malingering discrimination, and the physician-centred management of medically unexplained symptoms. essay and short note, recurring

Q **The suicide and self-harm band, the dominant essay theme.** "Enumerate various risk factors for suicide and describe the outline of management of attempted suicide", "Describe the management of the suicidal patient", "Discuss suicide and mental disorders" and "Discuss suicide with special reference to the Indian setting in detail" have all come as 25-mark essays, and "Differential diagnosis and synonym for deliberate self-harm" and the medico-legal "critical appraisal regarding attempted suicide as a crime" as 10-mark notes. Prepare the structured risk assessment and the limits of individual-risk prediction, safety planning and means restriction, the Mental Healthcare Act 2017 decriminalisation of attempted suicide under Section 115, and the anti-suicidal evidence for lithium and clozapine, all written with strict safe-messaging. essay and short note, recurring

Q **The sexual dysfunctions and paraphilias, as notes.** "Premature ejaculation" recurs as a 10-mark note across several sittings, and "Erectile dysfunction" and "Paraphilias" as 10-mark notes. Prepare the normal sexual response cycle, the phosphodiesterase-5-inhibitor-first management of erectile dysfunction, the

on-demand dapoxetine for premature ejaculation, and the consent, distress and harm framing of the paraphilic disorders. short note, recurring

Q **Dhat and the culture-bound presentations, as notes.** "Enumerate the culture-bound syndromes and describe Dhat syndrome in detail" and "Dhat syndrome" recur as 10-mark notes. Prepare Dhat as an Indian culture-bound sexual presentation, the semen-loss attribution, the cultural formulation, and the non-confrontational psychoeducational management. short note, recurring

Q **Consultation-liaison and psychosomatic medicine, as notes.** "Consultation liaison psychiatry" and "The role of the psychiatrist in psychosomatic medicine" have come as 10-mark notes. Prepare the psychosomatic concept and the biopsychosocial model here; the consultation-liaison service model and the disease-specific aspects are developed in the Consultation-liaison, geriatric and transcultural psychiatry notes. short note, recurring

Q **The impulse-control disorders, as an enumerate-and-describe essay.** "Enumerate the various impulse-control disorders and describe the etiology, clinical features and management of trichotillomania" gathers the group into one answer. Prepare intermittent explosive disorder, kleptomania, pyromania and the ICD-11 compulsive sexual behaviour disorder as a compact group, each a recurrent failure to resist an urge.

essay, enumerate

Prepare this material to be assessed and managed, not just recited: the somatic-symptom nosology and the factitious-versus-malingering discrimination, the erectile-dysfunction algorithm and the verified doses, the gender-incongruence affirming frame with its Indian legal basis, and the structured, safe suicide risk assessment and safety planning in these notes are built to the way the block is examined. The positive functional-neurological signs and the full management of conversion or functional neurological symptom disorder are developed in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes; the deep antidepressant and antipsychotic sexual-dysfunction pharmacology and the lithium and clozapine pharmacology in the Mood disorders: pharmacotherapy notes and the Schizophrenia: management, antipsychotics and adverse effects notes; the consultation-liaison service model and the disease-specific aspects in the Consultation-liaison, geriatric and transcultural psychiatry notes; and the population and public-health suicide prevention in the Community psychiatry and public health notes.

Quick review

SOMATIC SYMPTOM AND RELATED DISORDERS: THE NOSOLOGY, THE DISCRIMINATION, THE MANAGEMENT	
THE NOSOLOGY SHIFT	ICD-10 somatoform disorders collapse into DSM-5 **somatic symptom disorder**, which turns on the **positive psychological Criterion B** (excessive thoughts, feelings and behaviours about the symptoms), NOT on the symptom being "medically unexplained"; a medically explained symptom can qualify. Hypochondriasis becomes **illness anxiety disorder**. ICD-11 uses one graded **bodily distress disorder**.
THE DISCRIMINATION	Somatic symptom disorder and illness anxiety are **genuine, no external incentive**. Conversion or FND is not consciously feigned and is recognised by **positive signs** (taught in the Dissociative notes). **Factitious** = feigned, **no** external incentive (internal sick role; Munchausen, and by proxy). **Malingering** = feigned, **external** incentive; not a disorder. Secondary gain alone is not malingering.
THE SCALES	**PHQ-15**: 15 items 0 to 2, total out of a possible 30; bands 0-4, 5-9, 10-14, 15-30. **SSD-12**: 12 items 0 to 4, total out of a possible 48; cut-off **23 and above**.
MANAGEMENT (IPS 2020, WHO MHGAP)	**Physician-centred care**: one coordinating clinician, validate distress, restrict unnecessary testing, treat comorbid depression or anxiety, shift focus to functioning; reattribution; **CBT** first-line. Do NOT prescribe an antidepressant or anxiolytic for medically unexplained symptoms without specialist advice (mhGAP).
SEXUAL DYSFUNCTIONS, PARAPHILIAS AND GENDER	
RESPONSE CYCLE	Masters and Johnson four phases (excitement, plateau, orgasm, resolution); Kaplan adds **desire** (triphasic); Basson's circular model for responsive female desire.
ERECTILE DYSFUNCTION (ISSM, BSSM)	Screen cardiometabolic risk (an early vascular marker); confirm with the IIEF-5. **PDE5 inhibitor first-line**: sildenafil 25/50/100, tadalafil on-demand 10 or 20 and once-daily 2.5 or 5, vardenafil, avanafil. **Absolute contraindication with nitrates**. Test testosterone, replace only confirmed hypogonadism; no empirical testosterone in a eugonadal man. Second-line alprostadil or vacuum device; third-line prosthesis. IIEF-5 out of a possible 25 (22-25 none to 5-7 severe).
PREMATURE EJACULATION, HSDD	**Dapoxetine** on-demand 30 to 60 mg, 1 to 3 hours before activity, for premature ejaculation. **Flibanserin** 100 mg nightly for premenopausal HSDD (alcohol and hypotension cautions). Female dysfunctions: biopsychosocial and sex therapy first; local oestrogen for GSM dyspareunia; pelvic-floor physiotherapy and graded dilators for GPPPD.

SEXUAL DYSFUNCTIONS, PARAPHILIAS AND GENDER	
GENDER INCONGRUENCE	ICD-11 **depathologises** it: gender incongruence is a sexual-health condition, NOT a mental disorder (as homosexuality was earlier removed). Affirm; WPATH SOC-8 individualised informed-consent pathway. India: **NALSA 2014** and the **Transgender Persons Act 2019**. Trap: pathologising the identity.
DHAT; MEDICATION-INDUCED	**Dhat**: an Indian culture-bound presentation, symptoms attributed to semen loss; cultural formulation and psychoeducation, treat comorbid depression or anxiety. Medication-induced dysfunction: watchful waiting, dose reduction, switch (bupropion, mirtazapine, vortioxetine), or an adjunct; antipsychotic **hyperprolactinaemia** (worst with risperidone, least with aripiprazole).
PARAPHILIC DISORDERS	A paraphilia is a **disorder** only with distress or impairment, or harm or a non-consenting person. Types from exhibitionistic and voyeuristic to paedophilic disorder; psychological relapse-prevention, SSRI, and antiandrogen or GnRH-analogue in high-risk cases.
PSYCHOSOMATIC MEDICINE, CONSULTATION-LIAISON AND THE IMPULSE-CONTROL DISORDERS	
PSYCHOSOMATIC CONCEPT	Psychological factors affecting other medical conditions; the biopsychosocial model; stress-illness pathways (HPA, autonomic, immune). Taught compactly here.
CONSULTATION-LIAISON (CROSS-REF)	The C-L service model and the disease-specific aspects (psycho-oncology, HIV, endocrine, cardiac, transplant, surgical and ICU) are taught in the Consultation-liaison, geriatric and transcultural psychiatry notes.
IMPULSE-CONTROL DISORDERS	**Intermittent explosive disorder** (impulsive aggression out of proportion), **kleptomania** (stealing objects not needed), **pyromania** (fire-setting with fascination), and the ICD-11 **compulsive sexual behaviour disorder** (at the boundary with paraphilia and addiction). Each a recurrent failure to resist an urge.
SUICIDE AND SELF-HARM (THE HIGH-STAKES CORE)	
RISK ASSESSMENT	Weigh **static** factors (a previous attempt the strongest; mental illness; chronic illness; family history) against **dynamic** ones (hopelessness the leading one; ideation with plan and intent; a recent crisis; agitation; access to means at the category level) and the **protective** factors. Separate **chronic** from **acute** risk. The C-SSRS structures an ideation ladder plus a behaviour subscale.
LIMITS OF PREDICTION	The governing principle: **no tool predicts an individual suicide.** Risk-stratification scores must NOT decide who gets care; use a prevention-oriented formulation (risk status, risk state, resources, foreseeable changes). Assessment guides care, it does not forecast.

SUICIDE AND SELF-HARM (THE HIGH-STAKES CORE)	
MANAGEMENT AND SAFETY PLANNING	A compassionate, non-judgemental assessment (NICE); a **collaborative safety plan** (warning signs, coping, contacts, crisis lines, means-restriction counselling framed positively); the level of care (LOCUS); treat the disorder; active follow-up in the high-risk post-discharge window. A denied ideation is not a cleared risk.
DELIBERATE SELF-HARM (NICE NG225)	Self-harm is self-poisoning or self-injury **irrespective of intent**; NSSI is the intent-free subset. Every episode earns a full needs-based psychosocial assessment; **no risk-stratification tool** to predict or to ration care; apparent medical triviality is no reassurance; any self-harm raises future suicide risk.
PREVENTION AND ANTI-SUICIDAL DRUGS	**WHO LIVE LIFE**: limit access to means (highest-yield), responsible media, adolescent life skills, early identification. **Lithium** reduces suicide in mood disorders (Cipriani 2013, 48 RCTs); **clozapine** reduces suicidal behaviour in schizophrenia (InterSePT, Meltzer 2003). India: **Section 115 of the Mental Healthcare Act 2017** presumes severe stress and decriminalises an attempt.
THE GUIDELINE SPINE, THE INDIA CONTEXT AND THE TRAPS	
THE GUIDELINE ORDER	Guidelines-first on the where-to-treat, target-by-phase, modes-of-treatment spine: **NICE** leads the self-harm and suicide pathway, with **WHO LIVE LIFE** for prevention; **ISSM and BSSM** for the sexual dysfunctions; **WPATH SOC-8** for gender; the IPS somatoform guideline and WHO mhGAP for the somatic band.
THE INDIA CONTEXT	The Mental Healthcare Act 2017 **Section 115** (attempted suicide presumed under severe stress, not punished); the National Suicide Prevention Strategy 2022; **Dhat** as a culture-bound presentation; gender-affirmative care via **NALSA 2014** and the **Transgender Persons Act 2019**.
THE TRAPS	Calling somatic symptom disorder "medically unexplained"; confusing factitious (no incentive) with malingering (external incentive); empirical testosterone for a eugonadal man with ED; pathologising gender incongruence; reading a Dhat presentation without the cultural formulation; and the cardinal one, treating a denied ideation as a cleared risk, or a stratification score as a licence to discharge.

References

The somatic-symptom, sexual, impulse-control and suicide content is grounded in the standard psychiatry and psychopharmacology sources (the source hierarchy below), with Kaplan and Sadock and the Maudsley Prescribing Guidelines as the depth bar for the phenomenology, the neurobiology, the scale cut-offs and the doses, the New Oxford Textbook and the Companion for

the nosology, and Clinical Methods in Psychiatry for the Indian context. The management is guideline-first: NICE leads for the self-harm and suicide pathway, with WHO LIVE LIFE for prevention, ISSM and BSSM for the sexual dysfunctions, WPATH Standards of Care version 8 for gender-affirmative care, and the Indian Psychiatric Society somatoform guideline and WHO mhGAP for the somatic band. The Indian legal provisions (the Mental Healthcare Act 2017 Section 115, the NALSA judgment 2014 and the Transgender Persons (Protection of Rights) Act 2019) were checked against their primary texts. Every scale cut-off, dose, criterion and legal provision was checked against these sources; anything that could not be confirmed was omitted and flagged rather than guessed. Each journal identifier below was verified against PubMed this build and matched on title, first author and year; books, guidelines and statutes are cited by author or body, title and year.

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