

PSYCHIATRY POSTGRADUATE EXAMS

Weave Study Guides

Supplement, Requested Topics

Twenty-two guides across Papers I to IV, plus the cross-paper supplement. Study notes, model answers, mnemonics, high-yield comparisons, PYQ frequency, and quick review for every topic.

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PART V · SUPPLEMENT · REQUESTED TOPICS

22 Requested Topics

› Requested Topics

INDEX OF DISORDERS & TOPICS

PART V

Supplement Requested Topics

Cross-paper requested topics, gathered as the twenty-second guide.

Requested Topics

Supplement · Requested Topics. Six study modes, from notes to quick review.

Requested Topics

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01

Delirium & Consultation-Liaison Psychiatry

Pathophysiology, assessment tools, ICU complications, and the C-L consultation model

DELIRIUM: PATHOPHYSIOLOGY & ASSESSMENT

DEFINITION & EPIDEMIOLOGY

Delirium is an acute, fluctuating disturbance of attention, awareness, and cognition that develops over hours to days. It represents a **direct physiological consequence** of a medical condition, substance, or multiple aetiologies.

- Prevalence: 10–31% of hospitalised medical patients; up to **80% in ICU** patients on mechanical ventilation
- Mortality: Associated with 10–26% in-hospital mortality; each day of delirium independently increases mortality risk
- Post-operative delirium occurs in 15–53% of elderly surgical patients

PATHOPHYSIOLOGY: KEY HYPOTHESES

1. CHOLINERGIC DEFICIENCY HYPOTHESIS

The most established model. Acetylcholine (ACh) is critical for attention, arousal, and memory consolidation. Delirium is associated with:

- **Reduced cholinergic transmission**, anticholinergic medications are the most common pharmacological trigger
- Serum anticholinergic activity (SAA) correlates with delirium severity
- Physostigmine (AChE inhibitor) can transiently reverse anticholinergic delirium
- Reciprocal relationship: ACh ↓ and dopamine ↑ → produces the hyperactive delirium phenotype

2. NEUROINFLAMMATION HYPOTHESIS

Systemic inflammation leads to blood-brain barrier (BBB) breakdown and neuroinflammation:

- Pro-inflammatory cytokines (IL-1 β , IL-6, TNF- α) cross a compromised BBB
- Microglial activation → local neuroinflammation → neuronal dysfunction
- Endothelial activation and adhesion molecule upregulation → further BBB permeability
- Particularly relevant in **sepsis-associated delirium** and post-surgical delirium

3. OXIDATIVE STRESS HYPOTHESIS

Impaired oxidative metabolism (hypoxia, hypoglycaemia, thiamine deficiency) reduces acetylcholine synthesis and increases reactive oxygen species, contributing to neuronal injury.

4. NEUROTRANSMITTER IMBALANCE MODEL (INTEGRATED)

NEUROTRANSMITTER	CHANGE IN DELIRIUM	CLINICAL CORRELATE
Acetylcholine	↓ Decreased	Inattention, memory impairment
Dopamine	↑ Increased	Agitation, hallucinations, psychosis
GABA	↑ in hepatic / ↓ in withdrawal	Sedation (hepatic) or agitation (withdrawal)
Glutamate	↑ Excitotoxicity	Neuronal injury, cognitive sequelae
Serotonin	Variable	Perceptual disturbances
Melatonin	↓ Disrupted circadian	Sleep-wake cycle disturbance

EXAM PEARL

The cholinergic deficiency hypothesis is the most frequently tested model. Remember: **anticholinergic burden** is the single most modifiable risk factor. Always mention the ACh-dopamine reciprocal relationship in essay answers.

CONFUSION ASSESSMENT METHOD (CAM)

CAM: DIAGNOSTIC ALGORITHM

The CAM (Inouye et al., 1990) is the most widely validated bedside instrument for delirium detection. Sensitivity: 94–100%, Specificity: 90–95%.

FOUR CORE FEATURES

FEATURE	DESCRIPTION	HOW TO ASSESS
1. Acute onset & fluctuating course	Abrupt change from baseline mental status, symptoms wax and wane over 24 hours	Collateral history from nursing staff/ family; serial assessments
2. Inattention	Difficulty focusing, sustaining, or shifting attention	Digit span, serial 7s, days of week backward, spelling WORLD backward
3. Disorganised thinking	Rambling, incoherent speech, illogical flow of ideas, unpredictable topic switching	Ask simple questions: "Will a stone float on water?" "Are there fish in the sea?"
4. Altered level of consciousness	Any level other than normal alert, hyperalert, lethargic, stuporous, or comatose	RASS (Richmond Agitation-Sedation Scale) or clinical observation

DIAGNOSTIC RULE

CAM+ = Feature 1 + Feature 2 + (Feature 3 OR Feature 4)

Both Feature 1 (acute onset/ fluctuation) AND Feature 2 (inattention) must be present, PLUS either Feature 3 or Feature 4.

CAM-ICU (FOR INTUBATED/NON-VERBAL PATIENTS)

- Feature 1: Assessed via RASS fluctuation or GCS change
- Feature 2: Attention Screening Exam (ASE), visual (picture recognition) or auditory (letter 'A' squeeze test)
- Feature 3: Yes/ no questions + simple commands
- Feature 4: RASS score $\neq$ 0

DELIRIUM SUBTYPES

SUBTYPE	PREVALENCE	FEATURES	PROGNOSIS
Hyperactive	~25%	Agitation, restlessness, hallucinations, combativeness, pulling at lines	Better (detected earlier)

SUBTYPE	PREVALENCE	FEATURES	PROGNOSIS
Hypoactive	~25%	Lethargy, reduced motor activity, flat affect, withdrawal	Worse (often missed, higher mortality)
Mixed	~45%	Fluctuates between hyperactive and hypoactive features within hours	Intermediate
No motor subtype	~5%	CAM-positive but no motor disturbance	Variable

EXAM STRATEGY

Hypoactive delirium is the most commonly missed subtype and carries the worst prognosis. Examiners frequently ask why it is underdiagnosed, answer: mistaken for depression or fatigue, no behavioural disturbance to alert staff.

DELIRIUM MANAGEMENT ALGORITHM

STEP 1: IDENTIFY AND TREAT THE UNDERLYING CAUSE

MNEMONIC, I WATCH DEATH

I WATCH DEATH

Infection · Withdrawal · Acute metabolic · Trauma · CNS pathology · Hypoxia · Deficiencies · Endocrine · Acute vascular · Toxins/drugs · Heavy metals

STEP 2: NON-PHARMACOLOGICAL INTERVENTIONS (FIRST-LINE FOR ALL SUBTYPES)

- **Reorientation**, clocks, calendars, familiar objects, consistent caregivers, family presence
- **Sleep hygiene**, minimise night-time interventions, lights off, reduce noise, avoid sedating medications as sleep aids
- **Sensory optimisation**, ensure hearing aids and glasses are in place
- **Mobilisation**, early and frequent ambulation where medically safe
- **Hydration and nutrition**, correct dehydration, ensure adequate caloric intake
- **Minimise restraints**, physical restraints worsen agitation and prolong delirium
- **Medication review**, discontinue or reduce anticholinergics, benzodiazepines, opioids where possible

STEP 3: PHARMACOLOGICAL MANAGEMENT (WHEN NON-PHARMACOLOGICAL MEASURES INSUFFICIENT)

INDICATIONS FOR PHARMACOTHERAPY: SEVERE AGITATION THREATENING SAFETY, DISTRESSING HALLUCINATIONS/DELUSIONS, RISK OF SELF-HARM OR HARM TO STAFF

AGENT	DOSE	ROUTE	NOTES
Haloperidol	0.5–2 mg, repeat q30min PRN	PO/IM/IV	First-line for hyperactive delirium. Monitor QTc. Avoid in Parkinson's and DLB.
Quetiapine	12.5–50 mg BD	PO	Preferred in Parkinson's/DLB. Good for mixed subtype. Sedating at low doses.
Olanzapine	2.5–5 mg	PO/IM	Alternative to haloperidol. Avoid with benzodiazepines IM (respiratory depression).
Lorazepam	0.5–2 mg	PO/IM/IV	ONLY for alcohol/benzodiazepine withdrawal delirium and hepatic encephalopathy.
Dexmedetomidine	IV infusion 0.2–0.7 µg/kg/hr	IV	ICU setting only. α ₂ -agonist. Reduces delirium duration in ventilated patients.

EXAM PEARL

Benzodiazepines worsen delirium, except in alcohol/BZD withdrawal and hepatic encephalopathy. This is the most tested pharmacological principle in delirium management.

CONSULTATION-LIAISON MODEL

THE C-L CONSULTATION PROCESS

1. **Referral receipt**, clarify the question being asked (not just "psychiatry consult")
2. **Record review**, medications (anticholinergic burden), labs, vitals, nursing notes for fluctuation
3. **Patient assessment**, cognitive screening (CAM, MMSE/MoCA), mental status exam, physical exam for medical causes

4. **Collateral history**, baseline cognitive function, timeline of changes, substance use
5. **Formulation**, biopsychosocial, with emphasis on medical contributors
6. **Recommendations**, written clearly, prioritised, with specific medication doses and monitoring parameters
7. **Follow-up**, daily re-assessment until delirium resolves or patient is discharged

DIFFERENTIAL: DELIRIUM VS DEMENTIA VS DEPRESSION VS PSYCHOSIS

FEATURE	DELIRIUM	DEMENTIA	DEPRESSION	PSYCHOSIS
Onset	Acute (hours–days)	Insidious (months–years)	Weeks	Days–weeks
Course	Fluctuating	Progressive	Diurnal variation	Sustained
Attention	Markedly impaired	Relatively pre-served early	Mildly impaired	Variable
Consciousness	Altered (clouded)	Clear until late stages	Clear	Clear
Hallucinations	Visual (common)	Visual (DLB)	Rare (mood-congruent)	Auditory (common)
Reversibility	Usually reversible	Irreversible (mostly)	Treatable	Treatable
EEG	Diffuse slowing	Normal or mild slowing	Normal	Normal

ICU PSYCHIATRIC COMPLICATIONS

ICU DELIRIUM

ICU delirium affects up to **80% of mechanically ventilated patients** and is independently associated with longer ICU stay, higher mortality, and long-term cognitive impairment.

RISK FACTORS SPECIFIC TO ICU

- **Predisposing:** Age >65, pre-existing cognitive impairment, alcohol use disorder, high APACHE II score
- **Precipitating:** Sedation (especially benzodiazepines), mechanical ventilation, sleep deprivation, immobilisation, pain, sepsis

PREVENTION: ABCDEF BUNDLE (ICU LIBERATION)

LETTER	COMPONENT	ACTION
A	Assess, prevent, and manage pain	Regular pain assessment (BPS/CPOT); analgesia-first sedation

LETTER	COMPONENT	ACTION
B	Both SATs and SBTs	Daily spontaneous awakening + breathing trials
C	Choice of analgesia and sedation	Avoid benzodiazepines; prefer propofol or dexmedetomidine
D	Delirium: assess, prevent, manage	CAM-ICU every shift; non-pharmacological prevention
E	Early mobility and exercise	Progressive mobilisation from passive ROM to ambulation
F	Family engagement and empowerment	Open visitation, family involvement in care, orientation cues

CLINICAL ANCHOR

Agitation management in ICU: First rule out pain, full bladder, constipation, hypoxia, and drug withdrawal before reaching for sedation. Use the RASS to target light sedation (RASS 0 to -1) rather than deep sedation.

POST-ICU PSYCHIATRIC SEQUELAE

- **Post-intensive care syndrome (PICS):** cognitive impairment (25–80%), depression (30%), PTSD (10–50%), anxiety (70%)
- Duration of delirium correlates with severity of long-term cognitive impairment
- ICU diaries and psychological follow-up at 3 months recommended

Model Answer Outline, "Discuss the pathophysiology and management of delirium" (10 marks)

INTRODUCTION (1 MARK)

- Define delirium; state prevalence (10–31% medical, 80% ICU)

PATHOPHYSIOLOGY (3 MARKS)

- Cholinergic hypothesis, reduced ACh, anticholinergic burden as top modifiable risk factor
- Neuroinflammation, cytokines cross BBB, microglial activation
- Neurotransmitter imbalance, ACh ↓, DA ↑, GABA variable, melatonin disruption
- Oxidative stress, impaired oxidative metabolism

ASSESSMENT (2 MARKS)

- CAM algorithm, 4 features, diagnostic rule (1+2+3or4)
- Subtypes: hyperactive, hypoactive (worst prognosis, most missed), mixed

MANAGEMENT (3 MARKS)

- Identify and treat cause (I WATCH DEATH mnemonic)
- Non-pharmacological, reorientation, sleep hygiene, mobilisation, sensory optimisation
- Pharmacological, haloperidol first-line, quetiapine for PD/DLB, BZDs ONLY for withdrawal

PROGNOSIS (1 MARK)

- Usually reversible; persistent delirium associated with dementia risk; mention PICS for ICU delirium

02

Sexual Disorders & Gender Dysphoria

ICD-11 classification, medication-induced dysfunction, paraphilias, and gender incongruence

SEXUAL DYSFUNCTION: ICD-11 CLASSIFICATION

ICD-11 SEXUAL DYSFUNCTIONS (BLOCK 17: CONDITIONS RELATED TO SEXUAL HEALTH)

ICD-11 moved sexual dysfunctions out of the mental disorders chapter into **Conditions Related to Sexual Health**, a deliberate destigmatisation parallel to gender incongruence reclassification.

CATEGORY	MALE	FEMALE
Desire disorders	Hypoactive sexual desire dysfunction	Sexual interest/arousal dysfunction
Arousal disorders	Erectile dysfunction	(Merged with desire in female)
Orgasmic disorders	Male early ejaculation; Delayed ejaculation	Anorgasmia
Pain disorders		Sexual pain-penetration disorder

KEY ICD-11 VS DSM-5 DIFFERENCES

- ICD-11 merges female desire + arousal into one entity; DSM-5 does the same (FSIAD)
- ICD-11 requires the dysfunction to be **not better explained by** a medical condition, substance, or relationship distress
- ICD-11 uses "dysfunction" rather than "disorder", less pathologising language
- Duration criterion: several months (ICD-11); approximately 6 months (DSM-5)

ANTIPSYCHOTIC/SSRI-INDUCED SEXUAL DYSFUNCTION

HYPERPROLACTINAEMIA PATHWAY

Dopamine D2 blockade in the tuberoinfundibular pathway → **prolactin elevation** → downstream sexual effects:

- ↑ Prolactin → ↓ GnRH pulsatility → ↓ LH/FSH → ↓ testosterone/oestrogen
- **Men:** decreased libido, erectile dysfunction, gynaecomastia, galactorrhoea
- **Women:** amenorrhoea, galactorrhoea, decreased libido, anorgasmia
- Long-term: osteoporosis risk from chronic hypogonadism

PROLACTIN-SPARING VS PROLACTIN-RAISING ANTIPSYCHOTICS

HIGH PROLACTIN RISK	MODERATE	PROLACTIN-SPARING
Risperidone, Paliperidone, Amisulpride, Sulpiride, Haloperidol	Olanzapine, Ziprasidone	Aripiprazole (partial D2 agonist → may lower prolactin), Quetiapine, Cariprazine, Clozapine

SSRI-INDUCED SEXUAL DYSFUNCTION

- Prevalence: **30–70%** of patients on SSRIs (often under-reported)
- Mechanism: serotonin ↑ → inhibits dopamine and norepinephrine pathways involved in arousal and orgasm; 5-HT_{2A}/2C stimulation inhibits NO-mediated vasodilation
- Most common complaints: delayed orgasm/anorgasmia > decreased libido > erectile dysfunction
- **Post-SSRI Sexual Dysfunction (PSSD):** rare but recognised, persists after SSRI discontinuation

EXAM PEARL

Management of SSRI-induced sexual dysfunction: (1) Wait and watch, may improve over 1–3 months; (2) Dose reduction; (3) Drug holiday (weekend breaks, risky with short-half-life SSRIs); (4) Switch to bupropion or mirtazapine; (5) Augment with bupropion or sildenafil. **Bupropion** is the antidepressant with the lowest sexual side-effect profile.

PARAPHILIC DISORDERS & GENDER INCONGRUENCE

PARAPHILIC DISORDERS: ICD-11 CLASSIFICATION

ICD-11 distinguishes between **paraphilias** (atypical sexual interests, not disorders per se) and **paraphilic disorders** (cause distress OR involve non-consenting individuals).

DISORDER	FOCUS	FORENSIC RELEVANCE
Exhibitionistic	Exposing genitals to unsuspecting persons	IPC Section 354 (outraging modesty), POCSO if minor
Voyeuristic	Observing unsuspecting persons undressing/sexual activity	IPC Section 354C (voyeurism, added 2013)
Paedophilic	Prepubescent children	POCSO Act, IPC 376, mandatory reporting
Coercive sexual sadism	Non-consenting victims; physical/psychological suffering	IPC 354, 375, 376
Frotteuristic	Touching/rubbing against non-consenting person	IPC 354 (sexual harassment)
Other	Fetishistic, transvestic (only if causing distress)	Generally not forensic unless involving non-consent

EXAM STRATEGY

For forensic questions, always mention: (1) legal framework (IPC sections, POCSO Act), (2) fitness to stand trial, (3) criminal responsibility, (4) risk assessment (static + dynamic factors), (5) treatment (CBT, anti-androgens for severe cases, relapse prevention).

GENDER INCONGRUENCE: ICD-11 RECLASSIFICATION

THE LANDMARK CHANGE

ICD-11 moved gender identity conditions from "**Mental and Behavioural Disorders**" (ICD-10 F64) to a **new chapter: "Conditions Related to Sexual Health"**. This is one of the most significant reclassifications in ICD-11.

RATIONALE FOR RECLASSIFICATION

- **Destigmatisation**, being transgender is not a mental disorder
- **Evidence-based**, distress in gender incongruence largely arises from social stigma and barriers to care, not from the experience itself
- **Retained in ICD** (not removed entirely) to ensure access to healthcare services, hormonal treatment, and surgical care
- Parallel: homosexuality was removed from ICD-10 in 1990; gender incongruence retained but reclassified

ICD-11 TERMINOLOGY

ICD-10 (OLD)	ICD-11 (NEW)	CODE
Transsexualism (F64.0)	Gender incongruence of adolescence and adulthood	HA60
Gender identity disorder of childhood (F64.2)	Gender incongruence of childhood	HA61
Dual-role transvestism (F64.1)	Removed	

MANAGEMENT OF GENDER DYSPHORIA

- **Assessment:** Comprehensive psychiatric evaluation to rule out comorbid conditions, confirm persistent gender incongruence, assess capacity for informed consent
- **Psychological support:** Affirmative therapy approach; address minority stress, family dynamics, and social transition
- **Hormonal treatment:** Cross-sex hormones under endocrinology guidance (testosterone for trans men, oestrogen + anti-androgens for trans women)
- **Surgical:** Referral after sustained gender incongruence (WPATH Standards of Care 8, no mandatory time period)
- **Indian context:** Transgender Persons (Protection of Rights) Act, 2019, legal recognition, identity certificate, prohibition of discrimination

Model Answer Outline, "Discuss the ICD-11 reclassification of gender identity disorders" (10 marks)

ICD-10 CLASSIFICATION (2 MARKS)

- F64, Gender Identity Disorders under Mental and Behavioural Disorders; categories: transsexualism, dual-role transvestism, GID of childhood

ICD-11 RECLASSIFICATION (3 MARKS)

- Moved to "Conditions Related to Sexual Health", separate from mental disorders
- Renamed: Gender incongruence of adolescence/adulthood (HA60), childhood (HA61)
- Dual-role transvestism removed entirely

RATIONALE (3 MARKS)

- Destigmatisation, evidence that distress is largely from social factors, not intrinsic
- Retained in ICD for healthcare access (hormonal, surgical, psychological support)
- Follows pattern of homosexuality removal from ICD-10 in 1990

MANAGEMENT PRINCIPLES (2 MARKS)

- Affirmative approach, multidisciplinary care, WPATH SOC 8, Indian legal framework (TG Act 2019)

CULTURE-BOUND SYNDROMES & CULTURAL FORMULATION**CULTURE-BOUND SYNDROMES: KEY ENTITIES**

SYNDROME	REGION	CORE FEATURES	CLOSEST ICD-11 CATEGORY
Dhat syndrome	South Asia (India, Pakistan, Nepal, Bangladesh)	Preoccupation with semen loss via urine, nocturnal emissions, masturbation; somatic complaints (fatigue, weakness, anxiety)	Bodily distress disorder / Hypochondriasis
Koro	Southeast Asia, China, India (sporadic)	Acute anxiety that penis (or vulva/nipples) is retracting into body and will cause death	Other specified anxiety disorder
Latah	Malaysia, Indonesia	Exaggerated startle response → echolalia, echopraxia, automatic obedience	Dissociative disorder / Tic disorder (context-dependent)
Amok	Malaysia, Philippines	Sudden episode of indiscriminate violent attack, often preceded by brooding; followed by amnesia and exhaustion	Dissociative disorder / Intermittent explosive disorder
Susto	Latin America	"Soul loss" after frightening event; malaise, anorexia, insomnia, social withdrawal	Adjustment disorder / Depressive episode
Possession states	India (widespread), sub-Saharan Africa	Trance with identity replacement by deity/spirit; may include glossolalia, altered voice, amnesia	Dissociative trance disorder / Possession trance disorder

ICD-11 APPROACH TO CULTURAL FORMULATION

ICD-11 does not use the term "culture-bound syndrome." Instead, it recognises that cultural factors shape the **expression, interpretation, and help-seeking** of all mental disorders. The approach involves:

- **Cultural identity**, patient's ethnic, linguistic, religious identity; degree of acculturation

- **Cultural conceptualisation of distress**, how the patient understands and labels their symptoms (e.g., "Dhat" rather than "anxiety")
- **Psychosocial stressors and cultural features of vulnerability**, role of family, community, migration, discrimination
- **Cultural features of the relationship between patient and clinician**, language barriers, trust, explanatory model mismatch
- **Overall cultural assessment for diagnosis and care**, how cultural factors affect diagnosis, treatment planning, prognosis

EXAM PEARL

ICD-11 replaced "culture-bound syndromes" with a dimensional, formulation-based approach to culture. The Cultural Formulation Interview (CFI) from DSM-5 is a useful structured tool, 16 questions covering 4 domains. ICD-11's approach is conceptually similar but less structured.

POSSESSION STATES: DIFFERENTIAL DIAGNOSIS

DIAGNOSIS	KEY DISTINGUISHING FEATURES
Normative possession trance	Culturally sanctioned (temple rituals, festivals); not associated with distress or impairment; community-endorsed
Dissociative trance disorder	Involuntary, distressing, causes impairment; identity disruption; occurs outside sanctioned contexts
Psychotic disorder	Persistent delusions of being possessed (not episodic trance); auditory hallucinations (command type); deterioration in functioning; lack of episodic pattern
Temporal lobe epilepsy	Stereotyped episodes, aura, automatisms, post-ictal confusion; EEG abnormalities
Malingering	External incentive; inconsistent symptoms; symptom production under observation

DHAT SYNDROME: COMPREHENSIVE REVIEW

HIGH-FREQUENCY PG EXAMS TOPIC: ASKED REPEATEDLY IN SHORT NOTES AND LONG ANSWERS

DEFINITION

Dhat syndrome is a culture-bound condition prevalent in the Indian subcontinent, characterised by **excessive preoccupation with semen loss** and attribution of a wide range of somatic and psychological symptoms to this perceived loss.

ETYMOLOGY

"Dhat" derives from the Sanskrit *dhatu* (vital body fluid / essence). Rooted in Ayurvedic belief that semen is the most refined of the seven dhatus, and its loss leads to physical and mental deterioration.

EPIDEMIOLOGY

- Predominantly affects **young men (15–30 years)**, rural or semi-urban, lower socioeconomic status
- Prevalence: accounts for 10–30% of male attendees at psychiatric outpatients in India (Bhatia & Malik, 1991)
- Similar concepts: Shen-k'uei (China), Jiryan (Middle East), Prameha (Sri Lanka)

AETIOLOGY: BIOPSYCHOSOCIAL MODEL

DOMAIN	FACTORS
Biological	Comorbid depression, anxiety; prostatic/urethral pathology (rare); phosphaturia mistaken for semen
Psychological	Sexual guilt (often from religious / moral teachings about masturbation); performance anxiety; low sexual literacy; somatisation of distress
Sociocultural	Ayurvedic semen conservation belief; traditional healer reinforcement; family silence about sexuality; peer myths

CLINICAL PRESENTATION

- **Core complaint:** passage of "Dhat" in urine (whitish discolouration), nocturnal emissions, or semen loss during defecation
- **Somatic:** fatigue, body aches, weakness, weight loss, palpitations, poor appetite
- **Psychological:** anxiety, depressed mood, guilt, poor concentration, irritability
- **Sexual:** premature ejaculation, erectile dysfunction, loss of libido
- Patients often first consult **traditional healers or quacks**, may have received harmful "treatments"

MANAGEMENT: STEPPED APPROACH

1. **Therapeutic alliance**, take the complaint seriously; do not dismiss or ridicule; validate the distress
2. **Psychoeducation**, explain semen physiology (continuous production, not a finite resource); normalise nocturnal emissions; address myths about masturbation
3. **Cognitive restructuring**, challenge catastrophic beliefs about semen loss; graduated exposure to anxiety-provoking situations

4. **Treat comorbidities**, SSRIs for comorbid depression/anxiety; PE can be treated with dapoxetine or low-dose SSRI
5. **Cultural sensitivity**, use patient's explanatory model as a bridge; involve family if appropriate; consider referencing Ayurvedic framework in psychoeducation
6. **Address sexual knowledge gaps**, basic sex education, dispel myths, address performance anxiety

MNEMONIC, DHAT MANAGEMENT: SACRED

SACRED

Sympathetic listening · Assess comorbidities · Cognitive restructuring · Reassurance with education · Explanatory model bridging · Drug therapy if needed

Model Answer Outline, "Write a short note on Dhat syndrome with management" (10 marks)

DEFINITION & ETYMOLOGY (1.5 MARKS)

- Define Dhat syndrome; mention dhatu concept, cultural context in Indian subcontinent

EPIDEMIOLOGY (1 MARK)

- Young men, rural, lower SES; 10–30% of male OPD; cross-cultural parallels

AETIOLOGY (2 MARKS)

- Biopsychosocial: Ayurvedic beliefs, sexual guilt, somatisation, comorbid depression/anxiety

CLINICAL FEATURES (2 MARKS)

- Core complaint of semen loss + somatic symptoms + psychological symptoms + sexual dysfunction

MANAGEMENT (3 MARKS)

- Psychoeducation (semen physiology, nocturnal emission normalisation)
- CBT, cognitive restructuring of catastrophic beliefs
- Pharmacotherapy for comorbidities (SSRIs)
- Cultural sensitivity, use explanatory model as bridge, involve family

PROGNOSIS (0.5 MARKS)

- Good with psychoeducation and treatment of comorbidities; refractory cases often have untreated depression

CULTURAL ADAPTATION OF PSYCHOTHERAPY IN INDIA

WHY CULTURAL ADAPTATION MATTERS

Evidence-based therapies (CBT, IPT, DBT) were developed in Western, educated, industrialised contexts. Direct application in Indian settings faces barriers:

- **Collectivistic vs individualistic values**, Indian families often make treatment decisions jointly; "autonomy" is relational, not individual
- **Explanatory models**, karma, fate, divine punishment, evil eye, humoral imbalance (vata-pitta-kapha) coexist with biomedical understanding
- **Stigma**, "psychiatry = pagal" remains pervasive; somatisation is a preferred idiom of distress
- **Therapist shortage**, task-shifting to community health workers requires simplified, manualised protocols

BERNAL'S ECOLOGICAL VALIDITY FRAMEWORK: 8 DIMENSIONS OF ADAPTATION

DIMENSION	APPLICATION IN INDIAN CONTEXT
Language	Use local language; translate idioms accurately (not literally); use Hindi/regional metaphors for cognitive concepts
Persons	Match therapist-patient on language/culture where possible; use lay counsellors (MANAS trial model)
Metaphors	Use culturally resonant stories (e.g., Panchatantra, religious parables) to explain cognitive distortions
Content	Address culturally relevant stressors, dowry, in-law conflict, arranged marriage adjustment, caste-based discrimination
Concepts	Frame "thoughts-feelings-behaviour" triangle in local terms; use "man ki baat" (mind's talk) for automatic thoughts
Goals	Include family-level goals, not just individual; acceptance of family involvement in goal-setting
Methods	Activity scheduling can include temple visits, community events; behavioural activation can leverage joint family support
Context	Deliver in primary care (PHC/CHC), community settings, or via telehealth; address migration, urbanisation stress

INDIAN EVIDENCE FOR CULTURALLY ADAPTED INTERVENTIONS

- **MANAS trial (Patel et al., 2010, Lancet):** Collaborative stepped-care with lay counsellors in Goa PHCs, reduced depression and anxiety prevalence by 30–40% compared to enhanced usual care
- **PREMIUM (Patel et al., 2017, Lancet):** Healthy Activity Program (HAP), culturally adapted behavioural activation for depression; Counselling for Alcohol Problems (CAP), MI-based, lay counsellor-delivered
- **Thinking Healthy Programme (WHO, Rahman et al., 2008, Lancet):** CBT-based, pictorial aids, delivered by Lady Health Workers for perinatal depression in Pakistan, adapted and tested in India

EXAM PEARL

When writing about cultural adaptation, always cite the **MANAS trial** (Goa, Patel et al., Lancet 2010), it is the landmark Indian RCT for task-shifting in mental healthcare and is frequently examined. Also mention the **District Mental Health Programme (DMHP)** under NMHP as the national framework for community mental health.

04

Grief, Bereavement & Adjustment Disorders

Prolonged Grief Disorder, Worden's tasks, dual process model, and adjustment disorders

NORMAL GRIEF VS PROLONGED GRIEF DISORDER

NORMAL GRIEF: FEATURES

Grief is a **universal, adaptive response** to bereavement. It is NOT a disorder. Normal grief includes:

- **Emotional:** Sadness, yearning, anger, guilt, anxiety, loneliness
- **Cognitive:** Disbelief, preoccupation with the deceased, confusion, intrusive memories
- **Behavioural:** Crying, social withdrawal, restlessness, searching behaviour, visiting grave/memorials
- **Physical:** Fatigue, insomnia, appetite changes, somatic complaints (tightness in chest, hollow stomach)
- **Timeline:** Intensity typically peaks in first 6 months; gradual adaptation over 12 months; grief "pangs" may persist but decrease in frequency and intensity

PROLONGED GRIEF DISORDER (PGD): NEW IN ICD-11 AND DSM-5-TR

PGD is a **newly codified diagnostic entity** in both ICD-11 (6B42) and DSM-5-TR (added 2022). It represents grief that is persistent, pervasive, and functionally impairing beyond what is expected.

ICD-11 DIAGNOSTIC CRITERIA (6B42)

1. Death of a close person (partner, parent, child, or other person close to the bereaved)
2. **Persistent and pervasive longing** for OR **persistent and pervasive preoccupation** with the deceased
3. Accompanied by intense emotional pain (sadness, guilt, anger, denial, blame, difficulty accepting death, feeling part of self has died, inability to experience positive mood, emotional numbness, difficulty engaging in social/other activities)
4. Duration: persists for an **abnormally long period** after the loss, at least **6 months** (minimum); longer periods may be appropriate depending on cultural/contextual norms
5. The disturbance clearly exceeds expected social, cultural, or religious norms
6. Causes **significant impairment** in personal, family, social, educational, occupational, or other important areas

KEY DISTINCTION: NORMAL GRIEF VS PGD

FEATURE	NORMAL GRIEF	PROLONGED GRIEF DISORDER
Yearning	Present, diminishes over months	Persistent, pervasive, does not diminish
Duration	Months, with gradual adaptation	≥6 months (ICD-11) / ≥12 months adults, ≥6 months children (DSM-5-TR)
Functional impact	Temporary disruption, gradual return	Significant, persistent impairment across domains
Identity	Sense of self preserved	"Part of me died"; identity confusion; difficulty imagining future
Social engagement	Gradually resumes	Persistent avoidance or inability to re-engage
Positive emotions	Can experience moments of joy	Emotional numbness; anhedonia specific to post-loss life
Cultural norms	Within expected range	Exceeds cultural/religious expectations for mourning

EXAM PEARL

PGD is **NOT the same as depression**. In PGD, the yearning is focused on the deceased and the lost relationship. In depression, the low mood and anhedonia are pervasive and not specifically tied to the loss. Comorbidity is common (~30–50%), but they are distinct entities requiring different treatments.

BEREAVEMENT COUNSELLING & ADJUSTMENT DISORDERS

BEREAVEMENT COUNSELLING MODELS

WORDEN'S FOUR TASKS OF MOURNING

TASK	DESCRIPTION	THERAPEUTIC APPROACH
1. Accept the reality of the loss	Moving from denial to cognitive and emotional acknowledgment that the person is dead and will not return	Gently revisit the circumstances of death; use the deceased's name; avoid euphemisms
2. Process the pain of grief	Experience and work through the full range of grief emotions without avoidance	Normalise emotions; create safe space for expression; identify suppressed affect
3. Adjust to a world without the deceased	External adjustments (new roles, practical tasks), internal adjustments (identity, self-concept), spiritual adjustments (meaning-making)	Problem-solving for practical challenges; identity work; meaning-making
4. Find a way to maintain connection while embarking on a new life	Establish a continuing bond with the deceased while re-engaging with life	Rituals, legacy projects, permission to form new relationships

STROEBE & SCHUT'S DUAL PROCESS MODEL (DPM)

The DPM proposes that healthy grieving involves **oscillation** between two orientations:

- **Loss-oriented coping:** Processing the loss itself, crying, yearning, reminiscing, confronting the reality of death
- **Restoration-oriented coping:** Attending to life changes, learning new skills, developing new identity, forming new relationships, distraction from grief
- **Oscillation** between these two is normal and adaptive; getting "stuck" in either orientation is problematic

CLINICAL ANCHOR

Evidence-based treatment for PGD: Complicated Grief Treatment (CGT; Shear et al., 2005), a 16-session protocol combining elements of IPT and CBT with exposure (revisiting the death story) and restoration work. RCT evidence shows CGT superior to IPT alone for PGD.

RISK FACTORS FOR COMPLICATED GRIEF

- **Relationship:** Loss of child > spouse > parent; dependent or ambivalent relationship; caregiving burden
- **Circumstances:** Sudden/violent death, suicide, uncertain death (missing persons), multiple losses

- **Personal:** Pre-existing depression/anxiety, insecure attachment style, prior losses, social isolation
- **Social:** Disenfranchised grief (loss not socially recognised, e.g., extramarital partner, pet, perinatal loss), lack of social support

ADJUSTMENT DISORDERS: ICD-11

DEFINITION

Maladaptive reaction to an identifiable psychosocial stressor, characterised by **preoccupation with the stressor** and **failure to adapt**, leading to functional impairment.

ICD-11 KEY CHANGES

- ICD-11 introduces a **core symptom: preoccupation with the stressor** (recurrent distressing thoughts, constant worry, rumination about the stressor or its consequences)
- No subtypes in ICD-11 (unlike DSM-5 which retains subtypes: with depressed mood, with anxiety, with mixed anxiety and depressed mood, with disturbance of conduct, unspecified)
- Duration: symptoms arise within 1 month of stressor; resolve within 6 months of stressor cessation
- If symptoms meet criteria for another disorder (MDD, GAD), that diagnosis takes precedence

Model Answer Outline, "Distinguish between normal grief and prolonged grief disorder" (10 marks)

NORMAL GRIEF (3 MARKS)

- Adaptive response; emotional, cognitive, behavioural, somatic features
- Peaks in first 6 months; gradual adaptation; moments of positive emotion preserved
- Within cultural norms; functional recovery over time

PROLONGED GRIEF DISORDER (4 MARKS)

- New in ICD-11 (6B42) and DSM-5-TR, define with diagnostic criteria
- Persistent yearning/preoccupation + intense emotional pain ≥ 6 months
- Exceeds cultural norms; significant functional impairment
- Distinguish from depression (grief is relationship-focused, depression is pervasive)

RISK FACTORS FOR PGD (1.5 MARKS)

- Nature of relationship, circumstances of death, personal vulnerability, disenfranchised grief

MANAGEMENT (1.5 MARKS)

- CGT (Shear et al.), 16 sessions, exposure + restoration; Worden's tasks; Dual Process Model; SSRIs for comorbid depression

05

Somatic Symptom & Related Disorders

Bodily Distress Disorder, FNSD, factitious disorder, and stepped-care management

ICD-11 RECLASSIFICATION: SOMATIC SYMPTOM DISORDERS

THE PARADIGM SHIFT

ICD-11 made fundamental changes to the classification of somatic symptom disorders, moving away from the mind-body dualism that plagued ICD-10:

- **ICD-10:** "Somatoform disorders" (F45), defined by absence of organic pathology (diagnosis by exclusion)
- **ICD-11:** Focuses on **positive psychological and behavioural features**, excessive health-related cognitions/behaviours, regardless of whether a medical condition is present

ICD-11 CLASSIFICATION

BODILY DISTRESS DISORDER (BDD: 6C20)

Replaces most of ICD-10's somatoform categories (somatisation disorder, undifferentiated somatoform disorder, somatoform autonomic dysfunction).

- **Bodily symptoms** that are distressing and result in **excessive attention** directed towards the symptoms (repeated contact with health services, avoidance behaviours, constant symptom checking)
- Symptoms may or may not be attributable to a medical condition, **the diagnosis does not require absence of organic pathology**
- Severity qualifiers: **Mild** (one body system, intermittent) or **Moderate to severe** (multiple systems, persistent, significantly impairing)

COMPARISON: ICD-10 → ICD-11

ICD-10	ICD-11	KEY CHANGE
Somatisation disorder (F45.0)	Bodily Distress Disorder (6C20)	Unified under one category; no arbitrary symptom counts; positive criteria instead of exclusion-based
Undifferentiated somatoform disorder (F45.1)		
Somatoform autonomic dysfunction (F45.3)		
Hypochondriacal disorder (F45.2)	Hypochondriasis (6B23), under OCD-related disorders	Moved to OCD spectrum; reconceptualised as health anxiety

ICD-10	ICD-11	KEY CHANGE
Dissociative (conversion) disorders (F44)	Dissociative Neurological Symptom Disorder (6B60)	Now in dissociative disorders chapter; standalone entity
Pain disorder (F45.4)	Chronic primary pain (MG30.0)	Moved to pain chapter, no longer a "psychiatric" diagnosis

SOMATIC SYMPTOM DISORDER (DSM-5) VS BODILY DISTRESS DISORDER (ICD-11)

FEATURE	SSD (DSM-5)	BDD (ICD-11)
Name	Somatic Symptom Disorder	Bodily Distress Disorder
Focus	Disproportionate thoughts, feelings, behaviours about somatic symptoms	Excessive attention to bodily symptoms
Requires organic exclusion?	No	No
Severity	Mild, moderate, severe (specifier)	Mild vs moderate-to-severe
Duration	≥6 months	Several months
Body system specification	Not required	Yes (gastrointestinal, cardiopulmonary, etc.)

EXAM PEARL

The single most important change: **both ICD-11 and DSM-5 abandoned the requirement to prove absence of organic pathology.** A patient with diabetes AND excessive health-related distress can receive a diagnosis of BDD/SSD. The diagnosis rests on positive psychological criteria, not negative medical workup.

FNSD, FACTITIOUS DISORDER & MANAGEMENT

FUNCTIONAL NEUROLOGICAL SYMPTOM DISORDER (FNSD) / CONVERSION DISORDER

ICD-11 renames this as **Dissociative Neurological Symptom Disorder (6B60)** and places it under the dissociative disorders chapter, a significant reclassification.

KEY CLINICAL FEATURES

- **Motor symptoms:** weakness/paralysis, abnormal movements (tremor, dystonia, myoclonus), gait disorder, aphonia
- **Sensory symptoms:** anaesthesia, blindness, deafness
- **Seizure-like episodes:** Psychogenic Non-Epileptic Seizures (PNES), most common FNSD presentation in referral centres
- **Positive clinical signs** (demonstrate inconsistency, NOT absence of pathology):

SIGN	TEST	INTERPRETATION
Hoover's sign	Involuntary hip extension of "weak" leg when flexing contralateral leg against resistance	Positive = functional weakness
Drift without pronation	Arm drifts downward without pronation (organic UMN lesion → drift WITH pronation)	Functional weakness
Give-way weakness	Initial resistance followed by sudden collapse of effort	Functional (but not specific)
Tubular visual fields	Visual field testing at different distances, field stays same size (should expand with distance)	Functional blindness
PNES features	Asynchronous limb movements, eye closure during episode, prolonged duration, no post-ictal confusion, preserved corneal reflex	Psychogenic seizure (gold standard = video EEG)

FACTITIOUS DISORDER VS MALINGERING

FEATURE	FACTITIOUS DISORDER	MALINGERING
Motivation	Assume the sick role (internal psychological need)	External incentive (compensation, avoiding duty, legal advantage)
Symptom production	Deliberate fabrication or induction of symptoms	Deliberate fabrication or exaggeration
Awareness	Aware of producing symptoms; may not understand why	Fully aware and goal-directed
Diagnosis in ICD-11	Yes, Factitious Disorder (6D50)	Not a diagnosis, coded as Z-code (reason for encounter)
Munchausen's	Severe form, peregrinating, dramatic presentations, pathological lying (pseudologia fantastica)	N/A
By proxy	Factitious disorder imposed on another (Munchausen by proxy), child abuse	N/A

MANAGEMENT: STEPPED CARE FOR SOMATIC SYMPTOM DISORDERS

- Step 1, Primary care management:** Regular scheduled appointments (not symptom-driven), single treating physician, limit investigations, validate distress, physical activity prescription

2. **Step 2, Low-intensity psychological:** Psychoeducation, graded exercise therapy, guided self-help, relaxation training
3. **Step 3, Specialist psychological:** CBT (most evidence, addresses catastrophic cognitions, safety behaviours, attention to symptoms); ACT; brief psychodynamic therapy
4. **Step 4, Pharmacological:** SSRIs/SNRIs (for comorbid depression/anxiety and direct analgesic effects); low-dose TCAs for pain; avoid opioids and benzodiazepines

Model Answer Outline, "Discuss the ICD-11 approach to somatic symptom disorders" (10 marks)

ICD-10 LIMITATIONS (2 MARKS)

- Mind-body dualism; diagnosis by exclusion of organic pathology; arbitrary symptom counts; poorly reliable categories

ICD-11 RECLASSIFICATION (4 MARKS)

- Bodily Distress Disorder, positive criteria (excessive attention to symptoms), severity-based, body system specification
- Hypochondriasis → OCD spectrum
- FNSD → Dissociative Neurological Symptom Disorder in dissociative disorders chapter
- Pain disorder → Chronic primary pain in pain chapter

ADVANTAGES OF ICD-11 APPROACH (2 MARKS)

- No exclusion-of-organic-pathology requirement, reduces iatrogenic harm from excessive investigation
- Positive diagnostic criteria improve reliability and reduce stigma
- Can coexist with medical conditions, more clinically realistic

MANAGEMENT (2 MARKS)

- Stepped care: scheduled appointments → psychoeducation/graded exercise → CBT → SSRIs. Emphasise therapeutic alliance, limit investigations, single physician model.

06

Adolescent Psychiatry (Extended)

Early-onset psychosis, conduct disorder, tics, adolescent substance use, and safety planning

EARLY-ONSET PSYCHOSIS & CONDUCT DISORDER

EARLY-ONSET PSYCHOSIS (EOP) VS CHILDHOOD-ONSET SCHIZOPHRENIA (COS)

FEATURE	EARLY-ONSET PSYCHOSIS (EOP)	CHILDHOOD-ONSET SCHIZOPHRENIA (COS)
Age of onset	13–17 years	<13 years (very rare: 1 in 10,000–30,000)

FEATURE	EARLY-ONSET PSYCHOSIS (EOP)	CHILDHOOD-ONSET SCHIZOPHRENIA (COS)
Prevalence	~1–2% of all schizophrenia	<0.01% of children
Premorbid functioning	Variable	Often impaired: language delays, motor delays, social difficulties, ADHD-like features
Hallucinations	Predominantly auditory	Auditory + visual hallucinations more common than in adult-onset
Thought disorder	Present	Marked formal thought disorder; may be mistaken for developmental language disorder
Negative symptoms	Variable	Prominent and early; flat affect, avolition, social withdrawal
Prognosis	Intermediate	Worse than adult-onset; progressive grey matter loss on serial MRI; poor functional outcome

DIFFERENTIAL DIAGNOSIS: KEY CONSIDERATIONS

- **Autism spectrum disorder**, social withdrawal and odd behaviour may mimic negative symptoms; ASD does not have true hallucinations or delusions (distinguish from fantasy play)
- **Bipolar disorder with psychotic features**, episodic course, mood congruence, grandiosity
- **PTSD with dissociation**, flashbacks may be mistaken for hallucinations; trauma history crucial
- **Substance-induced psychosis**, cannabis, synthetic cannabinoids, stimulants; urine drug screen mandatory
- **Anti-NMDA receptor encephalitis**, young females, psychiatric symptoms preceding neurological; test for anti-NMDAR antibodies
- **22q11.2 deletion syndrome (DiGeorge)**, 25% develop psychosis; congenital cardiac anomalies, palatal abnormalities, learning difficulties

EXAM PEARL

Always mention **anti-NMDA receptor encephalitis** in the differential of first-episode psychosis in young people, it is potentially reversible and increasingly tested in exam vivas. Also mention **22q11.2 deletion syndrome** as the strongest genetic risk factor for schizophrenia.

CONDUCT DISORDER

ICD-11 CLASSIFICATION

Conduct-dissocial disorder (6C91), a pattern of repetitive and persistent behaviour that violates the basic rights of others or major age-appropriate societal norms.

CORE BEHAVIOURS (4 CLUSTERS)

1. **Aggression to people and animals**, bullying, fighting, use of weapons, physical cruelty, robbery
2. **Destruction of property**, fire-setting, deliberate vandalism
3. **Deceitfulness or theft**, lying, shoplifting, breaking into others' property
4. **Serious rule violations**, truancy, running away, staying out at night despite parental prohibition

CALLOUS-UNEMOTIONAL (CU) TRAITS: ICD-11 SPECIFIER

ICD-11 includes a "**with limited prosocial emotions**" specifier (equivalent to DSM-5's CU specifier). Features:

- Lack of remorse or guilt
- Callous, lack of empathy; unconcerned about others' feelings
- Unconcerned about performance, not bothered by poor schoolwork or disappointing authority figures
- Shallow or deficient affect, emotions appear insincere, used to manipulate

EXAM STRATEGY

CU traits are **the most important prognostic marker** in conduct disorder. They predict: (1) more severe and stable antisocial behaviour, (2) poorer treatment response to standard behavioural interventions, (3) higher risk of adult antisocial personality disorder. Always mention CU traits when discussing conduct disorder aetiology or prognosis.

CONDUCT DISORDER MANAGEMENT, TIC DISORDERS & ADOLESCENT SUBSTANCE USE

CONDUCT DISORDER: MANAGEMENT

PSYCHOSOCIAL INTERVENTIONS (FIRST-LINE)

INTERVENTION	TARGET AGE	DESCRIPTION	EVIDENCE LEVEL
Parent Management Training (PMT)	3–12 years	Teaching parents consistent discipline, positive reinforcement, effective commands, time-out techniques	Strong RCT evidence

INTERVENTION	TARGET AGE	DESCRIPTION	EVIDENCE LEVEL
Multisystemic Therapy (MST)	12–17 years	Intensive family- and community-based; addresses family, peer, school, and neighbourhood factors simultaneously	Strong; reduces incarceration
Functional Family Therapy (FFT)	11–18 years	Brief (8–12 sessions); targets family interaction patterns that maintain problem behaviour	Moderate-strong
Problem-Solving Skills Training (PSST)	7–14 years	CBT-based; teaches cognitive steps for interpersonal problem-solving; reduces hostile attribution bias	Moderate
Incredible Years Programme	3–8 years	Group-based parent training + child social skills; video-modelling of parenting techniques	Strong

PHARMACOLOGICAL (ADJUNCTIVE ONLY: NO DRUG IS FIRST-LINE FOR CONDUCT DISORDER)

- **Risperidone** (0.25–1.5 mg), for severe aggression unresponsive to psychosocial interventions; short-term use; monitor metabolic effects
- **Methylphenidate**, when comorbid ADHD is present (treating ADHD reduces ODD/CD severity by 30–50%)
- **Mood stabilisers** (lithium, valproate), for explosive aggression, limited evidence in children
- **No evidence for SSRIs** in conduct disorder without comorbid depression/anxiety

TIC DISORDERS & TOURETTE SYNDROME

CLASSIFICATION

DIAGNOSIS	CRITERIA	DURATION
Provisional tic disorder	Motor and/or vocal tics	<1 year
Chronic motor tic disorder	Motor tics only (no vocal)	≥1 year
Chronic vocal tic disorder	Vocal tics only (no motor)	≥1 year
Tourette syndrome	Multiple motor + ≥1 vocal tic (not necessarily concurrent)	≥1 year; onset <18 years

MANAGEMENT: STEPPED APPROACH

1. **Psychoeducation and watchful waiting**, many tics improve by late adolescence; explain waxing-waning course
2. **CBIT (Comprehensive Behavioural Intervention for Tics)**, first-line therapy; includes habit reversal training (awareness training + competing response training) + functional intervention; NNT ~3
3. **Pharmacological (moderate-severe)**: Alpha-2 agonists (clonidine 0.05–0.3 mg, guanfacine) → atypical antipsychotics (aripiprazole first choice, risperidone) → fluphenazine, pimozide (second-line, QTc monitoring)

ADOLESCENT SUBSTANCE USE: SCREENING & BRIEF INTERVENTION

CRAFT SCREENING TOOL (FOR AGES 12–21)

MNEMONIC, CRAFT

CRAFT

Car, Have you ridden in a CAR driven by someone (including yourself) who was high?

Relax, Do you use alcohol / drugs to RELAX, feel better, or fit in?

Alone, Do you ever use alcohol / drugs while you are ALONE?

Forget, Do you ever FORGET things you did while using?

Friends, Do FRIENDS or family tell you to cut down?

Trouble, Have you gotten into TROUBLE while using?

Scoring: ≥2 positive = significant problem; sensitivity 76%, specificity 94%

ADOLESCENT-SPECIFIC SAFETY PLANNING

- Screen every adolescent presenting with substance use for **suicidal ideation** (ASQ or Columbia Protocol)
- Involve parents / guardians unless safety concern (balance confidentiality with duty to protect)
- Safety plan components: warning signs, internal coping, social supports, adult contacts, restricting means, emergency numbers
- **Means restriction**, remove / lock medications, restrict access to lethal means (particularly relevant for pesticide ingestion in rural India)

Model Answer Outline, "Discuss the management of conduct disorder in adolescents" (10 marks)

DEFINITION & EPIDEMIOLOGY (1.5 MARKS)

- Define CD; prevalence 2–10%; male predominance; childhood-onset vs adolescent-onset

ASSESSMENT (1.5 MARKS)

- Multidomain: family (parenting style, conflict, abuse), peer (deviant peer group), school (academic failure), individual (CU traits, comorbid ADHD, substance use); functional

assessment of aggression triggers

PSYCHOSOCIAL INTERVENTIONS (4 MARKS)

- PMT for younger children (3–12), consistent discipline, positive reinforcement
- MST for adolescents, intensive, community-based, addresses all ecological systems
- FFT, brief, targets family interaction patterns
- PSST / CBT, reduces hostile attribution bias, problem-solving skills

PHARMACOLOGICAL (2 MARKS)

- Not first-line; risperidone for severe aggression (short-term); methylphenidate when comorbid ADHD
- No evidence for SSRIs in CD without depression

PROGNOSIS (1 MARK)

- CU traits predict worse outcome; childhood-onset worse than adolescent-onset; 40% develop ASPD in adulthood

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