

PSYCHIATRY POSTGRADUATE EXAMS

Weave Study Guides

Paper II, Clinical Psychiatry

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 II

Paper II

Clinical Psychiatry

Clinical psychiatry. The major syndromes from schizophrenia and mood through anxiety, substance use, and personality.

GUIDE 06 · PART II

Schizophrenia Spectrum

Paper II · Clinical Psychiatry. Six study modes, from notes to quick review.

Study Notes

1. EPIDEMIOLOGY

1.1 Prevalence and Incidence

PARAMETER	VALUE	NOTES
Lifetime prevalence	~1% (0.7-1.5%)	Remarkably consistent worldwide
Point prevalence	~0.3-0.5%	At any given time
Annual incidence	15-25 per 100,000	WHO 10-country study
Morbid risk	~0.85%	Lifetime probability of developing
DALYs ranking	Top 15 globally	Disproportionate to prevalence

1.2 Age of Onset

SEX	PEAK ONSET	SECOND PEAK
Males	18-25 years	
Females	25-35 years	40-45 years (perimenopausal)

- Mean onset: Males ~21 years, Females ~27 years
- **The estrogen hypothesis** explains the later female onset and second perimenopausal peak (loss of protective dopamine-modulating effects of estrogen)
- Very early onset (<13 years) = childhood-onset schizophrenia (rare, poor prognosis)
- Late-onset (>40 years) = more paranoid, better premorbid, more female, better outcome

1.3 Sex Differences

FEATURE	MALES	FEMALES
Onset	Earlier	Later
Premorbid functioning	Worse	Better
Negative symptoms	More prominent	Less prominent
Affective symptoms	Less	More
Response to treatment	Poorer	Better

FEATURE	MALES	FEMALES
Course	More chronic	More episodic
Structural brain changes	More prominent	Less prominent

1.4 Urban-Rural and Migration

- **Urban birth effect:** 2-3x higher incidence in urban settings (social drift + social causation)
- **Migration:** 2-3x increased risk, especially in ethnic minorities (social defeat hypothesis)
- **Season of birth:** Slight excess of winter / spring births in Northern Hemisphere (~5-8% excess)
- **Latitude effect:** Higher incidence at higher latitudes (vitamin D hypothesis)

EXAM PEARL

Clinical vignette: A 22-year-old male college dropout, living alone in the city, with progressive social withdrawal over 2 years followed by auditory hallucinations. Urban + male + early onset + poor premorbid = high-risk profile.

2. CLINICAL FEATURES

2.1 Positive Symptoms

HALLUCINATIONS

TYPE	FREQUENCY	FEATURES
Auditory	60-80%	Most common; voices discussing (third person), running commentary, command hallucinations
Visual	15-30%	Consider organic causes first
Tactile	5-15%	Formication, sexual sensations
Olfactory	5-10%	Often unpleasant
Gustatory	<5%	Rare

Key auditory hallucination features in schizophrenia:

- Third-person voices (two or more voices arguing about the patient), Schneider's FRS
- Running commentary (voice describing actions as they happen), Schneider's FRS
- Command hallucinations, assess always for dangerousness
- Thought echo (echo de la pensee), hearing own thoughts spoken aloud

- Location: typically perceived as external, in clear consciousness

DELUSIONS

TYPE	DESCRIPTION	EXAMPLE
Persecutory	Most common (~65%)	"The government is monitoring me through my phone"
Referential	Events have special personal meaning	"The newsreader is sending me coded messages"
Grandiose	Inflated self-importance	"I am the reincarnation of a god"
Religious	Religious themes	"I have been chosen by God for a special mission"
Somatic	Bodily function/ disease	"My organs are rotting inside"
Erotomaniac	Someone is in love with them	"The actress is secretly in love with me"
Nihilistic	Non-existence of self/ world	Less common, more in depression
Control/passivity	External control of thoughts/ actions	Schneider's FRS

Passivity phenomena (Schneider's FRS):

- Thought insertion, alien thoughts placed into mind
- Thought withdrawal, thoughts removed from mind
- Thought broadcasting, thoughts transmitted to others
- Made feelings/ impulses/ actions, will is replaced by external agency
- Somatic passivity, bodily sensations imposed from outside

FORMAL THOUGHT DISORDER (FTD)

TYPE · DESCRIPTION

Loosening of associations Ideas slip from one track to another with no logical connection

Tangentiality Responses obliquely related or unrelated to question

Derailment Gradual deviation from topic

Word salad (schizophasia) Incomprehensible mixture of words

Neologisms Made-up words

Clang associations Words connected by sound not meaning

Thought blocking Sudden interruption of train of thought

Circumstantiality Excessive detail but eventually reaches the point

Perseveration Inappropriate repetition of themes

Echolalia Repeating words/phrases of examiner

DISORGANIZED BEHAVIOR

- Unpredictable agitation
- Inappropriate sexual behavior
- Bizarre dress/appearance
- Disorganized goal-directed activity
- Catatonic features (see Section 5)

2.2 Negative Symptoms

Andreasen's Classification (Scale for Assessment of Negative Symptoms, SANS)

SYMPTOM	DEFINITION	CLINICAL PRESENTATION
Affective flattening	Reduced emotional expression	Unchanging facial expression, poor eye contact, reduced gestures, monotone voice
Alogia	Poverty of speech/content	Brief, empty replies; increased latency
Avolition/Apathy	Reduced motivation/initiation	Poor hygiene, inability to persist at work/school
Anhedonia/Asociality	Inability to experience pleasure; reduced social drive	No recreational interests, few/no friends, no sexual interest
Attentional impairment	Difficulty maintaining focus	Social inattentiveness, distractibility during interview

Primary vs Secondary Negative Symptoms:

FEATURE	PRIMARY	SECONDARY
Cause	Core illness process	Medication, depression, positive symptoms, understimulation
Onset	Insidious, from prodrome	After treatment initiation or during acute phase
Response to treatment	Poor	May improve by addressing cause

FEATURE	PRIMARY	SECONDARY
Neurobiological basis	Mesocortical dopamine hypofunction	Varies

Deficit syndrome (Carpenter & Kirkpatrick): Primary, enduring negative symptoms present during and between psychotic episodes. ~15-25% of schizophrenia cases. Associated with worse cognitive impairment and poorer outcome.

EXAM STRATEGY

When asked about negative symptoms, ALWAYS mention primary vs secondary distinction and deficit syndrome. This shows depth.

2.3 Cognitive Symptoms

Often the most disabling domain but historically overlooked.

DOMAIN	DEFICIT	FUNCTIONAL IMPACT
Attention	Sustained attention impaired	Can't follow conversations, read
Working memory	Verbal and spatial	Can't hold information to use it
Executive function	Planning, abstraction, set-shifting	Can't organize daily tasks
Processing speed	Slowed	Everything takes longer
Verbal memory	Encoding and retrieval	Learning new information difficult
Social cognition	Theory of mind, emotion recognition	Misreads social cues

- Present in 80% of patients
- Often predate psychosis onset by years
- Best predictor of functional outcome (more than positive or negative symptoms)
- Largely unresponsive to antipsychotics
- MATRICS Consensus Cognitive Battery (MCCB), 7 domains, gold standard assessment

2.4 Schneider's First Rank Symptoms (FRS)

Kurt Schneider (1959), proposed these as "first rank" because of their diagnostic weighting, NOT because of frequency or severity.

CATEGORY · SYMPTOMS

Auditory hallucinations Voices arguing (third person), Running commentary, Thought echo

Passivity experiences Made feelings, Made impulses, Made actions (volitional), Somatic passivity

Thought interference Thought insertion, Thought withdrawal, Thought broadcasting

Delusional perception Normal perception → immediate delusional significance (two-stage: perception + delusional interpretation)

Second Rank Symptoms:

- Other hallucinations (visual, tactile, olfactory)
- Perplexity
- Depressive and euphoric mood changes
- Emotional blunting
- Delusional intuition (sudden delusional idea, not based on perception)

Current Status of FRS:

- NOT pathognomonic for schizophrenia (found in 10-20% of bipolar, temporal lobe epilepsy, substance-induced psychosis)
- Removed as a special criterion in DSM-5 (previously, one FRS alone was sufficient in DSM-IV)
- ICD-11 also does not give FRS special diagnostic weight
- Useful clinically for phenomenological description
- Cross-cultural validity questioned (different rates across cultures)
- Still frequently asked in exams despite diminished diagnostic status

EXAM PEARL

Clinical vignette: A 28-year-old woman believes that her neighbor is inserting sexual thoughts into her mind (thought insertion) and that her thoughts are being broadcast on the radio (thought broadcasting). These are FRS, but they are not diagnostic of schizophrenia alone. Rule out mania, substance use, organic causes.

3. CLASSIFICATION

3.1 Historical Evolution

CONTRIBUTOR	YEAR	CONCEPT
Kraepelin	1896	Dementia praecox, distinguished from manic-depressive insanity by deteriorating course

CONTRIBUTOR	YEAR	CONCEPT
Bleuler	1911	Coined "schizophrenia" (split mind); described the 4 A's
Schneider	1959	First Rank Symptoms, phenomenological approach
Crow	1980	Type I (positive, good prognosis) vs Type II (negative, poor prognosis)
Andreasen	1982	Positive vs negative symptom dimensions
Liddle	1987	Three-factor model: psychomotor poverty, disorganization, reality distortion
DSM-5	2013	Dropped subtypes, dimensional assessment
ICD-11	2019	Dropped subtypes, added symptom qualifiers

Bleuler's 4 A's (Fundamental Symptoms):

1. Association disturbance (loosening), considered most fundamental
2. Affect disturbance (inappropriate, blunted)
3. Ambivalence (coexistence of opposite feelings)
4. Autism (withdrawal into inner world)

Accessory symptoms (not fundamental): Hallucinations, delusions

3.2 ICD-11 vs DSM-5

FEATURE	ICD-11 (6A20)	DSM-5 (295.90)
Duration	1 month of symptoms	6 months (including prodrome)
Subtypes	Dropped	Dropped
Symptom qualifiers	Yes, positive, negative, depressive, manic, psychomotor, cognitive	Dimensional assessment (CRDPSS)
Social/occupational dysfunction	Not required	Required
Schneider's FRS	No special weight	No special weight
Catatonia	Can be specified as qualifier	Separate specifier
First episode specifier	Yes	Yes

FEATURE	ICD-11 (6A20)	DSM-5 (295.90)
Course specifiers	First episode, multiple episodes, continuous	First episode, multiple episodes, continuous, unspecified

Key points for exam:

- ICD-11 requires only 1 month of symptoms; DSM-5 requires 6 months total
- Both dropped subtypes (paranoid, hebephrenic, etc.)
- ICD-11 uses symptom qualifiers for dimensional profiling
- DSM-5 requires functional impairment; ICD-11 does not mandate it
- Both removed special status of Schneider's FRS

3.3 Diagnostic Criteria: DSM-5

Criterion A (at least 2, for significant portion of 1 month, at least one must be 1, 2, or 3):

1. Delusions
2. Hallucinations
3. Disorganized speech
4. Grossly disorganized or catatonic behavior
5. Negative symptoms

Criterion B: Social/occupational dysfunction

Criterion C: Duration of at least 6 months (including prodrome/residual)

Criterion D: Schizoaffective and mood disorder excluded

Criterion E: Not due to substance/medical condition

Criterion F: If ASD or childhood communication disorder present, diagnosis only if prominent delusions/hallucinations for at least 1 month

4. SUBTYPES (HISTORICAL)

Why they were dropped: Low diagnostic reliability, limited clinical utility, patients shifted between subtypes over time, did not predict treatment response or outcome.

SUBTYPE	KEY FEATURES	NOTES
Paranoid	Prominent delusions (persecutory/grandiose) + auditory hallucinations; relatively preserved cognition and affect	Most common, best prognosis among subtypes

SUBTYPE	KEY FEATURES	NOTES
Hebephrenic (Disorganized)	Disorganized speech + behavior, inappropriate / flat affect, early onset	Poor prognosis, marked regression
Catatonic	Catatonic features dominant	Now a separate specifier; can occur in mood disorders, medical conditions
Simple	Insidious negative symptoms without prominent positive symptoms	Retained in ICD-10 (F20.6) as a separate entity; not in DSM-5
Residual	Past episode, currently mainly negative symptoms	Now covered by course specifiers
Undifferentiated	Meets criteria but no single subtype dominant	The "waste basket" category

EXAM STRATEGY

Know subtypes for ICD-10 questions (still appear in PYQs) but always mention they are dropped in ICD-11 / DSM-5.

5. CATATONIA

5.1 Clinical Features

Bush-Francis Catatonia Rating Scale (BFCRS), 23 items, most widely used.

Screening items (14 items):

Stupor No psychomotor activity, not actively relating to environment

Mutism No or minimal verbal response

Staring Fixed gaze, decreased blinking

Posturing Spontaneous maintenance of a position against gravity

Grimacing Maintenance of odd facial expressions

Echopraxia Mimicking examiner's movements

Echolalia Mimicking examiner's speech

Stereotypy Repetitive, non-goal-directed motor activity

Mannerisms Odd, purposeful movements (caricature of normal actions)

Verbigeration Repetition of phrases/sentences (like a stuck record)

Rigidity Maintenance of rigid posture despite attempts to be moved

Negativism Opposition or no response to external stimuli/instructions

Waxy flexibility Slight, even resistance to repositioning by examiner (like bending a candle)

Withdrawal Refusal to eat, drink, or make eye contact

Additional features:

- **Catalepsy**, maintaining an externally imposed posture
- **Ambitendency**, appearing "stuck" in indecisive, hesitant movements
- **Automatic obedience**, exaggerated cooperation
- **Mitgehen**, arm raised to light pressure despite instruction to resist
- **Gegenhalten**, resistance equal to force of stimulus
- **Grasp reflex**, grasps examiner's hand despite instruction not to

5.2 Catatonia in Current Classification

ASPECT	DSM-5	ICD-11
Status	Separate specifier applicable to multiple disorders	Symptom qualifier for schizophrenia; also standalone category (6A40)
Associated with	Schizophrenia, mood disorders, medical conditions, NMS	Same
Standalone diagnosis	Catatonic Disorder Due to Another Medical Condition (293.89)	Catatonia associated with another mental disorder; Catatonia induced by substances; Secondary catatonia

5.3 Medical Causes of Catatonia

- **Neurological:** Encephalitis (especially anti-NMDA receptor), stroke, seizures, brain tumors
- **Metabolic:** Hepatic encephalopathy, diabetic ketoacidosis, hypercalcemia, hyponatremia
- **Autoimmune:** SLE, anti-NMDA receptor encephalitis
- **Endocrine:** Thyroid storm, Addisonian crisis
- **Drug-induced:** NMS (consider as malignant catatonia), serotonin syndrome, phencyclidine
- **Infectious:** HIV, neurosyphilis

5.4 Lorazepam Challenge Test

STEP · DETAIL

Preparation Patient NPO (risk of aspiration), IV access

Dose Lorazepam 1-2 mg IV (some protocols use 2 mg)

Observation 5-15 minutes

Positive response Marked improvement in catatonic signs (reduced rigidity, starts speaking, moves)

Sensitivity ~70-80% for benzodiazepine-responsive catatonia

Duration of effect Usually temporary (hours); guides treatment approach

Treatment dose If positive: lorazepam 6-24 mg / day in divided doses

5.5 ECT for Catatonia

Indications for ECT:

- Malignant catatonia (fever, autonomic instability, elevated CK)
- Failed lorazepam challenge or inadequate response to benzodiazepines
- Life-threatening complications (dehydration, DVT, PE, pressure sores)
- Lethal catatonia / NMS unresponsive to medical management

Protocol:

- Bilateral ECT preferred
- Daily or alternate day in acute malignant catatonia
- Typically 6-10 sessions
- Response rate: 80-90% in catatonia (highest of any ECT indication)

EXAM PEARL

Clinical vignette: A 30-year-old male with known schizophrenia presents with mutism, posturing, waxy flexibility, and refusal to eat for 3 days. Vitals show low-grade fever and tachycardia. CK is elevated. This is malignant catatonia, life-threatening emergency. Start lorazepam challenge; if insufficient response, urgent bilateral ECT.

6. COURSE AND OUTCOME

6.1 Course Patterns

PATTERN	DESCRIPTION	PROPORTION
Single episode with full remission	One episode, no relapse	~15-20%
Episodic with inter-episode residual	Multiple episodes, incomplete recovery between	~35-40%
Episodic with progressive deficit	Each episode leaves increasing residual symptoms	~10-15%
Continuous	Persistent symptoms without clear episodic pattern	~10-15%
Episodic with stable deficit	Multiple episodes, stable residual between	~10-15%

General trajectory:

- Prodromal phase (months to years): Social withdrawal, decline in functioning, attenuated psychotic symptoms
- Active phase: Frank psychotic symptoms
- Residual phase: Negative symptoms predominate, some positive symptoms attenuated
- ~80% relapse within 5 years of first episode if untreated
- ~30% have a relatively good outcome, ~30% intermediate, ~30% poor, ~10% dead by suicide

6.2 Prognostic Factors

GOOD PROGNOSIS · POOR PROGNOSIS

Acute onset Insidious onset

Late onset Early onset

Female sex Male sex

Married/good social support Single/poor social support

Affective symptoms present Negative symptoms predominant

Good premorbid adjustment Poor premorbid adjustment

No family history Strong family history

Paranoid subtype Hebephrenic/disorganized subtype

Identifiable precipitant No precipitant

Developing country Developed country

No substance use Comorbid substance use

Good treatment response early Poor early treatment response

Preserved cognition Cognitive impairment

Normal brain imaging Structural brain abnormalities (enlarged ventricles)

WHO DOSMeD study finding: Better outcome in developing countries (India, Nigeria, Colombia) compared to developed countries, possibly due to extended family support, less expressed emotion, reintegration into community roles.

6.3 Andreasen's Remission Criteria (2005)

Requires ALL of the following for at least 6 months:

- Severity of $\leq$ mild (3 on PANSS 7-point scale) on EACH of these 8 items:
- Delusions (P1)
- Unusual thought content (G9)
- Hallucinatory behavior (P3)
- Conceptual disorganization (P2)
- Mannerisms/posturing (G5)
- Blunted affect (N1)
- Social withdrawal (N4)
- Lack of spontaneity (N6)

Key points:

- Both symptom severity AND time criteria must be met
- Remission $\neq$ recovery (recovery also requires functional improvement)
- Approximately 20-30% of patients meet remission criteria with treatment

6.4 Mortality and Suicide

- Life expectancy reduced by 15-20 years
 - Suicide: ~5% lifetime risk (previously cited as 10%, revised downward)
 - Highest risk: Young males, early illness, depression, previous attempts, substance use, high IQ (awareness of losses)
 - Cardiovascular disease is the leading cause of excess mortality
 - SMR (Standardized Mortality Ratio): 2-3x general population
-

7. ETIOLOGY

7.1 Dopamine Hypothesis

Evolution:

VERSION	CONCEPT	EVIDENCE
Original (Carlsson, 1960s)	Hyperdopaminergia	Amphetamine psychosis; antipsychotics block D2
Modified	Regional specificity	Mesolimbic hyperactivity → positive symptoms; Mesocortical hypoactivity → negative/cognitive symptoms
Version III (Howes & Kapur, 2009)	Aberrant salience	Presynaptic striatal dopamine dysregulation → aberrant salience assignment → psychotic experiences

Four dopamine pathways:

PATHWAY	FUNCTION	SCHIZOPHRENIA RELEVANCE
Mesolimbic	Reward, emotion	Hyperactive → positive symptoms
Mesocortical	Cognition, executive function	Hypoactive → negative + cognitive symptoms
Tuberoinfundibular	Prolactin inhibition	Blocked by antipsychotics → hyperprolactinemia
Nigrostriatal	Movement	Blocked by antipsychotics → EPS

7.2 Glutamate Hypothesis

- **NMDA receptor hypofunction**, PCP and ketamine (NMDA antagonists) produce positive AND negative symptoms and cognitive deficits
- More comprehensive than dopamine hypothesis (explains negative/cognitive symptoms)
- NMDA hypofunction on GABAergic interneurons → disinhibition of glutamate neurons → downstream dopamine dysregulation
- Glycine site modulators (D-serine, sarcosine), modest benefits in trials
- Links to neurodevelopmental model (NMDA receptors crucial for synaptic plasticity)

7.3 Serotonin Hypothesis

- 5-HT_{2A} receptor involvement (LSD, a 5-HT_{2A} agonist, causes hallucinations)

- SGAs have high 5-HT_{2A} / D₂ affinity ratio
- Pimavanserin (5-HT_{2A} inverse agonist) effective in Parkinson's psychosis
- Serotonin modulates dopamine release in mesolimbic and mesocortical pathways

7.4 Neurodevelopmental Model

EVIDENCE · DETAIL

Obstetric complications Hypoxia, maternal infection (influenza), Rh incompatibility

Minor physical anomalies Higher in schizophrenia

Premorbid abnormalities Delayed milestones, lower IQ in childhood

Structural brain changes Present at first episode (not progressive initially)

Synaptic pruning Excessive pruning in adolescence (Feinberg hypothesis)

Complement system C4 gene variants → excessive pruning (Sekar et al., 2016, Nature)

Two-hit model (Keshavan):

- **First hit:** Early neurodevelopmental insult (genetic + prenatal)
- **Second hit:** Adolescent/early adult triggers (stress, cannabis, excessive synaptic pruning)
- Disease manifests when second hit occurs on vulnerable substrate

7.5 Genetics

FINDING · DETAIL

Heritability ~80% (twin studies)

MZ concordance ~48%

DZ concordance ~17%

First-degree relative risk ~10%

Both parents affected ~46%

GWAS loci >270 identified (PGC3, 2022)

Key genes DISC1, NRG1, DTNBP1, COMT, C4A

Copy number variants 22q11.2 deletion (25-30% develop psychosis), 1q21.1, 15q13.3, 16p11.2

7.6 Cannabis and Psychosis

- Cannabis use in adolescence → 2-6x increased risk of schizophrenia
- Dose-response relationship (earlier use + higher potency = greater risk)
- THC → dopamine release in mesolimbic pathway
- CBD may have antipsychotic properties
- Gene-environment interaction: COMT Val/Val genotype + cannabis = highest risk
- Does NOT cause schizophrenia alone, requires genetic vulnerability

7.7 Other Etiological Factors

- **Expressed emotion (EE):** High EE (criticism, hostility, emotional over-involvement) → higher relapse rates (Vaughn & Leff, 1976)
- **Social isolation/defeat:** Social exclusion activates mesolimbic dopamine
- **Childhood adversity:** 2-3x increased risk with childhood trauma
- **Vitamin D deficiency:** Neonatal vitamin D levels inversely correlated with risk

8. MANAGEMENT

8.1 Antipsychotics: Overview

Mechanism: ALL effective antipsychotics block D2 receptors. The therapeutic window for D2 occupancy is **65-80%**.

D2 OCCUPANCY · EFFECT

<65% Subtherapeutic

65-80% Therapeutic range

>80% EPS/hyperprolactinemia

>90% Severe EPS, NMS risk

8.2 First-Generation Antipsychotics (FGA / "Typical")

DRUG	POTENCY	DOSE RANGE	KEY FEATURES
Chlorpromazine	Low	200-800 mg / day	Sedating, hypotensive, weight gain, historical gold standard
Haloperidol	High	5-20 mg / day	More EPS, less sedation/hypotension, available IV / IM
Trifluoperazine	High	5-20 mg / day	Widely used in India

DRUG	POTENCY	DOSE RANGE	KEY FEATURES
Fluphenazine	High	5-20 mg / day	Available as decanoate (LAI)
Thioridazine	Low	200-600 mg / day	QTc prolongation (restricted use), retinitis pigmentosa at >800mg
Flupentixol	Medium	3-18 mg / day	Available as decanoate; may have mild antidepressant properties
Pimozide	High	2-12 mg / day	Used in delusional disorder, monosymptomatic hypochondriasis
Zuclopenthixol	Medium	10-50 mg / day	Available as acetate (acute IM) and decanoate (maintenance LAI)

FGA Side Effects by Receptor:

RECEPTOR BLOCKED · SIDE EFFECTS

D2 (nigrostriatal) Acute dystonia, akathisia, parkinsonism, tardive dyskinesia

D2 (tuberoinfundibular) Hyperprolactinemia → galactorrhea, amenorrhea, sexual dysfunction, osteoporosis

H1 Sedation, weight gain

M1 (muscarinic) Dry mouth, constipation, urinary retention, blurred vision, cognitive impairment

Alpha-1 Postural hypotension, dizziness, sedation, reflex tachycardia

Extrapyramidal Symptoms (EPS), Timeline:

TIMING	EPS	FEATURES	TREATMENT
Hours-days	Acute dystonia	Torticollis, oculogyric crisis, trismus	IM/IV anticholinergic (promethazine, trihexyphenidyl)
Days-weeks	Akathisia	Subjective restlessness, inability to sit still	Propranolol, benzodiazepines, mirtazapine; reduce dose

TIMING	EPS	FEATURES	TREATMENT
Weeks-months	Drug-induced parkinsonism	Tremor, rigidity, bradykinesia	Trihexyphenidyl, reduce dose, switch to SGA
Months-years	Tardive dyskinesia	Orofacial choreoathetoid movements	Valbenazine, deutetabenazine (VMAT2 inhibitors); switch to clozapine

8.3 Second-Generation Antipsychotics (SGA / "Atypical")

Why "atypical"?

- Lower EPS risk at therapeutic doses
- 5-HT_{2A}/D₂ affinity ratio > 1 (Meltzer's hypothesis)
- Some efficacy for negative symptoms (debated)
- Fast dissociation from D₂ receptor (Kapur's hypothesis, applies to quetiapine, clozapine)

DRUG	DOSE RANGE	KEY FEATURES	MAIN SIDE EFFECTS
Risperidone	2-8 mg/day	D ₂ + 5-HT _{2A} ; dose-dependent EPS; most studied	Hyperprolactinemia (most of all SGAs), EPS at >6mg, weight gain
Olanzapine	5-20 mg/day	Broad receptor binding (MARTA); very effective	Worst metabolic profile (weight gain, diabetes, dyslipidemia)
Quetiapine	300-800 mg/day	Low D ₂ affinity, fast off; sedating	Sedation, weight gain, metabolic; minimal EPS/prolactin
Aripiprazole	10-30 mg/day	D ₂ partial agonist + 5-HT _{1A} partial agonist	Least metabolic effects; akathisia; activating
Ziprasidone	80-160 mg/day	Take with food (absorption); weight neutral	QTc prolongation (monitor), minimal metabolic
Paliperidone	3-12 mg/day	Active metabolite of risperidone; extended release	Similar to risperidone; less hepatic metabolism
Asenapine	10-20 mg/day	Sublingual; broad receptor profile	Oral hypoesthesia, weight gain
Lurasidone	40-160 mg/day	Take with food; cognitive benefits in trials	Akathisia, EPS; weight neutral; minimal metabolic

DRUG	DOSE RANGE	KEY FEATURES	MAIN SIDE EFFECTS
Iloperidone	12-24 mg / day	Slow titration needed; low EPS	Orthostatic hypotension, QTc
Brexiprazole	2-4 mg / day	D2 partial agonist (lower intrinsic activity than aripiprazole)	Less akathisia than aripiprazole; weight gain moderate
CLOZAPINE	150-900 mg / day	Gold standard for TRS, see Section 9	Agranulocytosis, metabolic, seizures, myocarditis

8.4 Newer Antipsychotics

DRUG	MECHANISM	KEY FEATURES	STATUS
Cariprazine	D3-preferring partial agonist + D2 partial agonist	Long half-life (2-4 weeks via active metabolites); superior for negative symptoms (RCT evidence vs risperidone); may help primary negative symptoms	Approved for schizophrenia, bipolar mania / depression
Lumateperone	5-HT2A antagonist + D2 partial agonist + glutamate modulation (GluN2B) + SERT inhibition	Low D2 occupancy (~40%); minimal metabolic / EPS; once daily; minimal prolactin	Approved for schizophrenia; also bipolar depression
Cobenfy (xanomeline-trospium)	M1 / M4 muscarinic agonist (xanomeline) + peripheral muscarinic antagonist (trospium)	FIRST non-D2 antipsychotic; novel mechanism; no EPS, no prolactin, no metabolic effects	FDA approved 2024; paradigm-shifting
Brexiprazole	D2 partial agonist + 5-HT1A partial agonist	Similar to aripiprazole but less akathisia	Approved; also for Alzheimer's agitation
Pimavanserin	5-HT2A inverse agonist	No D2 activity; approved for Parkinson's psychosis only	Not for schizophrenia as monotherapy

EXAM STRATEGY

Cariprazine and Cobenfy are very hot topics. For cariprazine, emphasize D3 preferring, negative symptoms, long half-life. For Cobenfy, emphasize muscarinic mechanism, no D2 blockade, no EPS / metabolic.

8.5 Long-Acting Injectables (LAI)

DRUG	FORMULATION	DOSING FREQUENCY	NOTES
Fluphenazine decanoate	FGA	Every 2-4 weeks	Oldest LAI; high EPS
Flupentixol decanoate	FGA	Every 2-4 weeks	Widely used in India
Zuclopenthixol decanoate	FGA	Every 2-4 weeks	For maintenance
Zuclopenthixol acetate	FGA	Every 2-3 days (acute)	For acute agitation (NOT for maintenance)
Haloperidol decanoate	FGA	Every 4 weeks	Common; high EPS
Paliperidone palmitate 1-monthly	SGA	Monthly	Most widely used SGA LAI
Paliperidone palmitate 3-monthly	SGA	Every 3 months	After stabilization on monthly
Paliperidone palmitate 6-monthly	SGA	Every 6 months	Newest; after 4+ months on monthly then 3-monthly
Aripiprazole monohydrate (Abilify Maintena)	SGA	Monthly	Overlap with oral aripiprazole for 14 days
Aripiprazole lauroxil (Aristada)	SGA	Every 4-8 weeks	Prodrug
Olanzapine pamoate (Zyprexa Relhyprev)	SGA	Every 2-4 weeks	Post-injection delirium/sedation syndrome (PDSS), monitor 3 hours
Risperidone LAI (Risperdal Consta)	SGA	Every 2 weeks	Microsphere technology; supplement with oral for 3 weeks

EXAM STRATEGY

LAI is a common question for compliance. Know paliperidone palmitate (1-monthly, 3-monthly, 6-monthly) and olanzapine pamoate PDSS.

8.6 Metabolic Monitoring

All patients on antipsychotics should have baseline and ongoing monitoring:

PARAMETER	BASELINE	4 WEEKS	8 WEEKS	12 WEEKS	ANNUALLY
Weight/BMI	Yes	Yes	Yes	Yes	Yes
Waist circumference	Yes			Yes	Yes
Blood pressure	Yes			Yes	Yes
Fasting glucose	Yes			Yes	Yes
Lipid profile	Yes			Yes	Yes (or every 5 years if normal)
HbA1c	Yes				Yes
Prolactin	Yes				If symptomatic

Metabolic Syndrome (ATP III criteria, any 3 of 5):

CRITERION · THRESHOLD

Waist circumference >102 cm (M), >88 cm (F)

Triglycerides >150 mg/dL

HDL cholesterol <40 mg/dL (M), <50 mg/dL (F)

Blood pressure >130/85 mmHg

Fasting glucose >100 mg/dL

Metabolic risk ranking (highest to lowest):

Clozapine = Olanzapine >> Quetiapine = Risperidone > Aripiprazole = Ziprasidone = Lurasidone

9. TREATMENT-RESISTANT SCHIZOPHRENIA (TRS)

9.1 Definition

International consensus (TRRIP Working Group, Howes et al., 2017):

- Failure of at least **2 adequate antipsychotic trials**
- Each trial: adequate dose (equivalent to 600 mg/day chlorpromazine), adequate duration (at least 6 weeks, ideally 6-8 weeks at optimal dose), adequate adherence confirmed

- At least 1 trial should be with an SGA
- Persistent moderate-severe positive symptoms despite treatment
- Significant functional impairment

Prevalence: ~30% of schizophrenia patients (one-third of all cases)

Pseudo-resistance (rule out first):

- Non-adherence (most common cause)
- Inadequate dose or duration
- Substance use (especially cannabis, stimulants)
- Pharmacokinetic factors (rapid metabolizers, check CYP1A2 for clozapine, smoking)
- Comorbid conditions (depression, OCD)

9.2 Clozapine: The Gold Standard for TRS

Evidence base:

- Kane et al. (1988), landmark trial demonstrating superiority over chlorpromazine in TRS
- Only antipsychotic with proven superiority in TRS
- Also reduces suicidality (InterSePT trial, only FDA-approved indication for suicide reduction)
- Effective for aggression and violence in schizophrenia

INITIATION PROTOCOL

DAY	DOSE	NOTES
Day 1	12.5 mg once or twice	Evening dosing preferred (sedation)
Day 2-7	Increase by 25-50 mg / day	If tolerated
Week 2-3	Increase by 50-100 mg / week	Target 300-450 mg / day
Week 4+	Adjust based on response / levels	Target plasma level >350 ng / mL
Maximum	900 mg / day	Rarely needed

Therapeutic plasma level: 350-600 ng / mL (trough level, 12 hours post-dose)

MONITORING PROTOCOL

PERIOD · WBC/ANC FREQUENCY

First 18 weeks Weekly

Weeks 18-52 Every 2 weeks (biweekly)

After 1 year Monthly

Stopping rules for neutropenia:

WBC	ANC	ACTION
>3500	>2000	Continue
3000-3500	1500-2000	Monitor twice weekly
<3000	<1500	STOP clozapine
<1000 (agranulocytosis)	<500	STOP permanently, isolate, G-CSF, antibiotics

SIDE EFFECTS

Common (>10%):

SIDE EFFECT	MECHANISM/NOTES	MANAGEMENT
Sedation	H1, M1 blockade	Give at bedtime, slow titration
Sialorrhea (hypersalivation)	M4 agonism (paradoxical)	Hyoscine patches, glycopyrrolate, atropine sublingual drops
Constipation	M1 blockade, can be FATAL (ileus)	Aggressive bowel regimen from day 1; fiber, lactulose, docusate
Weight gain	H1, 5-HT _{2C} blockade	Diet, exercise, metformin
Tachycardia	M1 blockade, alpha-1 blockade	Usually transient; propranolol if persistent
Orthostatic hypotension	Alpha-1 blockade	Slow titration, hydration
Metabolic syndrome	Multiple receptors	Monitor aggressively
Nocturnal enuresis	Unknown mechanism	Desmopressin, reduce evening fluids

Less common (1-10%):

SIDE EFFECT · NOTES

Seizures Dose-dependent (~3% at <300mg, ~5% at 300-600mg, ~8% at >600mg); add valproate prophylactically if dose >600mg

Fever Transient benign fever in first 2-3 weeks; distinguish from NMS/myocarditis/infection

OCD symptoms Due to 5-HT_{2A}/2C blockade; treat with fluvoxamine (also raises clozapine levels via CYP1A2 inhibition, dose adjust!)

Hepatotoxicity Transaminase elevation; usually transient

Rare but serious (<1%):

SIDE EFFECT	INCIDENCE	NOTES
Agranulocytosis	~0.8-1%	Highest risk in first 6 months; mandatory monitoring
Myocarditis	~1-3% (Australian data higher)	Usually first 4-6 weeks; troponin, CRP, echo if suspected
Cardiomyopathy	~0.1%	Dilated; can occur anytime
Pulmonary embolism	Increased risk	Immobility + sedation + weight gain + possibly clozapine itself
Ileus/bowel obstruction	~3% (often underdiagnosed)	Can be FATAL; prevent aggressively
NMS	Very rare with clozapine	But can occur

DRUG INTERACTIONS

INTERACTING DRUG	EFFECT ON CLOZAPINE LEVEL	MECHANISM
Smoking (tobacco)	Decreases (by 50%)	CYP1A2 induction by PAHs
Fluvoxamine	Increases (2-5x)	CYP1A2 inhibition
Caffeine	Increases (modestly)	CYP1A2 inhibition
Carbamazepine	Decreases + agranulocytosis risk	CYP3A4 induction + bone marrow
Valproate	Mild decrease in levels possible	May lower clozapine levels slightly
Ciprofloxacin	Increases	CYP1A2 inhibition

EXAM PEARL

Critical exam point: When a patient on clozapine STOPS SMOKING (e.g., hospitalization), clozapine levels can DOUBLE → toxicity risk. Reduce dose by 30-50% when patient stops smoking.

9.3 Augmentation Strategies (When Clozapine Alone Fails)

Evidence-based (some level of support):

STRATEGY	EVIDENCE LEVEL	NOTES
Amisulpride augmentation	Moderate (RCTs)	Targets presynaptic D2/D3 at low doses

STRATEGY	EVIDENCE LEVEL	NOTES
Aripiprazole augmentation	Moderate	May reduce metabolic effects of clozapine
ECT augmentation	Moderate	For persistent positive symptoms on clozapine
Lamotrigine augmentation	Moderate	Glutamate modulation; 200-400 mg/day
Lithium augmentation	Low-moderate	Mood stabilization + anti-suicidal
Cognitive remediation	Adjunctive	For cognitive symptoms

Ultra-treatment resistance: Failed adequate clozapine trial (plasma level >350 ng/mL for 8+ weeks) + augmentation attempts. Consider: clozapine + ECT, clozapine + amisulpride, clozapine + aripiprazole.

10. PSYCHOSOCIAL MANAGEMENT

10.1 Evidence-Based Psychosocial Interventions

INTERVENTION	EVIDENCE	KEY POINTS
CBTp (CBT for psychosis)	Strong (NICE first-line)	Targets distress associated with symptoms, not elimination; belief modification, behavioral experiments; 16+ sessions
Family interventions	Strong	Psychoeducation, communication skills, problem-solving, reducing EE; reduces relapse by ~50%; for ALL patients in contact with family
Social skills training	Moderate	Role-playing, modeling, feedback; improves social functioning
Cognitive remediation	Moderate-strong	Computer-based + strategic coaching; improves attention, memory, executive function; best when combined with vocational rehab
Supported employment (IPS model)	Strong	Individual Placement and Support; competitive employment with ongoing support; 2-3x more effective than traditional vocational rehab

INTERVENTION	EVIDENCE	KEY POINTS
Assertive Community Treatment (ACT)	Strong	Multidisciplinary team, low caseload (~10:1), community-based, 24/7 availability; for high-service-utilization patients
Token economy	Moderate	Operant conditioning in institutional settings
Art/music therapy	Low-moderate	Especially for negative symptoms, engagement
Illness management and recovery (IMR)	Moderate	Psychoeducation + relapse prevention + coping skills

10.2 Family Therapy in Schizophrenia

Rationale: High Expressed Emotion (EE) in family → 2-3x higher relapse rate (Vaughn & Leff, 1976).

Components of EE:

1. Critical comments
2. Hostility
3. Emotional over-involvement

Family intervention models:

MODEL · APPROACH

Psychoeducational (Anderson, Falloon) Education about illness, medication, early warning signs

Behavioral family management (Falloon) Communication training, problem-solving skills

Multiple family groups (McFarlane) Several families together; mutual support + problem-solving

Systemic family therapy Addressing family dynamics and relationship patterns

Evidence:

- Reduces relapse by 20-50% when combined with medication
- NNT ~6-7 (for preventing one relapse over 12 months)
- Should be offered to ALL patients living with or in close contact with family
- Minimum 10 sessions over 3+ months
- Single family > multiple family (for relapse prevention)

10.3 Rehabilitation

Principles:

- Recovery-oriented (focus on strengths, not deficits)
- Person-centered goals
- Graded, stepwise approach
- Community integration as ultimate goal

Components:

- Life skills training (cooking, budgeting, hygiene, transport)
- Vocational rehabilitation (sheltered workshops → supported employment → competitive employment)
- Supported housing (group homes → independent living with support)
- Peer support programs
- Relapse prevention planning (early warning signs identification, crisis plan)

10.4 Prodromal Phase Interventions

At-Risk Mental State (ARMS) / Ultra-High Risk (UHR):

CRITERION · DESCRIPTION

Attenuated psychotic symptoms (APS) Subthreshold positive symptoms

Brief limited intermittent psychotic symptoms (BLIPS) Full psychotic symptoms lasting <1 week, spontaneously resolve

Trait + state risk factors Family history + decline in functioning

Interventions for prodromal phase:

- **CBT**, strongest evidence (reduces transition to psychosis)
- **Omega-3 fatty acids**, early promising data (Amminger et al., 2010); later trial (NEURAPRO) negative
- **Low-dose antipsychotics**, effective but ethical concerns (treating people who may never develop psychosis; side effects)
- **Stress management, social support, substance use reduction**
- **Staged treatment**, begin with benign interventions, escalate if needed

Conversion rate: ~30% of UHR individuals develop psychosis within 3 years

10.5 Compliance/Adherence

Non-adherence rates: 40-60% within first year; up to 75% within 2 years

Causes of poor compliance:

CATEGORY · FACTORS

Patient-related Lack of insight (anosognosia), cognitive deficits, substance use, negative symptoms

Medication-related Side effects (EPS, sedation, weight gain, sexual dysfunction), complex regimens

Illness-related Paranoia about medication, denial of illness

Clinician-related Poor therapeutic alliance, inadequate psychoeducation

System-related Access to care, cost, stigma

Strategies to improve compliance:

- Long-acting injectables (LAIs)
 - Psychoeducation (patient + family)
 - Simplify regimen (once daily dosing, combination pills)
 - Motivational interviewing
 - Shared decision-making
 - Digital adherence monitoring (e.g., Abilify MyCite, aripiprazole with ingestible sensor)
 - Therapeutic alliance building
 - Address side effects proactively
 - Family involvement
 - Community mental health teams / ACT
-

11. SPECIAL TOPICS

11.1 Schizoaffective Disorder

- Concurrent mood episode + schizophrenia Criterion A symptoms
- Key: Delusions/hallucinations for 2+ weeks in ABSENCE of mood episode (differentiates from mood disorder with psychotic features)
- Bipolar type vs Depressive type
- Treatment: Antipsychotic + mood stabilizer / antidepressant

11.2 Schizotypal Personality Disorder

- In ICD-11: classified under schizophrenia spectrum (not personality disorders)
- Features: Ideas of reference, magical thinking, unusual perceptual experiences, odd thinking/speech, suspiciousness, inappropriate affect, odd behavior/appearance, few close relationships, excessive social anxiety

- Does NOT meet full psychotic criteria

11.3 Brief Psychotic Disorder / Acute and Transient Psychotic Disorder

- Duration: >1 day but <1 month (DSM-5) / variable (ICD-11)
- Full return to premorbid functioning
- With/without marked stressor
- Good prognosis

11.4 Delusional Disorder

- Non-bizarre delusions for >1 month
- Criterion A for schizophrenia never met (except tactile/olfactory hallucinations related to delusional theme)
- Functioning not markedly impaired
- Subtypes: Erotomanic, Grandiose, Jealous, Persecutory, Somatic, Mixed, Unspecified

11.5 Substance-Induced Psychosis vs Schizophrenia

FEATURE	SUBSTANCE-INDUCED	SCHIZOPHRENIA
Onset	Acute, temporal relationship to substance	Gradual prodrome
Visual hallucinations	More common	Less common
Orientation	Often impaired	Usually intact
Duration	Resolves within days-weeks of abstinence	Persistent
Insight	May improve with clearing	Often absent

Quick-Reference Summary Tables

Key Numbers to Remember

FACT · NUMBER

Lifetime prevalence ~1%

TRS prevalence ~30%

D2 occupancy therapeutic window 65-80%

Clozapine agranulocytosis risk ~1%

Clozapine therapeutic level >350 ng/mL

Suicide risk in schizophrenia ~5% lifetime

Life expectancy reduction 15-20 years

MZ twin concordance ~48%

Heritability ~80%

Relapse at 5 years (untreated) ~80%

Non-adherence rate (1 year) ~50%

UHR to psychosis conversion (3 years) ~30%

Family therapy NNT for relapse prevention ~6-7

Clozapine seizure threshold dose-dependent, ~3-8%

Model Answers

Q1. Describe positive and negative symptoms of schizophrenia. Discuss management of negative symptoms. [10 marks]: LONG ESSAY CANDIDATE

Exam Strategy: This is a high-frequency question. Split 3+3+4 (positive, negative, management of negative). Use tables. The management section is what differentiates a good answer, most students only write about antipsychotics.

Answer:

Introduction (0.5 marks)

Schizophrenia is a chronic psychotic disorder with a heterogeneous symptom profile. Eugen Bleuler first distinguished fundamental from accessory symptoms. The modern positive-negative dichotomy was formalized by Crow (1980) and elaborated by Andreasen (1982).

A. Positive Symptoms (3 marks)

Positive symptoms represent an excess or distortion of normal functions. They are generally responsive to antipsychotic medication and fluctuate with illness course.

SYMPTOM	DESCRIPTION	EXAMPLES
Hallucinations	Perceptions without external stimuli, in clear consciousness	Auditory (most common): third-person voices, running commentary, command hallucinations; also visual, tactile, olfactory
Delusions	Fixed, false beliefs not amenable to reason, incongruent with cultural background	Persecutory (most common), referential, grandiose, passivity phenomena (thought insertion/withdrawal/broadcasting)
Formal thought disorder	Disorganization of the form of thinking	Loosening of associations, tangentiality, derailment, word salad, neologisms, thought blocking
Disorganized behavior	Grossly disorganized or catatonic behavior	Unpredictable agitation, inappropriate behavior, bizarre dress

Assessment: Scale for Assessment of Positive Symptoms (SAPS, Andreasen), PANSS positive subscale.

B. Negative Symptoms (3 marks)

Negative symptoms represent a diminution or loss of normal functions. They are the primary determinant of long-term functional outcome.

Andreasen's SANS classification (5 domains):

SYMPTOM	DEFINITION	CLINICAL MANIFESTATION
Affective flattening	Reduced range and intensity of emotional expression	Unchanging facial expression, poor eye contact, decreased spontaneous movements, monotone voice
Alogia	Poverty of speech or content	Brief, laconic replies; increased response latency; poverty of content
Avolition/Apathy	Reduced motivation and goal-directed behavior	Poor self-care, inability to persist at work or education
Anhedonia/Asociality	Inability to experience pleasure; social withdrawal	No recreational activities, few or no relationships
Attentional impairment	Difficulty sustaining focus	Social inattentiveness, poor concentration

Critical distinction, Primary vs Secondary:

- Primary: Intrinsic to illness (deficit syndrome, Carpenter); poor treatment response
- Secondary: Due to medication (EPS, sedation), depression, positive symptoms (withdrawal due to paranoia), environmental deprivation
- Management hinges on this distinction, secondary causes must be identified and addressed first

C. Management of Negative Symptoms (4 marks)

Step 1: Address secondary causes

- Reduce/switch antipsychotic if EPS or sedation contributing
- Treat comorbid depression (add SSRI/SNRI)
- Address substance use
- Enhance environmental stimulation
- Assess medication adherence

Step 2: Pharmacological approaches

STRATEGY	EVIDENCE	NOTES
Cariprazine	Strongest evidence (RCT vs risperidone, Nemeth et al., 2017)	D3-preferring partial agonist; FDA-recognized benefit
Amisulpride (low dose, 50-300 mg)	Moderate	Preferential presynaptic D2/D3 blockade at low doses
Aripiprazole	Moderate	D2 partial agonism; less secondary negative symptoms
Clozapine	Moderate (for TRS)	May improve negative symptoms through overall illness improvement
Add-on antidepressants	Low-moderate	SSRIs, mirtazapine, for negative/depressive overlap
Cobenfy (xanomeline-trospium)	Emerging	Muscarinic agonist; early data suggest benefit

Step 3: Psychosocial interventions

INTERVENTION · TARGET

Social skills training Social withdrawal, asociality

Cognitive remediation Attention, executive function

Supported employment (IPS) Avolition, functional recovery

CBTp (adapted) Behavioral activation, activity scheduling

Art/music therapy Engagement, emotional expression

Physical exercise programs Motivation, cognitive function, metabolic health

Family interventions Reduce over-accommodation of negative symptoms

Conclusion: Management of negative symptoms requires a systematic approach: first rule out secondary causes, then use pharmacological strategies (cariprazine as first choice if available), and combine with psychosocial interventions targeting specific functional deficits.

Cross-reference: D1 Sections 2.2, 8.3, 8.4, 10.1

Q2. Define treatment-resistant schizophrenia (TRS). Discuss management protocol: pharmacological and psychological. [3+7 = 10 marks], LONG ESSAY CANDIDATE

Exam Strategy: Definition is 3 marks, be precise, include TRRIP criteria. Management is 7 marks, structure as algorithm. Clozapine is the bulk. Don't forget augmentation and psychosocial.

Answer:

A. Definition of TRS (3 marks)

Treatment-resistant schizophrenia (TRS) is defined as persistent, moderate-to-severe psychotic symptoms despite adequate antipsychotic treatment.

TRRIP Working Group Criteria (Howes et al., 2017):

1. Failure of at least **2 adequate antipsychotic trials** (at least 1 SGA)
2. Each trial at adequate dose (equivalent to ≥ 600 mg/day chlorpromazine)
3. Each trial for adequate duration (≥ 6 weeks at optimal dose)
4. Adequate adherence confirmed (plasma levels, LAI, supervised intake)
5. Persistent moderate-severe positive symptoms (PANSS total >80 or CGI-S ≥ 4)
6. Significant functional impairment

Prevalence: ~30% of schizophrenia patients

Pseudo-resistance must be excluded: non-adherence, inadequate dose/duration, substance use, rapid metabolism, comorbid conditions.

B. Management Protocol (7 marks)

Step 1: Confirm TRS diagnosis

- Review treatment history (doses, durations, adherence)
- Check plasma drug levels
- Screen for substance use (urine drug screen)
- Consider CYP genotyping if suspecting rapid metabolism
- Assess for comorbidities (depression, OCD, PTSD)

Step 2: Clozapine trial, Gold standard

Initiation:

- Start 12.5 mg OD or BD
- Increase by 25-50 mg/day over first week
- Increase by 50-100 mg/week from week 2
- Target dose: 300-450 mg/day (up to 900 mg/day)

- Target plasma level: >350 ng/mL (trough, 12h post-dose)

Monitoring:

- WBC / ANC: Weekly for 18 weeks → Biweekly for 34 weeks → Monthly thereafter
- Metabolic parameters: Weight, BMI, fasting glucose, lipids at baseline and 3-monthly
- Cardiac: ECG at baseline, troponin/CRP if fever or tachycardia in first month
- Seizure risk: Increases with dose; consider valproate prophylaxis if >600 mg/day

Adequate clozapine trial: Minimum 8 weeks at therapeutic plasma level (>350 ng/mL)

Step 3: Clozapine augmentation (if clozapine monotherapy fails)

AUGMENTATION	EVIDENCE	DOSE
Amisulpride	Moderate (RCTs)	400-800 mg/day
Aripiprazole	Moderate	10-15 mg/day; may reduce metabolic burden
ECT	Moderate	Bilateral, 12-20 sessions; often dramatic benefit
Lamotrigine	Moderate	200-400 mg/day; glutamate modulation
Lithium	Low-moderate	Target level 0.6-0.8 mEq/L

Step 4: Experimental/emerging

- Transcranial direct current stimulation (tDCS), for persistent auditory hallucinations
- Repetitive transcranial magnetic stimulation (rTMS), left temporo-parietal for hallucinations
- Electroconvulsive therapy (ECT), as augmentation

Psychological interventions for TRS:

INTERVENTION · ROLE

CBTp Reduces distress from residual symptoms; improves coping

Family intervention Psychoeducation about TRS; reduce EE; caregiver support

Cognitive remediation Addresses cognitive deficits

Social skills training Functional improvement

Supported employment/housing Recovery-oriented

ACT teams For complex, high-service-use patients

Algorithm summary:

Confirm TRS → Optimize current treatment → Start clozapine → Titrate to therapeutic level → Augment if needed (amisulpride / aripiprazole / ECT / lamotrigine) → Combine with psychosocial interventions throughout.

Cross-reference: D1 Sections 9.1-9.3, 10.1

Q3. Plan a clozapine trial. Discuss initiation, monitoring, and side effects (common, less common, rare). [10 marks]

Exam Strategy: Highly structured answer. Use tables for each section. This is a practical question, show you can actually do it. 3+3+4 split approximately.

Answer:

Introduction (0.5 marks)

Clozapine is the gold standard for treatment-resistant schizophrenia. It is the only antipsychotic with demonstrated superiority in TRS (Kane et al., 1988) and the only one approved for reducing suicidality (InterSePT trial).

A. Pre-initiation Assessment (1 mark)

- Confirm TRS (2 failed adequate antipsychotic trials)
- Baseline investigations: CBC with differential (WBC >3500, ANC >2000), metabolic panel (fasting glucose, lipids, HbA1c), LFT, RFT, ECG, weight/BMI/ waist circumference, blood pressure
- Cardiac assessment: Baseline troponin, CRP (for myocarditis monitoring)
- Inform patient and family: Written consent, side effect education, importance of monitoring compliance
- Register with clozapine monitoring service (REMS in US; equivalent in India)
- Stop medications that lower WBC (e.g., carbamazepine, contraindicated)

B. Initiation Protocol (2 marks)

TIMEFRAME	DOSING	KEY CONSIDERATIONS
Day 1	12.5 mg at night	Evening dosing (sedation)
Day 2	12.5 mg BD	Monitor for hypotension, sedation
Days 3-7	Increase by 25-50 mg / day	Slow titration reduces side effects
Week 2	Increase by 25-50 mg every 2 days	Target: 200-300 mg / day by end of week 2
Week 3-4	Increase by 50-100 mg / week	Target: 300-450 mg / day

TIMEFRAME	DOSING	KEY CONSIDERATIONS
Week 6+	Check plasma level (target >350 ng/mL)	If subtherapeutic, increase dose; if level adequate but no response, augment
Maximum dose	900 mg/ day	Seizure risk increases significantly above 600 mg

- Cross-taper previous antipsychotic over 1-2 weeks (do not abruptly stop)
- If patient smokes: will need higher doses (CYP1A2 induction)
- Divided dosing preferred initially (BD), can shift to mainly nocturnal once stable

C. Monitoring Protocol (3 marks)

PARAMETER	SCHEDULE	ACTION THRESHOLDS
WBC/ANC	Weekly x 18 weeks → Biweekly x 34 weeks → Monthly	WBC <3000 or ANC <1500: STOP
Weight/BMI	Monthly for 6 months, then 3-monthly	>7% gain: dietary intervention, consider metformin
Fasting glucose/HbA1c	Baseline, 3 months, then annually	>126 mg/dL or HbA1c >6.5%: diabetic workup
Lipid profile	Baseline, 3 months, then annually	Treat per guidelines
Troponin/CRP	Baseline; weekly for first 4 weeks (some protocols)	Rising troponin + fever + tachycardia: suspect myocarditis, STOP
ECG	Baseline, then annually	QTc >500ms: reassess
LFT	Baseline, then 6-monthly	Transient elevation common; stop if >3x ULN with symptoms
Clozapine plasma level	At steady state (after 1-2 weeks at stable dose)	Target >350 ng/mL; check if poor response or suspected non-adherence
Bowel function	Every visit (ask!)	Constipation can lead to fatal ileus

D. Side Effects (4 marks)

Common (>10%):

SIDE EFFECT	FREQUENCY	MECHANISM	MANAGEMENT
Sedation	40-60%	H1, M1 blockade	Nocturnal dosing, slow titration

SIDE EFFECT	FREQUENCY	MECHANISM	MANAGEMENT
Sialorrhea	30-50%	M4 agonism	Hyoscine, glycopyrrolate, sublingual atropine
Constipation	25-40%	Anticholinergic	Prophylactic laxatives from day 1 (lactulose, docusate)
Weight gain	30-50%	H1, 5-HT2C	Diet, exercise, metformin
Tachycardia	20-30%	Anticholinergic, alpha	Propranolol if persistent; usually settles
Hypotension	15-25%	Alpha-1 blockade	Slow titration, hydration
Dizziness	15-20%	Alpha-1 blockade	As above
Metabolic syndrome	20-40%	Multiple receptors	Aggressive monitoring and management
Nocturnal enuresis	10-20%	Unknown	Desmopressin, reduce evening fluids
Transient fever	5-15%	Immune	Distinguish from infection/ myocarditis; usually resolves

Less common (1-10%):

SIDE EFFECT	FREQUENCY	NOTES
Seizures	3-5% (dose-dependent)	<300mg: ~1%; 300-600: ~3%; >600: ~5-8%; prophylaxis with valproate
OCD symptoms/exacerbation	5-10%	5-HT2A/2C blockade; treat with fluvoxamine (CYP1A2 interaction!)
Hepatic transaminase elevation	5-10%	Usually transient
Neutropenia (non-agranulocytosis)	3-5%	WBC 3000-3500: monitor closely

Rare but serious (<1%):

SIDE EFFECT	FREQUENCY	PRESENTATION	ACTION
Agranulocytosis	0.8%	Sore throat, fever, infection; ANC <500	STOP permanently, isolate, G-CSF, broad-spectrum antibiotics
Myocarditis	1-3%	Fever, tachycardia, chest pain, dyspnea (first 4-6 weeks)	STOP, troponin/CRP/echo, cardiology referral
Cardiomyopathy	~0.1%	Heart failure symptoms; can occur anytime	STOP, cardiology
Ileus/bowel obstruction	~3%	Severe constipation, abdominal distension, absent bowel sounds	Surgical emergency if not caught early
Pulmonary embolism	Increased risk	DVT symptoms, sudden dyspnea	Standard PE management
NMS	Very rare	Rigidity, fever, autonomic instability, elevated CK	Standard NMS management

Conclusion: Clozapine remains the most effective antipsychotic for TRS but requires meticulous initiation, systematic monitoring, and proactive side effect management to ensure safety.

Cross-reference: D1 Section 9.2

Q4. Describe the course and outcome of schizophrenia. [10 marks]

Exam Strategy: Cover phases, course patterns, outcome statistics, mortality. Use WHO studies data. 2+3+3+2 split.

Answer:

Introduction (1 mark)

Kraepelin originally described schizophrenia (dementia praecox) as having a uniformly deteriorating course. Modern longitudinal studies show a heterogeneous course with outcomes ranging from full recovery to chronic disability.

A. Phases of Illness (2 marks)

PHASE	DURATION	FEATURES
Premorbid	Childhood-adolescence	Subtle deficits: developmental delays, social difficulties, cognitive deficits, schizoid traits

PHASE	DURATION	FEATURES
Prodromal	Months to years (avg 2-5 years)	Attenuated positive symptoms, functional decline, mood changes, social withdrawal, cognitive decline, sleep disturbance
Active/Psychotic	Variable (weeks-months per episode)	Frank positive symptoms (delusions, hallucinations, FTD), behavioral disturbance
Residual	Following active phase	Predominantly negative symptoms, attenuated positive symptoms, cognitive deficits, functional impairment
Stable/Recovery	Ongoing	Variable levels of remission and functioning

B. Course Patterns (3 marks)

PATTERN	FREQUENCY	DESCRIPTION
Single episode with full remission	15-20%	One episode, complete recovery, no relapse
Episodic with inter-episode residual symptoms	35-40%	Most common; incomplete recovery between episodes
Episodic with progressive deficit	10-15%	Stepwise deterioration with each episode
Continuous	10-15%	Persistent symptoms without episodic pattern
Episodic with stable deficit	10-15%	Episodes with consistent residual between

Rule of thirds (approximate):

- ~30% have a good outcome (remission, functional recovery)
- ~30% have an intermediate outcome (partial improvement, some functional impairment)
- ~30% have a poor outcome (chronic symptoms, significant disability)
- ~10% die by suicide

Key longitudinal studies:

STUDY · KEY FINDINGS

WHO IPSS (1973) Schizophrenia exists worldwide; better outcome in developing countries

WHO DOSMeD (1992) 2-year outcomes better in developing countries (India, Nigeria, Colombia)

Ciampi (1980), Zurich Long-term (36 years): 27% full recovery, 22% mild residual, 24% moderate, 18% severe, 9% died

Harding et al. (1987), Vermont 32-year follow-up: 62-68% showed significant improvement or recovery

Andreasen (2005) Remission criteria: sustained mild symptoms for 6+ months

C. Outcome Domains (2 marks)

DOMAIN · FINDING

Symptom remission 20-30% meet Andreasen's remission criteria

Functional recovery Only 14-20% achieve full functional recovery

Employment 10-20% in competitive employment

Independent living 50-60% with some form of support

Social relationships Frequently impoverished

Quality of life Significantly reduced

D. Mortality (2 marks)

- Life expectancy reduced by 15-20 years
- Suicide: ~5% lifetime risk; highest in young males, early illness, depression, substance use
- Natural causes: Cardiovascular disease is the leading cause of premature death (metabolic syndrome from antipsychotics + lifestyle factors + reduced healthcare access)
- SMR: 2-3x general population
- Mortality gap widening over time (general population health improving faster)

Conclusion: Schizophrenia has a heterogeneous course. While Kraepelin's view of inevitable deterioration is overly pessimistic, outcomes remain variable. Early intervention, sustained treatment, psychosocial rehabilitation, and metabolic monitoring are essential to optimize outcomes.

Cross-reference: D1 Section 6

Q5. Discuss prognostic factors and outcomes of schizophrenia. [10 marks]

Exam Strategy: Overlap with Q4. Emphasize prognostic factors (this is the focus). Table format is ideal. Add evidence base.

Answer:

Introduction (1 mark)

Prognosis in schizophrenia is determined by the interaction of illness characteristics, patient factors, treatment variables, and sociocultural context. Identifying prognostic factors guides treatment planning and helps set realistic expectations.

A. Good Prognostic Factors (3 marks)

CATEGORY · GOOD PROGNOSTIC FACTORS

Onset Acute onset, identifiable precipitant, late onset

Demographics Female sex, married / stable relationship, good social support

Premorbid Good premorbid adjustment (education, employment, social function)

Symptom profile Prominent affective symptoms, paranoid features, absence of negative symptoms

Family No family history of schizophrenia, low expressed emotion

Cognitive Preserved cognitive function

Treatment Early treatment response, good medication adherence, access to comprehensive care

Comorbidity No substance use disorder

Neuroimaging Normal brain structure

Sociocultural Developing country (WHO DOSMeD)

B. Poor Prognostic Factors (3 marks)

CATEGORY · POOR PROGNOSTIC FACTORS

Onset Insidious onset, no precipitant, early onset (especially childhood-onset)

Demographics Male sex, single / divorced, poor social support

Premorbid Poor premorbid adjustment, schizoid / schizotypal traits, developmental delays

Symptom profile Predominant negative symptoms, disorganization, deficit syndrome

Family Strong family history, high expressed emotion

Cognitive Marked cognitive impairment, low premorbid IQ

Treatment Delayed treatment (long DUP), poor early response, non-adherence

Comorbidity Comorbid substance use (especially cannabis, stimulants)

Neuroimaging Enlarged ventricles, reduced cortical volume

History Multiple relapses, previous suicide attempts

C. Evidence for Key Prognostic Factors (2 marks)

FACTOR · EVIDENCE

Duration of Untreated Psychosis (DUP) Longer DUP = worse outcome (meta-analysis: Marshall et al., 2005); Early intervention services reduce DUP and improve outcomes

Expressed Emotion Vaughn & Leff (1976): High EE = 50-65% relapse vs Low EE = 15-25% relapse at 9 months

Developing vs developed countries WHO DOSMeD: 2-year outcomes significantly better in developing countries (extended family, community reintegration, agricultural employment)

Cannabis use Henquet et al. (2005): Cannabis use associated with earlier onset, more relapses, poorer treatment response

Sex differences Better outcomes in females due to later onset, estrogen neuroprotection, more affective symptoms

D. Outcome Data (1 mark)

- Andreasen's remission: 20-30% achieve
- Functional recovery: 14-20%
- Competitive employment: 10-20%
- Suicide: ~5%
- Premature mortality from natural causes: 15-20 year life expectancy reduction

Cross-reference: D1 Section 6.2, Q4 above

Q6. Enumerate Schneider's first rank symptoms. Discuss their difference from second and third rank symptoms. Comment on their current status. [10 marks]

Exam Strategy: 4+3+3 split. Enumerate all FRS clearly. Second rank is less commonly asked, show depth. Current status = critical evaluation.

Answer:

Introduction (0.5 marks)

Kurt Schneider (1959) proposed certain symptoms as having special diagnostic importance for schizophrenia, terming them "first rank symptoms" (FRS). He emphasized their diagnostic utility, not their frequency or pathological significance.

A. First Rank Symptoms (4 marks)

Category 1: Auditory Hallucinations

FRS · DESCRIPTION

Voices arguing/discussing Two or more voices discussing the patient in third person

Running commentary Voice providing continuous commentary on patient's actions

Thought echo (Gedankenlautwerden) Patient hears own thoughts spoken aloud

Category 2: Passivity Experiences (made phenomena)

FRS · DESCRIPTION

Made feelings Emotions experienced as imposed from outside

Made impulses Urges/drives experienced as externally imposed

Made actions (made volitional acts) Body movements experienced as controlled externally

Somatic passivity Bodily sensations experienced as imposed from outside

Category 3: Thought Interference

FRS · DESCRIPTION

Thought insertion Alien thoughts placed into one's mind by an external agency

Thought withdrawal Thoughts removed from one's mind by an external force

Thought broadcasting Thoughts made accessible to others (transmitted, leaked)

Category 4: Delusional Perception

FRS · DESCRIPTION

Delusional perception Two-stage phenomenon: normal perception followed by delusional interpretation with personal significance, without any rational connection

Total: 11 first rank symptoms (some texts count slightly differently depending on grouping).

B. Second and Third Rank Symptoms (3 marks)

Second rank symptoms (less diagnostic specificity):

CATEGORY · SYMPTOMS

Hallucinations Other forms: visual, tactile, olfactory, gustatory hallucinations

Mood Perplexity, depressive mood changes, euphoric mood changes

Affect Emotional blunting

Delusions Delusional intuition (sudden delusional idea NOT arising from a perception, contrast with delusional perception which IS based on a perception)

Other Thought blocking (some classify here)

Difference between FRS and Second Rank:

FEATURE	FIRST RANK	SECOND RANK
Specificity	Higher for schizophrenia	Lower; found in many conditions
Diagnostic weight	Was given special diagnostic status	Supportive but not sufficient alone
Nature	Loss of ego boundaries (Ich-Störung)	Non-specific psychotic phenomena
Core feature	Passivity / loss of ownership of mental contents	Variable

Third rank is not a formal Schneiderian concept. Some texts use it informally to refer to non-specific symptoms such as disturbances in mood, psychomotor changes, and behavioral disturbances that can occur in multiple psychiatric conditions.

C. Current Status of FRS (3 marks)

ASPECT · CURRENT POSITION

DSM-5 FRS no longer given special diagnostic weight. Previously in DSM-IV, a single FRS was sufficient for Criterion A; DSM-5 requires 2 Criterion A symptoms, at least one being delusions, hallucinations, or disorganized speech

ICD-11 Also removed special diagnostic status of FRS; uses symptom qualifiers instead of FRS-based diagnosis

Specificity NOT pathognomonic: FRS found in 10-20% of bipolar disorder, temporal lobe epilepsy, substance-induced psychosis, and other conditions

Cross-cultural Variable rates across cultures; higher in some Western samples, lower in some non-Western samples

Sensitivity Only present in ~70% of schizophrenia cases; absent in 30% (especially negative-symptom-dominant, early illness)

Phenomenological value Remains useful for clinical description and phenomenological assessment, even if not diagnostically privileged

Neuroscience FRS map onto self-monitoring deficits (corollary discharge failures), provides neurobiological framework

Conclusion: While Schneider's FRS were historically central to schizophrenia diagnosis, modern classification systems have appropriately reduced their diagnostic privilege. They remain valuable as clinical descriptors and phenomenological markers but are neither specific to nor necessary for the diagnosis of schizophrenia.

Cross-reference: D1 Section 2.4

Q7. Discuss atypical antipsychotics. Why are they called "atypical"? Are they effective for negative symptoms? Discuss side effects. [10 marks]

Exam Strategy: 2+3+2+3 split. "Why atypical" is the conceptual hook. Negative symptom question requires nuance, don't just say "yes."

Answer:

A. Why "Atypical"? (2 marks)

The term "atypical" was introduced to distinguish second-generation antipsychotics (SGAs) from first-generation (FGA/typical) based on several properties:

FEATURE	TYPICAL (FGA)	ATYPICAL (SGA)
EPS at therapeutic dose	Frequent	Low / absent
D2 affinity	High, tight binding	Variable; many have fast dissociation
5-HT_{2A} antagonism	Minimal	Prominent

FEATURE	TYPICAL (FGA)	ATYPICAL (SGA)
5-HT_{2A}/D₂ ratio	<1	>1 (Meltzer's hypothesis)
Prolactin elevation	Marked	Less (except risperidone, paliperidone)
Tardive dyskinesia risk	Higher	Lower

Three hypotheses for "atypicality":

1. **Meltzer's serotonin-dopamine hypothesis:** 5-HT_{2A}/D₂ affinity ratio >1 → selective mesolimbic D₂ blockade, preserving nigrostriatal pathway
2. **Kapur's fast dissociation:** Rapid D₂ off-rate allows physiological dopamine surges to compete → less EPS (applies to clozapine, quetiapine)
3. **Limbic selectivity:** Preferential action on mesolimbic over nigrostriatal pathways

B. Individual Atypical Antipsychotics (3 marks)

DRUG	MECHANISM	KEY FEATURE
Clozapine	Multi-receptor (D ₁ , D ₂ , D ₄ , 5-HT _{2A} , 5-HT _{2C} , H ₁ , M ₁ , α)	Gold standard for TRS; anti-suicidal
Risperidone	D ₂ + 5-HT _{2A}	Most studied SGA; dose-dependent EPS
Olanzapine	MARTA (Multi-Acting Receptor Targeted Agent)	Highly effective; worst metabolic
Quetiapine	Low D ₂ , fast off; H ₁ , α-1, 5-HT _{2A}	Very sedating; versatile across disorders
Aripiprazole	D ₂ partial agonist, 5-HT _{1A} partial agonist	"Dopamine system stabilizer"; least metabolic
Ziprasidone	D ₂ + 5-HT _{2A} ; SERT/NET inhibition	Weight-neutral; QTc concern
Paliperidone	Active metabolite of risperidone	Extended release; less hepatic metabolism
Lurasidone	D ₂ + 5-HT _{2A} + 5-HT ₇ antagonist	Cognitive benefits; weight-neutral
Cariprazine	D ₃ -preferring partial agonist	Negative symptoms; very long half-life

C. Efficacy for Negative Symptoms (2 marks)

Nuanced answer required:

- SGAs are NOT consistently superior to FGAs for primary negative symptoms (CATIE trial showed no clear advantage)

- SGAs reduce **secondary** negative symptoms by causing fewer EPS and less sedation
- **Specific exceptions with evidence for primary negative symptoms:**
- Cariprazine: RCT evidence of superiority over risperidone for predominant negative symptoms (Nemeth et al., 2017)
- Amisulpride (low dose, 50-300 mg): Preferential presynaptic D2/D3 blockade → may enhance dopamine transmission
- Clozapine: Improves overall symptom burden including negative symptoms in TRS
- The "atypicals are better for negative symptoms" claim was largely a marketing narrative; rigorous evidence is limited
- Primary negative symptoms remain one of the greatest unmet needs in schizophrenia treatment

D. Side Effects (3 marks)

SIDE EFFECT	MOST ASSOCIATED DRUGS	MECHANISM
Metabolic syndrome (weight gain, diabetes, dyslipidemia)	Clozapine, olanzapine >> quetiapine, risperidone > aripiprazole, ziprasidone, lurasidone	H1, 5-HT2C, M3 blockade
Hyperprolactinemia	Risperidone, paliperidone (most); amisulpride	D2 blockade in tuberoinfundibular pathway
QTc prolongation	Ziprasidone, iloperidone (also thioridazine FGA)	Ion channel effects
Sedation	Clozapine, olanzapine, quetiapine	H1 blockade
EPS/Akathisia	Risperidone (high dose), aripiprazole (akathisia), lurasidone	D2 blockade in nigrostriatal pathway
Agranulocytosis	Clozapine (unique)	Immune-mediated
Seizures	Clozapine (dose-dependent)	Lowers seizure threshold
Myocarditis	Clozapine	Hypersensitivity
Post-injection delirium/sedation	Olanzapine pamoate (PDSS)	Accidental intravascular injection
Orthostatic hypotension	Clozapine, quetiapine, iloperidone	Alpha-1 blockade
Sialorrhea	Clozapine	M4 agonism (paradoxical)

Cross-reference: D1 Sections 8.3-8.4, D4 Table 3

Q8. Critical evaluation of typical antipsychotics in current practice. [10 marks]

Exam Strategy: Balanced answer. Don't just list disadvantages, show you understand their ongoing role. CATIE and CUtLASS data are key.

Answer:

Introduction (1 mark)

First-generation antipsychotics (FGAs/typicals) were the cornerstone of schizophrenia treatment from chlorpromazine's introduction (1952) until SGAs emerged in the 1990s. Their role in current practice requires a balanced evaluation considering efficacy, safety, cost, and access.

A. Mechanism and Classification (1 mark)

- Primary mechanism: D2 receptor antagonism
- High potency (haloperidol, fluphenazine, pimozide): More EPS, less sedation/autonomic
- Low potency (chlorpromazine, thioridazine): Less EPS, more sedation/hypotension/anticholinergic

B. Arguments for Continued Use (3 marks)

ARGUMENT · EVIDENCE/DETAIL

Comparable efficacy CATIE (2005): Perphenazine (FGA) was not significantly different from SGAs in all-cause discontinuation; CUtLASS (UK): FGAs non-inferior to SGAs

Cost-effectiveness FGAs are significantly cheaper; crucial for resource-limited settings (India, Africa)

Availability Widely available in developing countries; many SGAs not on essential drug lists

Injectable formulations Haloperidol IM (acute), fluphenazine/haloperidol decanoate (LAIs), well-established, reliable

Acute agitation IV/IM haloperidol remains standard for acute management

Specific indications Pimozide for delusional disorder; flupentixol for psychotic depression

Long clinical experience Decades of safety data; side effect profile well characterized

India-specific Trifluoperazine, fluphenazine, zuclopenthixol widely used and available

C. Arguments Against / Limitations (3 marks)

LIMITATION · DETAIL

EPS Acute dystonia (5-10%), akathisia (20-30%), parkinsonism (20-40%), tardive dyskinesia (5% per year cumulative)

Tardive dyskinesia Cumulative risk; often irreversible; major concern for long-term use

Hyperprolactinemia Galactorrhea, amenorrhea, sexual dysfunction, osteoporosis (long-term)

Subjective experience "Neuroleptic dysphoria", patients report feeling emotionally blunted, "zombie-like"

Negative/cognitive symptoms No benefit; may worsen via secondary negative symptoms (EPS, sedation)

NMS risk Higher with high-potency FGAs (haloperidol)

Sudden cardiac death Thioridazine, droperidol, QTc prolongation

Anticholinergic burden Low-potency FGAs contribute to cognitive decline, delirium risk in elderly

D. Current Guidelines and Role (2 marks)

GUIDELINE · POSITION

NICE (UK) SGA and FGA both acceptable first-line; choice based on individual patient factors

APA SGA generally preferred, but FGA acceptable; haloperidol for acute agitation

WHO Essential Medicines Chlorpromazine, fluphenazine, haloperidol on essential list

Indian practice FGAs still widely used due to cost and availability; gradual shift to SGAs

Appropriate current indications for FGAs:

1. Acute agitation/emergency (haloperidol IM/IV)
2. Resource-limited settings where SGAs are unavailable/unaffordable
3. Patients who have responded well to FGAs previously
4. LAI formulations when SGA LAIs unavailable
5. Specific conditions (pimozide for delusional disorder)
6. When SGA metabolic effects are a greater concern than EPS risk

Conclusion: FGAs remain relevant in current practice, particularly in emergency settings and resource-limited environments. The choice between FGA and SGA should be

individualized, weighing metabolic risks (SGAs) against EPS/TD risks (FGAs), patient preference, cost, and available formulations.

Cross-reference: D1 Section 8.2

Q9. Discuss psychosocial treatment and rehabilitation in schizophrenia. [5+5 = 10 marks]

Exam Strategy: Split clearly: psychosocial treatments (5 marks) and rehabilitation (5 marks). Mention evidence base for each.

Answer:

A. Psychosocial Treatments (5 marks)

Rationale: Antipsychotics alone achieve symptom remission in only 20-30%; functional recovery requires psychosocial interventions targeting residual symptoms, cognitive deficits, social skills, and vocational functioning.

INTERVENTION	DESCRIPTION	EVIDENCE
CBTp (CBT for psychosis)	Targets distress from symptoms (not symptom elimination); cognitive restructuring of delusional beliefs; behavioral experiments; coping strategies for hallucinations	NICE first-line recommendation; reduces distress, improves functioning; 16+ sessions; NNT ~7
Family interventions	Psychoeducation, communication skills, problem-solving, reducing expressed emotion (EE)	Reduces relapse by 20-50% (Pharoah et al., Cochrane); should be offered to ALL patients in contact with family; >10 sessions over 3+ months
Social skills training	Behavioral approach: role-playing, modeling, reinforcement, in-vivo practice	Improves social competence; evidence for generalization to community modest
Cognitive remediation	Computer-based exercises + strategic coaching targeting attention, memory, executive function	MATRICES-endorsed domains; effect size 0.45 for cognition; best when combined with vocational rehab
Supported employment (IPS)	Individual Placement and Support: rapid job search, competitive employment, ongoing support	2-3x more effective than traditional vocational rehab; RCT evidence across countries
ACT (Assertive Community Treatment)	Multidisciplinary team (psychiatrist, nurse, social worker, OT), low caseload (~10:1), community-based, 24/7	Reduces hospitalization, improves housing stability; for high-service-use patients

INTERVENTION	DESCRIPTION	EVIDENCE
Illness management and recovery (IMR)	Psychoeducation + relapse prevention + goal-setting + coping strategies	Structured program; improves self-management
Art/music therapy	Creative expression, group-based, targets engagement	Low-moderate evidence; useful for negative symptoms, engagement
Token economy	Operant conditioning: desired behaviors reinforced with tokens exchangeable for privileges	Effective in inpatient / institutional settings
Mindfulness-based interventions	Acceptance of psychotic experiences rather than elimination	Emerging evidence; may reduce distress from voices

B. Rehabilitation (5 marks)

Philosophy: Recovery model, focuses on living a meaningful life with or without symptoms, rather than "cure."

Principles:

- Person-centered (individual goals, preferences, strengths)
- Community-based (least restrictive environment)
- Graded and stepwise
- Holistic (biological + psychological + social + occupational)
- Rights-based (UNCPRD, United Nations Convention on Rights of Persons with Disabilities)

Components:

AREA · INTERVENTIONS

Life skills Self-care (hygiene, grooming), cooking, budgeting, shopping, transport use, medication management

Vocational Sheltered workshops → Transitional employment → Supported employment (IPS) → Competitive employment; Graded approach based on functioning level

Residential Halfway homes → Group homes → Supervised apartments → Independent living; Day care centers for those not ready for community living

Social Social clubs, peer support groups, self-help organizations, recreational activities

Educational Supported education programs; completion of interrupted education

Legal Advocacy, guardianship issues, rights protection, disability certification

Relapse prevention Identifying early warning signs, crisis plan, emergency contacts, advance directives

Indian context:

- DMHP (District Mental Health Programme): Community mental health services
- PG exams, CIP Ranchi, LGBRIMH: National rehabilitation centers
- Day care centers: Increasingly available in urban India
- Family as primary caregiver: Critical role in Indian context
- RPWD Act 2016: Disability certification, reservation in employment/education
- Limited access to specialized psychosocial interventions in rural areas

Phases of rehabilitation:

PHASE · FOCUS

Acute Symptom stabilization, crisis management, therapeutic alliance

Stabilization Medication optimization, psychoeducation, basic skills

Recovery Vocational planning, social reintegration, advanced skills

Maintenance Relapse prevention, ongoing support, community participation

Cross-reference: D1 Sections 10.1-10.3

Q10. Discuss the role of family therapy in schizophrenia. [10 marks]

Exam Strategy: Focused question. Cover rationale (EE research), models, evidence, practical components. Show clinical depth.

Answer:

Introduction (1 mark)

Family interventions in schizophrenia are among the most well-evidenced psychosocial treatments, consistently recommended by NICE, APA, and PORT guidelines. They address the family environment, which significantly impacts relapse rates and patient functioning.

A. Rationale, Expressed Emotion Research (2 marks)

Expressed Emotion (EE) concept developed by Brown, Birley, and Wing (1962); operationalized by Vaughn and Leff (1976).

EE COMPONENT · DESCRIPTION

Critical comments Negative remarks about patient's behavior

Hostility Generalized rejection of patient as a person

Emotional over-involvement Exaggerated emotional response, overprotectiveness, self-sacrifice

Key findings:

- High EE households: 50-65% relapse at 9 months vs 15-25% in low EE (Vaughn & Leff, 1976)
- Effect is dose-dependent: more face-to-face contact with high-EE relative = higher relapse
- Effect is cross-culturally replicated (though baseline EE levels differ)
- EE is modifiable through intervention
- EE is NOT blame, it is an understandable response to a distressing illness

B. Models of Family Intervention (3 marks)

MODEL	DEVELOPER	APPROACH
Psychoeducational	Anderson (1980), Goldstein	Education about illness, medication, prognosis, early warning signs, crisis management; structured, didactic
Behavioral family management	Falloon (1984)	Communication training + problem-solving skills; identify goals; reduce stress within family
Multiple family groups	McFarlane (1990)	5-7 families together; combines psychoeducation with mutual support and problem-solving; reduces isolation
Systemic/Milan	Systemic tradition	Circular questioning, family dynamics, neutrality; less evidence base for schizophrenia specifically
Cognitive-behavioral family intervention	Kuipers, Barrowclough	CBT techniques applied to family beliefs and attributions about the illness

Common elements across effective models:

1. Psychoeducation about the illness
2. Communication skills enhancement
3. Problem-solving training
4. Emotional processing and support

5. Relapse prevention planning

6. Crisis intervention strategies

C. Evidence Base (2 marks)

STUDY/META-ANALYSIS · FINDING

Pharoah et al. (Cochrane, 2010) Family intervention reduces relapse (RR 0.55 at 1 year); NNT ~7

Pilling et al. (2002) Reduces relapse by 20-50%; improves medication adherence

NICE (2014) Recommends for ALL patients in contact with or living with family; minimum 10 sessions over 3+ months

Leff et al. (1982) Low EE achieved in intervention group; relapse reduced to 9% vs 50% in control

Falloon et al. (1985) Behavioral family management: 6% relapse vs 44% in control at 9 months

Evidence summary:

- NNT to prevent one relapse: ~6-7
- Reduces hospitalization
- Improves medication adherence
- Improves patient social functioning
- Reduces family burden and distress
- Single-family format > multi-family for relapse prevention
- Multi-family format better for social network expansion

D. Practical Implementation (2 marks)

Session structure (typical):

- Duration: 10-20 sessions over 6-12 months
- Frequency: Fortnightly initially, then monthly
- Setting: Home-based preferred (more effective than clinic-based)
- Participants: Patient + key family members
- Therapist: Trained mental health professional (can be nurse or social worker with training)

Phase-wise approach:

PHASE	FOCUS	ACTIVITIES
Engagement	Alliance building	Meet family separately; assess EE, burden, coping
Psychoeducation	Knowledge about illness	Nature of schizophrenia, medication, prognosis, early warning signs
Communication training	Reduce EE	Active listening, expressing positive feelings, making positive requests, expressing negative feelings constructively
Problem-solving	Practical skills	Identify problem → brainstorm solutions → evaluate → implement → review
Relapse prevention	Sustained benefit	Early warning signs plan, crisis contacts, medication plan

Barriers in Indian context:

- Stigma around mental illness and family therapy
- Large joint families, who to include
- Patriarchal dynamics may complicate sessions
- Limited trained family therapists
- Transport and time constraints (especially rural)
- Cultural expectations of family obligation vs therapeutic boundaries

Conclusion: Family therapy is a first-line psychosocial intervention in schizophrenia with robust evidence for reducing relapse, improving adherence, and reducing family distress. It should be offered to all patients in contact with family, using a psychoeducational or behavioral model.

Cross-reference: D1 Section 10.2

Q11. Compare the ICD-10 and DSM-5 diagnosis of schizophrenia. How does the diagnosis differ? [10 marks]

Exam Strategy: Table-based answer. Note: question says ICD-10 not ICD-11, cover both ICD-10 and mention ICD-11 updates.

Answer:

Introduction (0.5 marks)

The diagnosis of schizophrenia has evolved across classification systems. Understanding differences between ICD-10 (still widely used in India) and DSM-5 is essential for clinical

practice and exam purposes.

A. Comparison Table (6 marks)

FEATURE	ICD-10 (F20)	DSM-5 (295.90)
Duration	1 month of symptoms	6 months total (including ≥1 month active symptoms + prodromal/residual)
Core symptoms required	At least 1 from Group 1 OR 2 from Group 2	At least 2 from Criterion A list (≥1 must be delusions, hallucinations, or disorganized speech)
Group 1 (ICD-10)	Thought echo, insertion, withdrawal, broadcasting; Passivity experiences; Third-person voices, running commentary; Persistent bizarre delusions	N/A, combined into single Criterion A list
Group 2 (ICD-10)	Persistent hallucinations + delusions; Neologisms, thought blocking; Catatonic behavior; Negative symptoms	N/A
Schneider's FRS	Given special diagnostic weight (Group 1 essentially = FRS)	NOT given special weight
Functional impairment	Not required	Required (Criterion B)
Subtypes	Yes (Paranoid, Hebephrenic, Catatonic, Simple, Residual, Undifferentiated, Post-schizophrenic depression)	Dropped (dimensional approach)
Mood disorder exclusion	Less explicit	Explicit (Criterion D: schizoaffective and mood disorder excluded)
Substance exclusion	Present	Present (Criterion E)
Catatonia	Subtype of schizophrenia	Separate specifier
Course specifiers	Continuous, episodic with progressive deficit, episodic with stable deficit, episodic remittent, incomplete remission, complete remission	First episode (currently in acute/partial/full remission), multiple episodes (same), continuous, unspecified

B. Key Diagnostic Differences (2 marks)

ISSUE · IMPACT

Duration threshold ICD-10 diagnoses schizophrenia 5 months earlier than DSM-5; what ICD-10 calls schizophrenia, DSM-5 may call schizophreniform disorder (<6 months)

FRS weighting ICD-10 gives FRS privileged status (single FRS sufficient); DSM-5 does not, requires 2 Criterion A symptoms

Functional criterion DSM-5 mandates social/occupational dysfunction; ICD-10 focuses purely on symptoms, a well-functioning person with symptoms qualifies in ICD-10 but might not in DSM-5

Subtypes ICD-10 retains subtypes including Simple Schizophrenia (F20.6); DSM-5 dropped all subtypes

Schizoaffective boundary DSM-5 has more explicit criteria for distinguishing; ICD-10 boundary is less clear

C. ICD-11 Updates (1.5 marks)

ICD-11 (implemented 2022) brings significant changes that align more closely with DSM-5:

CHANGE · DETAIL

Dropped subtypes Like DSM-5; uses symptom qualifiers instead

Symptom qualifiers Positive, Negative, Depressive, Manic, Psychomotor, Cognitive

FRS No longer given special diagnostic weight

Duration Still 1 month (shorter than DSM-5)

Catatonia Separate category (6A40) + qualifier

Simple schizophrenia Dropped

Dimensional approach Aligns with research (RDoC influence)

Conclusion: The key practical difference is duration (1 month vs 6 months). ICD-11 has modernized significantly, dropping subtypes and FRS privilege, but retains the shorter duration criterion. Clinicians should be familiar with both systems.

Cross-reference: D1 Section 3.2, D4 Table 1

Q12. What is metabolic syndrome? Discuss its relationship with antipsychotic drugs. [5+5 = 10 marks]

Exam Strategy: Clean split. Define metabolic syndrome (ATP III criteria) then link systematically to antipsychotics.

Answer:

A. Metabolic Syndrome (5 marks)

Definition: Metabolic syndrome is a cluster of interconnected metabolic abnormalities that increase the risk of cardiovascular disease (2x) and type 2 diabetes (5x).

Diagnostic Criteria (Modified ATP III/NCEP, 2005), any 3 of 5:

CRITERION · THRESHOLD

Central obesity (waist circumference) >102 cm (Males), >88 cm (Females) [South Asian: >90 cm (M), >80 cm (F)]

Elevated triglycerides ≥150 mg/dL (or on treatment)

Reduced HDL cholesterol <40 mg/dL (M), <50 mg/dL (F) (or on treatment)

Elevated blood pressure ≥130/85 mmHg (or on treatment)

Elevated fasting glucose ≥100 mg/dL (or on treatment)

Other criteria systems: IDF (2005) requires central obesity + 2 others; WHO (1998) requires insulin resistance.

Pathophysiology: Insulin resistance is the central mechanism → hyperinsulinemia → dyslipidemia → hypertension → pro-inflammatory state → prothrombotic state → accelerated atherosclerosis.

Prevalence in schizophrenia: 30-40% (2-3x general population rate).

B. Relationship with Antipsychotic Drugs (5 marks)

Mechanisms of antipsychotic-induced metabolic syndrome:

RECEPTOR	EFFECT	METABOLIC CONSEQUENCE
H1 blockade	Increased appetite, sedation (reduced activity)	Weight gain
5-HT_{2C} blockade	Increased appetite (leptin resistance)	Weight gain, insulin resistance
M3 blockade	Impaired insulin secretion from pancreatic beta cells	Diabetes (independent of weight gain)
D2 blockade	Altered reward pathways for food	Increased caloric intake
Direct adipocyte effects	Altered lipid metabolism	Dyslipidemia

Risk ranking by drug:

RISK LEVEL · DRUGS

Highest Clozapine, Olanzapine

High Quetiapine, Chlorpromazine

Moderate Risperidone, Paliperidone

Low Haloperidol, Aripiprazole, Brexpiprazole

Lowest Ziprasidone, Lurasidone, Cariprazine

Weight gain data (mean at 10 weeks):

DRUG · WEIGHT GAIN

Clozapine 4.5 kg

Olanzapine 4.2 kg

Quetiapine 2.3 kg

Risperidone 2.1 kg

Aripiprazole 0.7 kg

Ziprasidone 0.04 kg

Management:

STRATEGY · DETAILS

Prevention Choose metabolically safer antipsychotic; baseline metabolic screening before starting ANY antipsychotic

Monitoring Weight/BMI monthly for 3 months then quarterly; fasting glucose and lipids at baseline, 3 months, then annually; waist circumference; blood pressure

Lifestyle Diet counseling, structured exercise program (150 min/week moderate), smoking cessation

Pharmacological Metformin (best evidence; reduces weight by 2-3 kg, improves insulin sensitivity); Statins for dyslipidemia; Switch to metabolically neutral antipsychotic (aripiprazole, ziprasidone, lurasidone)

Switching Switch from olanzapine/clozapine to aripiprazole/ziprasidone if clinically feasible; use bridging strategy

Special note on Cobenfy (xanomeline-trospium): First antipsychotic with NO D2 blockade → no metabolic syndrome, no EPS, no prolactin elevation. May represent paradigm shift in metabolic safety.

Q13. Describe prodromal symptoms of schizophrenia. Discuss interventions in the prodromal phase. [10 marks]

Exam Strategy: 4+6 split. Describe prodrome thoroughly, then interventions with evidence.

Answer:

A. Prodromal Symptoms (4 marks)

Definition: The prodromal phase is the period between the first noticeable change from premorbid functioning and the onset of frank psychotic symptoms. Duration: months to years (mean 2-5 years).

Prodromal symptoms:

DOMAIN · SYMPTOMS

Attenuated positive Suspiciousness, ideas of reference, perceptual disturbances (illusions, brief hallucinations), magical thinking, unusual thought content

Negative-like Social withdrawal, reduced motivation, declining academic/occupational performance, anergia, reduced emotional expression

Cognitive Difficulty concentrating, memory problems, decline in school/work performance

Affective Depression, anxiety, irritability, mood swings, perplexity

Behavioral Sleep disturbance, change in appetite, decline in self-care, odd behavior, social role dysfunction

Neurotic-like Obsessive features, depersonalization, derealization

At-Risk Mental State (ARMS) / Ultra-High Risk (UHR) Criteria (Yung & McGorry, PACE criteria):

CRITERION	DESCRIPTION	DURATION
Attenuated Psychotic Symptoms (APS)	Subthreshold positive symptoms: unusual thought content, suspiciousness, perceptual abnormalities, disorganized speech, grandiosity	Present in past year, at least weekly for 1 month
Brief Limited Intermittent Psychotic Episodes (BLIPS)	Full psychotic symptoms that spontaneously resolve	Lasting <1 week, resolving spontaneously

CRITERION	DESCRIPTION	DURATION
Trait + State	Family history of psychotic disorder (FDR) OR schizotypal personality + functional decline (30% drop in GAF)	In past year

Conversion rate: ~30% of UHR individuals develop frank psychosis within 3 years (though rates declining in recent studies, possibly due to treatment effects or referral bias).

Assessment tools:

- CAARMS (Comprehensive Assessment of At-Risk Mental States)
- SIPS/SOPS (Structured Interview for Prodromal Syndromes)

B. Interventions in the Prodromal Phase (6 marks)

Philosophy: Staged treatment, begin with least harmful interventions; escalate if needed. Balance risk of transition to psychosis against risks of unnecessary treatment.

INTERVENTION	EVIDENCE	DETAILS
CBT	Strongest evidence	Morrison et al. (2004): CBT reduced transition rate to psychosis; targets distressing beliefs, anxiety, social withdrawal; 14-26 sessions; NICE-recommended for ARMS
Omega-3 fatty acids	Mixed	Amminger et al. (2010): Promising initial data (reduced transition from 28% to 5%); NEURAPRO multi-site trial (2017): did NOT replicate; currently not recommended as standalone
Low-dose antipsychotics	Effective but controversial	McGorry et al. (2002): Risperidone + CBT reduced transition; ethical concerns: treating people who may never develop psychosis (70% won't); side effects in young people (weight, metabolic, prolactin)
Antidepressants	Limited evidence	May help prodromal depression/anxiety; no clear evidence for preventing transition
Stress management	Supportive	Reduce environmental stressors; improve coping; sleep hygiene

INTERVENTION	EVIDENCE	DETAILS
Substance use reduction	Important	Cannabis cessation particularly important (reduces a modifiable risk factor)
Family support	Helpful	Psychoeducation about risk, monitoring for changes, reducing expressed emotion
Supported education/employment	Recovery-oriented	Prevent functional decline during prodrome
Physical exercise	Emerging	Neuroprotective effects; improves mood, cognition
Monitoring	Essential	Regular follow-up even without active treatment; transition to early intervention if psychosis develops

Current guidelines:

- NICE: CBT for all ARMS patients; do NOT use antipsychotics prophylactically
- APA: No specific guidelines for prodromal phase
- International Early Psychosis Association: Staged approach with regular monitoring

Ethical considerations:

- Labeling: Risk of stigma for individuals who may never develop psychosis
- False positives: 70% of UHR individuals do NOT transition
- Medication risks: Side effects of antipsychotics in a population that may not need them
- Right to know: Informing individuals about risk without causing undue anxiety
- Balanced approach: Staging model respects both autonomy and prevention

Cross-reference: D1 Section 10.4

Q14. Describe newer antipsychotics. Write a note on cariprazine. [10 marks]

Exam Strategy: 5+5 split. Cover 4-5 newer agents, then deep dive on cariprazine.

Answer:

A. Newer Antipsychotics (5 marks)

DRUG	YEAR APPROVED	MECHANISM	KEY FEATURES	SIDE EFFECTS
Cariprazine	2015	D3-preferring partial agonist + D2 partial agonist + 5-HT1A partial agonist	Superior for negative symptoms; very long half-life (2-4 weeks); approved for schizophrenia + bipolar mania / depression	Akathisia, EPS, insomnia; low metabolic risk
Brexiprazole	2015	D2 partial agonist + 5-HT1A partial agonist (lower intrinsic activity at D2 than aripiprazole)	Less akathisia than aripiprazole; also approved for Alzheimer's agitation	Weight gain (moderate), akathisia (less than aripiprazole)
Lumateperone	2019	5-HT2A antagonist + D2 partial agonist + D1 modulation + SERT inhibition + GluN2B modulation	Low D2 occupancy (~40%); minimal metabolic/EPS/prolactin; once daily 42 mg	Somnolence, sedation; weight-neutral; QTc caution
Pimavanserin	2016	5-HT2A inverse agonist (no D2 activity)	Only for Parkinson's disease psychosis; first antipsychotic without D2 blockade	QTc prolongation; not for schizophrenia monotherapy
Cobefny (xanomeline-trospium)	2024	M1/M4 muscarinic agonist (xanomeline) + peripheral muscarinic antagonist (trospium)	FIRST non-D2 antipsychotic for schizophrenia; no EPS, no metabolic, no prolactin	Nausea, dyspepsia, constipation (cholinergic peripheral effects managed by trospium)

Paradigm shifts:

- Lumateperone: Proves effective antipsychotic action at <65% D2 occupancy
- Cobefny: Proves antipsychotic effect WITHOUT D2 blockade (muscarinic pathway)
- Cariprazine: D3 pathway for negative symptoms

B. Cariprazine, Detailed Note (5 marks)

Pharmacology:

- D3 receptor partial agonist with high preferential binding (D3 affinity 6-8x higher than D2)

- D2 partial agonist
- 5-HT1A partial agonist
- 5-HT2B antagonist
- Low affinity for H1, M1, alpha-1 (low metabolic/sedative/hypotensive risk)

Pharmacokinetics:

PARAMETER · VALUE

Half-life (parent) 2-4 days

Active metabolites DDCAR (half-life 1-3 weeks), DCAR

Effective half-life 2-4 weeks (due to DDCAR)

Metabolism CYP3A4 (primary), CYP2D6

Time to steady state 4-8 weeks

Dose range 1.5-6 mg / day (schizophrenia: 1.5-6; bipolar mania: 3-6; bipolar depression: 1.5-3)

Clinical significance of long half-life:

- Missed doses have minimal impact on plasma levels (forgiveness factor)
- Effects and side effects persist for weeks after discontinuation
- Slower dose titration needed
- Takes 4-8 weeks to reach steady state

D3 Receptor and Negative Symptoms:

- D3 receptors concentrated in limbic and cortical areas (motivation, emotion, cognition)
- D3 partial agonism → enhancement of dopaminergic signaling in mesocortical pathway
- Nemeth et al. (2017), RCT: Cariprazine 4.5 mg vs Risperidone 4 mg for predominant negative symptoms over 26 weeks. Cariprazine showed statistically significant and clinically meaningful improvement on PANSS-FSNS (Factor Score for Negative Symptoms)
- First antipsychotic with RCT evidence specifically for primary negative symptoms

Indications:

1. Schizophrenia (positive and negative symptoms)
2. Bipolar I disorder, manic and mixed episodes
3. Bipolar I depression (monotherapy)

Side effects:

SIDE EFFECT · FREQUENCY

Akathisia 10-15% (most common; less than aripiprazole in some studies)

EPS (parkinsonism) 5-10%

Insomnia 5-10%

Headache 5-10%

Weight gain Minimal (~1 kg at 6 weeks)

Metabolic effects Minimal

Prolactin May decrease (partial agonist)

Sedation Low

Advantages over other antipsychotics:

- Evidence for primary negative symptoms (unique)
- Low metabolic burden
- Long half-life improves adherence forgiveness
- Broad indication (schizophrenia + bipolar mania + bipolar depression)
- Favorable prolactin profile

Limitations:

- Akathisia can be dose-limiting
- Very long washout period
- Not available in all countries
- Cost
- EPS still possible at higher doses

Cross-reference: D1 Sections 8.3, 8.4

Q15. Negative symptoms of schizophrenia: describe and discuss management. [3+3+4 = 10 marks]

Exam Strategy: Overlap with Q1 and Q7. Use mark allocation to guide depth. This is more focused on negative symptoms specifically.

Answer:

A. Description of Negative Symptoms (3 marks)

[Content substantially overlaps with Q1 Section B. Key points:]

Negative symptoms represent a diminution or absence of normal functions. The concept was formalized by Crow (1980) as Type II symptoms and further developed by Andreasen (SANS, 1982).

Andreasen's five domains:

1. **Affective flattening**, reduced emotional expression (facial, vocal, gestural)
2. **Alogia**, poverty of speech/content
3. **Avolition**, loss of motivation and drive
4. **Anhedonia/Asociality**, inability to experience pleasure; social withdrawal
5. **Attention impairment**, difficulty sustaining attention

Newer conceptualization (Kirkpatrick): Two factors:

- **Diminished expression** (affective flattening + alogia)
- **Avolition/apathy** (avolition + anhedonia + asociality)

Deficit syndrome (Carpenter): Primary, enduring negative symptoms present in stable periods; ~15-25% of patients; associated with worse outcome.

B. Primary vs Secondary Distinction (3 marks)

CAUSE OF SECONDARY NEGATIVE SYMPTOMS	MECHANISM	HOW TO IDENTIFY
Antipsychotic side effects	EPS (akinesia mimics apathy), sedation	Correlates with medication timing/dose; improves with dose reduction
Depression	Anhedonia, withdrawal, psychomotor retardation	Subjective sadness, guilt, hopelessness; responds to antidepressants
Positive symptoms	Withdrawal due to paranoia/fear	Improves when positive symptoms treated
Environmental understimulation	Institutionalization, lack of social contact	Improves with environmental enrichment
Substance use	Cannabis, alcohol → amotivation	History, drug screen
Medical comorbidity	Hypothyroidism, anemia, chronic illness	Investigations

Assessment approach: Rule out ALL secondary causes before labeling symptoms as primary.

Assessment tools: SANS, BNSS (Brief Negative Symptom Scale), CAINS (Clinical Assessment Interview for Negative Symptoms), PANSS negative subscale.

C. Management (4 marks)

Step 1: Address secondary negative symptoms

- Reduce antipsychotic dose if EPS/sedation contributing
- Switch to lower-EPS antipsychotic (aripiprazole, quetiapine)
- Treat depression (SSRI/SNRI/mirtazapine)
- Address substance use
- Improve environment (day programs, social contact)

Step 2: Pharmacological strategies for primary negative symptoms

STRATEGY	EVIDENCE	MECHANISM
Cariprazine	Strong (RCT)	D3 partial agonism → mesocortical dopamine enhancement
Amisulpride (50-300 mg)	Moderate	Low-dose presynaptic D2/D3 blockade → increased DA release
Aripiprazole	Moderate	D2 partial agonism; less secondary negative symptoms
Clozapine	Moderate (in TRS)	Overall symptom improvement including negative
Add-on antidepressants	Low-moderate	Mirtazapine, fluoxetine, for motivational/anhedonic component
Cobefy	Emerging	Muscarinic agonism; early data on negative symptoms
Modafinil/armodafinil	Low	For anergia/cognitive slowing (limited evidence)

Step 3: Psychosocial interventions

INTERVENTION · TARGETING

Social skills training Asociality, social withdrawal

Cognitive remediation Attention, executive function

Supported employment (IPS) Avolition, functional outcome

Behavioral activation (adapted CBTp) Avolition, anhedonia

Art/music therapy Engagement, emotional expression

Physical exercise programs Motivation, cognitive function

Environmental enrichment Understimulation

Family intervention Reduce over-accommodation

Emerging approaches:

- rTMS (repetitive transcranial magnetic stimulation), left DLPFC; modest evidence
- tDCS (transcranial direct current stimulation), emerging
- Oxytocin intranasal, social cognition; inconsistent results
- Glycine-site agonists (D-serine, sarcosine), glutamate modulation; modest

Cross-reference: D1 Section 2.2, Q1 above

Q16. Define compliance in psychiatry. Discuss causes of poor compliance. Discuss newer modalities for improving compliance in schizophrenia. [3+3+4 = 10 marks]

Exam Strategy: Define clearly (mention shift from compliance to adherence to concordance). Causes are systematic. Newer modalities = LAIs + digital tools.

Answer:

A. Definition (3 marks)

Compliance: The extent to which a patient's behavior (taking medication, following diet, lifestyle changes) coincides with medical advice. Term implies passive patient role.

Evolution of terminology:

TERM	IMPLICATION	MODEL
Compliance	Patient follows doctor's orders	Paternalistic
Adherence	Patient follows agreed-upon treatment plan	Partnership
Concordance	Shared decision-making; treatment plan mutually agreed	Collaborative

Types of non-adherence:

- Complete non-adherence (stopping medication entirely)
- Partial adherence (reducing dose, irregular intake, drug holidays)
- Covert non-adherence (claiming to take medication but not doing so)

Rates in schizophrenia: 40-60% non-adherent within first year; up to 75% by 2 years.

Impact: Non-adherence is the strongest predictor of relapse in schizophrenia. Each relapse potentially causes further neurobiological damage and functional decline.

B. Causes of Poor Compliance (3 marks)

CATEGORY · SPECIFIC FACTORS

Patient factors Lack of insight (anosognosia, most important), cognitive deficits (forgetting), substance use, negative symptoms (apathy), cultural beliefs about illness, perceived stigma

Medication factors Side effects (weight gain, EPS, sedation, sexual dysfunction, most cited by patients), complex regimen, polypharmacy, delayed onset of benefit, lack of perceived efficacy

Illness factors Paranoid delusions (about medication being poison), grandiosity (feeling well, don't need medication), disorganization

Clinician factors Poor therapeutic alliance, inadequate psychoeducation, not involving patient in decisions, not addressing side effects

System factors Cost of medication, distance to healthcare, lack of community follow-up, inadequate health insurance, stigma of attending psychiatric services

Social/family Lack of family support, family beliefs against medication, peer influence

C. Newer Modalities for Improving Compliance (4 marks)

1. Long-Acting Injectable Antipsychotics (LAIs):

LAI	FREQUENCY	ADVANTAGES
Paliperidone palmitate 1-monthly	Monthly	Most widely used SGA LAI
Paliperidone palmitate 3-monthly	Every 3 months	After stabilization on 1-monthly
Paliperidone palmitate 6-monthly	Every 6 months	Newest; maximum adherence convenience
Aripiprazole monohydrate	Monthly	Less metabolic effects
Aripiprazole lauroxil	Every 4-8 weeks	Prodrug
Olanzapine pamoate	Every 2-4 weeks	PDSS risk, 3-hour monitoring

Benefits: Guaranteed delivery, transparent adherence, eliminates daily decision, reduces relapse by 20-30% vs oral.

2. Digital Adherence Technologies:

TECHNOLOGY · DESCRIPTION

Abilify MyCite Aripiprazole tablet with ingestible sensor (IEM); detects ingestion via body patch → smartphone app → clinician portal

Medication Event Monitoring Systems (MEMS) Electronic cap records each bottle opening

Smartphone apps Medication reminders, symptom tracking, refill alerts

Telehealth/telemedicine Remote monitoring, video visits, check-ins

AI-based monitoring Predictive models for non-adherence risk

3. Psychoeducation and Motivational Approaches:

APPROACH · METHOD

Motivational interviewing Explore ambivalence about treatment; enhance intrinsic motivation

Shared decision-making Involve patient in choosing medication, discussing side effects

Psychoeducation (patient + family) Understanding illness, need for maintenance treatment, early warning signs

Peer support Recovered patients as role models

4. Simplified Regimens:

- Once-daily dosing (paliperidone ER, aripiprazole, lurasidone, cariprazine)
- Combination approaches reducing pill burden
- Extended-release formulations

5. Community-Based Approaches:

- ACT teams (Assertive Community Treatment): Bring medication to patient
- Community mental health workers (India: DMHP model)
- Directly Observed Therapy (DOT), adapted from TB model
- Home visits by community nurses

6. Side Effect Management:

- Proactive management of side effects BEFORE they cause non-adherence
- Switching to better-tolerated medication
- Addressing sexual dysfunction openly (often underreported)

Cross-reference: D1 Section 10.5

Q17. Discuss newer long-acting antipsychotics. Describe dosing and side effects of olanzapine pamoate. [10 marks]

Exam Strategy: 5+5 split. Cover all newer LAIs, then deep dive on olanzapine pamoate + PDSS.

Answer:

A. Newer Long-Acting Injectable Antipsychotics (5 marks)

LAI	MECHANISM	DOSE	FREQUENCY	KEY NOTES
Paliperidone palmitate 1-monthly (PP1M)	D2 + 5-HT2A antagonist	Initiation: 234 mg deltoid → 156 mg deltoid (day 8) → 117 mg monthly (deltoid / gluteal)	Monthly	Most widely prescribed SGA LAI; no oral supplementation needed
Paliperidone palmitate 3-monthly (PP3M)	Same	273-819 mg (based on PP1M dose)	Every 3 months	After ≥4 months on PP1M; gluteal only
Paliperidone palmitate 6-monthly (PP6M)	Same	1092 mg	Every 6 months	After adequate PP1M/PP3M; gluteal; longest interval available
Aripiprazole monohydrate (Abilify Maintena)	D2/5-HT1A partial agonist	400 mg (or 300 mg if side effects)	Monthly	Overlap with oral aripiprazole 10-20 mg for 14 days; deltoid or gluteal
Aripiprazole lauroxil (Aristada)	Same (prodrug)	441-882 mg	Every 4-8 weeks	Available with Aristada Initio (one-time injection for rapid initiation)
Olanzapine pamoate (Zyprexa Relhyprev)	MARTA	150-405 mg	Every 2-4 weeks	PDSS risk, see section B
Risperidone LAI (Risperdal Consta)	D2 + 5-HT2A	25-50 mg	Every 2 weeks	Microsphere technology; requires oral supplementation for 3 weeks
Risperidone ISM (Perseris)	Same	90-120 mg SC	Monthly	Subcutaneous; no oral supplementation needed

Advantages of newer LAIs:

- Extended dosing intervals (up to 6 months with PP6M)
- SGA profile (less EPS than FGA LAIs)
- Transparent adherence

- Reduced relapse rates vs oral equivalent
- Some require no oral supplementation (PP1M, aripiprazole lauroxil with Initio)

Comparison of LAI properties:

PROPERTY	PP1M	PP3M	ARIPRAZOLE MONOHYDRATE	OLANZAPINE PAMOATE
Injection site	Deltoid or gluteal	Gluteal only	Deltoid or gluteal	Gluteal only
Oral overlap needed	No	No	14 days	No
Storage	Room temperature	Room temperature	Room temperature	Room temperature
Post-injection monitoring	None	None	None	3 hours mandatory
Metabolic risk	Moderate	Moderate	Low	High

B. Olanzapine Pamoate, Dosing and Side Effects (5 marks)

Formulation: Olanzapine pamoate monohydrate (salt of olanzapine and pamoic acid); slow dissolution from injection site over weeks.

Dosing:

ORAL OLANZAPINE DOSE	LAI DOSE	FREQUENCY
10 mg/day	210 mg every 2 weeks OR 405 mg every 4 weeks	First 8 weeks: 210mg/2wks; Maintenance: 150mg/2wks or 300mg/4wks
15 mg/day	300 mg every 2 weeks (first 8 weeks) → 210 mg every 2 weeks or 405 mg every 4 weeks	As above
20 mg/day	300 mg every 2 weeks	May continue at this dose

- Maximum single injection: 405 mg
- Deep gluteal IM injection only (NOT deltoid, risk of too-rapid absorption)
- Use Z-track technique
- Reconstitute with provided diluent; use within 1 hour

Post-Injection Delirium/Sedation Syndrome (PDSS):

Mechanism Accidental intravascular injection → sudden spike in olanzapine blood levels

Incidence ~0.07% of injections (~2% of patients over time)

Onset Within 1-3 hours post-injection

Symptoms Sedation (ranging from mild to coma), confusion, disorientation, dysarthria, agitation, ataxia, aggression, dizziness, weakness, hypertension, seizure

Management Emergency medical support; no specific antidote; supportive care; activated charcoal not useful (IM route)

Recovery Complete recovery within 24-72 hours with supportive care

MANDATORY monitoring (REMS program):

- 3-hour observation at healthcare facility after EVERY injection
- Accompanied by healthcare provider or able to drive
- Monitor for sedation, confusion, altered consciousness
- If PDSS suspected: continuous monitoring in medical facility until fully recovered

Side effects (beyond PDSS):

SIDE EFFECT	FREQUENCY	NOTES
Injection site reactions	5-8%	Pain, induration, mass at injection site
Weight gain	Significant	Same metabolic profile as oral olanzapine
Metabolic syndrome	High risk	Dyslipidemia, hyperglycemia, weight gain
Sedation	Common	Expected with olanzapine; may be pronounced after injection
Hyperprolactinemia	Moderate	Less than risperidone LAI
EPS	Low	Typical olanzapine profile

Advantages:

- No oral overlap needed
- Effective for patients stabilized on oral olanzapine
- Transparent adherence

Disadvantages:

- PDSS risk requiring 3-hour monitoring (major logistical barrier)
- Gluteal only (no deltoid)
- High metabolic burden
- REMS registration required
- Limited prescribing due to monitoring requirements

Q18. Discuss non-pharmacological treatments in schizophrenia. Write about the role of family therapy. [5+5 = 10 marks]

Exam Strategy: Overlap with Q9 and Q10. Keep non-pharmacological section broader (include ECT, brain stimulation). Family therapy section per Q10.

Answer:

A. Non-Pharmacological Treatments (5 marks)

1. Neuromodulation:

MODALITY	INDICATION	EVIDENCE
ECT	Catatonia, TRS augmentation, acute psychosis with high suicidality	Strong for catatonia (80-90% response); moderate for TRS augmentation
rTMS	Auditory hallucinations (left temporo-parietal), negative symptoms (left DLPFC)	Moderate for hallucinations; emerging for negative symptoms
tDCS	Hallucinations, negative symptoms	Emerging evidence; accessible

2. Psychological Interventions:

INTERVENTION	KEY FEATURES	EVIDENCE LEVEL
CBTp	Distress reduction from psychotic symptoms; 16+ sessions; NICE first-line	Strong
Social skills training	Role-playing, behavioral rehearsal	Moderate
Cognitive remediation	Computer-based + coaching; MCCB domains	Moderate-Strong
Acceptance and Commitment Therapy (ACT)	Acceptance of voices rather than fighting; values-based living	Emerging
AVATAR therapy	Digital avatar representing voice; patient dialogues with avatar (therapist controls)	Craig et al. (2018, Lancet); promising for persistent hallucinations
Metacognitive training (MCT)	Targets cognitive biases (jumping to conclusions, theory of mind deficits)	Moderate

3. Vocational and Social:

INTERVENTION · KEY FEATURES

Supported employment (IPS) Competitive employment with support; 2-3x more effective than traditional

Assertive Community Treatment (ACT) Multidisciplinary, community-based, 24/7

Supported housing Graded from group homes to independent

Peer support Trained individuals with lived experience

4. Lifestyle:

AREA · INTERVENTION

Physical exercise 150 min/week; improves cognition, metabolic parameters, symptoms

Diet Mediterranean-style; address medication-induced cravings

Sleep hygiene Often disrupted in schizophrenia

Substance use Cannabis cessation; dual diagnosis programs

B. Role of Family Therapy (5 marks)

[Content per Q10, key points:]

Rationale: High Expressed Emotion (critical comments, hostility, emotional over-involvement) → 50-65% relapse rate vs 15-25% in low EE households (Vaughn & Leff, 1976).

Models: Psychoeducational (Anderson/Falloon), Behavioral family management, Multiple family groups (McFarlane), Cognitive-behavioral family approaches.

Evidence: Cochrane meta-analysis: Reduces relapse by 20-50%; NNT ~7. Recommended by NICE for ALL patients in contact with family. Minimum 10 sessions over 3+ months.

Components:

1. Psychoeducation about the illness
2. Communication skills training (active listening, positive requests, constructive feedback)
3. Problem-solving techniques
4. Early warning signs identification and crisis planning
5. Emotional support for carers
6. Reducing self-blame and guilt in families

Benefits beyond relapse prevention:

- Improved medication adherence

- Reduced family burden and burnout
- Better patient social functioning
- Reduced hospitalization
- Cost-effective

Indian context considerations:

- Joint family system, opportunity and challenge
- Stigma may prevent families from engaging
- Cultural sensitivity needed
- Limited trained therapists; task-shifting to community health workers possible

Cross-reference: D1 Sections 10.1-10.2, Q9, Q10

Mnemonics & Memory Tricks

EXAM PEARL

Total mnemonics: 20

Mnemonic 1: Schneider's First Rank Symptoms

"ATPD", Auditory, Thought, Passivity, Delusional perception

Expand each:

LETTER	CATEGORY	SYMPTOMS
A, Auditory hallucinations	3 types	Voices Arguing, Running Commentary, Thought Echo
T, Thought interference	3 types	Thought Insertion, Thought Withdrawal, Thought Broadcasting
P, Passivity experiences	4 types	Made Feelings, Made Impulses, Made Actions, Somatic Passivity
D, Delusional perception	1 type	Normal perception → delusional interpretation

Alternate mnemonic for all 11 FRS: "ABCDE FITS MP"

- Auditory hallucinations (arguing)
- Broadcasting (thought)
- Commentary (running)
- Delusional perception
- Echo (thought)
- Feelings (made)
- Impulses (made)
- Thought insertion
- Somatic passivity
- Made actions
- Pullout = withdrawal (thought)

Mnemonic 2: Bleuler's 4 A's (Fundamental Symptoms)

"4 A's"

A	SYMPTOM	KEY POINT
Association disturbance	Loosening of associations	Most fundamental per Bleuler
Affect disturbance	Inappropriate or blunted affect	Incongruent emotional response
Ambivalence	Coexisting opposite feelings/ thoughts	Simultaneous contradictions
Autism	Withdrawal into inner world	NOT same as ASD

Remember: "Accessory symptoms (Hallucinations, Delusions) are NOT the 4 A's", Bleuler considered them secondary.

Mnemonic 3: Positive vs Negative Symptoms

Positive = "DHFB", Delusions, Hallucinations, FTD, Bizarre behavior

(Think: "D-Hab FB" = bad Facebook posts)

Negative = "5 A's of ANDREASEN":

1. Affective flattening
2. Alogia
3. Avolition
4. Anhedonia/ Asociality
5. Attention impairment

Remember: Positive = too much of something; Negative = too little of something

Mnemonic 4: Good Prognostic Factors

"FAMILY LAMP"

LETTER · FACTOR

F Female sex

A Acute onset

M Married / good social support

I Identifiable precipitant

L Late onset

Y "Yes" affective symptoms (mood component)

L Limited family history (none)

A Adequate premorbid adjustment

M Minimal negative symptoms

P Paranoid subtype (historical)

Mnemonic 5: Poor Prognostic Factors

"MINUS HELP"

LETTER · FACTOR

M Male sex

I Insidious onset

N Negative symptoms predominant

U Unmarried / poor social support

S Substance use comorbid

H Heavy family history

E Early onset

L Low premorbid functioning

P Poor early treatment response

Mnemonic 6: Catatonia Features (Bush-Francis Key Items)

"SOME GRIPS WEEN"

LETTER · FEATURE

S Stupor

O Obedience (automatic)

M Mutism

E Echolalia / Echopraxia

G Grimacing

R Rigidity

I Immobility / Posturing

P Perseveration / Stereotypy

S Staring

W Waxy flexibility

E Excitement (catatonic)

E Excitement withdrawal

N Negativism

Extra features to add: Catalepsy, Ambitendency, Verbigeration, Mannerisms, Mitgehen, Gegenhalten

Mnemonic 7: Clozapine Side Effects

"CLOZAPINE SWAMPS"

LETTER	SIDE EFFECT	SEVERITY
C	Constipation (can → ileus, FATAL)	Common-Serious
L	Leukopenia / agranuLocytosis	Rare-Serious
O	Obesity / weight gain	Common
Z	"Zzz" = Sedation	Common
A	Agranulocytosis (1%)	Rare-Serious
P	Prolactin sparing (unlike others)	Note
I	Increased salivation (sialorrhea)	Common
N	Nocturnal enuresis	Common
E	Epileptic seizures (dose-dependent)	Less common
S	Sinus tachycardia	Common

LETTER	SIDE EFFECT	SEVERITY
W	Weight gain (worst of all antipsychotics)	Common
A	Anticholinergic effects	Common
M	Myocarditis (first 4-6 weeks)	Rare-Serious
P	Postural hypotension	Common
S	Sialorrhea (repeated because it's that important)	Common

Mnemonic 8: Clozapine Monitoring Schedule

"18 - 52 - LIFE"

PERIOD	FREQUENCY	DURATION
First 18 weeks	Weekly WBC/ANC	From initiation
Weeks 18-52	Biweekly (every 2 weeks)	Rest of first year
LIFETIME after year 1	Monthly	Indefinitely while on clozapine

Stopping rules, "3-1.5 STOP":

- WBC <3000 or ANC <1.500 → STOP clozapine
- ANC <500 (agranulocytosis) → STOP permanently, never rechallenge

Mnemonic 9: FGA vs SGA Comparison

"FGA = D2 Dominant, SGA = Serotonin-Dopamine Duo"

FEATURE	FGA MNEMONIC	SGA MNEMONIC
Mechanism	"Direct D2 block"	"Serotonin Gets Addressed" (5-HT _{2A} /D2 ratio >1)
Movement	"Frequent EPS"	"Spared from EPS" (mostly)
Metabolism	"Fewer metabolic"	"Serious metabolic" (olanzapine, clozapine)
Prolactin	"Full prolactin rise"	"Some spare prolactin" (except risperidone)

Mnemonic 10: D2 Occupancy Therapeutic Window

"65-80 SWEET SPOT"

OCCUPANCY · EFFECT

<65% Sub-therapeutic ("Under 65 = Under-treated")

65-80% Therapeutic range ("Sweet Spot")

>80% EPS and hyperprolactinemia ("Over 80 = Over-blocked")

>90% NMS territory

Mnemonic 11: Metabolic Syndrome Criteria (ATP III)

"BIG WAIST"

LETTER	CRITERION	THRESHOLD
B	Blood pressure	≥130/85 mmHg
I	Insulin/glucose (fasting glucose)	≥100 mg/dL
G	"Good cholesterol" low (HDL)	<40 (M), <50 (F) mg/dL
W	Waist circumference	>102 (M), >88 (F) cm
A	"A lot of" triglycerides	≥150 mg/dL
I	(included in B)	
S	(South Asian cutoffs: 90/80 cm)	Waist adjustment
T	Three of five needed	Diagnostic rule

Need ANY 3 of 5 criteria to diagnose.

Mnemonic 12: TRS Definition Criteria

"TWO-SIX-SIX"

NUMBER · CRITERION

TWO 2 adequate antipsychotic trials failed

SIX 6 weeks minimum per trial (at adequate dose)

SIX 600 mg chlorpromazine equivalent minimum dose

Plus: At least 1 trial must be an SGA. Adherence must be confirmed. Pseudo-resistance excluded.

Mnemonic 13: Andreasen's Remission Criteria

"DUH-CM-BSL", 8 PANSS items, all must be $\leq$ mild for ≥ 6 months

LETTER · PANSS ITEM

D Delusions (P1)

U Unusual thought content (G9)

H Hallucinatory behavior (P3)

C Conceptual disorganization (P2)

M Mannerisms/posturing (G5)

B Blunted affect (N1)

S Social withdrawal (N4)

L Lack of spontaneity (N6)

Duration: ≥ 6 months at mild or below.

Mnemonic 14: Dopamine Pathways

"MINT", 4 pathways

LETTER	PATHWAY	FUNCTION	SCHIZOPHRENIA RELEVANCE
M	Mesolimbic	Reward, emotion	Hyperactive $\rightarrow$ positive symptoms
I	Nigrostriatal	Movement	AP blockade $\rightarrow$ EPS
N	Mesocortical (to Neo-cortex)	Cognition, executive	Hypoactive $\rightarrow$ negative + cognitive
T	Tuberoinfundibular	Prolactin inhibition	AP blockade $\rightarrow$ hyperprolactinemia

Mnemonic 15: EPS Timeline

"Days-Weeks-Months-Years = DAPA-TARD"

TIME	EPS TYPE	MNEMONIC
Days	Dystonia (Acute)	"Days = Dystonia"
Weeks	Akathisia	"After a few weeks, can't sit still"

TIME	EPS TYPE	MNEMONIC
Weeks-Months	PArkinsonism	"PArkinsonism takes PAience to develop"
Months-Years	TARDive dyskinesia	"TARDy to arrive" (late-onset)

Mnemonic 16: Clozapine Drug Interactions

"SMOKE, COFFEE, FLUVOX raise or lower"

SUBSTANCE	EFFECT ON CLOZAPINE	MECHANISM	CLINICAL PEARL
Smoking (tobacco)	DECREASES levels by 50%	CYP1A2 induction	Stopping smoking → levels DOUBLE → toxicity
Coffee (caffeine)	INCREASES levels	CYP1A2 inhibition	Modest effect
Fluvoxamine	INCREASES levels 2-5x	CYP1A2 inhibition	Most important interaction
Carbamazepine	DECREASES + bone marrow risk	CYP3A4 + hematological	CONTRAINDICATED combo
Ciprofloxacin	INCREASES levels	CYP1A2 inhibition	Switch antibiotic

Key rule: "CYP1A2 is Clozapine's Achilles heel"

Mnemonic 17: Metabolic Risk Ranking of Antipsychotics

"COQR-AZL" (pronounced "cocker-azle")

From highest to lowest metabolic risk:

- Clozapine = Olanzapine (highest)
- Quetiapine = Risperidone (moderate)
- Aripiprazole = Ziprasidone = Lurasidone (lowest)

Mnemonic 18: Newer Antipsychotic Mechanisms

"C-L-B-C: The New Four"

LETTER	DRUG	KEY MECHANISM	UNIQUE FEATURE
C	Cariprazine	D3 partial agonist	Negative symptoms
L	Lumateperone	5-HT2A + low D2 + glutamate	Only 40% D2 occupancy works

LETTER	DRUG	KEY MECHANISM	UNIQUE FEATURE
B	Brexiprazole	D2 partial agonist (gentler)	Less akathisia than aripiprazole
C	Cobenfy	Muscarinic M1 / M4 agonist	No D2 at all, paradigm shift

Mnemonic 19: Expressed Emotion Components

"CHO", Criticism, Hostility, Over-involvement

COMPONENT · WHAT IT LOOKS LIKE

Critical comments "You never do anything right"; "You're so lazy"

Hostility Rejection of person (not just behavior); generalized negativity

Over-involvement Excessive emotional display, self-sacrifice, intrusive monitoring

High EE = 50-65% relapse. Low EE = 15-25% relapse.

Mnemonic 20: LAI Dosing Intervals

"1-2-3-6 Palmitate" for Paliperidone:

FORMULATION	FREQUENCY	MNEMONIC
PP1M	Every 1 month	Start here
PP3M	Every 3 months	After ≥4 months on PP1M
PP6M	Every 6 months	After adequate PP3M

Other intervals to know:

- Risperdal Consta: Every 2 weeks (the only biweekly SGA LAI)
- Aripiprazole (Abilify Maintena): **Monthly** (14-day oral overlap)
- Olanzapine pamoate: Every 2-4 weeks (3-hour watch for PDSS)

High-Yield Comparisons

Table 1: ICD-11 vs DSM-5: Schizophrenia Diagnosis

FEATURE	ICD-11 (6A20)	DSM-5 (295.90)
Minimum duration	1 month of symptoms	6 months total (≥1 month active + prodromal/residual)
Core symptoms	At least 1 core symptom (delusions, hallucinations, disorganized thinking, passivity/control experiences)	≥2 Criterion A symptoms; ≥1 must be delusions, hallucinations, or disorganized speech
Functional impairment	Not required for diagnosis	Required (Criterion B)
Subtypes	Dropped; replaced by symptom qualifiers	Dropped; dimensional approach (CRDPSS)
Symptom qualifiers	6 qualifiers: Positive, Negative, Depressive, Manic, Psychomotor, Cognitive	Clinician-Rated Dimensions of Psychosis Severity Scale (8 dimensions)
Schneider's FRS	No special diagnostic weight	No special diagnostic weight
Catatonia	Qualifier + separate category (6A40)	Separate specifier; can apply to multiple disorders
Course specifiers	First episode, Multiple episodes, Continuous	First episode, Multiple episodes, Continuous, Unspecified (each with acute/partial/full remission)
Mood exclusion	Must exclude schizoaffective and mood disorders	Explicit Criterion D: schizoaffective and mood disorders excluded
Substance exclusion	Yes	Yes (Criterion E)
Simple schizophrenia	Dropped	Not included
ASD provision	Not specified	Criterion F: if ASD/childhood communication disorder present, need prominent delusions/hallucinations ≥1 month

Key exam point: The biggest practical difference is DURATION, ICD-11 diagnoses schizophrenia 5 months earlier. What ICD-11 calls schizophrenia at 1 month, DSM-5 would call schizophreniform disorder until 6 months.

Table 2: Positive vs Negative Symptoms

FEATURE	POSITIVE SYMPTOMS	NEGATIVE SYMPTOMS
Definition	Excess or distortion of normal functions	Diminution or loss of normal functions
Examples	Hallucinations, delusions, FTD, disorganized behavior	Affective flattening, alogia, avolition, anhedonia, asociality
Crow's Type	Type I	Type II
Onset	Often acute	Often insidious, from prodrome
Course	Fluctuating, episodic	Persistent, stable
Response to antipsychotics	Good (especially FGAs and SGAs)	Poor (cariprazine has best evidence)
Neurobiological basis	Mesolimbic dopamine hyperactivity	Mesocortical dopamine hypoactivity; glutamate dysfunction
Structural correlates	Less prominent brain changes	Enlarged ventricles, cortical atrophy, reduced prefrontal volume
Impact on function	Acute impairment during episodes	Primary determinant of long-term functional outcome
Assessment scale	SAPS, PANSS positive subscale	SANS, BNSS, CAINS, PANSS negative subscale
Prognosis	Better (when predominant)	Worse (deficit syndrome = poorest)

Table 3: FGA vs SGA Antipsychotics

FEATURE	FGA (TYPICAL)	SGA (ATYPICAL)
Primary mechanism	D2 antagonism	5-HT2A/D2 antagonism (ratio >1); some are D2 partial agonists
D2 binding	Tight, slow dissociation	Variable; some fast dissociation (clozapine, quetiapine)
EPS risk	High (especially high-potency: haloperidol)	Low at therapeutic doses (except risperidone at high doses)
Tardive dyskinesia	Higher cumulative risk (~5% / year)	Lower (~1% / year); lowest with clozapine

FEATURE	FGA (TYPICAL)	SGA (ATYPICAL)
Prolactin elevation	Marked (especially haloperidol)	Variable: high (risperidone, paliperidone), low (aripiprazole, quetiapine, clozapine)
Metabolic effects	Less (except chlorpromazine)	Higher (worst: clozapine, olanzapine)
Efficacy for positive symptoms	Equivalent to SGAs	Equivalent to FGAs
Efficacy for negative symptoms	Poor; may worsen (secondary negatives)	Modest; cariprazine best evidence
Cost	Cheaper	More expensive (but generics available)
Examples	Chlorpromazine, haloperidol, trifluoperazine, fluphenazine, pimozide	Risperidone, olanzapine, quetiapine, aripiprazole, clozapine, cariprazine
Key evidence	Still first-line in resource-limited settings	CATIE: no clear superiority over FGAs overall; clozapine uniquely effective in TRS

Table 4: Good vs Poor Prognostic Factors

DOMAIN	GOOD PROGNOSIS	POOR PROGNOSIS
Onset	Acute, with identifiable precipitant	Insidious, no precipitant
Age	Late onset (>25 years)	Early onset (<20 years)
Sex	Female	Male
Marital status	Married, good social support	Single, divorced, isolated
Premorbid	Good adjustment (education, employment, social)	Poor adjustment, schizoid traits, developmental delays
Symptom profile	Affective symptoms, paranoid features	Negative symptoms predominant, disorganization
Family history	Absent for schizophrenia	Strong family history
Cognitive function	Preserved	Impaired
Substance use	None	Comorbid SUD (cannabis, stimulants)
Treatment response	Early good response	Poor early response, treatment resistance
DUP	Short (<3 months)	Long (>12 months)

DOMAIN	GOOD PROGNOSIS	POOR PROGNOSIS
Brain imaging	Normal	Enlarged ventricles, cortical atrophy
Sociocultural	Developing country (WHO DOSMeD)	Developed country
Expressed emotion	Low EE household	High EE household

Table 5: Schneider's First Rank vs Second Rank Symptoms

FEATURE	FIRST RANK SYMPTOMS	SECOND RANK SYMPTOMS
Diagnostic weight	Higher (historically); single FRS sufficient in ICD-10	Lower; not sufficient alone
Core concept	Loss of ego boundaries (Ich-Störung)	Non-specific psychotic/affective phenomena
Auditory hallucinations	Voices arguing (3rd person), Running commentary, Thought echo	Other hallucinations (visual, tactile, olfactory, gustatory)
Passivity	Made feelings, Made impulses, Made actions, Somatic passivity	Not included
Thought interference	Thought insertion, Thought withdrawal, Thought broadcasting	Thought blocking (some classify here)
Delusions	Delusional perception (perception → delusional meaning)	Delusional intuition (sudden delusional idea, NOT based on perception)
Mood	Not included	Perplexity, depressive changes, euphoric changes
Affect	Not included	Emotional blunting
Specificity for schizophrenia	~70% sensitivity; NOT pathognomonic (found in 10-20% bipolar, TLE, substance-induced)	Very low specificity
Current DSM-5 status	No special diagnostic privilege	Not formally categorized
Current ICD-11 status	No special diagnostic privilege	Not formally categorized

Table 6: Catatonia Features with Bush-Francis Scoring

Bush-Francis Catatonia Rating Scale (BFCRS), 23 items, rated 0-3

FEATURE	DEFINITION	SCORE 0	SCORE 3
Stupor	No psychomotor activity, not relating to environment	Normal responsiveness	Complete unresponsiveness
Mutism	No / minimal verbal response	Normal speech	No speech
Staring	Fixed gaze, poor blinking	Normal eye contact	Fixed stare, non-reactive
Posturing	Spontaneous maintenance of position against gravity	None	Maintained throughout interview
Grimacing	Odd facial expressions maintained	None	Constant
Echopraxia	Mimicking examiner's movements	None	Constant mimicry
Echolalia	Mimicking examiner's speech	None	Constant repetition
Stereotypy	Repetitive non-goal-directed movements	None	Continuous
Mannerisms	Odd purposeful movements	None	Continuous
Verbigeration	Repetition of phrases	None	Continuous
Rigidity	Resistance to passive movement	None	Extreme, unable to reposition
Negativism	Opposition to instructions / stimuli	Normal cooperation	Oppositional to all
Waxy flexibility	Slight resistance to repositioning (like bending candle)	None	Present throughout
Withdrawal	Refusal to eat / drink / eye contact	None	Complete refusal
Excitement	Extreme hyperactivity	None	Severe, purposeless
Impulsivity	Sudden inappropriate actions	None	Continuous
Automatic obedience	Exaggerated cooperation	None	Follows all commands robotically
Mitgehen	Raising arm to slight pressure despite instruction not to	None	Clearly present

FEATURE	DEFINITION	SCORE 0	SCORE 3
Gegenhalten	Resistance proportional to stimulus	None	Present
Ambitendency	Stuck in indecisive movements	None	Present throughout
Grasp reflex	Grasps examiner's hand despite instruction not to	None	Cannot release
Perseveration	Returning to same topic/movement	None	Persistent
Catalepsy	Maintains externally imposed posture	None	Maintains >1 minute

Screening: First 14 items form the screening tool. Score ≥ 2 = positive screen for catatonia.

Lorazepam challenge: 1-2 mg IV → observe 5-15 min → 50%+ improvement = positive.

Table 7: Antipsychotic Side Effects by Receptor

RECEPTOR BLOCKED	SIDE EFFECT	HIGH RISK DRUGS	LOW RISK DRUGS
D2 (nigrostriatal)	EPS: dystonia, akathisia, parkinsonism, TD	Haloperidol, risperidone (high dose), fluphenazine	Clozapine, quetiapine
D2 (tuberoinfundibular)	Hyperprolactinemia → galactorrhea, amenorrhea, sexual dysfunction, osteoporosis	Risperidone, paliperidone, amisulpride, haloperidol	Aripiprazole (partial agonist), quetiapine, clozapine
5-HT_{2C}	Weight gain, insulin resistance	Clozapine, olanzapine	Aripiprazole, ziprasidone, lurasidone
H1 (histamine)	Sedation, weight gain, increased appetite	Clozapine, olanzapine, quetiapine, chlorpromazine	Aripiprazole, haloperidol (less sedating despite other issues)
M1 (muscarinic)	Dry mouth, constipation, urinary retention, blurred vision, cognitive impairment, tachycardia	Clozapine, olanzapine, chlorpromazine, thioridazine	Risperidone, aripiprazole
Alpha-1 (adrenergic)	Orthostatic hypotension, dizziness, sedation, reflex tachycardia	Clozapine, chlorpromazine, quetiapine, iloperidone	Haloperidol, aripiprazole

RECEPTOR BLOCKED	SIDE EFFECT	HIGH RISK DRUGS	LOW RISK DRUGS
M3 (pancreatic)	Direct diabetes risk (independent of weight)	Clozapine, olanzapine	Aripiprazole, ziprasidone

Clinical application: When switching antipsychotics to manage side effects, choose a drug with a different receptor profile targeting the problematic receptor.

Table 8: Clozapine vs Other SGAs

FEATURE	CLOZAPINE	OTHER SGAs
TRS efficacy	ONLY antipsychotic with proven superiority	Not superior to each other in TRS
Anti-suicidal	ONLY drug with FDA-approved indication (InterSePT)	No specific anti-suicidal evidence
D2 binding	Low affinity, fast dissociation	Variable
Receptor profile	Broadest: D1, D2, D4, 5-HT _{2A} , 5-HT _{2C} , H1, M1, M4, alpha-1, alpha-2	Narrower profiles
EPS risk	Lowest of all antipsychotics	Low but present (especially risperidone at high doses)
TD risk	Virtually zero; treats existing TD	Low but possible
Agranulocytosis	~1% (unique)	Not a risk
Monitoring burden	Mandatory blood monitoring (weekly → biweekly → monthly) for life	Standard metabolic monitoring
Metabolic effects	Worst (with olanzapine)	Variable
Seizures	Dose-dependent (3-8%)	Very rare
Myocarditis	1-3% (first 4-6 weeks)	Not a concern
Sialorrhea	Very common (M4 agonism)	Not typical
Constipation/ileus	Serious risk, potentially fatal	Possible but rare
Smoking interaction	Levels decrease 50% (CYP1A2)	Less clinically significant
Use	Last resort but most effective	First/second line
LAI available	No	Yes (risperidone, paliperidone, aripiprazole, olanzapine)

FEATURE	CLOZAPINE	OTHER SGAS
Indication	TRS, suicidality in schizophrenia, treatment-resistant aggression	Broad (schizophrenia, bipolar, MDD augmentation, etc.)

Table 9: Newer Antipsychotics

FEATURE	CARIPRAZINE	LUMATEPERONE	BREXPIRAZOLE	COBENFY
Year approved	2015	2019	2015	2024
Primary mechanism	D3-preferring partial agonist + D2 partial agonist	5-HT _{2A} antagonist + D2 partial agonist + GluN2B + SERT	D2 partial agonist + 5-HT _{1A} partial agonist	M1 / M4 muscarinic agonist (xanomeline) + peripheral antagonist (trospium)
D2 involvement	Partial agonist	Partial agonist at ~40% occupancy	Partial agonist	NONE
Unique selling point	Superior for negative symptoms (RCT evidence)	Effective at sub-standard D2 occupancy; triple mechanism	Less akathisia than aripiprazole	First non-D2 antipsychotic
EPS risk	Moderate (akathisia)	Minimal	Low (less than aripiprazole)	None
Metabolic risk	Low	Minimal	Moderate	None
Prolactin	May decrease	Minimal elevation	May decrease	None
Sedation	Low	Moderate (somnolence)	Low-moderate	Low
Main side effects	Akathisia, EPS, insomnia	Somnolence, dizziness	Weight gain, akathisia	Nausea, dyspepsia, constipation (cholinergic)
Half-life	2-4 weeks (with active metabolites)	~18 hours	~91 hours	~12 hours (xanomeline); trospium ~18 hours
Dose	1.5-6 mg / day	42 mg / day	2-4 mg / day	Xanomeline 50-125mg + trospium 20mg BID
Other indications	Bipolar mania, bipolar depression	Bipolar depression (adjunct / mono)	MDD augmentation, Alzheimer's agitation	Schizophrenia only (currently)

FEATURE	CARIPRAZINE	LUMATEPERONE	BREXPIRAZOLE	COBENFY
Paradigm significance	D3 receptor as therapeutic target	Sub-65% D2 occupancy can be effective	Refined partial agonism	Proves antipsychotic effect without D2 blockade

PYQ Frequency Analysis

EXAM PEARL

Source: PG exams Dec 2011, Jun 2025 + PG exams 2013-2022

Executive Summary

Schizophrenia is the **single highest-yield topic in all of psychiatry exams**. With 49+ mentions across 28 PG exams sessions, it appears in virtually every exam, often multiple times across different papers. You WILL get at least one schizophrenia question. Probably two.

Topic-Level Frequency

TOPIC	EXAM MENTIONS	VERDICT
Management (antipsychotics, psychosocial, rehab)	15+	Every exam
TRS / Clozapine	6	Every 4-5 exams
Negative symptoms	5	Every 5-6 exams
Course and outcome / Prognosis	4	Every 6-7 exams
Classification / Diagnosis (ICD vs DSM, FRS)	4	Every 6-7 exams
Prodrome / Early intervention	3	Emerging
Psychosocial / Rehabilitation	6	Every 4-5 exams
Newer antipsychotics	3	Emerging
Metabolic syndrome	2	Linked to antipsychotics

Must-Prepare PYQ Templates

- "Describe positive and negative symptoms. Management of negative symptoms.", **perennial**
- "Define TRS. Management protocol (pharmacological + psychological).", **perennial**
- "Clozapine, initiation, monitoring, side effects.", **perennial**

4. "Course and outcome of schizophrenia." / "Prognostic factors.", **perennial**
 5. "First rank symptoms. Current status.", **classic**
 6. "Atypical antipsychotics, why atypical? Side effects.", **perennial**
 7. "Psychosocial treatment / rehabilitation in schizophrenia.", **perennial**
 8. "ICD-11 vs DSM-5 diagnosis of schizophrenia.", **emerging**
 9. "Prodromal symptoms. Early intervention.", **emerging**
 10. "Newer antipsychotics. Cariprazine.", **emerging hot topic**
-

Analysis based on PG exams Dec 2011, Jun 2025 + PG exams 2013-2022.

Quick Review

Vignette 1: First Episode Psychosis

Case: A 21-year-old male engineering student is brought by his parents with a 6-month history of declining academic performance, social withdrawal, and suspiciousness. Over the past 2 months, he has been talking to himself, laughing inappropriately, and locking himself in his room claiming that his classmates are plotting against him. He believes his thoughts are being broadcast through his laptop. He has no prior psychiatric history. There is no history of substance use. A paternal uncle was diagnosed with schizophrenia.

Q1. What is the most likely diagnosis? Justify with ICD-11 and DSM-5 criteria.

Answer:

Diagnosis: Schizophrenia, first episode, currently in acute episode.

ICD-11 justification:

- Duration >1 month of symptoms (2 months of frank psychosis)
- Core symptoms present: Persecutory delusions (classmates plotting), Thought broadcasting (FRS), Auditory hallucinations (talking to himself = likely responding to voices)
- Prodromal features for 4 months prior (social withdrawal, functional decline)

DSM-5 justification:

- Criterion A: ≥ 2 symptoms present, delusions, hallucinations, negative symptoms (withdrawal). At least 1 is delusion/hallucination
- Criterion B: Social/occupational dysfunction (academic decline)
- Criterion C: Total duration >6 months (4 months prodrome + 2 months active = 6 months)
- Criterion D: No mood disorder to account for symptoms
- Criterion E: No substance use

Note: Thought broadcasting is an FRS but does not receive special diagnostic weight in either DSM-5 or ICD-11.

Q2. What are the differential diagnoses?

Answer:

1. **Schizophreniform disorder**, if DSM-5 duration criterion not met (<6 months); here 6-month criterion is borderline

2. **Brief psychotic disorder**, duration <1 month; excluded here
3. **Substance-induced psychotic disorder**, cannabis/ stimulant use; excluded by history but urine drug screen warranted
4. **Bipolar disorder with psychotic features**, assess for mood episodes; no prominent mood symptoms here
5. **Delusional disorder**, but hallucinations and disorganization point toward schizophrenia
6. **Organic psychosis**, rule out with physical examination, basic labs, neuroimaging if indicated (first episode, seizures, etc.)
7. **Schizotypal personality disorder**, subthreshold, chronic; the acute psychotic presentation exceeds this

Q3. Outline the management plan.

Answer:

Immediate:

- Comprehensive assessment: MSE, physical examination, collateral from family
- Investigations: CBC, RFT, LFT, TFT, fasting glucose, lipid profile, urine drug screen, ECG
- Neuroimaging: MRI brain (first episode, to rule out organic causes)
- Risk assessment: Suicidality, harm to others, self-care capacity

Pharmacological:

- Start SGA: Risperidone 2 mg/ day or Aripiprazole 10-15 mg/ day (first episode patients respond at lower doses)
- Low starting dose (first-episode patients are medication-naïve and more sensitive)
- Target D2 occupancy 65-80%
- If agitation: Short-term benzodiazepine (lorazepam 1-2 mg)
- Avoid polypharmacy at this stage

Psychosocial:

- Psychoeducation: Patient and family, nature of illness, importance of treatment, prognosis
- Family intervention: Assess expressed emotion, educate about early warning signs
- Supported education: Facilitate return to studies if possible
- CBTp: Once acute symptoms settle
- Relapse prevention planning

Follow-up:

- Weekly initially, then fortnightly
- Metabolic monitoring from baseline

- Adequate trial: 4-6 weeks at therapeutic dose before considering switch
 - Minimum 1-2 years of treatment after first episode (some guidelines suggest longer)
-

Vignette 2: Treatment-Resistant Schizophrenia

Case: A 34-year-old male with a 12-year history of schizophrenia presents with persistent auditory hallucinations (commanding voices) and persecutory delusions despite treatment with risperidone 8 mg/day for 10 weeks followed by olanzapine 20 mg/day for 12 weeks. Medication adherence has been confirmed by family supervision and plasma level monitoring. He is unemployed, socially isolated, and his self-care is poor. He has been hospitalized 5 times in the past 8 years.

Q1. Does this patient meet criteria for treatment-resistant schizophrenia (TRS)? Define TRS.

Answer:

Yes, this patient meets TRS criteria.

TRRIP Working Group Definition (Howes et al., 2017):

1. ≥ 2 adequate antipsychotic trials, Yes (risperidone 8 mg, olanzapine 20 mg, both above 600 mg CPZ equivalents)
2. Adequate dose, Yes (risperidone 8 mg $\approx$ 800 CPZ; olanzapine 20 mg $\approx$ 600 CPZ)
3. Adequate duration, Yes (10 weeks and 12 weeks, both >6 weeks)
4. Adherence confirmed, Yes (family supervision + plasma levels)
5. At least 1 SGA trial, Yes (both are SGAs)
6. Persistent moderate-severe positive symptoms, Yes (commanding voices, persecutory delusions)
7. Significant functional impairment, Yes (unemployed, isolated, poor self-care)

Pseudo-resistance has been excluded (adherence confirmed, adequate doses/durations, no substance use mentioned).

Q2. What is the next step in management?

Answer:

Clozapine trial, the only evidence-based treatment for TRS.

Pre-initiation:

- Baseline: CBC with differential (WBC >3500 , ANC >2000), metabolic panel, LFT, ECG, troponin, CRP, weight/BMI/waist
- Stop/cross-taper olanzapine over 1-2 weeks
- Register with clozapine monitoring service
- Informed consent (patient + family)

Initiation:

- Day 1: 12.5 mg at night
- Increase by 25-50 mg/day over first week
- Increase by 50-100 mg/week from week 2
- Target: 300-450 mg/day
- Check plasma level at steady state: target >350 ng/mL

Monitoring:

- WBC/ANC: weekly for 18 weeks → biweekly for 34 weeks → monthly
- Metabolic: weight monthly; glucose, lipids at 3 months then annually
- Cardiac: troponin/CRP weekly for first 4 weeks (myocarditis screening)
- Bowel function: every visit

Q3. If clozapine alone is insufficient after an adequate trial, what augmentation strategies are available?

Answer:

An adequate clozapine trial = plasma level >350 ng/mL for ≥8 weeks.

STRATEGY	EVIDENCE	DOSE
Amisulpride augmentation	Moderate (RCTs)	400-800 mg/day
Aripiprazole augmentation	Moderate	10-15 mg/day; may also improve metabolic profile
ECT augmentation	Moderate	Bilateral, 12-20 sessions
Lamotrigine	Moderate	200-400 mg/day (glutamate modulation)
Lithium	Low-moderate	Target 0.6-0.8 mEq/L
CBTp	Adjunctive	Reduces distress from residual symptoms

Algorithm: Clozapine monotherapy → Clozapine + amisulpride → Clozapine + ECT → Clozapine + lamotrigine. Throughout: psychosocial interventions (CBTp, family, supported employment, ACT team).

Vignette 3: Catatonia

Case: A 30-year-old female with a known diagnosis of schizophrenia is brought to the emergency department after being found standing motionless in her room for 18 hours. On examination, she is mute, staring into space, with increased tone in all limbs. When the examiner positions her arm in an elevated position, she maintains it for over 2 minutes. She

is not eating or drinking. Temperature is 38.2 C, pulse 118/min, BP 150/95 mmHg. CK is elevated at 1200 IU/L.

Q1. What is the diagnosis? What features of catatonia are described?

Answer:

Diagnosis: Catatonia associated with schizophrenia. Given the fever, autonomic instability, and elevated CK, this is **malignant catatonia**, a life-threatening emergency.

Catatonic features identified:

1. **Stupor**, motionless for 18 hours, not relating to environment
2. **Mutism**, no verbal response
3. **Staring**, fixed gaze
4. **Rigidity**, increased tone in all limbs
5. **Waxy flexibility/Catalepsy**, maintains externally imposed arm position for >2 minutes
6. **Withdrawal**, not eating or drinking

Malignant features: Fever (38.2 C), tachycardia (118), hypertension (150/95), elevated CK (1200), suggestive of autonomic instability and muscle breakdown.

Q2. What is your differential diagnosis?

Answer:

1. **Malignant catatonia**, most likely given the presentation
2. **Neuroleptic Malignant Syndrome (NMS)**, must rule out; is she on antipsychotics? NMS presents with similar features (rigidity, fever, autonomic instability, elevated CK). Lead-pipe rigidity in NMS vs waxy flexibility in catatonia, though overlap exists
3. **Anti-NMDA receptor encephalitis**, young female, psychiatric symptoms, autonomic instability; check anti-NMDA receptor antibodies
4. **Serotonin syndrome**, if on serotonergic medications; clonus, hyperreflexia, diarrhea differentiate
5. **Encephalitis (viral)**, LP, MRI, EEG needed
6. **Metabolic derangement**, DKA, hepatic encephalopathy
7. **Status epilepticus (non-convulsive)**, EEG needed

Q3. Outline emergency management.

Answer:

Immediate (first hour):

- ABCs, IV access, monitoring (cardiac, SpO2, temperature)
- IV fluids for dehydration and rhabdomyolysis prevention
- Stop ALL antipsychotics (if NMS cannot be excluded)

- Investigations: CBC, CMP, CK, LFT, RFT, TFT, troponin, urine myoglobin, blood cultures, urine drug screen, LP (if infection suspected), MRI brain, EEG, anti-NMDA receptor antibodies

Lorazepam challenge test:

- IV lorazepam 1-2 mg
- Observe for 5-15 minutes
- Positive response (improvement in rigidity, movement, speech) confirms catatonia and guides treatment
- If positive: Continue lorazepam 8-24 mg/day in divided IV doses

If lorazepam insufficient or malignant features persist:

- **Emergency bilateral ECT**, daily or alternate day
- ECT is the definitive treatment for malignant catatonia (80-90% response)
- Typically 6-10 sessions

Supportive care:

- DVT prophylaxis (immobility)
- Pressure sore prevention
- Nutritional support (NG tube if needed)
- Temperature management
- Monitor CK, renal function (rhabdomyolysis → AKI)

If NMS confirmed: Dantrolene 1-2.5 mg/kg IV + bromocriptine 2.5 mg TDS + supportive care

Vignette 4: Comorbid Substance Use Disorder

Case: A 26-year-old male was diagnosed with schizophrenia at age 22. He has been smoking cannabis daily since age 17. Over the past year, his psychotic symptoms have worsened despite being on olanzapine 15 mg/day. He frequently misses appointments, his family reports that he sometimes skips medication, and his urine drug screen is positive for cannabis and amphetamines. He was recently involved in a physical altercation in his neighborhood.

Q1. What is the diagnosis? How does substance use impact schizophrenia?

Answer:

Diagnosis: Schizophrenia with comorbid substance use disorder (cannabis use disorder + amphetamine use disorder).

Impact of substance use on schizophrenia:

DOMAIN · EFFECT

Symptom exacerbation Cannabis and amphetamines directly worsen psychotic symptoms via dopaminergic enhancement

Treatment resistance Persistent substance use is a major cause of pseudo-resistance; may not respond despite adequate antipsychotic doses

Non-adherence Substance use strongly associated with medication non-adherence

Relapse Significantly increases relapse risk and hospitalization

Violence Substance use (especially stimulants) increases aggression risk in psychosis

Cognitive decline Additive cognitive impairment

Earlier onset Cannabis use in adolescence associated with 2-3 years earlier onset

Prognosis Poorer overall outcome across all domains

Q2. What are the differential diagnoses to consider?

Answer:

1. **Substance-induced psychotic disorder**, cannabis and amphetamine use can independently cause psychosis; however, persistent psychosis between periods of use + premorbid onset + 4-year history suggests primary schizophrenia with comorbid SUD
2. **Pseudo-resistance**, apparent treatment failure due to non-adherence + active substance use (NOT true TRS)
3. **Bipolar disorder with psychotic features + SUD**, amphetamine use may mimic mania
4. **Antisocial personality traits**, aggression + substance use; comorbid personality assessment needed

Q3. Outline a management plan.

Answer:

Immediate:

- Comprehensive assessment: substance use history (type, quantity, frequency, route, last use), psychiatric symptoms during periods of abstinence (if any), risk assessment (violence, self-harm)
- Urine drug screen confirmation and monitoring

Pharmacological:

- Address non-adherence FIRST, consider LAI (paliperidone palmitate or aripiprazole monohydrate)
- Avoid olanzapine if non-adherent (oral only); switch to LAI

- If continued psychosis despite abstinence + LAI: escalate as per TRS pathway
- Clozapine consideration if true TRS after establishing abstinence and adherence
- Clozapine may specifically reduce substance use in schizophrenia patients (some evidence)

Substance use treatment (integrated dual-diagnosis approach):

- Motivational interviewing for substance use
- Psychoeducation about cannabis / amphetamine effects on psychosis
- Harm reduction if abstinence not immediately achievable
- Consider referral to dual-diagnosis program
- No specific pharmacotherapy for cannabis use disorder; for amphetamine use disorder, no approved pharmacotherapy but behavioral approaches effective

Psychosocial:

- Integrated dual-diagnosis treatment (IDDT) model
- ACT team if available (for complex, high-service-use patients)
- Family intervention (high EE likely)
- Anger management
- Social skills training
- Supported employment when stable

Vignette 5: Elderly-Onset Psychosis

Case: A 62-year-old retired schoolteacher presents with a 3-month history of believing that her neighbors are stealing her belongings and poisoning her food. She hears voices of the neighbors planning against her. She lives alone after her husband died 2 years ago. She has well-controlled hypertension and type 2 diabetes. There is no prior psychiatric history. Cognitive screening (MMSE) is 27 / 30. MRI brain shows mild age-related atrophy.

Q1. What is the most likely diagnosis? What differentials should be considered in late-onset psychosis?

Answer:

Most likely diagnosis: Late-onset schizophrenia (sometimes called "late paraphrenia" historically, though this term is no longer in standard use). Alternatively, very late-onset schizophrenia-like psychosis (onset >60 years) as per the international consensus (Howard et al., 2000).

Differentials in late-onset psychosis:

DIFFERENTIAL · KEY FEATURES TO ASSESS

Dementia with psychotic features MMSE is 27/30, mild impairment could indicate early dementia; follow up with detailed neuropsychological testing (ACE-III, MoCA); Lewy body dementia particularly associated with visual hallucinations

Delirium Fluctuating consciousness, acute onset, identifiable precipitant; check for UTI, medications, metabolic derangement

Medication-induced psychosis Review medication list (steroids, anticholinergics, dopamine agonists)

Delusional disorder Systematized, non-bizarre delusions without prominent hallucinations; here hallucinations are present

Psychotic depression Bereavement context (husband died 2 years ago); assess for depressive symptoms, nihilistic/guilt delusions

Substance use Alcohol, OTC medications

Medical causes Hypothyroidism, B12 deficiency, neurosyphilis, cerebrovascular disease, brain tumor

Charles Bonnet syndrome Visual hallucinations in visually impaired elderly; usually with insight

Q2. How does late-onset schizophrenia differ from early-onset?

Answer:

FEATURE	LATE-ONSET (>40 YEARS)	EARLY-ONSET (<40 YEARS)
Sex	Female predominant	Male slightly predominant
Family history	Less likely	More likely
Premorbid functioning	Better	Worse
Symptom profile	Paranoid delusions + auditory hallucinations predominant	Full range of positive + negative + cognitive
Negative symptoms	Less prominent	More prominent
Cognitive decline	Less severe (though present)	More prominent
Structural brain changes	Less	More (enlarged ventricles, cortical atrophy)
Response to treatment	Good, at lower doses	Variable
Social isolation	Common precipitant	Common consequence

FEATURE	LATE-ONSET (>40 YEARS)	EARLY-ONSET (<40 YEARS)
Sensory deficits	Common (hearing loss may contribute)	Not typical

Q3. How would you manage this patient?

Answer:

Assessment:

- Detailed cognitive assessment (neuropsychological battery, not just MMSE)
- Sensory assessment (hearing, vision, sensory deprivation can precipitate/ maintain paranoia)
- Full medical workup: TFT, B12, folate, RPR/ VDRL, HIV, CRP, CBC, LFT, RFT, HbA1c
- Review all medications for psychotogenic potential
- Assess social isolation, bereavement, loneliness

Pharmacological:

- Start LOW, go SLOW, elderly are more sensitive to antipsychotics
- Risperidone 0.5-2 mg/ day or Aripiprazole 5-10 mg/ day (lower dose range)
- Avoid olanzapine (metabolic burden on existing diabetes)
- Avoid quetiapine at high doses (orthostatic hypotension fall risk)
- Monitor for metabolic effects (already has DM and HTN)
- QTc monitoring (cardiac risk)
- Fall risk assessment

Psychosocial:

- Address social isolation (day center, social group, befriending service)
- Bereavement support/ grief counseling if unresolved
- Hearing aid if hearing impairment detected
- Family/ neighbor engagement for support
- CBTp adapted for elderly
- Home-based care if possible

Considerations:

- Cerebrovascular risk: antipsychotics in elderly increase stroke risk (black box warning for dementia, relevant consideration here)
 - Falls risk: any sedating or hypotension-causing medication is dangerous
 - Follow up cognition: if MMSE declines, consider prodromal dementia with psychosis
-

Vignette 6: Negative-Symptom-Dominant Presentation

Case: A 28-year-old male is brought by his mother who reports that over the past 3 years, he has become progressively apathetic, stopped bathing, quit his job, has no friends, watches TV all day, and shows no emotional response even when family members are distressed. He rarely speaks beyond monosyllables. He had one psychotic episode at age 23 with persecutory delusions and auditory hallucinations, which resolved with treatment. He is currently on risperidone 4 mg/day. There are no current delusions or hallucinations.

Q1. What is the primary clinical issue? How would you classify these negative symptoms?

Answer:

Primary issue: Predominant negative symptoms in the residual phase of schizophrenia.

Negative symptoms present:

- Avolition (quit job, apathetic, watches TV all day)
- Affective flattening (no emotional response)
- Alogia (monosyllabic speech)
- Asociality (no friends)
- Poor self-care (stopped bathing)

Classification, Primary vs Secondary:

Must systematically assess whether these are primary (intrinsic to illness) or secondary (treatable cause):

POTENTIAL SECONDARY CAUSE · ASSESSMENT

EPS from risperidone Check for bradykinesia, rigidity (drug-induced parkinsonism can mimic negative symptoms)

Depression Screen for subjective sadness, guilt, hopelessness, sleep/appetite changes (PHQ-9)

Positive symptoms Currently absent, not driving withdrawal

Environmental understimulation Spending all day at home, no structured activity

Substance use Screen for cannabis (amotivational syndrome)

Medical TFT, B12, anemia

If all secondary causes excluded: **Deficit syndrome** (Carpenter), primary, enduring negative symptoms present during and between episodes. Prevalence: 15-25% of schizophrenia. Worst prognosis subgroup.

Q2. What changes would you make to medication?

Answer:

Step 1: Address potential secondary causes

- Reduce risperidone to 2 mg (minimum effective dose for relapse prevention), may reduce EPS-mediated negative symptoms
- OR switch to aripiprazole 10-15 mg (D2 partial agonist, less EPS, less prolactin, potentially activating)

Step 2: If primary negative symptoms persist after addressing secondary causes

- **Cariprazine 3-6 mg/day**, strongest RCT evidence for predominant negative symptoms (D3-preferring partial agonist)
- OR **Amisulpride 50-200 mg/day**, low-dose preferential presynaptic D2/D3 blockade
- Consider add-on antidepressant (mirtazapine 15-30 mg or fluoxetine 20 mg) for motivational / anhedonic overlap

Step 3: Monitor response over 8-12 weeks

- Use standardized assessment (BNSS or SANS) at baseline and follow-up

Q3. What psychosocial interventions would you recommend?

Answer:

1. **Behavioral activation** (adapted from CBTp), activity scheduling, graded task assignment, goal-setting
2. **Social skills training**, role-playing social interactions, conversation skills
3. **Cognitive remediation**, computer-based exercises for attention and executive function (best when combined with vocational rehab)
4. **Supported employment (IPS)**, structured activity and purpose; shown to improve motivation
5. **Art/music therapy**, non-verbal engagement, emotional expression
6. **Physical exercise program**, 150 min / week; evidence for cognitive improvement + motivation
7. **Family intervention**, educate family about negative symptoms (they are NOT laziness); reduce critical comments; prevent over-accommodation
8. **Environmental enrichment**, day program, social club, structured daily routine

Vignette 7: Metabolic Complications

Case: A 38-year-old female with a 10-year history of schizophrenia has been stable on olanzapine 20 mg / day for the past 4 years with good symptom control. She now weighs 98 kg (BMI 37), her waist circumference is 104 cm, fasting glucose is 142 mg / dL, HbA1c is

7.2%, triglycerides are 280 mg/dL, HDL is 38 mg/dL, and BP is 138/88 mmHg. She is concerned about her weight but is fearful of changing medication as she remembers her psychotic relapses.

Q1. What is the diagnosis? How many criteria for metabolic syndrome does she meet?

Answer:

Diagnosis: Metabolic syndrome secondary to olanzapine use, with new-onset type 2 diabetes mellitus.

ATP III criteria assessment:

CRITERION	HER VALUE	THRESHOLD	MET?
Waist circumference	104 cm	>88 cm (F)	YES
Triglycerides	280 mg/dL	≥150 mg/dL	YES
HDL	38 mg/dL	<50 mg/dL (F)	YES
Blood pressure	138/88	≥130/85 mmHg	YES
Fasting glucose	142 mg/dL	≥100 mg/dL	YES

She meets ALL 5 of 5 criteria, severe metabolic syndrome.

Additionally: HbA1c 7.2% confirms Type 2 Diabetes Mellitus (>6.5%).

Q2. What are the management options?

Answer:

Approach 1: Switch antipsychotic (preferred if feasible)

- Switch from olanzapine to a metabolically safer antipsychotic
- Options: Aripiprazole (best metabolic profile), Lurasidone (weight-neutral), Ziprasidone (weight-neutral), Cariprazine (low metabolic risk)
- Cross-titration strategy: Gradually increase new drug while slowly decreasing olanzapine over 4-6 weeks
- Close monitoring for psychotic relapse during switch
- Discuss risks vs benefits with patient, her fear of relapse is valid and must be addressed through shared decision-making

Approach 2: Add metformin (if switching not possible/patient refuses)

- Metformin 500 mg BD → 1000 mg BD
- Evidence: Reduces weight by 2-3 kg, improves insulin sensitivity
- Also directly addresses diabetes

Approach 3: Add aripiprazole to olanzapine

- Aripiprazole 5-10 mg/day added to olanzapine may improve metabolic parameters
- Evidence: Moderate; meta-analysis supports modest benefit

Metabolic management regardless of approach:

- Diabetes: Metformin +/- other antidiabetics; endocrine referral
- Dyslipidemia: Statin (atorvastatin); lifestyle modification
- Hypertension: Lifestyle first; if persistent, antihypertensive
- Lifestyle: Structured diet counseling, exercise program (150 min/week), smoking cessation if applicable
- Monitoring: Weight monthly, fasting glucose/HbA1c quarterly, lipids quarterly until stable

Q3. If you decide to switch to aripiprazole, how would you manage the transition?

Answer:

Cross-titration protocol:

WEEK	OLANZAPINE	ARIPIPRAZOLE
1	20 mg (continue)	5 mg
2	15 mg	10 mg
3	10 mg	15 mg
4	5 mg	20 mg
5	Stop	20-30 mg

Key considerations:

- Monitor for insomnia/akathisia (common with aripiprazole, especially activating early on)
- Monitor for psychotic relapse (weekly check-ins during transition)
- Olanzapine withdrawal symptoms: Insomnia, nausea, diaphoresis (cholinergic rebound), taper slowly
- If psychotic symptoms re-emerge during switch: Slow down taper, consider partial switch (lower olanzapine + aripiprazole)
- Family psychoeducation about the switch and early warning signs
- Reassure patient: The switch is being done carefully for her long-term health

Vignette 8: Clozapine Management

Case: A 40-year-old male with treatment-resistant schizophrenia has been on clozapine 450 mg/day for 2 years with good control of positive symptoms (plasma level: 420 ng/mL). He

smokes 20 cigarettes/day. He is admitted to hospital for an elective hernia surgery where smoking is not permitted. On post-operative day 2, he becomes excessively drowsy, drooling profusely, has a tonic-clonic seizure, and his blood pressure is 85/55 mmHg.

Q1. What is the most likely cause of this acute deterioration?

Answer:

Diagnosis: Clozapine toxicity secondary to abrupt smoking cessation.

Mechanism:

- Tobacco smoke (specifically polycyclic aromatic hydrocarbons, NOT nicotine) induces CYP1A2
- CYP1A2 is the primary enzyme metabolizing clozapine
- Abrupt cessation of smoking removes CYP1A2 induction
- Within 2-4 days, clozapine levels can DOUBLE
- His therapeutic level of 420 ng/mL may have risen to 700-800+ ng/mL
- Dose-dependent toxicity: seizures (>600 ng/mL risk increases significantly), sedation, hypotension, sialorrhea

Evidence supporting this diagnosis:

- Temporal relationship: Admitted → stopped smoking → day 2 symptoms
- Symptoms consistent with clozapine toxicity: excessive sedation, seizure (dose-dependent), hypotension, profuse drooling
- Known smoker (20/day) with CYP1A2 induction now removed

Q2. How should this be managed acutely?

Answer:

Immediate:

1. **Seizure management:** Lorazepam 2-4 mg IV; protect airway; do NOT give phenytoin (may interact)
2. **Hypotension:** IV fluids; avoid vasopressors initially; position patient flat
3. **Urgent clozapine level:** Confirm toxicity (expect >600 ng/mL)
4. **Hold clozapine** until seizure resolved and levels obtained
5. **Monitor:** Continuous cardiac monitoring, pulse oximetry, neurological observations

Once stable:

1. **Reduce clozapine dose by 30-50%:** From 450 mg → 225-300 mg/day
2. **Recheck plasma level** after dose reduction and once at new steady state (5-7 days)
3. **Target level:** 350-600 ng/mL

4. **Consider valproate** for seizure prophylaxis if maintaining dose >300 mg (dose-dependent seizure risk)

5. **Sialorrhea management:** Glycopyrrolate or hyoscine while profuse

Prevention for future:

- If patient resumes smoking after discharge: Will need dose RE-INCREASE (as CYP1A2 re-induced)
- Educate patient and surgical team about clozapine-smoking interaction
- NRT (nicotine replacement therapy) does NOT prevent clozapine level changes, it is the smoke, not the nicotine
- Document prominently in medical records

Q3. What other drug interactions with clozapine should clinicians be aware of?

Answer:

DRUG	EFFECT ON CLOZAPINE LEVEL	MECHANISM	CLINICAL ACTION
Fluvoxamine	Increases 2-5x	CYP1A2 inhibition	Reduce clozapine dose 50-75%; use for deliberate level-boosting if needed
Ciprofloxacin	Increases 1.5-2x	CYP1A2 inhibition	Use alternative antibiotic; if must use, reduce clozapine dose
Caffeine	Increases modestly	CYP1A2 inhibition	Monitor if patient changes caffeine intake dramatically
Carbamazepine	Decreases + agranulocytosis risk	CYP3A4 induction + bone marrow	CONTRAINDICATED combination
Valproate	May slightly decrease levels	Unclear mechanism	Monitor; used for seizure prophylaxis
Phenytoin	Decreases levels	CYP3A4 induction	Avoid if possible
Oral contraceptives	May increase levels	CYP1A2 inhibition	Monitor when starting/stopping
Omeprazole	May decrease levels	CYP1A2 induction	Monitor if starting/stopping

Vignette 9: Depot/LAI Considerations

Case: A 29-year-old male with schizophrenia has had 4 psychotic relapses in 3 years, each preceded by medication non-adherence confirmed by undetectable plasma levels. He

currently responds well to oral paliperidone 9 mg/day when he takes it consistently. His family is exhausted from trying to supervise his medication. He has poor insight and frequently says he does not need medication because "there is nothing wrong" with him.

Q1. What is the main barrier to treatment success? What intervention would you recommend?

Answer:

Main barrier: Medication non-adherence driven by poor insight (anosognosia).

Anosognosia is present in ~50% of schizophrenia patients and is NOT simply "denial", it is a neurobiological deficit (associated with right frontal lobe dysfunction).

Recommended intervention: Switch to Long-Acting Injectable (LAI) antipsychotic, specifically **Paliperidone Palmitate 1-monthly (PP1M)**, since he is already stable on oral paliperidone.

Rationale for LAI:

- Eliminates daily adherence decisions
- Transparent adherence (clinician/family knows when injection was given)
- Reduces relapse by 20-30% compared to oral equivalent
- Steady plasma levels (fewer peaks and troughs)
- Reduces caregiver burden
- Evidence that early LAI use (within first 2 years) improves long-term outcomes

Q2. Describe the initiation protocol for paliperidone palmitate.

Answer:

PP1M (1-monthly) initiation:

DAY	DOSE	SITE	NOTES
Day 1	234 mg (150 mg eq.)	Deltoid ONLY	Loading dose
Day 8 (±4 days)	156 mg (100 mg eq.)	Deltoid ONLY	Second loading dose
Month 2 onwards	117 mg (75 mg eq.) monthly	Deltoid OR Gluteal	Maintenance; adjust 39-234 mg based on response

- No oral supplementation needed (loading doses provide therapeutic levels)
- Can discontinue oral paliperidone after Day 1 injection
- Adjust maintenance dose based on response and tolerability
- If previous oral dose was 6 mg → maintenance 117 mg; if 9 mg → 156 mg; if 12 mg → 234 mg

Once stable on PP1M for ≥4 months, can transition to PP3M:

- PP3M dose = 3.5x the PP1M dose
- Example: PP1M 156 mg → PP3M 546 mg every 3 months

Eventually, PP6M:

- After adequate period on PP1M/PP3M
- PP6M 1092 mg every 6 months

Q3. How would you address the patient's poor insight?

Answer:

Pharmacological: LAI itself removes the daily insight-dependent decision.

Psychoeducation:

- Avoid direct confrontation about "being ill" (increases resistance)
- Motivational interviewing approach: explore discrepancy between patient's goals and current situation
- Reframe medication as "helping you stay out of hospital" rather than "because you are sick"
- Use relapse history: "Every time you stopped, you ended up in hospital, what was that like?"

Psychosocial:

- CBTp with specific focus on illness beliefs and health beliefs
- LEAP approach (Amador): Listen, Empathize, Agree (on shared goals), Partner
- Peer support from others with lived experience
- Family education about anosognosia (it's NOT willful denial)

Legal considerations (if insight remains absent and repeated relapses):

- Community Treatment Order (CTO) where legally available
- Supported decision-making
- Crisis plan and advance directives (when patient has partial insight)

Vignette 10: Forensic Considerations

Case: A 35-year-old male with paranoid schizophrenia was found standing over his neighbor's body holding a knife. He claims the neighbor was an alien who was going to abduct him. He has a 10-year history of schizophrenia with multiple admissions. His last psychiatric visit was 6 months ago; he stopped olanzapine 4 months ago. Urine drug screen is positive for methamphetamine.

Q1. What psychiatric and forensic issues need to be addressed?

Answer:

Psychiatric issues:

1. Acute psychotic episode with persecutory and bizarre delusions (neighbor = alien, abduction)
2. Medication non-adherence (stopped olanzapine 4 months ago)
3. Comorbid substance use (methamphetamine, worsens psychosis, increases violence risk)
4. Risk assessment: Has committed serious violence; ongoing risk while psychotic
5. Capacity assessment: Likely lacks capacity for treatment decisions currently

Forensic issues:

1. **Criminal responsibility:** Was the act committed under influence of psychotic symptoms? Assess for M'Naughtan rules applicability (did he know the nature and quality of his act? Did he know it was wrong?)
2. **Mental state at the time of offense:** Detailed assessment needed; collateral from witnesses; timeline of symptoms
3. **Fitness to stand trial:** Can he understand proceedings, instruct a lawyer?
4. **Section/involuntary admission:** Required for treatment (lacks insight, danger to others)
5. **Indian legal framework:** Section 84 IPC (now BNS Section 22), Act of a person of unsound mind; Mental Healthcare Act 2017, provisions for supported admission
6. **Substance intoxication vs psychosis:** Methamphetamine may have precipitated / worsened psychosis but underlying schizophrenia is the primary disorder

Q2. How would you assess his mental state at the time of the alleged offense?**Answer:****Retrospective assessment:**

- Detailed psychiatric interview (once stable enough)
- Timeline: When did he stop medication? When did symptoms worsen? What was his mental state in days/hours before the event?
- Nature of delusions: Were they commanding? Did he believe he was acting in self-defense against the "alien"?
- Substance use: When was the last use? What quantity? Does methamphetamine independently explain the behavior?
- Previous violence: Any pattern of violence during psychotic episodes?
- Collateral: Family, neighbors, previous psychiatrist, hospital records
- Physical evidence: Crime scene details, toxicology at time of arrest

Assessment of M'Naughtan criteria (Indian law, Section 84 IPC / BNS Section 22):

1. Did he know the NATURE of the act? (Did he know he was stabbing a person?), He may have known the physical act but believed the "person" was an alien

2. Did he know it was WRONG?, If he genuinely believed he was defending against an alien abduction, he may not have known it was wrong

Tools: HCR-20 (violence risk), PCL-R (if personality features), detailed MSE documentation

Q3. How would you manage this patient going forward?

Answer:

Acute management:

- Involuntary admission under Mental Healthcare Act 2017
- Restart antipsychotic, consider clozapine (TRS likely given multiple admissions + violence + non-adherence)
- Address methamphetamine: Detoxification, psychoeducation, relapse prevention
- 24/7 observation initially (continued violence risk)
- Physical examination and investigations

Medium-term:

- Stabilize on medication (clozapine if TRS; otherwise LAI to prevent future non-adherence)
- LAI strongly indicated: Paliperidone palmitate or zuclopenthixol decanoate
- Substance use treatment (integrated dual-diagnosis)
- Violence risk management (structured professional judgment, HCR-20)
- Fitness to stand trial assessment once stable
- Prepare psychiatric report for court

Long-term:

- Forensic community mental health follow-up
- Mandatory LAI (possibly via CTO)
- ACT team if available
- Substance use monitoring
- Family intervention
- Violence risk monitoring
- Supervised housing if no family support

Vignette 11: OCD Comorbidity with Schizophrenia

Case: A 32-year-old female with schizophrenia has been on clozapine 400 mg/day for 2 years with good control of psychotic symptoms. Over the past 8 months, she has developed repetitive handwashing (30+ times/day, hands are cracked and bleeding), checking rituals (locks, gas stove, 20+ minutes each), and distressing intrusive thoughts about

contamination. She recognizes these as excessive. Y-BOCS score is 28 (severe). She had no OCD symptoms before starting clozapine.

Q1. What is the likely diagnosis and its relationship to clozapine?

Answer:

Diagnosis: Clozapine-induced obsessive-compulsive symptoms (OCS) / OCD.

Relationship to clozapine:

- Prevalence: Clozapine-associated OCS occurs in 20-30% of patients; full OCD criteria in 5-10%
- Mechanism: 5-HT_{2A} and 5-HT_{2C} blockade by clozapine → serotonergic dysregulation → OC symptoms
- Dose-dependent: Higher clozapine doses associated with higher risk
- Temporal relationship: Symptoms developed after clozapine initiation; absent before
- She has good insight (recognizes symptoms as excessive), typical of OCS/OCD

Distinguishing from psychotic symptoms:

- OCD: Ego-dystonic, recognized as excessive, causes anxiety, resisted
- Delusions/psychotic rituals: Ego-syntonic, not resisted, no insight
- Her symptoms are clearly OCD (distressing, recognized as excessive)

Q2. How would you manage the OCD symptoms?

Answer:

Step 1: Do NOT stop clozapine, she is treatment-resistant and clozapine has controlled her psychosis. Stopping would risk psychotic relapse.

Step 2: Pharmacological options:

OPTION	DETAILS	CAUTION
Fluvoxamine (first choice)	50-200 mg/day; strongest evidence for clozapine-OCS	Potent CYP1A2 inhibitor, WILL increase clozapine levels 2-5x; MUST reduce clozapine dose by 50-75% and recheck levels
Aripiprazole add-on	5-10 mg/day	Partial D2 agonism may counteract clozapine's pro-OCD effect; some evidence
Sertraline/fluoxetine	Standard SSRI doses	Weaker CYP1A2 interaction than fluvoxamine; may be safer
CBT (ERP)	Exposure and Response Prevention	Should be offered alongside pharmacotherapy

Step 3: If adding fluvoxamine:

- Start fluvoxamine 25 mg/day
- Simultaneously reduce clozapine from 400 mg to 200 mg
- Monitor clozapine plasma level at 1-2 weeks
- Titrate fluvoxamine to 100-150 mg based on OCD response
- Target clozapine level: 350-600 ng/mL (may be achieved at lower clozapine dose due to interaction)

Step 4: CBT for OCD

- Exposure and Response Prevention (ERP), gold standard
- May need modification for patients with schizophrenia (simpler hierarchies, slower pace, repeated practice)

Q3. What clozapine dose adjustment is needed if fluvoxamine is started?

Answer:

Critical interaction: Fluvoxamine is the most potent CYP1A2 inhibitor among SSRIs. Adding it to clozapine without dose adjustment can cause clozapine toxicity (seizures, sedation, hypotension, agranulocytosis risk).

Protocol:

1. Before starting fluvoxamine: Check baseline clozapine level
2. Reduce clozapine dose by 50-75% (e.g., 400 mg → 100-200 mg)
3. Start fluvoxamine at low dose (25 mg)
4. Recheck clozapine level after 1-2 weeks
5. Target clozapine level 350-600 ng/mL
6. Titrate fluvoxamine slowly to effective dose for OCD (100-200 mg)
7. Repeat clozapine level after each fluvoxamine dose change
8. Monitor for toxicity signs: excessive sedation, seizures, hypotension, tachycardia

Note: This interaction can also be used therapeutically, in patients who are rapid metabolizers or smokers with low clozapine levels, adding low-dose fluvoxamine can deliberately boost clozapine levels ("fluvoxamine augmentation strategy").

Vignette 12: First-Episode Psychosis with Catatonic Features

Case: An 18-year-old female college student is brought to emergency by her roommate. Over 2 weeks, she became increasingly withdrawn, stopped attending classes, and was heard muttering to herself. Today, she was found lying in bed, unresponsive, maintaining her arms in odd positions. She is not speaking, not eating, and alternates between complete immobility and sudden bursts of purposeless agitation. No prior psychiatric history. Drug screen negative. No significant medical history.

Q1. What is the clinical presentation and likely diagnosis?

Answer:

Clinical presentation: First episode of psychosis with prominent catatonic features (retarded type with episodes of excited catatonia).

Catatonic features:

- Stupor (unresponsive, lying in bed)
- Mutism (not speaking)
- Posturing (maintaining arms in odd positions)
- Withdrawal (not eating)
- Excitement (sudden purposeless agitation)
- Possible alternation between retarded and excited catatonia

Psychotic features (preceding catatonia):

- Auditory hallucinations (muttering to herself)
- Social withdrawal (prodromal / active phase)
- Functional decline

Likely diagnosis: Schizophrenia, first episode with catatonia specifier (DSM-5) OR Schizophrenia with psychomotor disturbance qualifier (ICD-11).

Differentials:

1. Bipolar disorder with catatonia, assess for any mood episode history
2. Anti-NMDA receptor encephalitis, young female, psychiatric symptoms + catatonia (must rule out!)
3. Autoimmune encephalitis, antibody panel
4. Medical catatonia, drug screen negative but consider other causes
5. Brief psychotic disorder with catatonia

Q2. What investigations are essential in this case?

Answer:

Priority investigations:

INVESTIGATION · RATIONALE

Anti-NMDA receptor antibodies Young female + first episode psychosis + catatonia, MUST rule out; 70% of anti-NMDA receptor encephalitis cases initially present to psychiatry

MRI brain First episode; rule out structural lesion, encephalitis

EEG Rule out non-convulsive status epilepticus; anti-NMDA encephalitis shows extreme delta brush pattern

LP (CSF analysis) If encephalitis suspected; cell count, protein, glucose, oligoclonal bands, antibody panel

CBC, CMP, TFT, CRP Baseline + rule out metabolic/infectious causes

CK Elevated in malignant catatonia; rhabdomyolysis risk

Urine drug screen Already negative but confirm

Autoimmune panel ANA, anti-dsDNA if autoimmune encephalitis suspected

Ovarian ultrasound Anti-NMDA receptor encephalitis associated with ovarian teratoma in young women

Q3. How would you manage her?

Answer:

Immediate:

1. Admit; continuous monitoring
2. IV fluids, nutrition (NG tube if prolonged mutism/refusal)
3. DVT prophylaxis
4. Pressure sore prevention

Catatonia treatment:

1. **Lorazepam challenge:** 2 mg IV → observe 15 minutes
2. If positive: Lorazepam 8-24 mg/day IV in divided doses
3. If no response to lorazepam within 48-72 hours or if malignant features develop: **ECT**, bilateral, daily or alternate day

Once catatonia resolves and organic causes excluded:

- Start antipsychotic cautiously (catatonia can worsen with high-potency antipsychotics)
- Avoid haloperidol (can worsen catatonia)
- Use low-dose SGA: Olanzapine 5-10 mg or quetiapine 200-300 mg (lower D2 binding preferred)
- Continue benzodiazepine initially alongside antipsychotic

If anti-NMDA receptor encephalitis confirmed:

- First-line: IV methylprednisolone + IVIG or plasmapheresis
- Second-line: Rituximab, cyclophosphamide

- Tumor resection if teratoma found
 - Antipsychotics for symptom management (low-dose, preferably SGA)
-

Vignette 13: Schizophrenia with Pregnancy

Case: A 30-year-old female with stable paranoid schizophrenia on risperidone 4 mg/day discovers she is 8 weeks pregnant. This is a planned pregnancy discussed with her psychiatrist 6 months ago. She has had 2 psychotic episodes, the last being 3 years ago. She is currently symptom-free and functioning well. She is anxious about medication effects on the baby.

Q1. What are the key considerations regarding antipsychotic use in pregnancy?

Answer:

Risk-benefit analysis:

RISK OF CONTINUING MEDICATION · RISK OF STOPPING MEDICATION

Teratogenicity (generally low with SGAs) Psychotic relapse (50%+ risk in pregnancy if untreated)

Gestational diabetes (olanzapine > others) Risk to mother and fetus during psychosis

Neonatal complications (sedation, EPS in neonate) Poor prenatal care, nutrition, substance use during psychosis

Long-term neurodevelopmental effects (limited data) Harm to self/fetus, hospitalization, forced treatment

Key facts about antipsychotics in pregnancy:

- No antipsychotic is FDA category A (none proven safe)
- Most data on chlorpromazine, haloperidol (decades of use); SGAs, growing data but less
- No consistent pattern of major malformations with any antipsychotic
- Olanzapine and clozapine: highest risk of gestational diabetes
- Risperidone: Relatively well-studied; generally considered acceptable in pregnancy
- **General principle: The risk of untreated psychosis in pregnancy almost always outweighs the risk of medication**

Q2. What would you advise regarding her current medication?

Answer:

Recommendation: Continue risperidone at the lowest effective dose.

Rationale:

- She has had 2 previous episodes (relapse risk is high)

- Currently stable, do NOT change medication during pregnancy (avoid switches that add risk)
- Risperidone has a reasonable safety profile in pregnancy (Huybrechts et al., 2016)
- Dose reduction: Consider lowering to 3 mg if possible (minimum effective dose)
- Avoid adding/switching medications (each new drug adds unknown teratogenic risk)

Monitoring plan:

TRIMESTER · MONITORING

First Confirm medication continuation; folic acid; dating scan; glucose screening

Second Anomaly scan (18-20 weeks); glucose tolerance test; psychiatric review monthly

Third Growth scans; psychiatric review every 2 weeks; plan for delivery; neonatal team alert

Peripartum Continue medication; monitor for postpartum psychotic relapse (high risk); breastfeeding counseling

Neonatal Observe for neonatal adaptation syndrome (sedation, feeding difficulties, respiratory issues, EPS), usually self-limiting

Q3. What about breastfeeding?

Answer:

Risperidone and breastfeeding:

- Risperidone and its active metabolite (paliperidone) are excreted in breast milk
- Relative infant dose: ~4-5% (generally <10% considered acceptable)
- Paliperidone may be preferred over risperidone (lower milk-to-plasma ratio)
- Most guidelines: Breastfeeding can be considered with monitoring

General principles:

- Benefits of breastfeeding (bonding, nutrition, immunity) vs drug exposure
 - Shared decision-making with mother
 - If breastfeeding: Monitor infant for sedation, feeding difficulties, developmental milestones
 - Avoid polypharmacy during breastfeeding
 - Clozapine: Generally NOT recommended during breastfeeding (agranulocytosis risk to infant; high milk transfer)
 - Olanzapine: Limited transfer; considered compatible by many guidelines
-

Vignette 14: Tardive Dyskinesia

Case: A 55-year-old male with chronic schizophrenia has been on haloperidol 15 mg/day for 20 years with good symptom control. Over the past year, his family noticed repetitive lip-smacking, tongue protrusion, and jaw movements. He also makes purposeless hand movements. He is unaware of these movements. An attempt to reduce haloperidol led to worsening of these movements and re-emergence of psychotic symptoms.

Q1. What is the diagnosis? Describe the features.

Answer:

Diagnosis: Tardive Dyskinesia (TD) secondary to chronic haloperidol use.

Features described:

- Orofacial movements: lip-smacking, tongue protrusion, jaw movements (classic TD triad)
- Choreiform hand movements
- Involuntary, purposeless, repetitive
- Patient unaware (common in TD, impaired awareness)
- Worsened on dose reduction (unmasking phenomenon)

TD characteristics:

- Onset: After months-years of antipsychotic use (typically >3 months in adults, >1 month in elderly)
- Risk: ~5% per year with FGAs; cumulative risk 25-30% after 5 years; lower with SGAs (~1%/year)
- Irreversible in 50-70% of cases
- Risk factors: Older age, female sex, longer duration/higher dose of FGA, diabetes, mood disorder, early EPS
- Assessment: Abnormal Involuntary Movement Scale (AIMS), standard monitoring tool
- Pathophysiology: D2 receptor supersensitivity from chronic blockade

Q2. How would you manage the tardive dyskinesia?

Answer:

Step 1: Switch from haloperidol (FGA) to SGA

- Cross-taper to quetiapine (least D2 blockade among common SGAs) or clozapine (only antipsychotic that can treat TD)
- Clozapine is the preferred switch if psychotic symptoms are difficult to control (he relapsed on dose reduction)
- Slow cross-taper to minimize psychotic relapse

Step 2: VMAT2 inhibitors (FDA-approved for TD)

DRUG	DOSE	MECHANISM	SIDE EFFECTS
Valbenazine	40-80 mg / day	Vesicular monoamine transporter 2 (VMAT2) inhibitor; reduces dopamine release	Sedation, fatigue, QTc prolongation
Deutetrabenazine	12-48 mg / day (BID)	VMAT2 inhibitor (deuterated form of tetrabenazine, longer half-life)	Depression, suicidality (black box), parkinsonism

- Both FDA-approved specifically for tardive dyskinesia
- VMAT2 inhibition → decreased dopamine packaging into vesicles → reduced dopaminergic transmission
- Valbenazine generally preferred (once daily, less depression risk)

Step 3: Adjunctive

- Benzodiazepines (clonazepam 0.5-2 mg / day), may provide modest benefit
- Vitamin E, weak evidence; may slow progression (not reverse)
- Ginkgo biloba, some RCT evidence in Chinese populations

Q3. What preventive measures should have been taken?

Answer:

1. **Use SGAs preferentially** (lower TD risk than FGAs)
2. **Lowest effective dose** of any antipsychotic
3. **Regular AIMS screening:** Every 6 months for FGAs; annually for SGAs; every 3 months in high-risk groups (elderly, female)
4. **Early detection:** Educate patient and family about early signs
5. **Avoid anticholinergics long-term** (may mask early TD; increase risk)
6. **Consider LAI SGAs** for non-adherent patients (more predictable dosing)
7. **Document baseline AIMS** before starting any antipsychotic
8. **If early TD detected:** Immediate switch to lower-risk antipsychotic (clozapine, quetiapine); start VMAT2 inhibitor

Vignette 15: Ultra-High Risk / Prodromal Phase

Case: A 17-year-old male high school student is referred by his school counselor. Over 6 months, his grades have dropped from A's to D's, he has stopped playing basketball, withdrawn from friends, and sleeps excessively. He tells the counselor he sometimes feels like people are watching him, hears faint whispers that he cannot make out, and has

become convinced that certain numbers have special meaning for him. His mother has schizophrenia. He does not meet criteria for a psychotic disorder as symptoms are below threshold for frank psychosis.

Q1. What clinical category does this patient fall under? What are the criteria?

Answer:

Category: At-Risk Mental State (ARMS) / Ultra-High Risk (UHR) for psychosis.

He meets TWO UHR criteria:

1. Attenuated Psychotic Symptoms (APS):

- Suspiciousness (feels people watching him), sub-threshold persecutory ideation
- Perceptual disturbances (faint whispers), sub-threshold hallucinations
- Magical thinking (numbers have special meaning), sub-threshold delusional ideation
- Present for >1 week in past year

2. Trait + State risk:

- Genetic risk: First-degree relative with schizophrenia (mother)
- Functional decline: Grades dropped from A to D (>30% drop in GAF equivalent)

Assessment tools:

- CAARMS (Comprehensive Assessment of At-Risk Mental States), structured interview
- SIPS/SOPS (Structured Interview for Prodromal Syndromes)

Conversion risk: ~30% will develop frank psychosis within 3 years. This means 70% will NOT, critical for management decisions.

Q2. What are the differential diagnoses?

Answer:

DIFFERENTIAL · KEY ASSESSMENT

Major depressive disorder Sleep changes, withdrawal, grade decline could be depression; assess mood, anhedonia, guilt, suicidality

Social anxiety disorder "People watching him" could be social anxiety rather than paranoia; assess for fear of evaluation vs persecutory belief

Cannabis use Common at age 17; can cause attenuated psychotic symptoms; drug screen

ADHD Grade decline + sleep issues; but attenuated psychotic symptoms go beyond ADHD

Autism spectrum Social withdrawal, unusual interests (numbers); but temporal change from premorbid argues against

Normal adolescent development Some suspiciousness and magical thinking can be normative; severity and functional decline suggest otherwise

Schizotypal personality traits Chronic sub-threshold features; but acute decline suggests something more dynamic

Q3. How would you manage this patient?

Answer:

Assessment:

- Comprehensive psychiatric evaluation (CAARMS/SIPS)
- Cognitive assessment (baseline, important for monitoring)
- Urine drug screen (cannabis, stimulants)
- Physical examination, basic blood work, TFT
- Family history details (mother's illness course)
- Risk assessment (suicidality, self-harm)
- GAF/SOFAS for functional monitoring

Management (staged approach):

Stage 1, Low-risk interventions (start here):

- **CBT** (NICE-recommended for ARMS): 14-26 sessions targeting distressing beliefs, anxiety, social withdrawal, functional recovery
- **Psychoeducation:** Age-appropriate, non-alarmist explanation of risk; involve family
- **Stress management:** Sleep hygiene, study skills, social engagement
- **Substance use:** If cannabis used, motivational interviewing for cessation
- **Family support:** Mother's illness means he has witnessed psychosis; address his fears and genetic concerns
- **School engagement:** Liaise with school counselor for academic support

Stage 2, If symptoms worsen:

- Consider low-dose antipsychotic **ONLY** if transitioning to frank psychosis
- Omega-3 fatty acids (EPA + DHA 1-2g/day), low risk, some supportive data
- Antidepressant if depressive symptoms prominent

Monitoring:

- Monthly initially; assess for progression to psychosis
- Repeat CAARMS/SIPS at 3-6 month intervals
- Cognitive monitoring annually

- If frank psychosis develops: Immediate referral to early intervention in psychosis (EIP) service; initiate treatment as per first-episode protocol (low-dose SGA)

Key ethical consideration: Communicate risk proportionately. 70% chance he will NOT develop psychosis. Avoid labeling. Use terms like "stress vulnerability" rather than "pre-schizophrenia." Involve him in all decisions (age-appropriate shared decision-making).

GUIDE 07 · PART II

Mood Disorders

Paper II · Clinical Psychiatry. Six study modes, from notes to quick review.

Study Notes

SECTION 1: BIPOLAR DISORDER

1.1 Epidemiology

PARAMETER · VALUE

Lifetime prevalence BP-I 1-1.5%

Lifetime prevalence BP-II 0.4-1.1%

Bipolar spectrum (including sub-threshold) 2.4-4.7%

Male:Female ratio BP-I 1:1

Male:Female ratio BP-II Female predominance

Mean age of onset 20-25 years (BP-I), slightly later for BP-II

Delay to correct diagnosis Average 5-10 years

Comorbidity rate >60% have at least one comorbid condition

Suicide rate 15-20x general population; ~25-50% attempt; 10-15% complete

Key Indian data:

- NMHS (2016): mood disorders prevalence ~5.25% (includes depression)
- Urban > rural prevalence in most studies
- Significant treatment gap (>70% for mood disorders)

1.2 Classification: BP-I vs BP-II vs Cyclothymia

FEATURE	BP-I	BP-II	CYCLOTHYMIA
Manic episodes	Yes (at least 1)	No (only hypomania)	No
Hypomanic episodes	May occur	Yes (at least 1)	Hypomanic symptoms (sub-threshold)
Major depressive episodes	Common but NOT required for diagnosis	Yes (at least 1)	Depressive symptoms (sub-threshold)
Duration requirement	Mania >= 7 days (or hospitalization)	Hypomania >= 4 consecutive days	>= 2 years (1 year in adolescents)

FEATURE	BP-I	BP-II	CYCLOTHYMIA
Functional impairment	Marked (mania)	Hypomania: no marked impairment; depression: significant	Persistent mild instability
Psychotic features	Can occur in mania or depression	Only in depression (if present, reclassify to BP-I)	No
Hospitalization	Common in mania	Less common	Rare

EXAM PEARL

If a patient presenting with hypomania develops even one psychotic feature, the diagnosis is upgraded to BP-I.

1.3 Clinical Features of Mania: DIGFAST

LETTER	FEATURE	CLINICAL DETAIL
D	Distractibility	Attention drawn to irrelevant stimuli
I	Indiscretion / Impulsivity	Reckless behavior, spending sprees, sexual indiscretions, foolish investments
G	Grandiosity	Inflated self-esteem, may reach delusional proportions
F	Flight of ideas	Racing thoughts, rapid topic shifting, clang associations
A	Activity increase	Psychomotor agitation, increased goal-directed activity
S	Sleep deficit	Decreased need for sleep (NOT insomnia, patient feels rested with 2-3 hours)
T	Talkativeness	Pressured speech, difficult to interrupt, loud

DSM-5 Criteria for Manic Episode:

- Abnormally and persistently elevated, expansive, or irritable mood AND abnormally and persistently increased goal-directed activity or energy
- Duration: ≥ 7 days (or any duration if hospitalization required)
- At least 3 of DIGFAST symptoms (4 if mood is only irritable)
- Marked functional impairment, psychotic features, or hospitalization

- Not attributable to substance/medical condition

1.4 Hypomania vs Mania

FEATURE	HYPOMANIA	MANIA
Duration	≥ 4 consecutive days	≥ 7 days (or any duration if hospitalized)
Severity	Observable change in functioning, NOT marked impairment	Marked impairment in functioning
Psychotic features	NEVER present	Can be present
Hospitalization	NOT required	May be required
Functional impact	May actually improve functioning transiently	Causes significant dysfunction
Social/occupational disruption	Mild	Severe
Subjective experience	Often ego-syntonic ("I feel great")	May lose insight entirely

EXAM PEARL

If any psychotic feature is present during a "hypomaniac" episode, it is mania by definition, and the diagnosis is BP-I.

1.5 Mixed Features (DSM-5 Specifier)

DSM-5 replaced the old "mixed episode" (DSM-IV) with a **specifier** that can be applied to either manic/hypomaniac or depressive episodes.

Manic/hypomaniac episode with mixed features (≥ 3 depressive symptoms):

- Depressed mood, diminished interest, psychomotor retardation, fatigue, worthlessness/guilt, suicidal ideation

Depressive episode with mixed features (≥ 3 manic/hypomaniac symptoms):

- Elevated mood, grandiosity, pressured speech, flight of ideas, increased energy/activity, decreased need for sleep, risk-taking

Clinical significance:

- Mixed features = higher suicide risk
- Mixed features = poorer response to antidepressants (may worsen)
- Valproate and atypical antipsychotics preferred over lithium for mixed presentations
- Mixed features may predict bipolar course in unipolar depression patients

1.6 Rapid Cycling

Definition: ≥ 4 mood episodes (any combination of mania, hypomania, depression) in a 12-month period. Episodes demarcated by either remission (≥ 2 months) or switch to opposite polarity.

Risk factors:

- Female sex (more common in women)
- Hypothyroidism (must screen thyroid function)
- Antidepressant use (classic trigger, particularly TCAs, less so SSRIs)
- Substance use
- BP-II $>$ BP-I
- Neurological conditions

Management considerations:

- Withdraw antidepressants
- Optimize mood stabilizers (valproate often preferred)
- Check and correct thyroid function
- Lithium less effective in rapid cycling than in classic episodic BPAD
- Consider combination mood stabilizers

1.7 Course and Outcome

KRAEPELIN'S ORIGINAL OBSERVATIONS

- Kraepelin (1921) described the longitudinal course extensively
- Noted increasing frequency of episodes over time with shortening of inter-episode intervals
- Recognized that some patients achieved full inter-episode recovery while others developed chronic residual symptoms

MODERN COURSE DATA

PARAMETER · DATA

Average number of lifetime episodes 8-12

Mean episode duration (mania) 4-6 months (untreated)

Mean episode duration (depression) 6-12 months

Inter-episode interval Decreases over time (kindling)

First episode polarity ~50% depressive, ~50% manic/mixed

Time spent in depression vs mania Depression predominates (3:1 ratio in BP-I, higher in BP-II)

Full inter-episode recovery Only ~40-50% achieve complete functional recovery

Subsyndromal symptoms between episodes Present in ~50-60%

Cognitive impairment Documented even in euthymia, executive function, verbal memory

Occupational disability ~30-40% experience significant occupational impairment

PROGNOSTIC FACTORS

GOOD PROGNOSIS · POOR PROGNOSIS

Late onset Early onset

Few episodes Frequent episodes

Male sex (some studies) Rapid cycling

Absence of mixed features Mixed features

Good inter-episode functioning Poor inter-episode functioning

Compliance with treatment Comorbid substance use

Good social support Comorbid personality disorder

Absence of psychotic features Psychotic features

Classical euphoric mania Mood-incongruent psychotic features

1.8 Genetics of Bipolar Disorder

PARAMETER · DATA

Heritability ~85% (one of the most heritable psychiatric disorders)

First-degree relative risk 5-10x general population

MZ twin concordance 40-70%

DZ twin concordance 5-10%

Adoption studies Support genetic contribution over environment

Overlap with schizophrenia genetics Significant, shared risk loci at CACNA1C, ANK3, ODZ4

Polygenic risk Hundreds of common variants, each small effect

Key genes/loci:

- **CACNA1C**, voltage-gated calcium channel (shared with schizophrenia)
- **ANKK3**, ankyrin G, involved in axon initial segment function
- **CLOCK genes**, circadian rhythm (CLOCK, ARNTL, PER3, CRY1)
- **BDNF**, Val66Met polymorphism associated with bipolar risk
- **DISC1**, Disrupted in Schizophrenia 1 (shared with schizophrenia)
- **DGKH**, diacylglycerol kinase eta
- **NRG1**, neuregulin 1 (shared with schizophrenia)

Genetic overlap:

- Bipolar shares more genetic architecture with schizophrenia than with unipolar depression
 - Cross-Disorder Group of the PGC identified shared loci across bipolar, schizophrenia, MDD, ADHD, and ASD
 - This supports a dimensional rather than categorical view of psychosis spectrum
-

1.9 Neurobiology of Bipolar Disorder

KINDLING MODEL (POST & WEISS)

- Initial episodes require environmental stressors (psychosocial triggers)
- Repeated episodes lead to neurobiological sensitization
- Later episodes become increasingly autonomous (occur without clear triggers)
- Parallels seizure kindling in epilepsy
- Supports early and aggressive treatment to prevent progression
- Explains why inter-episode intervals decrease over time

CIRCADIAN RHYTHM DISRUPTION

- **CLOCK** gene polymorphisms associated with bipolar risk
- Social rhythm disruption can trigger episodes (Ehlers et al., Social Zeitgeber Theory)
- Sleep deprivation can trigger mania
- Light therapy effective in bipolar depression
- Melatonin pathway abnormalities documented
- Interpersonal and Social Rhythm Therapy (IPSRT) targets this mechanism

MITOCHONDRIAL DYSFUNCTION

- Altered mitochondrial function in bipolar disorder

- Decreased ATP production, altered calcium signaling
- Explains multi-system involvement (brain, kidney, thyroid)
- Lactate elevation in brain imaging studies
- mtDNA mutations and deletions described
- Lithium has mitochondrial protective effects (one proposed mechanism)

OTHER NEUROBIOLOGICAL MODELS

- **Monoamine dysregulation:** Catecholamine excess in mania, deficit in depression (oversimplified but foundational)
- **Second messenger systems:** Abnormal phosphoinositide signaling, PKC overactivity in mania
- **Neuroinflammation:** Elevated pro-inflammatory cytokines (IL-6, TNF-alpha) in mood episodes
- **Glutamate:** Elevated in mania, altered in depression
- **HPA axis:** Cortisol dysregulation, dexamethasone non-suppression
- **Neuroplasticity:** Reduced BDNF, hippocampal volume loss, prefrontal cortical thinning

SECTION 2: BIPOLAR DISORDER MANAGEMENT

2.1 Lithium

MECHANISM OF ACTION

- **GSK-3beta inhibition**, the most well-established mechanism; GSK-3beta is involved in apoptosis, circadian regulation, neurogenesis
- **Inositol depletion**, lithium inhibits inositol monophosphatase, reducing phosphoinositide signaling (Berridge hypothesis)
- **Neuroprotection**, increases Bcl-2 (anti-apoptotic), increases gray matter volume
- **Modulates glutamate**, reduces excitatory glutamatergic transmission
- **Serotonergic enhancement**, increases tryptophan uptake, enhances 5-HT release
- **Anti-suicidal effect**, specific to lithium, not shared by other mood stabilizers

THERAPEUTIC RANGE AND MONITORING

Acute mania 0.8-1.2 mEq/L

Maintenance 0.6-1.0 mEq/L (0.6-0.8 for elderly)

Toxicity threshold >1.5 mEq/L

Time to steady state 5-7 days

Blood level timing 12 hours post last dose (trough level)

MONITORING SCHEDULE

TEST	BASELINE	EVERY 6 MONTHS	AS INDICATED
Serum lithium	Yes	Yes (after stabilization, q3-6 months)	After dose change (5-7 days later)
Renal function (creatinine, eGFR)	Yes	Yes	Symptoms of renal impairment
Thyroid (TSH, free T4)	Yes	Yes	Symptoms of hypothyroidism
Calcium/parathyroid	Yes	Yes (annually minimum)	Symptoms of hypercalcemia
ECG	Yes (>40 years or cardiac history)	Annual if elderly	Cardiac symptoms
Weight/BMI	Yes	Yes	
Pregnancy test	Yes (women of child-bearing age)		Before starting
FBC	Yes	Annually	

SIDE EFFECTS

Early/common:

- Fine tremor (beta-blockers can help)
- Polyuria/polydipsia (nephrogenic diabetes insipidus, lithium impairs aquaporin-2)
- GI upset (nausea, diarrhea)
- Weight gain
- Cognitive dulling ("lithium fog")
- Acne, psoriasis exacerbation

Long-term:

- **Hypothyroidism** (up to 20-30%, more in women; treat with levothyroxine, do NOT stop lithium)
- **Nephrogenic diabetes insipidus** (reduced concentrating ability; amiloride may help)
- **Chronic kidney disease** (controversial; risk increases with duration; monitor eGFR)
- **Hyperparathyroidism/hypercalcemia** (monitor calcium)
- **Cardiac**, T-wave flattening/inversion, sinus node dysfunction (rare)
- **Ebstein's anomaly**, cardiac teratogenicity (1:1000 risk, much lower than historically feared but still significant; relative risk ~20-40x)

LITHIUM TOXICITY

SEVERITY	LEVEL (MEQ/L)	FEATURES
Mild	1.5-2.0	Coarse tremor, nausea, diarrhea, drowsiness, muscular weakness
Moderate	2.0-2.5	Ataxia, dysarthria, confusion, hyperreflexia, myoclonic jerks
Severe	>2.5	Seizures, coma, renal failure, cardiovascular collapse, death

Management of lithium toxicity:

1. STOP lithium immediately
2. IV normal saline (0.9% NaCl), aggressive hydration
3. Monitor serum lithium levels every 2-4 hours
4. Monitor renal function, electrolytes
5. Correct dehydration and sodium depletion
6. Avoid diuretics (especially thiazides, increase lithium reabsorption)
7. **Hemodialysis** if: level >4.0 mEq/L, level >2.5 with severe symptoms, renal failure, deteriorating despite supportive care
8. Whole bowel irrigation if recent ingestion of sustained-release preparation
9. Monitor for rebound, redistribution from tissues can cause level to rise again after dialysis

Common precipitants of toxicity:

- Dehydration (vomiting, diarrhea, fever, hot weather)
- NSAIDs (reduce renal clearance)
- ACE inhibitors / ARBs
- Thiazide diuretics (NOT loop diuretics, loop diuretics are safer)
- Reduced sodium intake
- Renal impairment
- Drug interactions

2.2 Valproate (Sodium Valproate / Divalproex)

MECHANISM

- Enhances GABAergic transmission (inhibits GABA transaminase, enhances GABA synthesis)
- Blocks voltage-gated sodium channels

- Inhibits histone deacetylase (HDAC), epigenetic effects
- Modulates PKC signaling

THERAPEUTIC RANGE

- 50-125 mcg/mL for acute mania
- Trough levels

MONITORING

- LFTs at baseline and periodically (first 6 months critical)
- FBC and platelets (thrombocytopenia)
- Coagulation screen
- Amylase/lipase if abdominal symptoms
- Weight, metabolic parameters
- Menstrual history in women

SIDE EFFECTS

- **Hepatotoxicity**, idiosyncratic, highest risk in children <2 years on polytherapy; monitor LFTs
- **Pancreatitis**, rare but potentially fatal
- **PCOS/menstrual irregularities**, weight gain, hyperandrogenism, menstrual dysfunction; mechanism involves enzyme induction affecting androgen metabolism
- **Teratogenicity**, NEURAL TUBE DEFECTS (spina bifida ~1-2%), decreased IQ in offspring (8-9 points), higher rate of autism; absolute contraindication in pregnancy / women of childbearing age per many recent guidelines
- **Thrombocytopenia**, dose-related
- **Weight gain**, significant
- **Tremor, hair loss (usually reversible), GI upset**
- **Hyperammonemia**, can occur without hepatic dysfunction; may present as confusion

VALPROATE IN WOMEN OF CHILDBEARING AGE

EXAM PEARL

Recent guideline changes, EMA (2018): Banned valproate in pregnancy and women of childbearing age unless Pregnancy Prevention Programme in place. MHRA (UK): Similar restrictions. NICE: Recommends against valproate in women/girls of childbearing potential. Indian guidelines increasingly following this trend. If absolutely necessary: informed consent, effective contraception, regular pregnancy tests, folic acid (5mg).

2.3 Carbamazepine

MECHANISM

- Voltage-gated sodium channel blocker (primary mechanism)
- Also affects calcium channels
- Reduces glutamate release

KEY PHARMACOLOGICAL FEATURES

- **Autoinduction**, carbamazepine induces its own metabolism (CYP3A4); levels drop over first 2-4 weeks; need to recheck levels and often increase dose
- **Potent CYP inducer**, induces CYP3A4, CYP2C9, CYP1A2; reduces levels of many drugs (OCs, warfarin, other anticonvulsants, antipsychotics)
- Therapeutic level: 4-12 mcg/mL

HLA-B*1502 SCREENING

- **HLA-B*1502** allele associated with carbamazepine-induced Stevens-Johnson Syndrome (SJS) and toxic epidermal necrolysis (TEN)
- Highest prevalence in Southeast Asian populations (Han Chinese, Thai, Malaysian, Indian)
- FDA mandates screening before prescribing in at-risk populations
- If positive, DO NOT prescribe carbamazepine (or oxcarbazepine)

SIDE EFFECTS

- CNS: dizziness, ataxia, diplopia, drowsiness
- Hyponatremia (SIADH)
- Agranulocytosis/aplastic anemia (rare but serious, monitor FBC)
- Hepatotoxicity
- SJS/TEN (see HLA-B*1502 above)
- Teratogenic (NTDs, but less than valproate)

DRUG INTERACTIONS (MAJOR EXAM TOPIC)

- Reduces levels of: haloperidol, olanzapine, clozapine, lamotrigine, oral contraceptives, warfarin, theophylline, doxycycline, valproate
- Levels increased by: erythromycin, cimetidine, fluoxetine, fluvoxamine, grapefruit juice
- Levels decreased by: phenytoin, phenobarbital

2.4 Lamotrigine

KEY FEATURES

- **Primary role: bipolar depression prevention** (maintenance)

- NOT effective for acute mania
- Effective for preventing depressive relapses in BPAD
- Glutamate modulator (reduces glutamate release via sodium channel blockade)

TITRATION: SJS RISK (CRITICAL)

- **Must titrate slowly**, rapid titration increases SJS/TEN risk
- Standard titration: 25mg/day weeks 1-2 → 50mg/day weeks 3-4 → 100mg/day week 5 → target 200mg/day by week 6-7
- If co-prescribed with valproate (which DOUBLES lamotrigine levels): halve the titration rate, start at 12.5mg
- If co-prescribed with carbamazepine (which halves lamotrigine levels): may need higher doses

SIDE EFFECTS

- **SJS/TEN**, risk highest in first 8 weeks, with rapid titration, and with concurrent valproate
- Rash (any rash during titration = STOP and evaluate)
- Headache, dizziness, nausea
- Insomnia
- Generally well-tolerated, no weight gain, no cognitive dulling
- Favorable metabolic profile

2.5 Atypical Antipsychotics in Bipolar Disorder

DRUG	ACUTE MANIA	BIPOLAR DEPRESSION	MAINTENANCE	NOTES
Quetiapine	Yes	Yes (FDA approved)	Yes	Only drug approved for both poles + maintenance
Olanzapine	Yes	Yes (olanzapine-fluoxetine combo)	Yes	Significant metabolic effects
Aripiprazole	Yes	Limited evidence	Yes	Weight-neutral, akathisia common
Lurasidone	No	Yes (FDA approved)		Take with food (>350 kcal), no metabolic burden

DRUG	ACUTE MANIA	BIPOLAR DEPRESSION	MAINTENANCE	NOTES
Cariprazine	Yes	Yes (FDA approved)	Yes	D3-preferring partial agonist; emerging evidence
Risperidone	Yes	No	Limited	More EPS than others
Ziprasidone	Yes	No	Limited	QTc monitoring
Asenapine	Yes	No	Yes	Sublingual administration

2.6 Acute Mania: Treatment Algorithm

Step 1: Assess severity, rule out medical causes, assess safety

- Discontinue antidepressants
- Check substance use, thyroid, medical causes

Step 2: First-line pharmacotherapy

- **Mild-moderate mania:** Lithium or valproate monotherapy
- **Severe mania/psychotic mania:** Atypical antipsychotic (quetiapine, olanzapine, aripiprazole, risperidone) + lithium/valproate
- **Agitation:** Short-term benzodiazepines (lorazepam) + antipsychotic

Step 3: If inadequate response (2-4 weeks):

- Switch to alternative first-line agent
- Combine lithium + valproate
- Combine mood stabilizer + atypical antipsychotic

Step 4: Treatment-resistant mania:

- ECT (highly effective, especially with catatonic features or life-threatening mania)
- Clozapine (limited evidence but used in refractory cases)

2.7 Bipolar Depression: Management (HIGH-YIELD EXAM TOPIC)

EXAM PEARL

Do NOT give antidepressant monotherapy in bipolar depression, risk of manic switch and cycle acceleration.

First-line options:

1. Quetiapine monotherapy (300mg, the BOLDER trials)
2. Lithium monotherapy (evidence moderate)
3. Lamotrigine (better for prevention than acute treatment, but used)
4. Lurasidone (with or without lithium/valproate)
5. Cariprazine (1.5mg, recent FDA approval)
6. Olanzapine-fluoxetine combination (OFC)

If adding antidepressant (not first-line):

- ALWAYS combine with mood stabilizer
- SSRIs or bupropion preferred (lower switch risk)
- AVOID TCAs and SNRIs (higher switch risk)
- Short duration, withdraw antidepressant as soon as depression remits

Other options:

- ECT, effective for severe/refractory bipolar depression, psychotic features, suicidality
- Interpersonal and Social Rhythm Therapy (IPSRT)
- CBT adapted for bipolar

2.8 Maintenance Treatment

Goal: Prevent relapse of both manic and depressive episodes

AGENT	PREVENTS MANIA	PREVENTS DEPRESSION	ANTI-SUICIDE
Lithium	+++	++	Yes (unique)
Valproate	+++	+	No
Lamotrigine	+	+++	No
Quetiapine	++	++	No
Aripiprazole	++	+	No
Olanzapine	++	+	No
Carbamazepine	++	+	No

Lithium remains the gold standard for maintenance, especially for classical euphoric mania, strong family history, good inter-episode functioning. Its anti-suicidal effect is unique and well-established (Cipriani et al., 2013 meta-analysis).

SECTION 3: DEPRESSION

3.1 Epidemiology

PARAMETER · VALUE

Lifetime prevalence 10-20% (varies by population)

Point prevalence 5-8%

Female:Male ratio 2:1

Mean age of onset 25-35 years (getting younger in recent cohorts)

Recurrence risk 50% after first episode, 80% after second, 90% after third

Global disease burden #1 cause of disability worldwide (WHO)

Indian data (NMHS 2016) ~5.25% prevalence for mood disorders

3.2 Diagnostic Criteria

ICD-11 DEPRESSIVE EPISODE

Core symptoms (at least 1 required):

1. Depressed mood
2. Diminished interest or pleasure (anhedonia)

Accessory symptoms:

1. Reduced concentration and attention
2. Reduced self-esteem and self-confidence
3. Ideas of guilt and unworthiness
4. Bleak and pessimistic views of the future
5. Ideas or acts of self-harm or suicide
6. Disturbed sleep
7. Diminished appetite

Severity grading:

- Mild: Core + enough accessory for minimum of 5 total; mild functional impairment
- Moderate: Core + enough for minimum of 6-7 total; considerable difficulty in functioning
- Severe: Core + most accessory symptoms; marked distress / agitation or retardation; major functional impairment

DSM-5 MAJOR DEPRESSIVE EPISODE: SIG E CAPS + DEPRESSED MOOD

Five or more of the following for ≥ 2 weeks (must include depressed mood or anhedonia):

MNEMONIC · SYMPTOM

S Sleep disturbance (insomnia or hypersomnia)

I Interest loss (anhedonia)

G Guilt (excessive or inappropriate)

E Energy loss / fatigue

C Concentration difficulty / indecisiveness

A Appetite change (increase or decrease) / weight change

P Psychomotor agitation or retardation

S Suicidal ideation

+ Depressed mood (most of the day, nearly every day)

3.3 Subtypes of Depression

MELANCHOLIC FEATURES

- Loss of pleasure in almost all activities (distinct quality of depressed mood, qualitatively different from grief)
- Lack of reactivity to pleasurable stimuli
- Worse in the morning (diurnal variation)
- Early morning awakening (>2 hours before usual)
- Marked psychomotor retardation or agitation
- Significant anorexia or weight loss
- Excessive guilt
- **Better response to biological treatments (TCAs, ECT)**
- Strong HPA axis dysregulation

ATYPICAL FEATURES

- **Mood reactivity** (mood brightens in response to positive events), required criterion
- Significant weight gain or increased appetite ("comfort eating")
- Hypersomnia (leaden paralysis)
- Long-standing pattern of interpersonal rejection sensitivity
- **Better response to MAOIs and SSRIs than TCAs**

- More common in BP-II depression

PSYCHOTIC FEATURES (DEPRESSION WITH PSYCHOTIC SYMPTOMS)

- Delusions and/or hallucinations during depressive episode
- **Mood-congruent:** themes of guilt, worthlessness, disease, death, nihilism, punishment (Cotard syndrome, nihilistic delusions)
- **Mood-incongruent:** persecutory, referential, thought insertion/broadcasting (worse prognosis)
- Requires antidepressant + antipsychotic OR ECT
- Antidepressant monotherapy inadequate
- ECT highly effective

SEASONAL AFFECTIVE DISORDER (SAD)

- Recurrent episodes with seasonal pattern (typically winter)
- Atypical features common (hypersomnia, weight gain, carbohydrate craving)
- Light therapy (10,000 lux for 30 min each morning) is first-line
- SSRIs also effective
- Latitude-dependent prevalence

PERIPARTUM DEPRESSION

- Onset during pregnancy or within 4 weeks postpartum (DSM-5 specifier), clinically, up to 1 year recognized
- Prevalence: 10-15% of pregnancies
- Screen using Edinburgh Postnatal Depression Scale (EPDS)
- Risk factors: prior depression, lack of social support, unplanned pregnancy, domestic violence
- Treatment: psychotherapy first-line for mild-moderate; SSRIs (sertraline preferred, low breast milk transfer) for moderate-severe
- **Brexanolone** (IV allopregnanolone), FDA approved for postpartum depression (2019)
- **Zuranolone**, oral neurosteroid, FDA approved 2023

3.4 Biological Basis of Depression

MONOAMINE HYPOTHESIS (CLASSIC)

- Depression results from deficiency of serotonin (5-HT), norepinephrine (NE), and/or dopamine (DA)
- Evidence: reserpine causes depression; antidepressants increase monoamine availability; tryptophan depletion can trigger relapse

- Limitations: antidepressants increase monoamines within hours, but clinical effect takes weeks; direct monoamine depletion does not consistently cause depression in healthy subjects

HPA AXIS DYSREGULATION

- Hyperactivity of HPA axis in depression
- Elevated cortisol levels, enlarged adrenals
- Dexamethasone non-suppression (DST, ~50% sensitivity, poor specificity)
- CRH hypersecretion
- Glucocorticoid receptor resistance
- Chronic stress → HPA dysregulation → hippocampal damage → perpetuation of depression

NEUROPLASTICITY AND BDNF

- Brain-Derived Neurotrophic Factor (BDNF) reduced in depression
- Reduced hippocampal volume (neuroimaging studies)
- Antidepressants increase BDNF and promote neurogenesis
- Stress reduces neuroplasticity; antidepressants reverse this
- "Neuroplasticity hypothesis", depression as a disorder of synaptic plasticity

NEUROINFLAMMATION

- Elevated pro-inflammatory cytokines (IL-1, IL-6, TNF-alpha, CRP) in depression
- Inflammatory conditions (RA, IBD, MS) have high depression comorbidity
- Interferon-alpha treatment causes depression in ~30% of patients
- Anti-inflammatory agents (celecoxib, minocycline) show antidepressant effects in trials
- Links inflammation → tryptophan degradation via IDO enzyme → reduced serotonin

GLUTAMATE MODEL

- Glutamate/GABA imbalance in depression
- Ketamine (NMDA antagonist) produces rapid antidepressant effects
- Supports role of glutamatergic dysfunction
- Esketamine (intranasal), FDA approved for TRD

SECTION 4: TREATMENT-RESISTANT DEPRESSION (TRD)

4.1 Definition

TRD = failure to achieve adequate response after ≥ 2 adequate antidepressant trials from different pharmacological classes, each at adequate dose and duration ($\geq 6-8$ weeks at therapeutic dose).

"Adequate trial" means:

- Therapeutic dose reached
- Minimum 6-8 weeks at therapeutic dose
- Confirmed adherence

Pseudo-resistance (rule out first):

- Inadequate dose or duration
- Non-adherence
- Misdiagnosis (bipolar depression, medical cause, substance use, personality disorder)
- Comorbidity not addressed (anxiety, substance use, medical illness)

4.2 TRD Staging

THASE-RUSH STAGING MODEL

STAGE · FAILED TREATMENT

Stage I Failure of ≥ 1 adequate SSRI trial

Stage II Failure of ≥ 1 adequate trial of different class (e.g., SNRI, TCA)

Stage III Failure of adequate TCA trial

Stage IV Failure of adequate MAOI trial

Stage V Failure of bilateral ECT course

MASSACHUSETTS GENERAL HOSPITAL (MGH) STAGING MODEL

- Quantitative scoring system
- Points assigned for: number of failed trials, optimization attempts, augmentation trials, ECT
- More granular than Thase-Rush
- Better for research quantification

4.3 TRD Management Algorithm

Step 1, Optimization:

- Ensure current antidepressant is at maximum tolerated dose
- Ensure adequate duration ($\geq 6-8$ weeks at therapeutic dose)
- Confirm adherence
- Address comorbidities

Step 2, Switching:

- Switch to a different class (SSRI → SNRI → TCA → MAOI)
- Within-class switch if partial response (e.g., escitalopram → sertraline)
- Switching preferred when there is NO response to current agent

Step 3, Augmentation:

- **Lithium augmentation**, most evidence-based augmentation strategy; 0.6-0.8 mEq/L; response in 2-4 weeks
- **Atypical antipsychotic augmentation**, aripiprazole (2-5mg), quetiapine (50-300mg), olanzapine (2.5-10mg), brexpiprazole, cariprazine
- **Thyroid augmentation**, T3 (liothyronine 25-50 mcg/day) even in euthyroid patients; better evidence than T4
- Augmentation preferred when there IS partial response to current agent

Step 4, Combination:

- SSRI/SNRI + mirtazapine ("California rocket fuel" = venlafaxine + mirtazapine)
- SSRI/SNRI + bupropion
- Avoid SSRI + MAOI (serotonin syndrome)

Step 5, Esketamine (Spravato):

- Intranasal esketamine (FDA approved 2019 for TRD)
- Used with concurrent oral antidepressant
- Must be administered in certified healthcare settings (REMS program)
- Monitoring for 2 hours post-administration
- Rapid onset (within hours to days)

Step 6, ECT:

- Gold standard for severe/refractory depression
- 70-90% response rate in TRD
- Bilateral > unilateral for efficacy (but more cognitive side effects)
- Indicated especially for: psychotic depression, severe suicidality, catatonia, treatment resistance, pregnancy (relatively safe)

Step 7, Neuromodulation and emerging:

- **rTMS**, repetitive Transcranial Magnetic Stimulation; left DLPFC high-frequency stimulation; FDA approved; 4-6 week course
 - **VNS**, Vagus Nerve Stimulation; implanted device; FDA approved for chronic TRD
 - **DBS**, Deep Brain Stimulation; experimental; targets include subcallosal cingulate (Cg25), nucleus accumbens
 - **Psilocybin**, emerging evidence for TRD; phase II trials promising; not yet approved
-

SECTION 5: DEPRESSION IN THE ELDERLY

5.1 Vascular Depression Hypothesis (Alexopoulos)

- Late-onset depression (>60 years) often associated with cerebrovascular disease
- White matter hyperintensities on MRI correlate with depression severity
- Vascular risk factors (hypertension, diabetes, smoking) increase depression risk
- Executive dysfunction prominent
- Poor response to antidepressants compared to early-onset depression
- Higher risk of progression to dementia
- "Depression-executive dysfunction syndrome", apathy, psychomotor retardation, poor insight

5.2 Pseudodementia vs Dementia

FEATURE	PSEUDODEMENTIA (DEPRESSION)	DEMENTIA
Onset	Relatively acute, dateable	Insidious, family notices before patient
Cognitive complaints	Emphasized by patient ("I can't remember anything")	Minimized by patient, noticed by family
Effort on testing	"Don't know" answers, gives up easily	Tries hard, confabulates, near-miss answers
Mood	Pervasive sadness, anhedonia	Mood may be labile or apathetic but not pervasively sad
Diurnal variation	Often present (worse in morning)	Sundowning (worse in evening)
Sleep	Early morning awakening	Fragmented, reversed sleep-wake
Memory pattern	Inconsistent; similar impairment for recent and remote	Recent > remote memory loss (temporal gradient)
History	Psychiatric history, psychosocial stressors	May have vascular risk factors, gradual decline
Response to treatment	Improves with antidepressants	Does not improve
Neuroimaging	May be normal or show white matter changes	Atrophy, especially hippocampal

EXAM PEARL

Pseudodementia is a major risk factor for developing true dementia later (30-50% within 5 years).

5.3 Management in Elderly

Principles:

- "Start low, go slow, but go", lower starting doses, slower titration, but aim for therapeutic doses
- SSRIs preferred (escitalopram, sertraline, fewer interactions)
- Avoid TCAs, anticholinergic effects (confusion, urinary retention, falls, cardiac), orthostatic hypotension
- Avoid benzodiazepines (falls, confusion, dependence)
- Mirtazapine useful (appetite stimulation, sedation can help insomnia)
- Consider drug interactions (elderly on multiple medications)
- Monitor sodium (SSRIs can cause SIADH/hyponatremia, higher risk in elderly)
- ECT safe and effective in elderly, may be preferred for severe depression
- Psychotherapy effective, CBT, IPT, problem-solving therapy

SECTION 6: SUICIDE

6.1 Epidemiology: India

PARAMETER · DATA

Suicide rate (India, NCRB 2021) ~12 per 100,000 population

India's share of global suicides ~36% of female suicides, ~24% of male suicides worldwide

Peak age group 18-30 years (India's suicides skew younger than Western countries)

Male:Female ratio (completed suicide) ~1.7:1 (narrower gap than Western countries)

Most common method Hanging (most common), poisoning (pesticides, significant in rural India)

Farming suicides Major public health concern; linked to debt, crop failure, drought

Student suicides Rising trend; academic pressure, competitive exams

Section 309 IPC Decriminalized (Mental Healthcare Act 2017, Section 115), attempted suicide presumed to be under severe stress; person to be treated, not prosecuted

6.2 Risk Factors: SAD PERSONS

LETTER	FACTOR	DETAIL
S	Sex	Male (completed), Female (attempted)

LETTER	FACTOR	DETAIL
A	Age	Elderly (>65) and young adults (15-30)
D	Depression	Major depression, bipolar, schizophrenia
P	Previous attempt	STRONGEST predictor of future suicide
E	Ethanol/substance abuse	Acute intoxication + chronic use
R	Rational thinking loss	Psychosis, severe cognitive distortion
S	Social supports lacking	Isolation, living alone, unemployment
O	Organized plan	Specific method, access to means, preparation
N	No spouse	Single, divorced, widowed
S	Sickness	Chronic illness, chronic pain, terminal illness

Additional risk factors:

- Family history of suicide
- Access to lethal means (firearms, pesticides)
- Recent discharge from psychiatric hospitalization (first 1-4 weeks = highest risk)
- Hopelessness (Beck Hopelessness Scale, more predictive than depression severity)
- Impulsivity/aggression traits
- LGBTQ+ youth
- Recent loss (bereavement, relationship, financial)

6.3 Assessment

COLUMBIA SUICIDE SEVERITY RATING SCALE (C-SSRS)

- Standardized, FDA-endorsed tool
- Assesses suicidal ideation (types 1-5) and suicidal behavior (types 1-5)
- Ideation severity: wish to be dead → non-specific active suicidal thoughts → active ideation with method → active ideation with intent → active ideation with plan and intent
- Behavior: preparatory acts → aborted attempt → interrupted attempt → actual attempt → completed suicide

- Can track changes over time

ASSESSMENT FRAMEWORK (EVERY EVALUATION SHOULD INCLUDE)

1. **Ideation:** Passive ("wish I were dead") vs active ("want to kill myself")
2. **Plan:** Specific method? Timeframe?
3. **Means:** Access to method? (firearms, medications, pesticides)
4. **Intent:** How strong is the desire? What are the reasons for living?
5. **Preparedness:** Giving away possessions, writing notes, saying goodbyes
6. **Risk factors:** Inventory of SAD PERSONS + additional
7. **Protective factors:** Reasons for living, children, religious beliefs, social connectedness, therapeutic alliance
8. **Recent changes:** Sudden calm after agitation (may indicate decision made), recent discharge, recent loss

6.4 Protective Factors

- Strong social support and connectedness
- Children in the home (especially for women)
- Religious/spiritual beliefs that discourage suicide
- Reasons for living (future-oriented thinking)
- Therapeutic alliance and engagement in treatment
- Limited access to lethal means
- Problem-solving skills
- Cultural and community belonging

6.5 Management and Prevention

INDIVIDUAL LEVEL

- **Safety planning** (Stanley-Brown Safety Planning Intervention), NOT a "no-suicide contract"
- Warning signs
- Internal coping strategies
- Social contacts for distraction
- Family/friends to contact
- Professionals/agencies to contact
- Making the environment safe (means restriction)
- **Means restriction**, most effective single intervention (locking medications, removing firearms, pesticide regulation)
- **Pharmacological:**

- Clozapine, ONLY medication with FDA indication for suicide reduction (in schizophrenia; InterSePT trial)
- Lithium, reduces suicide risk in mood disorders (Cipriani meta-analysis)
- Ketamine/ esketamine, rapid reduction in suicidal ideation (hours to days)
- Treat underlying disorder aggressively
- **Psychotherapy:** DBT (strongest evidence for repeated self-harm/BPD), CBT for suicide prevention (CBT-SP), CAMS (Collaborative Assessment and Management of Suicidality)
- **Follow-up:** Contact within 48 hours of discharge from ER/ psychiatric unit; WHO SUPRE studies show brief contact interventions reduce reattempts

POPULATION LEVEL

- Means restriction (pesticide bans, Sri Lanka model; bridge barriers; medication packaging)
- Gatekeeper training programs (QPR, ASIST, Mental Health First Aid)
- Media guidelines (Papageno effect vs Werther effect)
- School-based programs
- Crisis helplines (iCall, Vandrevalla Foundation, AASRA in India)
- Training primary care physicians in depression detection

SECTION 7: ANTIDEPRESSANTS

7.1 Classification and Mechanisms

SSRIS (SELECTIVE SEROTONIN REUPTAKE INHIBITORS)

DRUG · UNIQUE FEATURES

Fluoxetine Long half-life (2-6 days, norfluoxetine 4-16 days); activating; CYP2D6 inhibitor; only SSRI approved for children

Sertraline Mild DA reuptake inhibition; preferred in cardiac patients (SADHART trial); preferred in breastfeeding

Paroxetine Most anticholinergic SSRI; potent CYP2D6 inhibitor; worst discontinuation syndrome; teratogenic (cardiac defects, avoid in pregnancy)

Fluvoxamine CYP1A2 inhibitor; primarily for OCD; significant drug interactions

Escitalopram S-enantiomer of citalopram; cleanest side effect profile; most selective SSRI

Citalopram QTc prolongation at high doses; dose cap 40mg (20mg in elderly)

Class side effects: GI (nausea, diarrhea), sexual dysfunction (30-40%), headache, insomnia or sedation, weight gain (long-term), activation / anxiety initially, hyponatremia (SIADH, especially elderly), bleeding risk (platelet serotonin depletion)

SNRIS (SEROTONIN-NOREPINEPHRINE REUPTAKE INHIBITORS)

DRUG · UNIQUE FEATURES

Venlafaxine Dose-dependent: serotonergic at low dose (<150mg), noradrenergic at higher dose; dose-dependent hypertension; lethal in overdose (relative to SSRIs)

Duloxetine Also approved for neuropathic pain, fibromyalgia, GAD; hepatotoxicity risk; avoid in liver disease

Desvenlafaxine Active metabolite of venlafaxine; fewer drug interactions; more predictable pharmacokinetics

Milnacipran Primarily for fibromyalgia (India); more NE than 5-HT effect

Class side effects: Similar to SSRIs + hypertension (NE effect), sweating, dry mouth, constipation, urinary retention

TCAS (TRICYCLIC ANTIDEPRESSANTS)

DRUG	RELATIVE ACTIVITY	UNIQUE FEATURES
Amitriptyline	5-HT > NE	Most sedating; used for pain, migraine prophylaxis
Imipramine	5-HT ≈ NE	Also used for enuresis; active metabolite = desipramine
Clomipramine	5-HT >> NE	Most serotonergic TCA; gold standard for OCD
Nortriptyline	NE > 5-HT	Least hypotensive TCA; therapeutic window (50-150 ng/mL)
Desipramine	NE >> 5-HT	Most noradrenergic; least sedating / anticholinergic

Class side effects: Anticholinergic (dry mouth, constipation, urinary retention, blurred vision, cognitive impairment), cardiac (QTc prolongation, arrhythmias, lethal in overdose), sedation (H1 blockade), orthostatic hypotension (alpha-1 blockade), weight gain, seizure threshold lowering

TCA overdose: lethal due to cardiac toxicity (widened QRS, arrhythmias); management = sodium bicarbonate IV, cardiac monitoring, activated charcoal if early

MAOIS (MONOAMINE OXIDASE INHIBITORS)

DRUG	TYPE	FEATURES
Phenelzine	Non-selective, irreversible	Most studied; effective for atypical depression
Tranylcypromine	Non-selective, irreversible	Amphetamine-like structure; more activating
Isocarboxazid	Non-selective, irreversible	Less commonly used
Moclobemide	Selective MAO-A, reversible (RIMA)	Fewer dietary restrictions; less potent; available in India
Selegiline patch	Selective MAO-B at low dose	Transdermal; bypasses first-pass → fewer dietary restrictions at low dose

Tyramine reaction ("cheese effect"): Inhibition of MAO-A in gut allows tyramine from fermented foods to enter circulation → NE release → hypertensive crisis. Avoid: aged cheese, wine, beer, fermented foods, fava beans, cured meats.

Serotonin syndrome: Combination of MAOI + serotonergic drug (SSRI, SNRI, meperidine, tramadol, dextromethorphan, St. John's wort) → hyperthermia, agitation, myoclonus, hyperreflexia, diarrhea, autonomic instability, potentially fatal. Washout period: 2 weeks for most SSRIs; 5 weeks for fluoxetine (long half-life) before starting MAOI.

ATYPICAL/OTHER ANTIDEPRESSANTS

DRUG	MECHANISM	KEY FEATURES
Mirtazapine	NaSSA (alpha-2 antagonist + 5-HT _{2A} /2C/3 blockade + H ₁ blockade)	Sedating (especially <15mg due to H ₁ predominance); weight gain; no sexual dysfunction; no nausea; good for elderly with insomnia/poor appetite
Bupropion	NDRI (NE + DA reuptake inhibition)	Activating; no sexual dysfunction; helps with smoking cessation; CONTRAINDICATED in eating disorders and seizure disorders; weight neutral
Vortioxetine	Multimodal: 5-HT reuptake inhibition + 5-HT _{1A} agonism + 5-HT ₃ /7 antagonism	Pro-cognitive effects; may improve cognitive symptoms of depression; less sexual dysfunction; GI side effects

DRUG	MECHANISM	KEY FEATURES
Agomelatine	MT1/MT2 melatonin agonist + 5-HT2C antagonist	Circadian rhythm resynchronization; no sexual dysfunction; no weight gain; monitor LFTs (hepatotoxicity risk); not available in USA
Tianeptine	Glutamate modulator (previously thought to enhance 5-HT reuptake)	Opioid mu-receptor effects discovered; abuse potential; effective but controlled in some countries
Trazodone	SARI (5-HT2A antagonist + 5-HT reuptake inhibitor)	Very sedating at low dose, used primarily as hypnotic; priapism risk (rare); orthostatic hypotension

NEWER/EMERGING AGENTS

DRUG	MECHANISM	STATUS	KEY FEATURES
Esketamine (Spravato)	NMDA receptor antagonist (S-enantiomer of ketamine)	FDA approved (2019) for TRD	Intranasal; rapid onset (hours); REMS program; dissociative side effects; used with oral antidepressant
Brexanolone (Zulresso)	GABA-A receptor positive allosteric modulator (allopregnanolone)	FDA approved (2019) for postpartum depression	IV infusion over 60 hours; very expensive; limited access; REMS
Zuranolone (Zurzuvae)	Oral neuroactive steroid (GABA-A modulator)	FDA approved (2023) for PPD	Oral, 14-day course; first oral medication specifically for PPD
Psilocybin	5-HT2A agonist (psychedelic)	Phase II/III trials	Breakthrough therapy designation for TRD; single or two doses with psychotherapy; not yet approved
MDMA-assisted therapy	Serotonin/dopamine/NE release	FDA review (2024) for PTSD	Primarily PTSD, not depression per se; controversial FDA decision
Dextromethorphan/bupropion (Auvelity)	NMDA antagonist/sigma-1 agonist + CYP2D6 inhibitor	FDA approved (2022) for MDD	Rapid onset; novel glutamate mechanism; not specifically for TRD

7.2 Side Effect Profiles: Quick Reference

SIDE EFFECT	HIGHEST RISK	LOWEST RISK
Sexual dysfunction	Paroxetine, SSRIs, SNRIs	Bupropion, mirtazapine, agomelatine, vortioxetine
Weight gain	Mirtazapine, TCAs, paroxetine	Bupropion, fluoxetine (short-term)
Sedation	Mirtazapine, TCAs (amitriptyline), trazodone	Bupropion, fluoxetine, venlafaxine
Insomnia	Fluoxetine, bupropion, venlafaxine	Mirtazapine, trazodone
GI upset	SSRIs, SNRIs, vortioxetine	Mirtazapine (5-HT3 blockade = antiemetic)
QTc prolongation	Citalopram (>40mg), TCAs	SSRIs (except citalopram), bupropion
Seizure risk	Bupropion (dose-related), TCAs	SSRIs
Hypertension	Venlafaxine (dose-related), duloxetine	SSRIs
Discontinuation syndrome	Paroxetine, venlafaxine	Fluoxetine (long half-life, self-tapering)

7.3 Serotonin Syndrome (CRITICAL EXAM TOPIC)

Triad: Mental status changes + Autonomic instability + Neuromuscular hyperactivity

SYSTEM • FEATURES

Mental Agitation, confusion, delirium, anxiety

Autonomic Hyperthermia, tachycardia, diaphoresis, hypertension, diarrhea, mydriasis

Neuromuscular Myoclonus, hyperreflexia (especially lower limbs), clonus, tremor, rigidity

Hunter Criteria (diagnostic): Must have serotonergic agent PLUS one of:

1. Spontaneous clonus
2. Inducible clonus + agitation or diaphoresis
3. Ocular clonus + agitation or diaphoresis
4. Tremor + hyperreflexia
5. Hypertonia + temperature >38C + ocular or inducible clonus

Vs Neuroleptic Malignant Syndrome (NMS):

FEATURE	SEROTONIN SYNDROME	NMS
Onset	Hours (rapid)	Days to weeks (slow)
Causative agents	Serotonergic drugs	Dopamine antagonists
Neuromuscular	Myoclonus, hyperreflexia, CLONUS	Lead-pipe rigidity, bradyreflexia
Pupils	Mydriasis	Normal
GI	Diarrhea	Normal or ileus
Resolution	Hours to days (if drug stopped)	Days to weeks
CK elevation	Mild	Markedly elevated
Treatment	Cyproheptadine (5-HT _{2A} antagonist)	Dantrolene, bromocriptine

Management:

1. Stop all serotonergic agents
2. Supportive care (IV fluids, cooling, monitoring)
3. Benzodiazepines for agitation
4. Cyproheptadine (4-8mg PO, repeat q2h, max 32mg / day), specific antidote
5. Severe: intubation, paralysis, ICU

SECTION 8: PERSISTENT MOOD DISORDERS

8.1 Persistent Depressive Disorder (Dysthymia)

DSM-5 Criteria:

- Depressed mood for most of the day, more days than not, for ≥ 2 years (1 year in children/adolescents)
- Plus ≥ 2 of: poor appetite / overeating, insomnia / hypersomnia, low energy / fatigue, low self-esteem, poor concentration / indecisiveness, hopelessness
- Never without symptoms for >2 months during the 2-year period
- No manic / hypomanic episode ($\rightarrow$ cyclothymia or bipolar)
- "Double depression" = persistent depressive disorder + superimposed major depressive episode

Management: SSRIs / SNRIs + psychotherapy (CBT, CBASP, Cognitive Behavioral Analysis System of Psychotherapy specifically designed for chronic depression)

8.2 Cyclothymic Disorder

DSM-5 Criteria:

- ≥ 2 years of numerous periods of hypomanic symptoms and depressive symptoms that do NOT meet criteria for hypomanic or major depressive episode
- Never symptom-free for >2 months
- Never met criteria for a major depressive, manic, or hypomanic episode
- Significant distress or impairment

Clinical relevance:

- ~15-50% develop BP-I or BP-II over time
- Often diagnosed as "personality disorder" or "temperament"
- Treat with mood stabilizers if significant impairment; avoid antidepressant monotherapy

8.3 Disruptive Mood Dysregulation Disorder (DMDD): DSM-5

Rationale for creation: Address overdiagnosis of pediatric bipolar disorder in the USA. DMDD captures children with severe, chronic irritability who were being misclassified as bipolar.

Criteria:

- Severe recurrent temper outbursts (verbal / behavioral) grossly out of proportion
- Outbursts ≥ 3 times per week
- Mood between outbursts: persistently irritable or angry most of the day, nearly every day, observable by others
- Duration: ≥ 12 months (no period of ≥ 3 consecutive months without all criteria)
- Present in ≥ 2 of 3 settings (home, school, peers) and severe in ≥ 1
- Age at diagnosis: 6-18 years; age at onset: before 10 years
- Cannot coexist with ODD, intermittent explosive disorder, or bipolar disorder
- If criteria for both DMDD and ODD are met, DMDD diagnosis takes precedence

Key distinction from pediatric bipolar:

- DMDD = chronic, non-episodic irritability (the child's baseline)
- Bipolar = episodic irritability with distinct mood episodes
- DMDD prognostic trajectory → unipolar depression and anxiety in adulthood (NOT bipolar)

Management:

- Parent management training, CBT
- Stimulants (if comorbid ADHD)
- SSRIs (for irritability / depression)
- Atypical antipsychotics (risperidone, aripiprazole, for severe aggression, last resort)
- No mood stabilizer evidence

SECTION 9: RATING SCALES QUICK REFERENCE

SCALE	TYPE	ITEMS	RATER	KEY DETAILS
HAM-D (HDRS)	Depression severity	17 (original) or 21	Clinician	Gold standard for research; item 3 = suicide; score: 0-7 normal, 8-16 mild, 17-23 moderate, >24 severe
MADRS	Depression severity	10	Clinician	More sensitive to change; better for antidepressant trials; less somatic focus than HAM-D
PHQ-9	Depression screening + severity	9	Self-report	Mirrors DSM-5 criteria; score: 5-9 mild, 10-14 moderate, 15-19 moderately severe, 20-27 severe
BDI-II	Depression severity	21	Self-report	Cognitive focus; widely used in CBT research
YMRS	Mania severity	11	Clinician	Young Mania Rating Scale; gold standard for manic episodes
EPDS	Peripartum depression screening	10	Self-report	Edinburgh Postnatal Depression Scale; cutoff 10-13
GDS	Geriatric depression	15 or 30	Self-report	Geriatric Depression Scale; yes/no format; avoids somatic items that confound in elderly
C-SSRS	Suicide assessment	Structured interview	Clinician	Columbia; ideation + behavior subscales

SCALE	TYPE	ITEMS	RATER	KEY DETAILS
SAD PERSONS	Suicide risk	10 items	Clinician	Screening mnemonic-based; low predictive value individually

SECTION 10: ECT IN MOOD DISORDERS

Indications in Mood Disorders

1. **Severe depression with psychotic features**, response rate >80%
2. **Severe depression with suicidality**, fastest acting treatment
3. **Treatment-resistant depression**, after failed adequate pharmacotherapy
4. **Catatonia** (including depressive catatonia)
5. **Severe mania** (refractory to medications, delirious mania)
6. **Bipolar depression**, effective, especially with psychotic features
7. **Peripartum depression**, when pharmacotherapy risky or ineffective
8. **Nutritional compromise**, patient refusing food/fluids due to depression

Key Facts for Exam

- **Mechanism:** Not fully understood; enhances monoaminergic transmission, promotes neuroplasticity (BDNF increase), normalizes HPA axis, anticonvulsant theory (seizure threshold increases with successive treatments)
- **Electrode placement:** Bilateral (bifrontotemporal) > right unilateral for efficacy; right unilateral > bilateral for cognitive side effects
- **Course:** Typically 6-12 sessions, 2-3 times per week
- **Maintenance ECT:** For relapse prevention in patients who responded to ECT; typically weekly → biweekly → monthly
- **Absolute contraindication:** Raised intracranial pressure (pheochromocytoma is also frequently cited)
- **Side effects:** Headache (most common), nausea, muscle soreness, confusion (transient), retrograde amnesia (short-term), anterograde amnesia (transient); cognitive effects less with unilateral placement
- **Mortality:** ~0.002% per treatment (very safe; anesthesia risk)
- **Pregnancy:** Safe in all trimesters; one of the safest options for severe depression in pregnancy

Cross-references: NB-06 (Schizophrenia, for overlap on antipsychotics, metabolic syndrome)

Model Answers

QUESTION 1: "Pharmacological management of Bipolar Disorders." (10 marks / 20 marks LONG ESSAY)

EXAM STRATEGY

10-mark: Introduction (1), acute mania (3), bipolar depression (3), maintenance (2), special populations (1). 20-mark: All of the above expanded + algorithm flowchart + evidence citations + neurobiology of drug action + emerging treatments. Tables are your friend here, examiners can scan them fast. MUST mention lithium's anti-suicidal property, it's a differentiator answer.

Model Answer (10-mark version)

Introduction (1 mark)

Bipolar disorder is a chronic relapsing condition requiring distinct pharmacological strategies for each phase: acute mania, acute bipolar depression, and maintenance/prophylaxis. Treatment goals include symptom remission, functional recovery, and relapse prevention. The pharmacological armamentarium includes mood stabilizers (lithium, valproate, carbamazepine, lamotrigine) and atypical antipsychotics.

Acute Mania Management (3 marks)

SEVERITY	FIRST-LINE	SECOND-LINE
Mild-moderate	Lithium or valproate monotherapy	Carbamazepine, atypical antipsychotic monotherapy
Severe / psychotic	Atypical antipsychotic + lithium or valproate	ECT (if refractory or catatonic)
Acute agitation	Short-term benzodiazepines (lorazepam) + antipsychotic	IM olanzapine or haloperidol

- Discontinue antidepressants if present
- Quetiapine, olanzapine, risperidone, aripiprazole all have RCT evidence for acute mania
- Typical antipsychotics (haloperidol) still used widely but metabolic and neurological concerns
- Response assessment at 2-4 weeks; switch or combine if inadequate
- ECT for treatment-resistant mania, delirious mania, catatonic features

Bipolar Depression Management (3 marks)

This is the most challenging phase. Cardinal rule: NO antidepressant monotherapy (risk of manic switch and cycle acceleration).

AGENT	EVIDENCE LEVEL	NOTES
Quetiapine (300mg)	Strong (BOLDER trials)	Only drug with monotherapy evidence for both poles
Lithium	Moderate	Anti-suicidal benefit
Lamotrigine	Moderate	Better for prevention than acute; slow titration
Lurasidone	Strong (FDA approved)	With or without Li/VPA; take with food
Cariprazine (1.5mg)	Emerging (FDA approved)	D3-preferring partial agonist
OFC (olanzapine-fluoxetine)	Strong	Metabolic concerns

If antidepressant added: ALWAYS with mood stabilizer cover; prefer SSRIs or bupropion; short course; avoid TCAs/venlafaxine.

Maintenance Treatment (2 marks)

DRUG	PREVENTS MANIA	PREVENTS DEPRESSION	SPECIAL PROPERTIES
Lithium	+++	++	Anti-suicidal (unique); gold standard
Valproate	+++	+	Preferred in rapid cycling, mixed states
Lamotrigine	+	+++	Preferred for depressive-predominant course
Quetiapine	++	++	Adjunctive or monotherapy
Aripiprazole	++	+	Weight-neutral option

- Continuation for minimum 2 years after acute episode; lifelong in most patients after 2+ episodes
- Lithium remains gold standard, meta-analyses confirm unique anti-suicidal effect (Cipriani et al., 2013)
- Monitor adherence, non-adherence is the leading cause of relapse

Special Populations (1 mark)

- **Pregnancy:** Avoid valproate (NTD, cognitive impairment in offspring). Lithium with caution (Ebstein's risk ~0.1%); use lowest effective dose; level monitoring monthly and weekly near delivery. Lamotrigine relatively safer. ECT safe.

- **Elderly:** Lower doses, monitor renal function closely, drug interactions
 - **Rapid cycling:** Withdraw antidepressants, optimize mood stabilizers (valproate often preferred), check thyroid
 - **Children/adolescents:** Lithium, aripiprazole FDA-approved; valproate and quetiapine also used
-

Model Answer (20-mark LONG ESSAY expansion: additional sections)

Neurobiology of Drug Action (3 marks)

- Lithium: GSK-3 β inhibition, inositol depletion (Berridge hypothesis), neuroprotection via Bcl-2, modulation of glutamate, serotonin enhancement
- Valproate: GABAergic enhancement, sodium channel blockade, HDAC inhibition (epigenetic), PKC modulation
- Carbamazepine: Sodium channel blockade, reduces glutamate release
- Lamotrigine: Sodium channel-dependent glutamate modulation; why it works in depression but not mania remains debated
- Atypical antipsychotics: D2 antagonism + 5-HT_{2A} antagonism; quetiapine's NE reuptake inhibition (norquetiapine) may explain depression efficacy

Treatment Algorithm, Flowchart Format (2 marks)

1. Confirm diagnosis (rule out medical, substance-induced, unipolar)
2. Determine phase (mania / mixed → depression → maintenance)
3. Phase-specific treatment (as above)
4. Assess response at 2-4 weeks
5. Non-response → switch within class or add agent
6. Refractory → ECT or clozapine (mania) / esketamine or ECT (depression)

Emerging and Novel Treatments (2 marks)

- Cariprazine: D₃-preferring partial agonist; evidence for bipolar depression and maintenance
- Lumateperone: Novel mechanism (5-HT_{2A} antagonist, D₂ partial agonist, SERT inhibitor); recent trials in bipolar depression
- rTMS/TMS: Limited evidence in bipolar depression
- Ketamine/esketamine: Emerging evidence for acute bipolar depression/suicidality
- Pramipexole: Dopamine agonist with some evidence for bipolar depression

Evidence Base (2 marks)

- BALANCE trial: Lithium + valproate combination superior to valproate alone for maintenance

- BOLDER trials: Quetiapine monotherapy effective for bipolar depression
- STEP-BD: Antidepressant adjunctive therapy no better than placebo for bipolar depression (controversial finding)
- InterSePT: Clozapine reduces suicidality (in schizophrenia; extrapolated to refractory bipolar)

QUESTION 2: "Course and outcome of Bipolar Affective Disorder." (10 marks)

EXAM STRATEGY

Kraepelin's observations (1), episode characteristics (2), longitudinal course (3), prognostic factors (2), functional outcomes (1), impact of treatment (1). Use a table for prognostic factors, always impressive.

Model Answer

Historical Context (1 mark)

Kraepelin (1921) first systematically described the longitudinal course of manic-depressive illness, noting increasing episode frequency over time, shortening of inter-episode intervals, and variable inter-episode recovery. His observations laid the groundwork for the kindling model.

Episode Characteristics (2 marks)

PARAMETER · DATA

Average number of lifetime episodes 8-12

Manic episode duration (untreated) 4-6 months

Depressive episode duration 6-12 months

First episode polarity ~50% depressive

Time spent in depression vs mania 3:1 (BP-I), higher in BP-II

Polarity predominance Most patients have a predominant polarity (depressive > manic)

Longitudinal Course (3 marks)

- **Kindling model (Post & Weiss):** Early episodes triggered by stressors; later episodes become autonomous. Inter-episode interval shortens. This supports early intervention.
- **Episode frequency:** Tends to increase over time, especially without treatment. Average cycle length shortens from ~4 years (early) to <1 year (late).

- **Inter-episode functioning:** Only 40-50% achieve full functional recovery between episodes. Subsyndromal symptoms present in 50-60% (Judd et al., 2002, NIMH-CDS). Cognitive impairment (executive function, verbal memory) persists even in euthymia.
- **Rapid cycling:** Develops in 15-20% of patients, more common in women, associated with hypothyroidism and antidepressant use.
- **Mixed features:** Increasingly recognized as common (30-40% of episodes have some mixed features). Associated with worse outcomes.

Prognostic Factors (2 marks)

GOOD PROGNOSIS · POOR PROGNOSIS

Late onset Early onset (<25 years)

Few episodes Multiple episodes (>5)

Classical euphoric mania Mixed features

Good inter-episode functioning Residual symptoms between episodes

Treatment adherence Comorbid substance use (40-60% lifetime)

Good social support Personality disorder comorbidity

Absence of psychotic features Mood-incongruent psychotic features

Male sex (some studies) Rapid cycling

Functional and Psychosocial Outcomes (1 mark)

- 30-40% experience significant occupational disability
- Divorce rates 2-3x general population
- Reduced life expectancy by 10-15 years (cardiovascular disease, suicide)
- Suicide attempt rate 25-50%; completion rate 10-15%
- Economic burden substantial (direct + indirect costs)

Impact of Treatment on Course (1 mark)

- Lithium maintenance reduces relapse rate by 30-40%
 - Treatment adherence is the strongest modifiable prognostic factor
 - Psychoeducation (Colom et al.) significantly improves outcomes
 - Early intervention programs show promise in reducing episode burden
 - Adjunctive psychotherapy (CBT, IPSRT, FFT) improves adherence and reduces relapses
-

QUESTION 3: "Classify BPAD and discuss recent advances in treatment." (10 marks / 20 marks LONG ESSAY)

EXAM STRATEGY

Classification (3), recent advances in pharmacotherapy (4), neuromodulation (1), psychotherapy (1), biomarkers/precision medicine (1). For 20-mark: expand each section + add emerging compounds + staging models.

Model Answer (10 marks)

Classification of Bipolar Disorder (3 marks)

ICD-11 Classification:

- 6A60 Bipolar type I disorder
- 6A61 Bipolar type II disorder
- 6A62 Cyclothymic disorder

DSM-5 Classification:

- Bipolar I Disorder (at least one manic episode)
- Bipolar II Disorder (at least one hypomanic + one major depressive episode)
- Cyclothymic Disorder (chronic fluctuating mood, sub-threshold)
- Substance/Medication-Induced Bipolar
- Bipolar Due to Another Medical Condition
- Other Specified / Unspecified Bipolar

DSM-5 Specifiers: With anxious distress, with mixed features, with rapid cycling, with melancholic features, with atypical features, with mood-congruent/incongruent psychotic features, with catatonia, with peripartum onset, with seasonal pattern.

Key DSM-5 change from DSM-IV: "Mixed episode" replaced by "with mixed features" specifier applicable to any episode.

Recent Advances in Pharmacotherapy (4 marks)

ADVANCE · DETAIL

Cariprazine D3-preferring partial agonist; FDA approved for bipolar depression (2019), mania, and maintenance; emerging as versatile agent

Lumateperone Novel mechanism (5-HT_{2A}/D₂/SERT); Positive phase III trials for bipolar depression as adjunct to Li/VPA

Lurasidone FDA approved for bipolar depression (2013); favorable metabolic profile; take with food (350+ kcal)

Dextromethorphan/bupropion Glutamatergic mechanism; FDA approved for MDD (2022); trials ongoing for bipolar depression

Long-acting injectables Aripiprazole LAI (Abilify Maintena) and risperidone LAI for maintenance; improves adherence

Valproate restriction EMA/MHRA/NICE restrict valproate in women of childbearing age due to teratogenicity and neurodevelopmental effects

Esketamine/ketamine Emerging evidence for acute bipolar depression and suicidality; not yet approved for bipolar specifically

Lithium precision dosing Pharmacogenomic-guided lithium dosing; predicting response based on genetic markers

Advances in Neuromodulation (1 mark)

- rTMS: Growing evidence for bipolar depression (left DLPFC stimulation)
- Theta-burst stimulation (TBS): Accelerated protocols (Stanford SAINT protocol, 5 days vs 4-6 weeks)
- DBS: Experimental targets (subcallosal cingulate, ventral capsule/ventral striatum)
- ECT remains most effective physical treatment; ultrabrief pulse reduces cognitive effects

Advances in Psychotherapy (1 mark)

- Family-Focused Therapy (FFT, Miklowitz): Reduces relapse in early-course bipolar
- IPSRT (Frank): Social rhythm stabilization reduces recurrence
- CBT for bipolar (Lam et al.): Reduces depressive relapses
- Psychoeducation (Colom & Vieta): Group psychoeducation is evidence-based for relapse prevention
- Digital interventions: Smartphone-based mood monitoring (MONARCA, SIMPLe)

Advances in Understanding and Biomarkers (1 mark)

- Staging models (Berk, Kapczinski): Stage 0-4 progression; early intervention focus
 - Neuroimaging biomarkers: Amygdala hyperactivity, PFC hypoactivity; potential for treatment response prediction
 - Polygenic risk scores: Improving but not yet clinical utility
 - Circadian rhythm interventions: Dark therapy, blue-light blocking for acute mania
 - Inflammatory markers: CRP, IL-6 as potential predictors of treatment response
 - Metabolomics/proteomics: Emerging signatures for bipolar subtypes
-

QUESTION 4: "Genetics of Bipolar disorders." (10 marks)

EXAM STRATEGY

Heritability data (2), family / twin / adoption studies (3), molecular genetics (3), genetic overlap (1), clinical implications (1).

Model Answer

Heritability (2 marks)

Bipolar disorder is one of the most heritable psychiatric conditions, with estimated heritability of approximately 85% from twin studies. This is higher than most other psychiatric disorders (schizophrenia ~80%, MDD ~40%, anxiety disorders ~30-40%).

Family Studies (1 mark)

- First-degree relatives have 5-10x risk of developing BPAD compared to general population
- Risk of any mood disorder in first-degree relatives: ~25%
- First-degree relatives also at increased risk for unipolar depression, schizoaffective disorder
- The closer the genetic relationship, the higher the concordance

Twin Studies (1 mark)

- Monozygotic concordance: 40-70% (varies by study)
- Dizygotic concordance: 5-10%
- MZ concordance <100% confirms environmental factors play a role despite high heritability
- The gap between MZ and DZ concordance provides the strongest evidence for genetic contribution

Adoption Studies (1 mark)

- Biological relatives of bipolar adoptees have higher rates of mood disorders than adoptive relatives
- Mendlewicz & Rainer (1977): foundational adoption study showing genetic transmission
- Environmental contribution exists but is smaller than genetic contribution

Molecular Genetics (3 marks)

Genome-Wide Association Studies (GWAS):

- Bipolar Disorder Working Group of PGC: identified 64+ genome-wide significant loci (latest meta-analysis)

- Polygenic architecture: hundreds of common variants, each with small effect (OR 1.05-1.15)

Key genes and pathways:

GENE/LOCUS	FUNCTION	NOTES
CACNA1C	Voltage-gated calcium channel (L-type)	Most replicated; shared with schizophrenia and MDD
ANK3	Ankyrin G, axon initial segment	Critical for action potential generation
CLOCK genes (CLOCK, PER3, CRY1)	Circadian rhythm regulation	Links to circadian disruption in bipolar
BDNF	Neurotrophic factor	Val66Met polymorphism; affects neuroplasticity
DISC1	Centrosome function, neurodevelopment	Shared with schizophrenia
DGKH	Diacylglycerol kinase	Second messenger pathway (lithium target)
NRG1	Neuregulin, synaptic function	Shared with schizophrenia
ODZ4	Cell-cell signaling	Identified in PGC GWAS

Rare variants: Copy number variants (CNVs), duplications at 16p11.2, deletions at 3q29, 15q11.2, overlap with schizophrenia and ASD.

Genetic Overlap with Other Disorders (1 mark)

- Bipolar shares more genetic variance with schizophrenia than with unipolar depression
- Cross-Disorder Group of PGC: identified shared loci across BPAD, schizophrenia, MDD, ADHD, ASD
- Genetic correlation: BPAD-SCZ ~0.68, BPAD-MDD ~0.44
- Supports dimensional/spectrum models rather than strict categorical boundaries
- Schizoaffective disorder may represent genetic intermediate

Clinical Implications (1 mark)

- Genetic counseling: Empirical risk figures for families (10-15% risk for offspring of one affected parent; 50-70% if both parents affected)
- Pharmacogenomics: HLA-B*1502 screening before carbamazepine; CYP2D6/CYP2C19 for antidepressant metabolism
- Polygenic risk scores: Not yet clinical utility but improving; may predict illness course, treatment response

- Gene-environment interaction: Childhood adversity + genetic vulnerability = highest risk
- Ethical considerations: Genetic testing, discrimination, insurance implications

QUESTION 5: "Persistent mood disorders." (10 marks)

EXAM STRATEGY

Classification (2), dysthymia/PDD (3), cyclothymia (3), DMDD (2). Common exam question, comprehensive coverage expected.

Model Answer

Classification (2 marks)

Persistent (chronic) mood disorders are characterized by prolonged, sub-threshold mood disturbance. DSM-5 includes:

1. **Persistent Depressive Disorder (PDD)**, replaces DSM-IV dysthymic disorder; also incorporates chronic major depression
2. **Cyclothymic Disorder**, chronic fluctuation between sub-threshold hypomania and depression
3. **Disruptive Mood Dysregulation Disorder (DMDD)**, DSM-5 addition; children with chronic severe irritability

ICD-11: Dysthymic disorder (6A72), Cyclothymic disorder (6A62)

Persistent Depressive Disorder / Dysthymia (3 marks)

Criteria: Depressed mood more days than not for ≥ 2 years (1 year in children/adolescents), plus ≥ 2 of: appetite changes, sleep changes, low energy, low self-esteem, poor concentration, hopelessness. Never symptom-free for >2 months.

DSM-5 specifiers: With anxious distress, with mixed features, with melancholic features, with atypical features, with mood-congruent/incongruent psychotic features, in partial/full remission, early/late onset, with pure dysthymic syndrome vs with persistent major depressive episode vs with intermittent major depressive episodes.

Epidemiology: Lifetime prevalence 3-6%; F:M = 2:1; onset often insidious in adolescence or early adulthood.

Double depression: PDD + superimposed major depressive episode. Present in ~75% of PDD patients. Worse prognosis than MDE alone, higher relapse rates, poorer treatment response.

Management:

- SSRIs/SNRIs first-line (similar efficacy to MDE treatment)

- Psychotherapy: CBT, CBASP (McCullough, Cognitive Behavioral Analysis System of Psychotherapy, specifically designed for chronic depression), IPT
- Combined pharmacotherapy + psychotherapy superior to either alone (Keller et al., 2000, nefazodone + CBASP study)
- Duration of treatment: prolonged; high relapse risk on discontinuation

Cyclothymic Disorder (3 marks)

Criteria: $\geq$ 2 years (1 year in adolescents) of numerous periods with hypomanic symptoms and depressive symptoms not meeting full criteria for hypomanic or major depressive episode. Never symptom-free for >2 months. No full manic, hypomanic, or major depressive episode during initial 2-year period.

Epidemiology: Lifetime prevalence 0.4-1%; equal sex distribution; onset usually adolescence/early adulthood.

Course: 15-50% progress to BP-I or BP-II. Chronic and fluctuating. Often misdiagnosed as personality disorder (especially borderline, the affective instability overlap).

Relationship to bipolar: Considered part of the bipolar spectrum. Family studies show increased bipolar disorder in relatives. Temperament theories (Akiskal) place cyclothymia on a continuum with bipolar.

Management:

- Mood stabilizers (lithium, valproate) if significant functional impairment
- Avoid antidepressant monotherapy (risk of destabilization/switch)
- Psychoeducation about mood patterns
- Social rhythm stabilization
- Psychotherapy supportive but limited evidence

Disruptive Mood Dysregulation Disorder, DMDD (2 marks)

Rationale: Created in DSM-5 to address the overdiagnosis of pediatric bipolar disorder. Captures children with severe chronic irritability who were being misclassified.

Criteria: Severe recurrent temper outbursts $\geq$ 3x/week; persistently irritable/angry mood between outbursts (most of day, nearly every day); present $\geq$ 12 months (no $\geq$ 3-month symptom-free period); in $\geq$ 2/3 settings; age 6-18 years, onset before 10 years.

Key distinction: DMDD = chronic non-episodic irritability (baseline state) vs pediatric bipolar = episodic mood changes with discrete episodes.

Prognostic trajectory: Develops into unipolar depression and anxiety disorders in adulthood, NOT bipolar disorder. This distinguishes it fundamentally from true pediatric bipolar.

Management: Parent management training, CBT; stimulants if comorbid ADHD; SSRIs for depression/anxiety component; atypical antipsychotics for severe aggression as last resort.

QUESTION 6: "Recent advances in management of mood disorders." (10 marks)

EXAM STRATEGY

Cover BOTH bipolar and unipolar; pharmacological (4), neuromodulation (2), psychotherapy (1), digital/precision (1), guidelines (1), emerging (1).

Model Answer

Pharmacological Advances (4 marks)

Bipolar disorder:

- Cariprazine: D3-preferring partial agonist; first drug showing superiority for both bipolar mania and depression with maintenance approval
- Lumateperone: Novel multimodal mechanism; adjunctive for bipolar depression
- Lurasidone: Established for bipolar depression; favorable metabolic profile
- Long-acting injectable antipsychotics: Aripiprazole LAI for bipolar maintenance, addresses adherence
- Valproate restrictions: EMA/MHRA guidelines restricting use in women of childbearing age

Unipolar depression:

- Esketamine (Spravato): Intranasal NMDA antagonist; FDA approved for TRD (2019) and MDD with suicidal ideation (2020); rapid onset within hours
- Brexanolone (Zulresso): IV allopregnanolone; FDA approved for postpartum depression (2019); GABA-A modulation
- Zuranolone (Zurzuvae): Oral neuroactive steroid; FDA approved for PPD (2023); 14-day course
- Dextromethorphan/bupropion (Auvelity): Glutamate modulation; FDA approved for MDD (2022); faster onset than traditional antidepressants
- Psilocybin: Breakthrough therapy designation for TRD; phase II/III trials showing sustained response after 1-2 sessions with psychotherapy support

Neuromodulation Advances (2 marks)

- Accelerated TMS protocols: Stanford SAINT protocol, 10 sessions/day for 5 days; rapid response in TRD (remission rates ~80% in initial trials)
- Theta-burst stimulation (TBS): Equivalent efficacy to standard rTMS in shorter session time
- MST (Magnetic Seizure Therapy): Aims to produce ECT-like efficacy with fewer cognitive effects; under investigation

- tDCS (Transcranial Direct Current Stimulation): Modest evidence for depression; home-based protocols being tested
- Focused ultrasound: Experimental; non-invasive deep brain stimulation

Psychotherapy Advances (1 mark)

- Internet-delivered CBT (iCBT): Non-inferior to face-to-face for mild-moderate depression; scalable
- Behavioral activation: Standalone treatment non-inferior to CBT for depression (COBRA trial)
- CBASP: Specifically designed for chronic depression; superior to antidepressant alone when combined
- Digital phenotyping: Smartphone sensors detecting mood episodes in bipolar (passive monitoring)

Precision Medicine and Biomarkers (1 mark)

- Pharmacogenomics: CYP2D6 / CYP2C19 testing for antidepressant selection (GeneSight, etc.)
- Inflammatory biomarkers: CRP-guided treatment selection, high CRP may predict better response to anti-inflammatory or duloxetine
- Staging models: Berk staging for bipolar; early intervention paradigm
- Machine learning: Predicting treatment response from clinical + neuroimaging data

Guideline Updates (1 mark)

- CANMAT 2018: Updated bipolar treatment guidelines; lurasidone and cariprazine incorporated
- NICE 2023 update: Valproate restrictions formalized
- APA Practice Guidelines: Updated depression management incorporating esketamine
- WHO mhGAP: Simplified algorithms for non-specialist settings (relevant to Indian context)

Emerging Paradigms (1 mark)

- Chronotherapy: Combined sleep deprivation + light therapy + sleep phase advance, rapid antidepressant effect
 - Anti-inflammatory approaches: Celecoxib, minocycline augmentation trials positive
 - Gut-brain axis: Probiotics ("psychobiotics") as adjunctive treatment, early evidence
 - Ketamine clinics: Growing off-label IV ketamine use for TRD worldwide
-

QUESTION 7: "Disruptive Mood Dysregulation Disorder." (10 marks)

EXAM STRATEGY

Rationale (2), criteria (3), differential (2), course (1), management (2).

Model Answer

Background and Rationale (2 marks)

DMDD was introduced in DSM-5 (2013) to address the dramatic increase in pediatric bipolar disorder diagnoses in the United States (a 40-fold increase between 1994-2003). Leibenluft et al. at NIMH identified that many children diagnosed with bipolar actually had severe chronic irritability (termed "Severe Mood Dysregulation" in research) rather than episodic mood disturbance. Follow-up studies showed these children developed unipolar depression and anxiety in adulthood, NOT bipolar disorder, confirming they were a distinct clinical entity.

Diagnostic Criteria (3 marks)

1. Severe recurrent temper outbursts manifested verbally (rage) and/or behaviorally (physical aggression) grossly out of proportion in intensity or duration to the situation
2. Temper outbursts inconsistent with developmental level
3. Outbursts occur ≥ 3 times per week on average
4. Mood between outbursts is persistently irritable or angry most of the day, nearly every day, and observable by others (parents, teachers, peers)
5. Criteria present for ≥ 12 months; no period of ≥ 3 consecutive months without all symptoms
6. Present in ≥ 2 of 3 settings (home, school, peers) and severe in at least one
7. Age 6-18 years at diagnosis; onset of symptoms before age 10 years
8. Never a distinct period lasting >1 day meeting full criteria for manic/hypomanic episode
9. Not better explained by another mental disorder; does not coexist with ODD, intermittent explosive disorder, or bipolar disorder

Differential Diagnosis (2 marks)

CONDITION · DISTINGUISHING FEATURE

Bipolar disorder (pediatric) Episodic mood changes with distinct elevated/expansive mood; episodes demarcated from baseline

ODD Less severe; primarily defiant/vindictive; does NOT require persistent irritable mood between outbursts; DMDD takes precedence if both criteria met

Intermittent Explosive Disorder Impulsive outbursts only; does NOT require chronic irritable mood between episodes

ADHD Impulsivity and emotional dysregulation common but not the defining feature; frequently comorbid with DMDD

ASD Meltdowns often sensory / routine disruption; consider broader developmental picture

Conduct Disorder Deliberate, planned antisocial behavior vs reactive outbursts

Course and Prognosis (1 mark)

- Prevalence: 2-5% of children (community studies)
- Develops into unipolar depression and anxiety disorders in adulthood (NOT bipolar, this is the critical distinction)
- High comorbidity with ADHD, ODD (when diagnosed separately), anxiety disorders
- Associated with significant academic and social impairment
- Suicide risk elevated compared to non-DMDD peers

Management (2 marks)

Psychosocial (first-line):

- Parent management training (structured behavioral interventions)
- CBT (anger management, emotion regulation skills)
- School-based interventions

Pharmacological:

- Stimulants: If comorbid ADHD (common); methylphenidate may improve irritability and outbursts
 - SSRIs: For underlying depressive / anxious component
 - Atypical antipsychotics: Risperidone, aripiprazole for severe aggression / irritability, should be last resort due to metabolic effects in children
 - NO mood stabilizer evidence for DMDD (unlike bipolar)
-

QUESTION 8: "Define TRD. Discuss staging and management." (10 marks / 20 marks LONG ESSAY)

EXAM STRATEGY

Definition + pseudo-resistance (2), staging models (3), management algorithm (4), emerging treatments (1). For 20 marks: expand each step with evidence, add neuromodulation details, precision medicine.

Model Answer

Definition (2 marks)

Treatment-Resistant Depression (TRD) is defined as failure to achieve adequate clinical response (typically <50% improvement in depression rating scale scores) after at least 2 adequate antidepressant trials from different pharmacological classes, each at adequate dose for adequate duration (minimum 6-8 weeks at therapeutic dose with confirmed adherence).

Pseudo-resistance (must exclude before diagnosing TRD):

- Inadequate dose or duration of current trial
- Non-adherence (common, up to 50% of patients)
- Misdiagnosis: bipolar depression, medical causes (hypothyroidism, anemia, B12 deficiency, Cushing's, cerebral lesion), substance use, personality disorder
- Untreated comorbidities: anxiety disorders, substance use, chronic pain, sleep disorders

Staging Models (3 marks)

Thase-Rush Model (most commonly cited):

STAGE · DEFINITION

Stage I Failure of ≥ 1 adequate SSRI trial

Stage II Failure of ≥ 1 adequate trial from a different class (e.g., SNRI)

Stage III Failure of adequate tricyclic antidepressant trial

Stage IV Failure of adequate MAOI trial

Stage V Failure of bilateral ECT course

Massachusetts General Hospital (MGH) Staging Method:

- Quantitative scoring system: 1 point per failed adequate trial, 0.5 points for optimization, 0.5 for augmentation, 3 points for ECT failure
- Total score represents overall treatment resistance burden
- More granular and better suited for research

European Staging Model (Souery):

- Considers number of failed adequate trials and augmentation strategies
- Integrates chronicity as a factor

Management Algorithm (4 marks)

Step 1, Optimization (confirm adequacy):

- Maximize dose to highest tolerated

- Ensure ≥ 8 weeks at therapeutic dose
- Verify adherence (serum levels if available, e.g., TCA levels)
- Address comorbidities (anxiety, substance use, sleep, medical)
- Psychotherapy if not already engaged

Step 2, Switching:

- Indicated when NO response to current agent
- Within class: escitalopram $\rightarrow$ sertraline (limited evidence for benefit)
- Between classes: SSRI $\rightarrow$ SNRI $\rightarrow$ bupropion $\rightarrow$ TCA $\rightarrow$ MAOI
- Allow adequate washout for MAOI (2 weeks from most drugs; 5 weeks from fluoxetine)

Step 3, Augmentation:

- Indicated when PARTIAL response (some improvement but inadequate)
- **Lithium augmentation:** Most evidence-based; 0.6-0.8 mEq/L; response in 2-4 weeks; Bauer et al. meta-analysis confirms efficacy
- **Atypical antipsychotic augmentation:** Aripiprazole (2-5mg), quetiapine (50-300mg), olanzapine (2.5-10mg), brexpiprazole (1-3mg), all FDA-approved for augmentation
- **Thyroid augmentation:** T3 (liothyronine) 25-50 mcg/day; even in euthyroid patients; accelerates and augments response

Step 4, Combination antidepressants:

- SSRI/SNRI + mirtazapine ("California rocket fuel" = venlafaxine + mirtazapine)
- SSRI + bupropion (complementary mechanisms, may reduce sexual side effects)
- NEVER combine SSRI + MAOI or SNRI + MAOI

Step 5, Esketamine:

- Intranasal (Spravato); FDA approved 2019
- Used WITH concurrent oral antidepressant
- REMS program: administer in certified setting, monitor 2 hours
- Rapid onset (hours to days); dosing: 56mg or 84mg twice weekly initially $\rightarrow$ weekly $\rightarrow$ biweekly
- Side effects: dissociation, dizziness, sedation, nausea, increased BP

Step 6, ECT:

- Gold standard for severe/refractory depression
- 70-90% response rate in TRD
- Bilateral > right unilateral for efficacy
- Especially indicated: psychotic features, severe suicidality, catatonia, nutritional compromise

- Maintenance ECT for sustained benefit

Step 7, Experimental/emerging:

- rTMS (left DLPFC high-frequency or right DLPFC low-frequency)
- VNS (implanted; FDA approved for chronic TRD unresponsive to ≥ 4 treatments)
- DBS (investigational; subcallosal cingulate)
- Psilocybin-assisted therapy (phase II/III)
- IV ketamine (off-label)

Emerging Treatments (1 mark)

- Accelerated TMS (SAINT protocol): 5-day course; promising open-label and RCT data
- Psilocybin: Phase II data showing sustained antidepressant effects after 1-2 sessions
- MDMA-assisted therapy: Primarily PTSD; some MDD overlap data
- Anti-inflammatory augmentation: Celecoxib, minocycline in clinical trials
- Zuranolone: Oral neuroactive steroid; approved for PPD, trials in MDD ongoing

QUESTION 9: "Depression in the elderly: clinical features, differential diagnosis, management." (10 marks)

EXAM STRATEGY

Epidemiology / context (1), clinical features (3), differential diagnosis (3), management (3).

Model Answer

Epidemiology and Context (1 mark)

Depression affects 10-15% of community-dwelling elderly and up to 40% of those in institutional care. Late-onset depression (onset >60 years) may represent a distinct clinical entity linked to cerebrovascular disease (vascular depression hypothesis, Alexopoulos, 1997). Depression in elderly is underdiagnosed and undertreated due to diagnostic complexity and attribution of symptoms to "normal aging."

Clinical Features (3 marks)

Differences from younger adults:

FEATURE	ELDERLY DEPRESSION	YOUNGER ADULT DEPRESSION
Mood complaint	May deny sadness; presents with apathy, withdrawal	Overt sadness more common
Somatic symptoms	Prominent, pain, fatigue, GI complaints, weight loss	Less prominent

FEATURE	ELDERLY DEPRESSION	YOUNGER ADULT DEPRESSION
Cognitive complaints	Common, "pseudodementia"; memory, concentration	Present but less prominent
Anxiety	Very common (up to 50% comorbidity)	Common
Psychotic features	More common in elderly depression (hallucinations, delusions of guilt/poverty/nihilism)	Less common
Psychomotor changes	Retardation often prominent	Variable
Hypochondriasis	Common	Less common
Executive dysfunction	Prominent in vascular depression	Variable
Suicidal behavior	Higher completion rate; less warning; more lethal methods	More attempts, lower completion

Vascular depression features:

- Late onset (no prior psychiatric history)
- Executive dysfunction out of proportion
- Psychomotor retardation
- Apathy > sadness
- White matter hyperintensities on MRI
- Associated vascular risk factors (HTN, DM, smoking)
- Poorer response to antidepressants
- Greater disability

Differential Diagnosis (3 marks)

CONDITION · KEY DIFFERENTIATORS

Dementia See pseudodementia vs dementia table; gradual onset, confabulation, temporal gradient, no diurnal variation

Medical illness Hypothyroidism, B12/folate deficiency, Parkinson's, stroke, cancer, chronic pain; always screen medically

Medication-induced Beta-blockers, corticosteroids, interferon, benzodiazepines, anticholinergics; temporal relationship

Bereavement/adjustment Temporal relationship to loss; grief has waves rather than persistent flatness

Apathy syndrome Motivational deficit without sadness or guilt; can be independent of depression

Substance use Alcohol use disorder in elderly often hidden; benzodiazepine dependence

Delirium Acute onset, fluctuating course, altered consciousness; always consider in elderly

Pseudodementia vs dementia, key distinguishing features:

- Pseudodementia: rapid onset, "don't know" answers, inconsistent memory, psychiatric history, responds to antidepressants
- Dementia: insidious onset, near-miss answers / confabulates, progressive, temporal gradient memory loss, does not respond to antidepressants
- CRITICAL: pseudodementia patients have 30-50% risk of developing true dementia within 5 years

Management (3 marks)

Pharmacological:

- "Start low, go slow, but go", aim for therapeutic doses
- **SSRIs preferred:** Escitalopram, sertraline (fewest drug interactions)
- **Mirtazapine:** Good choice if insomnia, poor appetite, weight loss (sedating, appetite-stimulating)
- **Duloxetine:** Useful if comorbid neuropathic pain
- **Venlafaxine:** Effective but monitor BP
- **Avoid TCAs:** Anticholinergic effects (confusion, falls, urinary retention, constipation, cardiac); orthostatic hypotension
- **Avoid benzodiazepines:** Falls, confusion, paradoxical disinhibition, dependence
- **Monitor for hyponatremia:** SSRIs / SNRIs cause SIADH; higher risk in elderly; check sodium at 1-2 weeks
- **ECT:** Safe and highly effective in elderly; may be first-line for severe depression with psychotic features, suicidality, or inability to tolerate medications

Psychotherapy:

- CBT adapted for elderly (slower pace, written materials, involve family)
- Problem-solving therapy
- Interpersonal therapy (IPT)
- Behavioral activation
- Reminiscence therapy (group or individual)

Other considerations:

- Address social isolation, bereavement, loss of role
- Treat comorbid medical conditions
- Optimize sensory function (hearing aids, glasses)
- Exercise (moderate evidence for depression improvement)
- Suicide risk: higher in elderly men; always assess access to means; lethal methods more common in elderly

QUESTION 10: "Suicide: risk factors, assessment scales, prevention strategies." (10 marks)

EXAM STRATEGY

Epidemiology (1), risk factors (3), assessment scales (3), prevention (3). Indian data expected, include NCRB, MHCA Section 115.

Model Answer

Epidemiology (1 mark)

Globally: ~800,000 deaths by suicide annually (WHO). India: ~12/100,000 population (NCRB 2021), accounting for ~36% of global female suicides and ~24% of male suicides. Peak age: 18-30 years (younger than Western countries). Suicide decriminalized in India under Mental Healthcare Act 2017, Section 115, attempted suicide presumed to be under severe stress.

Risk Factors (3 marks)

SAD PERSONS mnemonic:

- Sex: Male (completed); Female (attempted)
- Age: >65 years and 15-30 years
- Depression/psychiatric illness
- Previous attempt (STRONGEST single predictor)
- Ethanol/substance abuse
- Rational thinking loss (psychosis)
- Social supports lacking
- Organized plan with intent
- No spouse (single / divorced / widowed)
- Sickness (chronic illness, chronic pain)

Additional risk factors:

- Family history of suicide
- Access to lethal means (firearms, pesticides)

- Recent psychiatric discharge (first 1-4 weeks highest risk)
- Hopelessness (Beck Hopelessness Scale, more predictive than depression severity alone)
- Impulsivity/aggression traits
- History of childhood abuse/neglect
- LGBTQ+ identity (especially youth)
- Media contagion (Werther effect)

Indian-specific:

- Farming suicides (debt, crop failure, drought, NCRB reports)
- Student suicides (academic pressure, competitive exam failure)
- Dowry-related deaths
- Common methods: hanging, poisoning (organophosphates, rural), self-immolation

Assessment Scales (3 marks)

SCALE	TYPE	KEY FEATURES
Columbia Suicide Severity Rating Scale (C-SSRS)	Clinician-rated	Gold standard; rates ideation (5 levels) + behavior (5 levels); FDA endorsed; tracks change over time
SAD PERSONS	Clinician checklist	Mnemonic-based; 10 risk factors scored 0 or 1; guides disposition; low individual predictive value
Beck Scale for Suicide Ideation (BSI)	Self-report (19 items)	Measures intensity, frequency, duration of ideation; good for research
Beck Hopelessness Scale (BHS)	Self-report (20 items)	Measures hopelessness, more predictive of suicide than depression severity
PHQ-9 Item 9	Screening	Single item on suicidal thoughts; sensitivity for screening in primary care
MINI Suicidality Module	Clinician-rated	Part of MINI structured interview; quick categorization
Nurses' Global Assessment of Suicide Risk (NGASR)	Nursing tool	15-item inpatient risk assessment

Clinical assessment framework (beyond scales):

1. Ideation: passive vs active
2. Plan: specific method, timeframe

3. Means: access to intended method
4. Intent: strength of desire, reasons for living
5. Preparedness: giving possessions, writing notes
6. Protective factors: children, religion, social support
7. Recent changes: sudden calm, recent discharge, recent loss

Prevention Strategies (3 marks)

Universal (population-level):

- Means restriction: Most effective single intervention, pesticide bans (Sri Lanka model reduced suicides by 50%), bridge barriers (Golden Gate Bridge), medication blister packs, firearm storage laws
- Media guidelines: WHO recommendations; Papageno effect (responsible reporting reduces imitation) vs Werther effect (sensationalized reporting increases imitation)
- School-based programs: Signs of Suicide (SOS), Yellow Ribbon
- Gatekeeper training: QPR (Question, Persuade, Refer), ASIST, Mental Health First Aid, training non-mental health professionals to identify and refer
- Crisis helplines: iCall, Vandrevalla Foundation, AASRA (India)

Selective (targeted to high-risk groups):

- Screening in primary care (PHQ-9, EPDS for peripartum)
- Training primary care physicians in depression detection and management
- Follow-up contact after suicide attempt (WHO BIC study, brief intervention and contact)
- Military / police / medical professional targeted programs

Indicated (individual-level):

- Safety planning (Stanley-Brown model, NOT "no-suicide contracts" which are ineffective)
 - Pharmacotherapy: Clozapine (FDA-approved for suicide reduction in schizophrenia, InterSePT trial), Lithium (reduces suicide in mood disorders, Cipriani meta-analysis), Ketamine / esketamine (rapid reduction in suicidal ideation)
 - Psychotherapy: DBT (gold standard for repeated self-harm/BPD), CBT-SP (CBT for Suicide Prevention), CAMS (Collaborative Assessment and Management of Suicidality)
 - Post-discharge follow-up: Contact within 48 hours; bridging appointments; intensive outpatient
-

QUESTION 11: "Newer antidepressants: classification and clinical applications." (10 marks)

EXAM STRATEGY

Define "newer" broadly (2), classify by mechanism (3), clinical applications (3), evidence summary (2).

Model Answer

Introduction (1 mark)

"Newer antidepressants" encompasses agents developed beyond the traditional SSRI/SNRI/TCA/MAOI framework, with novel mechanisms of action. These include multimodal serotonergic agents, glutamate modulators, neurosteroids, and psychedelic-assisted therapies.

Classification by Mechanism (4 marks)

DRUG	MECHANISM	YEAR APPROVED	PRIMARY INDICATION
Vortioxetine	Multimodal: SERT inhibition + 5-HT1A agonist + 5-HT3/7 antagonist + 5-HT1B partial agonist	2013	MDD (especially with cognitive symptoms)
Agomelatine	MT1/MT2 melatonin agonist + 5-HT2C antagonist	2009 (EU)	MDD (circadian-related)
Bupropion/dextrometorphan (Auvelity)	NMDA antagonist + sigma-1 agonist + NDRI	2022	MDD
Esketamine (Spravato)	NMDA receptor antagonist	2019	TRD, MDD with suicidal ideation
Brexanolone (Zulresso)	GABA-A PAM (allopregnanolone analog)	2019	Postpartum depression
Zuranolone (Zurzuvae)	Oral GABA-A PAM (neuroactive steroid)	2023	Postpartum depression
Cariprazine	D3-preferring partial agonist + D2 partial agonist + 5-HT1A partial agonist	2019 (bipolar depression)	Bipolar depression, schizophrenia
Psilocybin	5-HT2A agonist (classical psychedelic)	Breakthrough therapy (not yet approved)	TRD (investigational)

Clinical Applications (3 marks)

Vortioxetine:

- Unique pro-cognitive effects, improves processing speed, executive function, attention in MDD
- Useful when cognitive symptoms are prominent
- Better sexual dysfunction profile than SSRIs
- Dose-related GI side effects (nausea)
- 5-20mg/day

Agomelatine:

- Resynchronizes circadian rhythm (MT1/MT2 agonism)
- No sexual dysfunction, no weight gain, no discontinuation syndrome
- Monitor LFTs (hepatotoxicity risk, check at baseline, 3, 6, 12, 24 weeks)
- Useful for depression with sleep-wake cycle disruption
- Not available in USA; available in India
- 25-50mg at bedtime

Esketamine:

- Rapid onset (hours); glutamate-mediated neuroplasticity
- For TRD (with concurrent oral antidepressant) and MDD with acute suicidal ideation
- Nasal spray; REMS program, administer in clinic, monitor 2 hours
- Side effects: dissociation, dizziness, sedation, nausea, BP elevation
- Dosing: 56mg or 84mg; twice weekly (wk 1-4) → weekly (wk 5-8) → weekly or biweekly

Brexanolone:

- First drug specifically approved for postpartum depression
- IV infusion over 60 hours; inpatient setting
- Rapid response (within 24-48 hours)
- Very expensive; limited availability
- REMS: sedation monitoring, pulse oximetry

Zuranolone:

- First ORAL medication for postpartum depression
- 14-day course (50mg nightly)
- Rapidly effective
- Can be taken at home (unlike brexanolone)

Bupropion/dextromethorphan:

- Glutamate mechanism + monoaminergic effects

- Faster onset than traditional antidepressants (1 week in trials)
- For MDD (not specifically TRD)

Evidence Summary and Clinical Decision-Making (2 marks)

- Cognitive symptoms dominant → vortioxetine
- Circadian disruption/sleep issues → agomelatine
- Treatment-resistant → esketamine (with oral antidepressant)
- Postpartum depression → brexanolone (severe, inpatient) or zuranolone (oral)
- Rapid onset needed → esketamine or bupropion/dextromethorphan
- Bipolar depression → cariprazine, lurasidone
- These agents expand the toolkit but do NOT replace first-line SSRIs/SNRIs for initial treatment of MDD

QUESTION 12: "Lithium: mechanism of action, monitoring, side effects, toxicity and management." (10 marks)

EXAM STRATEGY

Mechanism (3), monitoring (2), side effects (2), toxicity + management (3). This is a standalone lithium question, go deep, use tables.

Model Answer

Mechanism of Action (3 marks)

MECHANISM · DETAIL

GSK-3beta inhibition Most established mechanism. GSK-3beta regulates apoptosis, glycogen metabolism, circadian rhythms, neurogenesis. Inhibition → neuroprotection, circadian stabilization

Inositol depletion Berridge hypothesis: lithium inhibits inositol monophosphatase → depletes free inositol → reduces phosphoinositide (PI) turnover → dampens overactive receptor-mediated signaling. Effect is greatest in most active neurons.

Neuroprotection Increases Bcl-2 (anti-apoptotic protein), increases NAA, increases gray matter volume on MRI. May prevent or slow neurodegeneration.

Glutamate modulation Reduces excitatory glutamatergic transmission; protects against glutamate excitotoxicity

Serotonergic enhancement Increases tryptophan uptake, enhances 5-HT release, modifies 5-HT receptor sensitivity

CREB and BDNF Increases CREB (transcription factor) and BDNF expression → enhanced synaptic plasticity

Anti-suicidal effect Specific to lithium; mechanism incompletely understood; may involve serotonergic enhancement + reduced impulsivity / aggression

Monitoring Schedule (2 marks)

TEST	BASELINE	REGULAR MONITORING	NOTES
Serum lithium level	Before starting, then 5-7 days after initiation/dose change	Every 3-6 months (stable), every 1-3 months (unstable)	12-hour post-dose trough
Renal function (creatinine, eGFR, urine osmolality)	Yes	Every 6 months	Monitor for nephrogenic DI and CKD
Thyroid (TSH +/- free T4)	Yes	Every 6 months	Hypothyroidism in 20-30%
Calcium + parathyroid hormone	Yes	Every 12 months (6 monthly if abnormal)	Hyperparathyroidism
ECG	Yes (>40 years or cardiac history)	Annual in elderly	T-wave flattening, sinus node dysfunction
FBC	Yes	Annual	
Weight/BMI	Yes	Every visit	Weight gain common
Pregnancy test	Yes (women of child-bearing age)	As needed	Teratogenicity
Electrolytes (Na, K)	Yes	With renal function	

Therapeutic ranges:

- Acute mania: 0.8-1.2 mEq/L
- Maintenance: 0.6-1.0 mEq/L
- Elderly: 0.4-0.8 mEq/L (more sensitive)
- Toxicity concern: >1.5 mEq/L

Side Effects (2 marks)

TIMING	SIDE EFFECT	MANAGEMENT
Early	Fine tremor	Propranolol 10-20mg; reduce dose if possible
Early	GI (nausea, diarrhea)	Take with food; switch to slow-release formulation

TIMING	SIDE EFFECT	MANAGEMENT
Early	Polyuria / polydipsia	Nephrogenic DI; amiloride may help; ensure hydration
Ongoing	Weight gain	Diet, exercise counseling
Ongoing	Cognitive dulling ("lithium fog")	Dose reduction; assess if depression vs medication effect
Ongoing	Acne, psoriasis exacerbation	Dermatological management
Long-term	Hypothyroidism	Levothyroxine supplementation; do NOT stop lithium
Long-term	Hyperparathyroidism	Monitor calcium; endocrine referral if persistent
Long-term	Chronic kidney disease	Monitor eGFR; controversial whether lithium directly causes vs risk factor; minimize other nephrotoxins
Long-term	Cardiac (T-wave changes, sinus node dysfunction)	ECG monitoring; usually benign
Teratogenicity	Ebstein's anomaly	Risk ~0.1% (lower than historically thought but 20-40x baseline); level II ultrasound + fetal echocardiography

Toxicity and Management (3 marks)

Clinical features by severity:

LEVEL (MEQ/L)	SEVERITY	FEATURES
1.5-2.0	Mild	Coarse tremor (replacing fine), nausea, vomiting, diarrhea, drowsiness, muscular weakness, fasciculations
2.0-2.5	Moderate	Ataxia, dysarthria, confusion, myoclonic jerks, hyperreflexia, nystagmus, EEG changes
2.5-3.0	Severe	Seizures (generalized tonic-clonic), impaired consciousness, coma
>3.0	Life-threatening	Renal failure, cardiovascular collapse, irreversible cerebellar damage, death

Common precipitants:

- Dehydration (fever, diarrhea, vomiting, reduced intake, hot weather)
- NSAIDs (ibuprofen, naproxen, reduce renal lithium clearance)
- ACE inhibitors / ARBs (reduce GFR)
- Thiazide diuretics (increase lithium reabsorption in proximal tubule)
- Sodium restriction / depletion
- Acute renal impairment from any cause

Management:

1. **Stop lithium immediately**
2. **IV normal saline 0.9%**, aggressive rehydration to restore volume and sodium; enhances renal lithium excretion
3. **Serial lithium levels** every 2-4 hours until consistently declining
4. **Monitor:** renal function, electrolytes, ECG, neurological status
5. **Correct precipitating cause** (stop offending drugs, treat infection / dehydration)
6. **Avoid further nephrotoxins**
7. **Hemodialysis**, indications:
 8. Serum lithium >4.0 mEq/L (regardless of symptoms)
 9. Serum lithium >2.5 mEq/L with severe symptoms
10. Renal failure (impaired excretion)
11. Deterioration despite supportive care
12. Post-dialysis: monitor for rebound (lithium redistributes from tissue compartments; may need repeat dialysis)
13. **Whole bowel irrigation** if recent ingestion of sustained-release formulation (activated charcoal does NOT bind lithium)
14. **Benzodiazepines** for seizures
15. **ICU admission** for severe toxicity

QUESTION 13: "Describe negative symptoms in schizophrenia and discuss management." (10 marks)

EXAM STRATEGY

Cross-reference NB-06 (Schizophrenia). If this appears in Paper II as a mood disorder overlap, focus on the mood disorder intersection. This overlaps with depression (avolition, anhedonia, flat affect), differential between negative symptoms and depression is important. See NB-06-P2-Schizophrenia files for full model answer.

Brief note for mood disorder context:

Negative symptoms (avolition, anhedonia, flat affect, alogia, asociality) overlap significantly with depressive features. In schizoaffective disorder, distinguishing "primary" negative symptoms from depressive episodes is critical for treatment planning. If depression is contributing: treat depression aggressively (antidepressants with mood stabilizer cover). If primary negative symptoms: cariprazine (D3 partial agonism), amisulpride (low dose), consider psychosocial interventions.

QUESTION 14: "What is metabolic syndrome and its relationship with psychotropic medications?" (10 marks)

EXAM STRATEGY

Definition/ criteria (2), pathophysiology (1), psychotropics involved (4), monitoring (2), management (1).

Model Answer

Definition and Criteria (2 marks)

Metabolic syndrome is a cluster of interconnected metabolic abnormalities that increase cardiovascular disease and type 2 diabetes risk. Defined by NCEP ATP-III criteria (3 of 5):

COMPONENT · THRESHOLD

Waist circumference >102 cm (M) / >88 cm (F); modified for Asian: >90 cm (M) / >80 cm (F)

Triglycerides $\geq$ 150 mg/dL

HDL cholesterol <40 mg/dL (M) / <50 mg/dL (F)

Blood pressure $\geq$ 130/85 mmHg or on treatment

Fasting glucose $\geq$ 100 mg/dL or on treatment

Prevalence in psychiatric populations: 2-3x general population (30-50% in schizophrenia/bipolar).

Pathophysiology (1 mark)

- Central insulin resistance
- Visceral adiposity $\rightarrow$ inflammation $\rightarrow$ endothelial dysfunction
- Psychotropic medications compound inherent metabolic vulnerability
- Shared genetic/environmental risk factors between severe mental illness and metabolic disease
- Sedentary lifestyle, poor diet, smoking more common in psychiatric patients

- Illness-intrinsic factors (HPA axis dysregulation, inflammation)

Psychotropic Medications and Metabolic Risk (4 marks)

DRUG	WEIGHT GAIN	DIABETES RISK	DYSLIPIDEMIA	RANKING
Clozapine	++++	++++	++++	Highest risk
Olanzapine	++++	+++	+++	Very high risk
Quetiapine	++	++	++	Moderate-high
Risperidone	++	+	+	Moderate
Paliperidone	++	+	+	Moderate
Aripiprazole	+/-	+/-	+/-	Low
Ziprasidone	+/-	+/-	+/-	Low
Lurasidone	+/-	+/-	+/-	Low
Cariprazine	+/-	+/-	+/-	Low

Mood stabilizers:

- Valproate: Significant weight gain, insulin resistance, PCOS
- Lithium: Moderate weight gain, hypothyroidism
- Carbamazepine: Mild weight gain
- Lamotrigine: Weight-neutral

Antidepressants:

- Mirtazapine: Weight gain (H1 blockade)
- TCAs (amitriptyline): Weight gain
- Paroxetine: Weight gain (most among SSRIs)
- SSRIs: Generally mild or weight-neutral short-term; some long-term gain
- Bupropion: Weight-neutral to mild loss

Monitoring (2 marks)

Per ADA / APA consensus guidelines (2004, updated):

WIDE COMPARISON · LANDSCAPE

PARAMETER	BASELINE	4 WEEKS	8 WEEKS	12 WEEKS	QUARTERLY	ANNUALLY	5-YEARLY
Weight/BMI	Yes	Yes	Yes	Yes	Yes		
Waist circumference	Yes			Yes		Yes	
Blood pressure	Yes			Yes		Yes	
Fasting glucose	Yes			Yes		Yes	
Fasting lipid profile	Yes			Yes			Yes
Personal/family history	Yes					Yes	

Management (1 mark)

- Lifestyle intervention first: diet, exercise, smoking cessation
- Switch to lower metabolic risk medication if clinically feasible (e.g., olanzapine → aripiprazole)
- Metformin: Best-studied pharmacological intervention for antipsychotic-associated weight gain (500-2000mg / day)
- Topiramate: Adjunctive for weight reduction
- GLP-1 receptor agonists (liraglutide, semaglutide): Emerging evidence in psychiatric populations
- Treat individual components: statins for dyslipidemia, antihypertensives, glucose management
- Regular cardiovascular risk assessment

QUESTION 15: "Discuss the role of ECT in treatment of mood disorders." (10 marks)

EXAM STRATEGY

Indications (3), mechanism (1), procedure highlights (2), evidence (2), side effects (1), special populations (1).

Model Answer

Indications in Mood Disorders (3 marks)

Depression:

1. Severe depression with psychotic features, response rate >80% (superior to pharmacotherapy alone)
2. Severe depression with acute suicidality, fastest acting antidepressant treatment
3. Treatment-resistant depression (Thase-Rush Stage V)
4. Catatonic depression
5. Depression with nutritional compromise (refusing food / fluids)
6. Depression in pregnancy, when pharmacotherapy is ineffective or poses unacceptable risk
7. Depression in elderly, safe, effective, sometimes preferred over polypharmacy

Bipolar disorder:

1. Acute mania (refractory to medications, delirious mania, catatonic features)
2. Bipolar depression (severe, psychotic, or refractory)
3. Mixed states with severe features

4. Rapid cycling (as adjunct to mood stabilizers)

Mechanism (1 mark)

The precise mechanism remains incompletely understood. Leading theories:

- **Monoamine enhancement:** Increases 5-HT, NE, DA neurotransmission
- **Neuroplasticity:** Increases BDNF, promotes hippocampal neurogenesis
- **HPA axis normalization:** Reduces cortisol hypersecretion
- **Anticonvulsant theory:** Progressive increase in seizure threshold across sessions reflects GABAergic enhancement
- **Neuroinflammatory modulation:** Reduces pro-inflammatory cytokines
- **Connectivity changes:** Normalizes default mode network hyperconnectivity in depression

Procedure Key Points (2 marks)

- **Pre-ECT workup:** Physical exam, blood tests (FBC, electrolytes, blood sugar), ECG (>40 yrs), chest X-ray (if indicated), anesthesia assessment, informed consent, dental assessment
- **Electrode placement:** Bilateral (bifrontotemporal) vs right unilateral, bilateral more effective but more cognitive side effects; ultra-brief pulse right unilateral optimizes the balance
- **Anesthesia:** Short-acting agents, thiopental / propofol (induction) + succinylcholine (muscle relaxation) + atropine / glycopyrrolate (to reduce secretions)
- **Seizure adequacy:** Motor seizure $\geq$ 20 seconds (15 seconds by EEG); subthreshold → re-stimulate with higher charge
- **Course:** 6-12 sessions; 2-3 times per week; depression typically responds by 6-8 sessions
- **Stimulus parameters:** Charge (millicoulombs), pulse width (brief 0.5-2ms vs ultrabrief 0.25-0.3ms), frequency (Hz), current (mA), duration
- **Maintenance ECT:** For relapse prevention; typically weekly → biweekly → monthly; indicated when pharmacotherapy alone insufficient to prevent relapse

Evidence Base (2 marks)

- **UK ECT Review Group (2003):** ECT superior to sham ECT and pharmacotherapy for depression
- **CORE study:** Bilateral ECT + nortriptyline superior to either alone for psychotic depression
- **Kellner et al. (2006):** ECT + nortriptyline + lithium maintenance reduces relapse after successful ECT
- **Efficacy rates:** 70-90% for severe depression; 80%+ for psychotic depression; 50-60% for TRD

- **Speed of response:** Faster than any pharmacological intervention, significant improvement often within 1-2 weeks
- **Mania:** Limited RCT data but clinical experience supports efficacy; particularly for delirious mania and treatment-resistant mania
- **Comparison with pharmacotherapy:** ECT superior to pharmacotherapy for severe depression, psychotic depression, and acutely suicidal patients; generally reserved as later-line due to logistics and stigma

Side Effects (1 mark)

- **Most common:** Headache (most frequent), nausea, muscle soreness (from succinylcholine)
- **Cognitive:** Transient confusion (minutes to hours post-treatment), anterograde amnesia (usually resolves weeks after course), retrograde amnesia (for events around treatment period; usually resolves, but some patients report persistent autobiographical memory gaps)
- **Cardiovascular:** Transient bradycardia (vagal stimulation during seizure) followed by tachycardia; usually benign
- **Cognitive effects less with:** Right unilateral > bilateral, ultrabrief > brief pulse, lower stimulus intensity
- **Mortality:** ~0.002% per treatment (comparable to general anesthesia risk)
- **Absolute contraindication:** Raised intracranial pressure (risk of herniation); pheochromocytoma (hypertensive crisis)
- **Relative contraindications:** Recent cerebral hemorrhage, unstable aneurysm, recent MI, retinal detachment

Special Populations (1 mark)

- **Elderly:** Safe and effective; may be preferred over polypharmacy; post-ECT delirium risk higher
 - **Pregnancy:** Safe in all trimesters; one of the safest treatments for severe depression in pregnancy; monitor fetal heart, avoid aortocaval compression (left lateral tilt), obstetric standby in third trimester
 - **Adolescents:** Used in treatment-resistant cases; similar efficacy to adults; parental consent required
 - **Medical comorbidity:** Generally safe with appropriate anesthetic management; cardiac patients need cardiology clearance
-

Mnemonics & Memory Tricks

MNEMONIC 1: DIGFAST: Symptoms of Mania

DIGFAST

MNEMONIC

DIGFAST

EXAM PEARL: Symptoms of a manic episode, need ≥ 3 (or 4 if mood is only irritable, not elevated/expansive).

LETTER	SYMPTOM	ANCHOR
D	Distractibility	Can't hold attention, drawn to every irrelevant stimulus
I	Indiscretion / Impulsivity	Spending sprees, sexual indiscretions, reckless investments
G	Grandiosity	"I am God" / "I will solve world hunger", inflated self-esteem
F	Flight of ideas	Racing thoughts, jumping topics, clang associations
A	Activity increase	Goal-directed hyperactivity, psychomotor agitation
S	Sleep decreased	Feels rested after 2-3 hours, NOT insomnia (patient doesn't WANT more sleep)
T	Talkativeness	Pressured speech, loud, hard to interrupt

EXAM PEARL

Need ≥ 3 of DIGFAST (or 4 if mood is only irritable, not elevated/expansive) for manic episode diagnosis.

MNEMONIC 2: SIG E CAPS: Depression (DSM-5 Criteria)

SIG E CAPS

MNEMONIC

SIG E CAPS

EXAM PEARL: Think of it as a doctor writing a prescription (Sig: E Caps), "Prescribe Energy Capsules"

LETTER	SYMPTOM	DETAIL
S	Sleep	Insomnia (especially early morning awakening) or hypersomnia
I	Interest	Loss of interest or pleasure in activities (anhedonia)
G	Guilt	Excessive or inappropriate guilt; worthlessness
E	Energy	Fatigue, loss of energy
C	Concentration	Decreased concentration, indecisiveness
A	Appetite	Decreased or increased; weight change (>5% in one month)
P	Psychomotor	Agitation or retardation (observable by others)
S	Suicidal ideation	Thoughts of death, suicidal ideation, plan, attempt

Plus depressed mood (most of day, nearly every day)

EXAM PEARL

>= 5 of 9 for >= 2 weeks. MUST include either depressed mood OR anhedonia.

MNEMONIC 3: Bipolar I vs II vs Cyclothymia: "The 1-2-C Rule"

The 1-2-C Rule

MNEMONIC

The 1-2-C Rule

EXAM PEARL: Bipolar I = 1 manic episode is enough; Bipolar II = 2 types needed; Cyclothymia = Chronic sub-threshold.

Bipolar I = 1 manic episode is enough (depression not required for diagnosis)

- Mania $\geq$ 7 days OR hospitalization
- Psychotic features CAN occur
- Marked functional impairment

Bipolar II = 2 types needed (hypomania + depression BOTH required)

- Hypomania $\geq$ 4 days
- NEVER a full manic episode
- If psychosis occurs, it's BP-I by definition

Cyclothymia = Chronic but sub-threshold (never meets full criteria for either pole)

- $\geq$ 2 years of fluctuation
- Sub-threshold hypomania + sub-threshold depression
- Never symptom-free for >2 months

Memory trick: "One episode of mania = BP-I, Two poles required = BP-II, Chronically cycling below threshold = Cyclothymia"

MNEMONIC 4: Lithium Side Effects: "LITHIUM"

LITHIUM

MNEMONIC

LITHIUM

EXAM PEARL: Each letter of the drug name maps to a key side effect.

LETTER	SIDE EFFECT	DETAIL
L	Leukocytosis	Benign; can mask infection

LETTER	SIDE EFFECT	DETAIL
I	Insipidus (diabetes)	Nephrogenic DI; polyuria / polydipsia; impaired aquaporin-2
T	Tremor	Fine tremor (therapeutic); coarse tremor = toxicity
H	Hypothyroidism	20-30%; more in women; treat with levothyroxine, continue lithium
I	Increased weight	Common; diet counseling
U	Upset stomach	Nausea, diarrhea; take with food
M	Morphological (cardiac)	Ebstein's anomaly (teratogen); T-wave changes on ECG

EXAM PEARL

"Lithium loves the thyroid and kidneys, it won't leave them alone" (hypothyroidism, nephrogenic DI, chronic kidney disease, hyperparathyroidism)

MNEMONIC 5: Lithium Monitoring: "RENAL ThyCa" (Renal, Thyroid, Calcium)

RENAL ThyCa

MNEMONIC

RENAL ThyCa

EXAM PEARL: Baseline investigations before starting lithium.

Baseline before starting lithium:

- Renal function (creatinine, eGFR)
- ECG (if >40 or cardiac history)
- Nutrition / weight (BMI)
- And electrolytes
- Lithium level (then 5-7 days after starting)
- Thyroid (TSH, free T4)
- Calcium (+ PTH)
- Pregnancy test (women of childbearing age)

- FBC

Every 6 months (stable patient):

- Lithium level
- Renal function
- Thyroid function
- Calcium (annually minimum)

After every dose change: Recheck lithium level 5-7 days later

MNEMONIC 6: Lithium Toxicity Features: "TOXIC SALT"

TOXIC SALT

MNEMONIC

TOXIC SALT

EXAM PEARL: Arranged by severity (mild → moderate → severe).

LETTER	FEATURE	SEVERITY
T	Tremor (COARSE, replacing fine)	Mild (1.5-2.0)
O	Output increased (vomiting, diarrhea)	Mild
X	eXhaustion (drowsiness, muscular weakness)	Mild
I	Incoordination (ataxia)	Moderate (2.0-2.5)
C	Confusion, dysarthria	Moderate
S	Seizures	Severe (>2.5)
A	Arrhythmias, cardiovascular collapse	Severe
L	Loss of consciousness (coma)	Severe
T	Terminal renal failure	Severe

EXAM PEARL

Precipitants, "DNS" (Dehydration, NSAIDs, Sodium depletion/ diuretics)

MNEMONIC 7: Valproate Side Effects: "VALPROATE"

VALPROATE

MNEMONIC

VALPROATE

EXAM PEARL: Each letter of the drug name maps to a key side effect.

LETTER · SIDE EFFECT

- V Vomiting / GI upset
- A Alopecia (hair loss, usually reversible)
- L Liver toxicity (hepatotoxicity, monitor LFTs; highest risk: children <2 on polytherapy)
- P Pancreatitis (rare but potentially fatal)
- R Reproductive (PCOS, menstrual irregularities, teratogenicity, NTDs)
- O Obesity (weight gain)
- A Ammonia elevated (hyperammonemia, can cause confusion even without liver failure)
- T Thrombocytopenia (dose-related)
- E Ebstein's? No, that's lithium! Neural tube defects + decreased IQ + autism risk

EXAM PEARL

Valproate is effectively CONTRAINDICATED in women of childbearing age (EMA 2018, MHRA, NICE)

MNEMONIC 8: TRD Staging (Thase-Rush): "S-S-T-M-E"

S-S-T-M-E

MNEMONIC

Some Students Try Making Electricity

EXAM PEARL: Five stages of treatment-resistant depression.

STAGE	FAILED TREATMENT	MEMORY AID
I	SSRI	"Start with SSRI"

STAGE	FAILED TREATMENT	MEMORY AID
II	Switch to different class (SNRI, etc.)	"Switch classes"
III	TCA (tricyclic)	"Try Tricyclics"
IV	MAOI	"MAOI Maneuver"
V	ECT (bilateral)	"ECT, End of the line"

MNEMONIC 9: Antidepressant Classes: "SSN-TMA"

SSN-TMA

MNEMONIC

SSN-TMA

EXAM PEARL: Quick reference for antidepressant class recall.

ABBREVIATION	CLASS	EXAMPLES
SS	SSRIs	Fluoxetine, Sertraline, Paroxetine, Escitalopram, Fluvoxamine, Citalopram
N	SNRIs	Venlafaxine, Duloxetine, Desvenlafaxine, Milnacipran
T	TCAs	Amitriptyline, Imipramine, Clomipramine, Nortriptyline, Desipramine
M	MAOIs	Phenelzine, Tranylcypromine, Moclobemide, Selegiline
A	Atypicals / Others	Mirtazapine (NaSSA), Bupropion (NDRI), Vortioxetine, Agomelatine, Trazodone (SARI)

EXAM PEARL

SSRIs order by selectivity: "Escitalopram is the most selective; Paroxetine is the most problematic (anticholinergic, worst discontinuation, teratogenic)"

SSRI memory for unique features, "FSPFEC":

- Fluoxetine = longest half-life (no discontinuation syndrome), CYP2D6 inhibitor
- Sertraline = safest in cardiac, breastfeeding

- Paroxetine = worst discontinuation, most anticholinergic, avoid in pregnancy
- Fluvoxamine = OCD specialist, CYP1A2 inhibitor
- Escitalopram = cleanest profile, most selective
- Citalopram = QTc at high doses, dose cap 40mg

MNEMONIC 10: SAD PERSONS: Suicide Risk Factors

SAD PERSONS

MNEMONIC

SAD PERSONS

EXAM PEARL: Ten risk factors for suicide, guides risk stratification and disposition.

LETTER	FACTOR	HIGH-RISK QUALIFIER
S	Sex	Male (completed); Female (attempted)
A	Age	>65 or 15-30
D	Depression	Any psychiatric illness, especially mood disorders
P	Previous attempt	Strongest single predictor
E	Ethanol / drugs	Active use; intoxication at time
R	Rational thinking loss	Psychosis, command hallucinations
S	Social supports lacking	Isolation, unemployment
O	Organized plan	Specific, lethal, access to means
N	No spouse	Single, divorced, widowed
S	Sickness	Chronic pain, terminal illness

Scoring: 0-2 = outpatient, 3-4 = close follow-up, 5-6 = consider hospitalization, 7-10 = hospitalize

EXAM PEARL

SAD PERSONS has poor predictive validity individually but is a useful clinical framework. Emphasize that "previous attempt" is the strongest predictor, and "hopelessness" (not in the mnemonic) is more predictive than depression severity.

MNEMONIC 11: Serotonin Syndrome: "HAM" Triad

HAM Triad

MNEMONIC

HAM

EXAM PEARL: Three-system triad of serotonin syndrome.

TRIAD COMPONENT · FEATURES

H, Hyperactivity (neuromuscular) Myoclonus, hyperreflexia, clonus (especially lower limbs), tremor, rigidity

A, Autonomic instability Hyperthermia, tachycardia, diaphoresis, hypertension, diarrhea, mydriasis

M, Mental status changes Agitation, confusion, delirium

EXAM PEARL

Distinguishing feature from NMS: Serotonin syndrome = CLONUS + hyperreflexia (fast onset, hours). NMS = lead-pipe RIGIDITY + bradyreflexia (slow onset, days-weeks).

CLINICAL ANCHOR

Treatment: "Stop the serotonin, Cool the patient, Cyproheptadine"

MNEMONIC 12: SSRI Side Effects: "SING SAD"

SING SAD

MNEMONIC

SING SAD

EXAM PEARL: Common SSRI side effects.

LETTER · SIDE EFFECT

S Sexual dysfunction (30-40%, most common reason for discontinuation)

I Insomnia (or sedation depending on agent)

N Nausea (GI, most common early side effect)

G GI disturbance (diarrhea)

S SIADH/hyponatremia (especially elderly)

A Activation/ anxiety (initial worsening, first 1-2 weeks)

D Discontinuation syndrome (paroxetine, venlafaxine worst; "FINISH" = Flu-like, Insomnia, Nausea, Imbalance, Sensory disturbance [electric shocks], Hyperarousal)

MNEMONIC 13: Switching vs Augmentation Decision: "No Response = Switch; Partial Response = Augment"

Switch vs Augment

MNEMONIC

No Response = Switch; Partial Response = Augment

EXAM PEARL: Decision rule for TRD management.

SCENARIO	STRATEGY	WHY
NO response after adequate trial	SWITCH to different class	Current mechanism isn't working at all
PARTIAL response	AUGMENT (add second agent)	Current mechanism partially working; build on it

EXAM PEARL

Top augmentation agents, "LAT" (Lithium, Atypicals, Thyroid): - Lithium: Most evidence-based; 0.6-0.8 mEq/L - Atypical antipsychotics: Aripiprazole (2-5mg), quetiapine (50-300mg) - Thyroid: T3 (liothyronine) 25-50mcg even in euthyroid patients

Top combination strategies, "California Rocket Fuel":

- Venlafaxine + mirtazapine = serotonin + norepinephrine boost from both sides
 - SSRI + bupropion = serotonin + dopamine/NE
-

MNEMONIC 14: Lamotrigine Titration: "Slow and Low Saves Skin"

Slow and Low Saves Skin

MNEMONIC

Slow and Low Saves Skin

EXAM PEARL: Rapid titration = Stevens-Johnson Syndrome risk.

Standard titration (NO valproate):

- Weeks 1-2: 25 mg / day
- Weeks 3-4: 50 mg / day
- Week 5: 100 mg / day
- Week 6-7: Target 200 mg / day

With VALPROATE (doubles lamotrigine levels):

- HALVE all doses: Start 12.5 mg → 25 mg → 50 mg → target 100 mg
- "Valproate = Very careful lamotrigine dosing"

With CARBAMAZEPINE (halves lamotrigine levels):

- May need HIGHER target doses (300-400 mg)

EXAM PEARL

ANY rash during titration = STOP immediately and evaluate. Better to restart from scratch than risk SJS.

MNEMONIC 15: Good vs Poor Prognosis in BPAD: "LATE GOOD vs EARLY BAD"**LATE GOOD vs EARLY BAD****MNEMONIC****LATE GOOD vs EARLY BAD**

EXAM PEARL: Prognostic factors in bipolar affective disorder.

Good prognosis, "LATE GOOD":

- Late onset
- Adherence to treatment
- Typical euphoric mania (not mixed)
- Episodes few in number
- Good inter-episode functioning
- One mood pole predominant (not mixed)
- Outside support (social network)
- Disorder without psychosis

Poor prognosis, "EARLY BAD":

- Early onset (<25 years)

- Abuse of substances (comorbid)
 - Rapid cycling
 - Lack of compliance
 - Years of illness (chronicity)
 - Bizarre psychotic features (mood-incongruent)
 - Associated personality disorder
 - Depressive predominance with residual symptoms
-

MNEMONIC 16: Bipolar Depression: "NO Antidepressant Monotherapy!" (NOAM)

NOAM

MNEMONIC

NOAM, NO Antidepressant Monotherapy

EXAM PEARL: First-line treatment options for bipolar depression.

What TO use, "QLLOC":

- Quetiapine (BOLDER trials, 300mg)
- Lithium (also anti-suicidal)
- Lamotrigine (better for prevention)
- OFC (olanzapine-fluoxetine combination)
- Cariprazine / Lurasidone (newer options)

What NOT to do:

- NO antidepressant monotherapy (manic switch risk)
 - NO TCAs/venlafaxine if adding antidepressant (higher switch risk)
 - If antidepressant must be used: SSRI or bupropion + mood stabilizer, SHORT course
-

MNEMONIC 17: Depression Rating Scales: "How Many Patients Burned out?"

How Many Patients Burned out?

MNEMONIC

How Many Patients Burned out?

EXAM PEARL: H=HAM-D, M=MADRS, P=PHQ-9, B=BDI-II.

INITIAL	SCALE	REMEMBER
H	HAM-D	Hamilton = clinician-rated, 17 items, gold standard research
M	MADRS	Montgomery-Asberg = clinician-rated, 10 items, sensitive to change
P	PHQ-9	Patient Health Questionnaire = self-report, 9 items, mirrors DSM-5
B	BDI-II	Beck Depression Inventory = self-report, 21 items, cognitive focus

EXAM PEARL

Clinician-rated = H and M (Hamilton, MADRS); Self-report = P and B (PHQ-9, BDI)

MNEMONIC 18: Mania vs Hypomania: "Duration, Destruction, Delusions"

Duration, Destruction, Delusions

MNEMONIC

Duration, Destruction, Delusions

EXAM PEARL: Three Ds that distinguish mania from hypomania.

FEATURE	HYPOMANIA	MANIA
Duration	≥ 4 days	≥ 7 days (or any if hospitalized)
Destruction (functional impairment)	NOT marked	MARKED
Delusions (psychotic features)	NEVER	Can occur

EXAM PEARL

"If there are any D's that are BIG, it's mania, not hypomania."

MNEMONIC 19: Drugs That Precipitate Lithium Toxicity: "DNATA"

DNATA

MNEMONIC

DNATA

EXAM PEARL: Drug and non-drug causes that raise lithium levels to toxic range.

LETTER · DRUG/CAUSE

D Dehydration (any cause, fever, vomiting, diarrhea, hot weather)

N NSAIDs (ibuprofen, naproxen, reduce renal lithium clearance)

A ACE inhibitors / ARBs

T Thiazide diuretics (NOT loop diuretics, loop are safer)

A Any sodium depletion (low-salt diet, excessive sweating)

EXAM PEARL

"DNATA causes lithium to rise", Lithium is handled like sodium by the kidney. Anything that depletes sodium or reduces GFR raises lithium levels.

MNEMONIC 20: Discontinuation Syndrome Features: "FINISH"

FINISH

MNEMONIC

FINISH

EXAM PEARL: Six features of antidepressant discontinuation syndrome.

LETTER · SYMPTOM

F Flu-like symptoms

I Insomnia

N Nausea

I Imbalance (dizziness, vertigo)

S Sensory disturbances (electric shock sensations, "brain zaps")

H Hyperarousal (anxiety, agitation, irritability)

EXAM PEARL

Worst offenders: Paroxetine and venlafaxine (short half-lives). Never happens with: Fluoxetine (long half-life = self-tapering). Prevention: Gradual taper over 2-4 weeks minimum.

High-Yield Comparisons

TABLE 1: Bipolar I vs Bipolar II vs Cyclothymia

FEATURE	BIPOLAR I	BIPOLAR II	CYCLOTHYMIA
Defining episode	At least 1 manic episode	At least 1 hypomanic + 1 major depressive	Chronic sub-threshold fluctuation both poles
Mania	Yes (≥ 7 days or hospitalization)	Never	Never
Hypomania	May occur	Yes (≥ 4 days)	Sub-threshold hypomanic symptoms
Major depression	Common but NOT required for diagnosis	Required (at least 1 episode)	Never meets full MDE criteria
Duration requirement	Mania ≥ 7 days	Hypomania ≥ 4 days + MDE ≥ 2 weeks	≥ 2 years (1 year in adolescents)
Psychotic features	Can occur (mania or depression)	Only in depression; if in "hypomania" $\rightarrow$ re-classify to BP-I	Never
Functional impairment	Marked during mania	Hypomania: NOT marked; Depression: significant	Mild but chronic
Hospitalization	Common during mania	Rare for hypomania	Very rare
Prevalence	1-1.5%	0.4-1.1%	0.4-1%
Sex ratio	M = F	F > M	M = F
Suicide risk	10-15% lifetime	Comparable to BP-I	Lower but elevated vs general population
Course	Episodic; depression predominates 3:1	Depression predominates even more	Chronic fluctuating; 15-50% convert to BP-I or II
Treatment	Mood stabilizers + antipsychotics	Mood stabilizers; lamotrigine for depression prevention	Mood stabilizers if impaired; avoid antidepressant monotherapy

TABLE 2: Mania vs Hypomania

FEATURE	MANIA	HYPOMANIA
Duration	>= 7 days (or any duration if hospitalized)	>= 4 consecutive days
Symptom threshold	>= 3 DIGFAST (4 if only irritable)	Same symptom criteria
Functional impairment	Marked impairment OR hospitalization	Change in functioning observable by others, NOT marked impairment
Psychotic features	CAN be present	NEVER present (if present → mania → BP-I)
Insight	Often completely lost	Partially preserved
Need for hospitalization	Frequently required	Not required
Subjective experience	May feel invincible; sometimes dysphoric	Often ego-syntonic ("best I've ever felt"); may be productive
Danger to self/others	Significant risk (reckless behavior, aggression)	Mild risk; less dangerous behavior
Impact on diagnosis	Defines BP-I (one episode sufficient)	Defines BP-II (only with MDE)
Observable by others	Clearly abnormal to anyone	Represents clear change from baseline; others notice
Response to treatment	Requires acute pharmacotherapy (mood stabilizers + antipsychotics)	May not need acute treatment; prophylaxis focus

EXAM PEARL

The distinction is primarily one of SEVERITY and CONSEQUENCES. Same symptoms, different magnitude. When in doubt, assess functional impact and ask collateral sources.

TABLE 3: Unipolar Depression vs Bipolar Depression: Clinical Clues

FEATURE	UNIPOLAR DEPRESSION	BIPOLAR DEPRESSION
Age of onset	25-35 years (average)	Earlier: 15-25 years
Family history	Depression in family	Bipolar disorder in family
Episode characteristics	Variable; insomnia common	Hypersomnia, hyperphagia, leaden paralysis (atypical features more common)

FEATURE	UNIPOLAR DEPRESSION	BIPOLAR DEPRESSION
Psychomotor	Agitation or retardation	Retardation more prominent
Episode duration	6-12 months	Often shorter (3-6 months)
Number of episodes	Fewer lifetime episodes	More frequent episodes
First episode age	Later	Often <25 years
Postpartum onset	Less common association	Strong association with bipolar
Psychotic features	Less common	More common (especially mood-congruent)
Antidepressant response	Good response to monotherapy	May switch to mania; rapid cycling; poor sustained response
Treatment-resistance	Less likely to be TRD	More likely (may be unrecognized bipolar)
Mood lability	Less	More (mood swings within episode)
Substance use	Comorbid but less strongly	Strongly associated
Suicidal behavior	Elevated	MORE elevated than unipolar
Response to mood stabilizers	Limited	Good (lithium, lamotrigine)
Antidepressant monotherapy	First-line treatment	CONTRAINDICATED without mood stabilizer cover

CLINICAL ANCHOR

Any patient presenting with "TRD" should be carefully screened for bipolarity (MDQ, HCL-32, or clinical interview for past hypomania). Unrecognized bipolar depression is the most common reason for apparent treatment resistance.

TABLE 4: Lithium vs Valproate vs Carbamazepine vs Lamotrigine

FEATURE	LITHIUM	VALPROATE	CARBAMAZEPINE	LAMOTRIGINE
Primary mechanism	GSK-3beta inhibition, inositol depletion	GABA enhancement, Na channel block	Na channel blockade	Glutamate modulation via Na channels
Acute mania	+++	+++	++	NOT effective
Bipolar depression	++	+	+	++ (better for prevention)

FEATURE	LITHIUM	VALPROATE	CARBAMAZEPINE	LAMOTRIGINE
Maintenance (mania prevention)	+++	+++	++	+
Maintenance (depression prevention)	++	+	+	+++
Anti-suicidal effect	YES (unique)	No	No	No
Therapeutic level	0.6-1.2 mEq/L	50-125 mcg/mL	4-12 mcg/mL	Not level-monitored
Renal effects	Nephrogenic DI, CKD	None significant	None significant	None significant
Thyroid effects	Hypothyroidism (20-30%)	None	None	None
Hepatic effects	None	Hepatotoxicity	Hepatotoxicity	Rare
Hematologic	Leukocytosis (benign)	Thrombocytopenia	Agranulocytosis (rare)	None
Teratogenicity	Ebstein's anomaly (~0.1%)	NTDs (1-2%), decreased IQ, autism, WORST teratogen	NTDs (less than valproate)	Relatively safest; possible cleft palate (low risk)
Weight gain	Moderate	Significant	Mild	None (weight-neutral)
Cognitive effects	Dulling ("lithium fog")	Mild	Mild	None
Skin	Acne, psoriasis	Hair loss (reversible)	SJS/TEN (HLA-B*1502)	SJS/TEN (slow titration critical)
Drug interactions	NSAIDs, ACEi, thiazides	Enzyme inhibitor	Potent enzyme inducer + autoinduction	Levels doubled by valproate, halved by CBZ
Women of child-bearing age	Caution (Ebstein's); usable with precautions	CONTRAINDICATED (recent guidelines)	Caution (teratogenic)	Preferred option
Rapid cycling	Less effective	Often preferred	Limited data	May help
Mixed states	Less effective	Often preferred	Limited data	Limited data
Overdose lethality	Significant (toxicity protocol)	Moderate	Moderate	Low

FEATURE	LITHIUM	VALPROATE	CARBAMAZEPINE	LAMOTRIGINE
Best for	Classic euphoric mania, maintenance, anti-suicide	Acute mania, mixed episodes, rapid cycling	Carbamazepine-responsive patients, mania	Bipolar depression prevention

TABLE 5: SSRIs vs SNRIs vs TCAs vs MAOIs

FEATURE	SSRIS	SNRIS	TCAS	MAOIS
Mechanism	Block SERT (serotonin reuptake)	Block SERT + NET (serotonin + norepinephrine)	Block SERT + NET + anti-cholinergic + antihistamine + alpha-1	Inhibit monoamine oxidase (MAO-A and/or MAO-B)
Efficacy (mild-moderate)	First-line	First-line	Effective but not first-line	Effective but not first-line
Efficacy (severe/melancholic)	Good	Good-very good	Very good (gold standard historically)	Very good
Efficacy (atypical depression)	Good	Good	Poor	Best (gold standard)
Sexual dysfunction	30-40% (common)	30-40% (common)	Common	Less than SSRIs
Weight gain	Variable; long-term possible	Variable	Significant	Significant
Sedation	Low (except paroxetine)	Low	High (H1 blockade)	Variable
Anticholinergic	Minimal	Minimal	Significant (dry mouth, constipation, urinary retention, blurred vision)	Low
Cardiac safety	Good (except citalopram QTc)	Good (monitor BP with venlafaxine)	POOR (QTc prolongation, lethal in overdose)	Good
Overdose safety	Safe	Relatively safe (venlafaxine less so)	LETHAL (cardiac arrhythmias)	LETHAL (hypertensive crisis)

FEATURE	SSRIS	SNRIS	TCAS	MAOIS
Drug interactions	CYP450 (fluoxetine, paroxetine = 2D6 inhibitors)	Fewer than SSRIs	Multiple (CYP, anticholinergic summation)	SEROTONIN SYNDROME with serotonergics; TYRAMINE reaction with fermented foods
Dietary restrictions	None	None	None	YES (tyramine-free diet required for irreversible MAOIs)
Discontinuation syndrome	Moderate (worst: paroxetine)	Severe (worst: venlafaxine)	Moderate	Moderate
First-line status	YES (most guidelines)	YES (equal to SSRIs in most guidelines)	NO (second / third-line due to side effects and over-dose risk)	NO (third / fourth-line due to dietary restrictions and interactions)
Special indications	Depression, anxiety disorders, OCD	Depression + pain (duloxetine), GAD	Pain syndromes, enuresis (imipramine), OCD (clomipramine)	Atypical depression, TRD
Key examples	Fluoxetine, sertraline, escitalopram, paroxetine	Venlafaxine, duloxetine, desvenlafaxine	Amitriptyline, imipramine, clomipramine, nortriptyline	Phenelzine, tranylcypromine, moclobemide

TABLE 6: TRD Staging: Thase-Rush and MGH Models

Thase-Rush Model

STAGE	DEFINITION	CLINICAL MEANING
Stage I	Failed ≥ 1 adequate SSRI trial	First-level resistance
Stage II	Stage I + failed ≥ 1 trial from a different class (SNRI, bupropion)	Cross-class resistance
Stage III	Stage II + failed adequate TCA trial	Moderate resistance
Stage IV	Stage III + failed adequate MAOI trial	High resistance

STAGE	DEFINITION	CLINICAL MEANING
Stage V	Stage IV + failed bilateral ECT course	Maximal resistance

MGH Staging Method

COMPONENT · POINTS

Each failed adequate antidepressant trial 1 point

Optimization of dose/duration within a trial 0.5 points

Each augmentation strategy tried 0.5 points

Each combination strategy tried 0.5 points

Failed ECT course 3 points

Total score Sum = overall TRD burden

Comparison

FEATURE	THASE-RUSH	MGH
Type	Categorical (stages)	Quantitative (continuous score)
Granularity	Low (5 stages)	High (decimal scoring)
Includes augmentation/optimization	No	Yes
Research utility	Moderate	High (better for comparing across studies)
Clinical utility	Good (intuitive)	Good (more precise)
Limitation	Implies fixed treatment order (SSRI → TCA → MAOI) which doesn't reflect modern practice	More complex to calculate

TABLE 7: Pseudodementia vs Dementia

FEATURE	PSEUDODEMENTIA (DEPRESSION)	DEMENTIA
Onset	Relatively rapid, dateable	Insidious, gradual
Duration before seeking help	Short (weeks to months)	Long (months to years)
Who complains	PATIENT emphasizes cognitive deficits	FAMILY notices; patient minimizes or is unaware

FEATURE	PSEUDODEMENTIA (DEPRESSION)	DEMENTIA
Effort on cognitive testing	"Don't know" answers; gives up easily; poor effort	Tries hard; near-miss answers; confabulates
Mood	Pervasively depressed; guilt, hopelessness	May be apathetic or labile; not pervasively sad
Diurnal variation	Present (worse in morning)	Sundowning (worse in evening)
Sleep pattern	Early morning awakening	Fragmented; reversed sleep-wake cycle
Memory pattern	Inconsistent; recent = remote	Temporal gradient (recent >> remote)
Attention/concentration	Often intact but "can't be bothered"	Genuinely impaired
Orientation	Usually preserved	Impaired (especially time → place → person)
Language	Normal (may be slow)	Anomia, word-finding difficulty, paraphasias
Psychiatric history	Often present (prior depression)	Usually absent
Vegetative symptoms	Prominent (appetite, sleep, energy, libido)	Less prominent
Response to antidepressants	YES, cognition improves	NO
Neuroimaging	Normal or white matter hyperintensities	Cortical atrophy (especially hippocampal); specific patterns per dementia type
Progression	Does NOT progress to worsening dementia acutely; BUT 30-50% develop true dementia within 5 years	Progressive decline

TABLE 8: Normal Grief vs Major Depression vs Prolonged Grief Disorder

FEATURE	NORMAL GRIEF	MAJOR DEPRESSION	PROLONGED GRIEF DISORDER (ICD-11)
Trigger	Identifiable loss (bereavement)	May or may not have clear trigger	Bereavement (death of close person)

FEATURE	NORMAL GRIEF	MAJOR DEPRESSION	PROLONGED GRIEF DISORDER (ICD-11)
Duration	Weeks to months; gradually improves	≥ 2 weeks (DSM-5 criterion)	≥ 6 months (ICD-11); ≥ 12 months (DSM-5-TR)
Core emotional experience	Yearning, longing, sadness in waves; triggered by reminders of deceased	Pervasive depressed mood, anhedonia; persistent, not in waves	Intense longing / yearning for the deceased; persistent preoccupation with the deceased
Self-esteem	Preserved	Worthlessness, excessive guilt	Preserved (guilt related to the death if present, not global worthlessness)
Positive emotions	CAN experience joy / humor (in between grief waves)	Pervasive inability to experience pleasure	Emotional numbness; difficulty experiencing positive emotions
Suicidal ideation	Rare; if present, typically "wish to join deceased"	Common; active suicidal ideation / plans	May occur (wanting to die to join deceased)
Functional impairment	Temporary; gradually improves	Sustained; does not spontaneously improve	Sustained; centered on inability to move forward
Identity disruption	May question identity briefly	Persistent feelings of worthlessness	Difficulty reintegrating identity without the deceased
Avoidance	Minimal	Social withdrawal (general)	Specific avoidance of reminders of the death or the loss
Course	Gradually improving; adapts to loss	Persistent without treatment	Persistent WITHOUT the gradual adaptation seen in normal grief
Treatment	Support, psychoeducation; usually self-resolving	Antidepressants, psychotherapy	Complicated grief therapy (Shear); limited evidence for antidepressants alone

EXAM PEARL

The "bereavement exclusion" was removed in DSM-5. MDD can be diagnosed during bereavement if full criteria met. Prolonged Grief Disorder was added in DSM-5-TR (2022) as a new diagnosis.

TABLE 9: Depression Rating Scales

SCALE	HAM-D (HDRS)	MADRS	PHQ-9	BDI-II
Full name	Hamilton Depression Rating Scale	Montgomery-Asberg Depression Rating Scale	Patient Health Questionnaire-9	Beck Depression Inventory-II
Rater	Clinician	Clinician	Self-report	Self-report
Items	17 (original); 21, 24 versions exist	10	9	21
Time to administer	15-20 minutes	10-15 minutes	5 minutes	5-10 minutes
Scoring	0-52 (17-item)	0-60	0-27	0-63
Severity cutoffs	0-7 normal; 8-16 mild; 17-23 moderate; ≥ 24 severe	0-6 normal; 7-19 mild; 20-34 moderate; ≥ 35 severe	0-4 none; 5-9 mild; 10-14 moderate; 15-19 moderate-severe; 20-27 severe	0-13 minimal; 14-19 mild; 20-28 moderate; 29-63 severe
Domain focus	Somatic/vegetative symptoms weighted heavily	Inner feelings, pessimism, suicidality, more psychological	Mirrors DSM-5 criteria directly	Cognitive symptoms of depression
Sensitivity to change	Good	BETTER than HAM-D (designed for this)	Good	Good
Research gold standard	YES (most widely used in RCTs)	Increasingly used; preferred by FDA in some trials	Primary care screening standard	CBT research standard
Suicide assessment	Item 3 (single item)	Item 10 (suicidal thoughts)	Item 9 ("thoughts of self-harm")	Item 9
Limitations	Overweights somatic symptoms; insomnia/anxiety inflate score; inter-rater variability	Requires trained rater	Self-report bias; doesn't assess psychomotor change	Self-report bias; cognitive focus may miss somatic depression
Best used for	Clinical trials; treatment response monitoring	Clinical trials; sensitive outcome measure	Screening in primary care; treatment monitoring; population screening	CBT treatment monitoring; research

Additional scales worth knowing:

- **YMRS** (Young Mania Rating Scale): 11-item clinician-rated; gold standard for mania severity
- **EPDS** (Edinburgh Postnatal Depression Scale): 10-item self-report; perinatal screening
- **GDS** (Geriatric Depression Scale): 15/30-item self-report; yes/no format avoids somatic confounds in elderly
- **C-SSRS** (Columbia Suicide Severity Rating Scale): Structured clinical interview for suicide ideation + behavior

TABLE 10: Newer Antidepressants Comparison

FEATURE	ESKETAMINE (SPRAVATO)	BREXANOLONE (ZULRESSO)	VORTIOXETINE (BRINTELLIX)	AGOMELATINE (VALDOXAN)	CARIPRAZINE (VRAYLAR)
Mechanism	NMDA receptor antagonist (S-enantiomer of ketamine)	GABA-A receptor PAM (allopregnanolone analog)	Multimodal: SERT inhibition + 5-HT1A agonist + 5-HT3/7 antagonist	MT1/MT2 agonist + 5-HT2C antagonist	D3-preferring partial agonist + D2 partial agonist + 5-HT1A partial agonist
Primary indication	TRD; MDD with suicidal ideation	Postpartum depression	MDD (especially with cognitive symptoms)	MDD	Bipolar depression; schizophrenia; bipolar I mania
Route	Intranasal (in clinic)	IV infusion (60 hours; inpatient)	Oral	Oral	Oral
Onset of action	Hours to days (rapid)	24-48 hours	2-4 weeks (standard)	2-4 weeks	2-4 weeks
Dosing	56-84mg twice weekly → weekly → biweekly	60-hour continuous IV	5-20mg once daily	25-50mg at bedtime	1.5-3mg once daily (bipolar depression)
Key advantage	Fastest acting; works in TRD	First drug for PPD; very rapid	Pro-cognitive effects; less sexual dysfunction	No sexual dysfunction; no weight gain; circadian rhythm	Effective across bipolar phases; weight-neutral

FEATURE	ESKETAMINE (SPRAVATO)	BREXANOLONE (ZULRESSO)	VORTIOXETINE (BRINTELLIX)	AGOMELATINE (VALDOXAN)	CARIPRAZINE (VRAYLAR)
Key disadvantage	REMS; dissociation; BP elevation; requires clinic visit; expensive	IV only; 60-hour infusion; REMS; extremely expensive	GI side effects (nausea); expensive	Hepatotoxicity risk (monitor LFTs); not available in USA	Akathisia; long half-life (active metabolite 2-4 weeks)
Sexual dysfunction	Minimal	None reported	Less than SSRIs	None	Minimal
Weight effects	Neutral	Neutral	Neutral	Neutral	Neutral
Monitoring	BP during administration; dissociation monitoring for 2 hours	Pulse oximetry; sedation monitoring	Standard	LFTs at baseline, 3, 6, 12, 24 weeks	Metabolic parameters (standard antipsychotic monitoring)
REMS program	Yes	Yes	No	No	No
Availability in India	Limited	Not available	Available	Available	Available
Year approved	2019 (FDA)	2019 (FDA)	2013 (FDA)	2009 (EU; not FDA)	2015 (FDA; bipolar depression 2019)

Also notable:

- **Zuranolone (Zurzuvae):** Oral GABA-A PAM; FDA approved 2023 for PPD; 14-day course; first oral PPD-specific treatment
- **Dextromethorphan/bupropion (Auvelity):** NMDA antagonist + NDRI; FDA approved 2022 for MDD; faster onset than traditional antidepressants
- **Psilocybin:** 5-HT_{2A} agonist; breakthrough therapy designation for TRD; not yet approved; 1-2 sessions with psychotherapy support

PYQ Frequency Analysis

Executive Summary

Mood disorders rival schizophrenia as the **highest-yield clinical topic** with 50+ mentions across 28 sessions. Bipolar disorder and depression together guarantee at least 1-2 questions per exam. The spread is roughly equal between bipolar and depression, with pharmacotherapy dominating.

Topic-Level Frequency

TOPIC	EXAM MENTIONS	VERDICT
Bipolar, pharmacological management	8	Nearly every exam
Bipolar, course and outcome	4	Every 6-7 exams
Bipolar, genetics	3	Every 8-10 exams
Bipolar, classification (recent advances)	3	Emerging
Depression, TRD	4	Every 6-7 exams
Depression, elderly	3	Every 8-10 exams
Depression, biological basis	3	Every 8-10 exams
Lithium (pharmacology, monitoring, toxicity)	6	Every 4-5 exams
Antidepressants (classification, newer agents)	5	Every 5-6 exams
Suicide (assessment, risk factors, prevention)	5	Every 5-6 exams
Persistent mood disorders (dysthymia, cyclothymia)	2	Occasional
Recent advances (esketamine, brexanolone, newer agents)	3	Rising

Must-Prepare PYQ Templates

EXAM STRATEGY

Prioritise these 10 templates, they cover ~90% of mood disorder exam exposure. Prepare a structured answer skeleton for each before the exam.

1. "Pharmacological management of bipolar disorder.", **perennial**
2. "Course and outcome of BPAD.", **perennial**
3. "Classify BPAD. Recent advances in treatment.", **perennial**
4. "Lithium, mechanism, monitoring, side effects, toxicity.", **perennial**
5. "Define TRD. Staging. Management strategies.", **perennial**
6. "Depression in the elderly, features, differential, management.", **perennial**
7. "Suicide, risk factors, assessment, prevention strategies.", **perennial**
8. "Newer antidepressants. Esketamine.", **emerging**
9. "Genetics of bipolar disorder.", **moderate**
10. "Persistent mood disorders.", **occasional**

Analysis based on PG exams Dec 2011, Jun 2025 + PG exams 2013-2022.

Quick Review

Vignette 1: First Episode Mania

Case:

A 22-year-old male engineering student is brought by his parents with a 10-day history of decreased sleep (3-4 hours, feels well-rested), talking excessively about starting multiple business ventures, spending his semester's tuition fees on stock trading, and confrontational behavior with faculty. He has no prior psychiatric history. He believes he has "cracked the code to becoming a billionaire" and is irritable when challenged.

Q1. What is the most likely diagnosis? Justify with diagnostic criteria.

Answer: Bipolar I Disorder, current episode manic, severe, with possible mood-congruent psychotic features (grandiose delusions). Criteria met: (1) Elevated/expansive and irritable mood with increased energy for >7 days; (2) DIGFAST symptoms present, Decreased sleep need (S), Grandiosity (G), Talkativeness/pressured speech (T), Activity increase (A), Indiscretion/reckless spending (I); (3) Marked functional impairment (academic, financial); (4) No substance use indicated. The "cracking the code" belief may represent a grandiose delusion if firmly held despite evidence.

Q2. Outline your initial management plan.

Answer:

1. **Safety assessment:** Assess for aggression risk, suicidality, capacity for informed consent
2. **Investigations:** CBC, RFT, LFT, TFT (rule out hyperthyroidism), blood glucose, urine drug screen, HIV / VDRL (if indicated), ECG
3. **Acute pharmacotherapy:** Atypical antipsychotic (olanzapine 10-15mg or risperidone 2-4mg) for rapid symptom control + mood stabilizer (lithium 600-900mg or valproate 750-1000mg). Short-term benzodiazepine (lorazepam 2-4mg / day) for agitation and insomnia
4. **Inpatient admission** given severity and functional impairment
5. **Psychoeducation** for family regarding illness, treatment, and prognosis once acute phase stabilizes

Q3. What are the poor prognostic factors in this case?

Answer: Early onset (22 years), potential psychotic features (mood-congruent grandiosity if delusional), first episode already showing significant impairment. However, no substance use and family brought him early (supportive family) are positive. Adherence will be the key modifiable factor, young males are at highest risk for non-adherence once they feel better.

Vignette 2: Bipolar Depression Trap

Case:

A 34-year-old woman presents with persistent low mood, hypersomnia (sleeping 12-14 hours/day), increased appetite with 6 kg weight gain over 2 months, leaden paralysis, and difficulty functioning at work. She has had two prior "depressive episodes" at ages 25 and 29, each treated with SSRIs with initial improvement but subsequent worsening and "irritable episodes." Her mother has bipolar disorder.

Q1. What diagnosis should you strongly consider, and why?

Answer: Bipolar II Disorder, current episode depressive, with atypical features. Red flags for bipolarity: (1) Atypical depression features (hypersomnia, hyperphagia, leaden paralysis); (2) Early onset of depressive episodes (age 25); (3) Family history of bipolar disorder in first-degree relative; (4) Past "irritable episodes" following SSRI treatment, likely unrecognized hypomanic episodes or antidepressant-induced mood destabilization; (5) Recurrent episodes with poor sustained response to SSRIs. Screen formally with MDQ or HCL-32. Take detailed history of past mood elevations, including collateral from family.

Q2. What is the critical treatment error to avoid?

Answer: **Antidepressant monotherapy.** This patient has likely been repeatedly destabilized by SSRI monotherapy (the "irritable episodes" were probably antidepressant-induced hypomania or mixed features). In bipolar depression, antidepressant monotherapy carries risks of: (1) Manic/hypomanic switch; (2) Mixed state induction; (3) Rapid cycling induction; (4) Cycle acceleration. First-line treatment: quetiapine 300mg, OR lithium, OR lamotrigine, OR lurasidone. If antidepressant is added, it **MUST** be with mood stabilizer cover, using SSRI or bupropion (not SNRI/TCA), for the shortest effective duration.

Q3. What maintenance strategy would you recommend?

Answer: Lamotrigine (effective for preventing depressive relapses in BP-II, which is the predominant polarity), titrated slowly (25mg weeks 1-2, 50mg weeks 3-4, target 200mg by week 6-7). Alternatively, lithium (also provides anti-suicidal benefit). Quetiapine if used acutely can be continued for maintenance. Long-term psychoeducation and social rhythm therapy (IPSRT) as adjuncts. Monitor for any hypomanic switches. Indefinite maintenance given 3+ episodes.

Vignette 3: Lithium Toxicity

Case:

A 48-year-old man with Bipolar I Disorder on lithium 900mg/day for 3 years (stable, level 0.8 mEq/L last checked 2 months ago) presents with 3 days of vomiting and diarrhea due

to gastroenteritis. Today he is drowsy, has a coarse tremor, slurred speech, and unsteady gait. His wife reports increasing confusion over the past 24 hours.

Q1. What is your immediate clinical concern and what investigations would you order?

Answer: **Lithium toxicity** precipitated by dehydration from gastroenteritis. The clinical features (coarse tremor replacing fine, dysarthria, ataxia, confusion) correspond to moderate toxicity (expected level 2.0-2.5 mEq/L). Investigations: (1) STAT serum lithium level; (2) Serum creatinine and eGFR (assess renal function); (3) Serum electrolytes (Na, K, dehydration-related derangements); (4) CBC; (5) ECG (cardiac effects of toxicity); (6) Blood glucose (exclude hypoglycemia as contributing factor).

Q2. Outline the management of this patient step by step.

Answer:

1. **Stop lithium immediately**
2. **IV normal saline 0.9%**, aggressive rehydration (200-300 mL/hour initially, titrate to clinical response and urine output); corrects dehydration AND enhances renal lithium excretion
3. **Monitor lithium levels** every 2-4 hours until consistently declining
4. **Monitor renal function and electrolytes** serially
5. **Neurological monitoring**, GCS, reflexes, seizure watch
6. **ECG monitoring**, continuous cardiac telemetry
7. **Antiemetics** for ongoing vomiting (ondansetron, avoid metoclopramide if possible in psychiatric patients)
8. **Hemodialysis criteria:** If lithium level >2.5 mEq/L with these severe symptoms (ataxia, confusion, dysarthria), hemodialysis is indicated. Also indicated if level >4.0 (regardless of symptoms), or if renal failure develops, or if clinical deterioration despite supportive care
9. **Post-dialysis:** Monitor for rebound (lithium redistributes from tissue compartments; repeat level 6-8 hours post-dialysis; may need repeat dialysis)
10. **Once recovered:** Reintroduce lithium at lower dose after complete recovery; consider whether valproate or alternative mood stabilizer more appropriate given toxicity history. Educate extensively about fluid intake, sick day rules, and drugs to avoid (NSAIDs, dehydration)

Q3. What are the "sick day rules" you would educate this patient about to prevent recurrence?

Answer: (1) If vomiting, diarrhea, or fever develop: maintain fluid intake, drink extra water; (2) If unable to keep fluids down for >24 hours: STOP lithium and seek medical attention; (3) Avoid NSAIDs (ibuprofen, naproxen); use paracetamol for pain/fever; (4) Avoid new medications without checking with prescriber (especially ACE inhibitors, ARBs, thiazides);

(5) Avoid excessive sweating without fluid replacement (hot weather, intense exercise); (6) Do not reduce salt intake without medical advice; (7) Carry a lithium alert card; (8) Know early toxicity signs: increased tremor, nausea, diarrhea, drowsiness, stop lithium and contact doctor if these develop.

Vignette 4: Treatment-Resistant Depression Workup

Case:

A 42-year-old school teacher presents with persistent depressive symptoms for 18 months. She has tried escitalopram 20mg for 12 weeks (minimal improvement), then duloxetine 120mg for 10 weeks (slight improvement in energy but mood unchanged). She reports pervasive low mood, anhedonia, insomnia, fatigue, concentration difficulty, and passive suicidal ideation ("I wish I wouldn't wake up"). She is adherent to medications. No substance use.

Q1. Does this patient meet criteria for TRD? Stage the resistance.

Answer: Yes, this meets TRD criteria: failure of 2 adequate antidepressant trials from different classes (SSRI: escitalopram 20mg x 12 weeks; SNRI: duloxetine 120mg x 10 weeks) at adequate doses for adequate duration with confirmed adherence. **Thase-Rush staging: Stage II** (failed SSRI + failed different class). **MGH score: 2 points** (2 failed adequate trials). Before confirming TRD, rule out pseudo-resistance: (1) Confirm adherence (serum levels if available); (2) Screen for bipolarity (MDQ, clinical history); (3) Screen for comorbidities (thyroid, B12, anemia, diabetes, chronic pain, substance use, sleep apnea); (4) Assess personality pathology; (5) Assess psychosocial stressors.

Q2. Outline the next steps in her management algorithm.

Answer: Given PARTIAL response to duloxetine (slight energy improvement):

1. **Augmentation preferred** (partial response = augment; no response = switch)
2. **First-line augmentation:** Lithium (0.6-0.8 mEq/L, response expected 2-4 weeks) OR atypical antipsychotic (aripiprazole 2-5mg is best-tolerated; alternatives: quetiapine 50-150mg, brexpiprazole 1-2mg)
3. **If lithium/atypical augmentation fails:** Try T3 augmentation (liothyronine 25-50 mcg/day even if euthyroid)
4. **If augmentation fails:** Combination strategy, add mirtazapine to duloxetine ("California rocket fuel" variant) or switch entirely to venlafaxine + mirtazapine
5. **If combinations fail:** Esketamine (intranasal, with oral antidepressant), FDA approved for TRD
6. **If above fails:** ECT referral (especially given passive suicidal ideation)
7. **Concurrent:** Psychotherapy (CBT or CBASP for chronic depression); address sleep hygiene; graded exercise

Q3. The patient asks about ketamine clinics she read about online. How do you counsel her?

Answer: Esketamine (Spravato) is the FDA-approved form for TRD, administered intranasally in certified clinical settings with 2-hour monitoring. It has strong evidence for rapid antidepressant effect (hours to days) and is used with a concurrent oral antidepressant. IV ketamine (offered in private clinics) has growing evidence but is off-label for depression, not FDA-approved, variable quality control, and insurance may not cover it. Both have similar side effects: dissociation, dizziness, sedation, nausea, BP elevation. She should discuss with her psychiatrist whether she has met the criteria to warrant esketamine (failed ≥ 2 adequate trials, which she has). IV ketamine clinics vary in quality; ensure any clinic has psychiatric oversight, monitoring protocols, and a plan for maintenance treatment.

Vignette 5: Elderly Depression vs Dementia

Case:

A 72-year-old retired professor is brought by his son with a 3-month history of forgetfulness, social withdrawal, poor appetite with 5 kg weight loss, and loss of interest in reading (his lifelong passion). When asked about his memory, he says "I can't remember anything anymore. My brain is finished." He appears sad, makes poor eye contact, and repeatedly answers "I don't know" to cognitive screening questions. His wife died 6 months ago.

Q1. What is your differential diagnosis, and which diagnosis is most likely?

Answer: Differential: (1) **Major Depressive Disorder (most likely)**, presenting as pseudodementia; (2) Dementia (Alzheimer's or vascular); (3) Prolonged grief disorder; (4) Medical cause (hypothyroidism, B12 deficiency, cerebral pathology). **Pseudodementia is most likely** because: (a) Relatively acute onset (3 months) with dateable precipitant (wife's death); (b) Patient emphasizes cognitive deficits ("I can't remember anything") rather than minimizing them; (c) "Don't know" answers on testing (poor effort rather than genuine failure); (d) Prominent vegetative symptoms (appetite, weight loss, anhedonia); (e) Sad affect with loss of interest in previously valued activities.

Q2. How would you systematically differentiate pseudodementia from early dementia?

Answer:

- 1. Cognitive testing:** Apply MMSE/MoCA, in pseudodementia, patient gives up easily, says "I don't know," scores may fluctuate; in dementia, patient tries hard, near-miss answers, more consistent pattern of deficit
- 2. Detailed history (from son):** Onset (acute vs gradual), temporal relationship to bereavement, any confabulation, any language decline, any getting lost, any personality change

3. **Formal neuropsychological testing:** Inconsistent performance in pseudodementia; consistent pattern of decline in dementia
4. **Investigations:** TFT, B12, folate, CBC, ESR/CRP, RFT, LFT, blood glucose, VDRL, calcium, urine analysis
5. **Neuroimaging:** MRI brain, assess for hippocampal atrophy (dementia), white matter hyperintensities (vascular depression), cortical atrophy
6. **Depression-specific scales:** GDS (Geriatric Depression Scale), HAM-D
7. **Therapeutic trial:** If depression suspected, start antidepressant; improvement in cognition supports pseudodementia
8. **Caveat:** Pseudodementia increases risk of developing true dementia (30-50% within 5 years); longitudinal follow-up essential

Q3. How would you manage this patient?

Answer:

1. **Pharmacotherapy:** SSRI first-line, escitalopram 5mg initially, titrate to 10-20mg ("start low, go slow, but go"); alternatively, mirtazapine 7.5-15mg at bedtime if insomnia, poor appetite, and weight loss are prominent (appetite-stimulating, sedating)
2. **Avoid:** TCAs (anticholinergic effects in elderly, confusion, falls, urinary retention, cardiac risk); benzodiazepines (falls, paradoxical confusion, dependence)
3. **Monitor:** Sodium level at 1-2 weeks (SSRI-induced hyponatremia risk higher in elderly); response assessment at 4-6 weeks
4. **Psychotherapy:** Supportive therapy, grief counseling; CBT adapted for elderly; behavioral activation
5. **ECT:** Consider if severe (significant weight loss with nutritional compromise), suicidal, or medication-refractory; safe and effective in elderly
6. **Social interventions:** Engage son and family; assess social isolation; day center or community activities; ensure basic needs met
7. **Follow-up:** Regular; repeat cognitive testing in 3-6 months to confirm improvement; remain vigilant for emergent dementia

Vignette 6: Peripartum Depression

Case:

A 28-year-old primigravida, 2 weeks postpartum, is brought by her husband. She has been persistently tearful, unable to bond with her baby, experiencing intrusive thoughts of harming the infant ("What if I drop the baby from the balcony?"), severe insomnia despite the baby sleeping, loss of appetite, and guilt ("I'm the worst mother"). She has no prior psychiatric history. She is breastfeeding.

Q1. What is the diagnosis? How do you differentiate from "baby blues"?

Answer:Major Depressive Disorder with peripartum onset (peripartum depression).

Differentiation from baby blues:

FEATURE	BABY BLUES	PERIPARTUM DEPRESSION
Timing	Days 3-10 postpartum	Onset within 4 weeks postpartum (DSM-5); clinically up to 1 year
Duration	Self-resolving within 2 weeks	Persistent (>2 weeks)
Severity	Mild mood lability, tearfulness	Full depressive syndrome (meets MDE criteria)
Functioning	Intact; can care for baby	Impaired; difficulty with infant care
Prevalence	50-80% of postpartum women	10-15%
Bonding	Usually preserved	Impaired
Intrusive thoughts	Absent	May be present

This patient has full MDE criteria (>2 weeks duration, persistent low mood, anhedonia, insomnia, appetite loss, guilt) with peripartum onset. The intrusive ego-dystonic thoughts of harm ("What if I drop the baby?") are consistent with peripartum depression/OCD, these are NOT psychotic and the patient is distressed by them (ego-dystonic). However, must screen for peripartum psychosis (does she hear voices? does she believe she SHOULD harm the baby? is there bizarre behavior?).

Q2. How do you assess and manage the intrusive thoughts?

Answer:The intrusive thoughts of harming the infant are ego-dystonic (she is horrified by them, not acting on them). This pattern is consistent with peripartum OCD-like phenomena (common in peripartum depression). Key assessment: (1) Are thoughts ego-dystonic (distressing) vs ego-syntonic (rational/justified)? (2) Any command hallucinations? (3) Any delusional beliefs about the baby? (4) Any history of violence? (5) Is she avoiding the baby out of fear (protective behavior, actually a GOOD sign of intact maternal concern)?

If ego-dystonic with no psychotic features: reassure that intrusive thoughts are common and do NOT indicate danger. They indicate anxiety and hypervigilance, not intent. Support adequate supervision of mother-infant dyad without separating them (separation worsens bonding and depression).

Q3. What is your treatment plan considering she is breastfeeding?

Answer:

1. **Mild-moderate:** CBT/IPT first-line (strong evidence in perinatal depression)

2. **Moderate-severe (this patient):** SSRI + psychotherapy. **Sertraline preferred**, lowest breast milk transfer of all SSRIs; extensive safety data in breastfeeding. Start 25-50mg, titrate to 100-150mg
 3. **Severe/refractory:** Consider brexanolone (IV, inpatient, rapid but expensive/limited access) or zuranolone (oral, 14-day course, newer option)
 4. **ECT:** If severe with suicidality, psychotic features, or inability to care for self/baby; safe postpartum
 5. **Practical:** Ensure adequate sleep (partner/family to take some nighttime feeds, expressed milk or formula for one feed); social support; screen for domestic violence; EPDS screening
 6. **Safety:** Supervise mother-infant interactions supportively (not punitively); involve family; safety plan if suicidality present
 7. **Avoid:** Paroxetine (cardiac teratogenicity if future pregnancy), fluoxetine (higher breast milk levels), benzodiazepines (sedation in infant)
-

Vignette 7: Suicide Risk Assessment

Case:

A 55-year-old farmer from rural Karnataka presents to the emergency department after his family found him with a bottle of organophosphate pesticide, which he had not yet ingested. He recently lost his crop to drought, is Rs. 5 lakh in debt, his wife left him 3 months ago, and he has been drinking heavily (quarter of whisky daily for 2 months). He says "there is no point in living" and "my family would be better off without me." He appears calm and cooperative.

Q1. Perform a structured suicide risk assessment for this patient.

Answer: SAD PERSONS scoring:

- Sex: Male (+1)
- Age: 55 (+1)
- Depression: Likely present (+1)
- Previous attempt: Assess, this appears to be a first but interrupted attempt (+1 for current attempt/preparatory behavior)
- Ethanol: Active heavy use (+1)
- Rational thinking loss: Assess for psychosis (likely intact but cognitively narrowed) (+0 or +1)
- Social supports lacking: Wife left, likely isolated (+1)
- Organized plan: Had method (pesticide), had access (+1)
- No spouse: Separated (+1)

- Sickness: Assess, farming work, chronic pain? (need to assess)

Score: 7-9/10 = HIGH RISK, hospitalize.

C-SSRS assessment:

- Ideation: Active suicidal ideation with plan and intent (Level 5, most severe)
- Behavior: Preparatory act (obtained pesticide); interrupted attempt (found before ingestion)
- Frequency / duration: Likely persistent given circumstances

RED FLAGS: Calm demeanor after a near-attempt (may indicate decision made / relief at having a plan); access to lethal means (farmer = pesticides readily available); hopelessness ("no point"); perceived burdensomeness ("family better off without me", Joiner's interpersonal theory); multiple simultaneous stressors (financial, relational, substance use); Indian male farmer = high-risk demographic.

Q2. What immediate management is required?

Answer:

1. **Safety:** Admit to psychiatric inpatient unit; 1:1 observation; remove all means (belt, sharp objects, medications)
2. **Means restriction:** Counsel family to remove ALL pesticides and dangerous substances from the home and lock them securely; this is the single most effective intervention
3. **Medical clearance:** Assess for organophosphate exposure even if he says he didn't ingest (check for cholinergic symptoms); blood glucose, LFT, RFT
4. **Assess for depression:** Full mental status examination; HAM-D / PHQ-9; likely Major Depressive Disorder
5. **Assess for alcohol use disorder:** CAGE / AUDIT; medical detoxification if indicated (risk of withdrawal seizures)
6. **Pharmacotherapy:** Start SSRI (sertraline 50mg) for depression; manage alcohol withdrawal with benzodiazepine protocol if needed; consider short-term sedation for acute distress
7. **Legal considerations:** Under Mental Healthcare Act 2017, Section 115, attempted suicide is NOT a criminal offense; patient to be treated, not prosecuted; document this
8. **Psychosocial:** Social work consultation for financial counseling, legal aid for debt; connect with agricultural support schemes; family counseling

Q3. What population-level intervention is most relevant to preventing farming suicides in India?

Answer: Means restriction, specifically pesticide regulation. The Sri Lanka model demonstrated that banning the most toxic pesticides (WHO Class I) reduced national suicide rate by ~50% without increasing suicides by other methods (substitution did not

fully occur). In India, the evidence supports: (1) Banning highly hazardous pesticides (HHPs), endosulfan already banned; expand to others; (2) Locked pesticide storage (community storage models); (3) Promoting less toxic alternatives (integrated pest management); (4) Training primary healthcare workers in rural areas to detect depression (mhGAP model); (5) Addressing root causes, crop insurance, debt relief, irrigation. The Pesticides Management Bill in India has been debated; implementing it would save lives.

Vignette 8: SSRI Non-Response

Case:

A 38-year-old IT professional was started on escitalopram 10mg for Major Depressive Disorder (moderate severity) 6 weeks ago. He reports "no change" in mood. He takes the medication "most days" and occasionally skips weekends. His PHQ-9 score is 18 (unchanged from baseline of 19).

Q1. Before diagnosing treatment failure, what factors should you assess?

Answer:Pseudo-resistance checklist:

1. **Adherence:** He admits to skipping weekends, this is intermittent non-adherence. Escitalopram has a 27-32 hour half-life; weekend skipping may significantly reduce effectiveness. Ask specifically about missed doses, pill counts, pharmacy records
2. **Adequate dose:** 10mg is the starting / therapeutic dose; may need optimization to 15-20mg before calling this a failure
3. **Adequate duration:** 6 weeks is borderline adequate; some patients respond at 8-12 weeks
4. **Diagnosis review:** Re-screen for bipolarity (MDQ), check for comorbidities (thyroid, B12, substance use, sleep disorder, personality pathology)
5. **Psychosocial factors:** Ongoing stressors? Work stress in IT sector? Relationship issues? Sleep hygiene?
6. **Comorbid anxiety:** May need higher dose or adjunctive treatment
7. **Substance use:** Screen for alcohol, cannabis, other substances

Q2. What is your management plan?

Answer:

1. **Optimize current trial first:** Increase escitalopram to 20mg; address adherence (psychoeducation about daily dosing, set phone reminders, identify barriers to adherence); reassess in 4 weeks
2. **If still no response at 20mg after 4 additional weeks:** This constitutes a true Thase-Rush Stage I failure

3. **Next step, SWITCH** (since no response rather than partial response): Switch to SNRI (venlafaxine XR 75mg → titrate to 150-225mg) or duloxetine (60-120mg)
4. **Concurrent psychotherapy:** CBT, strong evidence as adjunct; behavioral activation if initiation of CBT is delayed
5. **Sleep hygiene and exercise:** Both have evidence as adjuncts to pharmacotherapy
6. **If partial response to SNRI:** Augment with aripiprazole 2-5mg or lithium 600-900mg

Q3. He asks if he can stop medication once he feels better. How do you counsel him?

Answer: After remission is achieved: continue at the same dose for at least 6-12 months (continuation phase) to prevent relapse. This is his first episode, so after 6-12 months of sustained remission, gradual taper over 4-8 weeks is reasonable. If he has recurrent episodes (2+ episodes, or 1 severe episode with suicidality / psychosis), maintenance treatment may be indefinite. Abrupt discontinuation risks: (1) Relapse; (2) Discontinuation syndrome (dizziness, sensory disturbances, GI upset, less common with escitalopram vs paroxetine / venlafaxine but still possible). Educate that antidepressants are NOT addictive (common misconception) but should not be stopped abruptly.

Vignette 9: Mixed Features

Case:

A 30-year-old woman with known Bipolar I Disorder presents with 5 days of elevated mood, decreased sleep (4 hours / night, feels energized), grandiose plans to start a restaurant chain, and pressured speech. However, she simultaneously reports feeling "desperately sad inside," frequent crying episodes, and suicidal thoughts. She says "I have all this energy but I want to die."

Q1. What DSM-5 specifier applies to this presentation?

Answer: Manic episode with mixed features. She meets full criteria for a manic episode (elevated mood, decreased sleep need, grandiosity, pressured speech, >7 days if criteria have been met). The mixed features specifier requires ≥ 3 concurrent depressive symptoms during the manic episode: (1) Depressed mood ("desperately sad inside"); (2) Diminished interest is not clearly mentioned but crying episodes suggest; (3) Suicidal ideation. Three depressive symptoms during mania qualifies for the "with mixed features" specifier.

Clinical significance: Mixed features = HIGHER suicide risk than pure mania. The combination of manic energy / impulsivity with depressive hopelessness / suicidality is particularly dangerous.

Q2. How does this presentation change your treatment approach compared to pure mania?

Answer: Key treatment differences:

1. **Avoid antidepressants entirely**, worse outcomes in mixed states
2. **Lithium may be LESS effective** in mixed presentations compared to pure euphoric mania
3. **Valproate often preferred**, better evidence for mixed states than lithium
4. **Atypical antipsychotics**: Olanzapine, quetiapine, aripiprazole, asenapine all have evidence for mixed mania
5. **Combination therapy often needed earlier**: Mood stabilizer + atypical antipsychotic
6. **Higher suicide monitoring**: Mixed features = highest suicide risk in bipolar; consider inpatient admission; 1:1 observation if actively suicidal; remove means

Q3. What is the suicide risk profile of this patient?

Answer: VERY HIGH. Risk factors: (1) Active suicidal ideation during a mood episode; (2) Mixed features (energy + impulsivity to act on suicidal thoughts); (3) Female (more attempts); (4) Known bipolar disorder (15-20x general population suicide rate); (5) Current acute episode. Protective factor assessment needed: children? social support? religious beliefs? Immediate safety planning required. This patient should be hospitalized until the mixed episode resolves. The lethality of mixed states is that patients have the depressive wish to die AND the manic energy to act on it, the most dangerous combination in all of psychiatry.

Vignette 10: Rapid Cycling

Case:

A 40-year-old woman with Bipolar II Disorder has had 6 mood episodes in the past 12 months (4 depressive, 2 hypomanic). She is currently on lithium 900mg/day (level 0.7 mEq/L) and sertraline 100mg/day. Her last TSH was 8.2 mIU/L (elevated; reference 0.5-4.5).

Q1. What pattern does this represent, and what are the likely contributing factors?

Answer: **Rapid cycling**, defined as ≥ 4 mood episodes in 12 months. She has had 6 episodes, qualifying clearly. Contributing factors in this case:

1. **Antidepressant use (sertraline)**, classic trigger for rapid cycling; antidepressants can accelerate cycle frequency, particularly in bipolar
2. **Hypothyroidism (TSH 8.2)**, unrecognized or undertreated hypothyroidism is a well-established risk factor for rapid cycling and medication resistance
3. **Female sex**, rapid cycling is 2-3x more common in women
4. **BP-II**, rapid cycling is more common in BP-II than BP-I

Q2. What immediate changes would you make to her treatment?

Answer:

1. **Taper and discontinue sertraline**, antidepressant withdrawal is the first and most critical intervention in rapid cycling. Taper over 2-4 weeks
2. **Treat hypothyroidism**, start levothyroxine (50-75 mcg initially; titrate to normalize TSH). This alone may significantly reduce cycling. Target TSH in lower normal range
3. **Optimize/switch mood stabilizer**: Lithium may be LESS effective in rapid cycling. Options: (a) Add valproate to lithium (combination therapy); or (b) Switch to valproate monotherapy if lithium has been clearly ineffective; or (c) Lamotrigine addition for depression prevention (given 4 depressive vs 2 hypomanic episodes, her predominant polarity is depressive)
4. **Monitor closely**, mood charting (daily mood diary) is essential for tracking response

Q3. After 3 months, thyroid is normalized, sertraline discontinued, and she is on lithium + valproate. She develops a depressive episode. How do you manage it?

Answer: Bipolar depression in a rapid-cycling patient, this is among the hardest presentations to treat:

1. **Do NOT reintroduce antidepressants**, high risk of re-inducing rapid cycling
2. **First-line:** Quetiapine 300mg (evidence for bipolar depression; won't destabilize cycling)
3. **Alternative/adjunct:** Lamotrigine (titrate slowly; better for prevention but can be trialed acutely)
4. **Lurasidone:** FDA approved for bipolar depression; add to lithium / valproate
5. **Optimize existing mood stabilizers:** Ensure valproate level in upper therapeutic range
6. **If refractory:** ECT (effective in bipolar depression, no cycling risk)
7. **Psychotherapy:** CBT adapted for bipolar, IPSRT for rhythm stabilization
8. **Lifestyle:** Sleep hygiene (critical in rapid cycling, any sleep disruption can trigger episodes), regular social rhythms, exercise

Vignette 11: Valproate in Women of Childbearing Age

Case:

A 26-year-old newly married woman with Bipolar I Disorder has been stable on valproate 1000mg / day for 3 years (no episodes since starting). She comes for preconception counseling as she and her husband wish to start a family within the year.

Q1. What are the teratogenic risks of valproate, and why is this an ethical dilemma?

Answer: Valproate teratogenicity:

1. **Neural tube defects:** 1-2% risk (10x general population), spina bifida most common
2. **Neurodevelopmental effects:** Average 8-9 IQ point reduction in children exposed in utero (NEAD study); higher rates of autism spectrum disorder, developmental delay, and learning disabilities

3. **Other malformations:** Craniofacial, cardiac, limb defects

4. **Dose-dependent:** Higher doses = greater risk; no established safe threshold

The ethical dilemma: She has been stable for 3 years on this medication. Switching introduces: (1) Risk of relapse during switch; (2) Risk of relapse during pregnancy if alternative is less effective; (3) Manic or depressive episodes during pregnancy are themselves harmful to fetus and mother. However, continuing valproate exposes the fetus to serious, well-documented harm. Recent EMA/MHRA guidelines effectively contraindicate valproate in women of childbearing age unless no alternative exists and a Pregnancy Prevention Programme is in place. The principle of informed consent and shared decision-making is critical.

Q2. What is your management plan?

Answer:

1. **Switch medication BEFORE conception**, do NOT wait until pregnancy is confirmed (NTD risk is highest in first 4-6 weeks, often before pregnancy is recognized)
2. **Switch to safer mood stabilizer:** Lamotrigine is the preferred option, best safety data in pregnancy (minimal teratogenicity); effective for bipolar maintenance, especially depression prevention. Titrate lamotrigine UP while tapering valproate DOWN (cross-titration). Note: valproate inhibits lamotrigine metabolism, so lamotrigine levels will RISE as valproate is withdrawn
3. **Alternative:** Lithium, effective for maintenance; Ebstein's anomaly risk is ~0.1% (lower than historically cited); requires level monitoring through pregnancy; dose adjustments needed (GFR increases in pregnancy → clearance increases → may need dose increase; then after delivery → GFR drops → risk of toxicity); fetal echocardiography at 16-20 weeks
4. **High-dose folic acid:** 5mg / day (not standard 400mcg), start at least 1 month before conception; continue through first trimester
5. **Contraception until switch is complete and stable** (3-6 months on new medication without relapse before attempting conception)
6. **Multidisciplinary team:** Psychiatrist + obstetrician + genetic counselor
7. **Monitor closely during switch and pregnancy**, pregnancy is a high-risk period for relapse

Q3. She asks if she can "just stop all medications during pregnancy." How do you counsel her?

Answer: Stopping all medication is NOT recommended for Bipolar I Disorder. Relapse rates off medications during pregnancy are very high, up to 70% (Viguera et al., 2007). A manic episode during pregnancy poses serious risks: poor self-care, risky behavior, substance use, impaired judgment, nonadherence to prenatal care, and postpartum psychosis risk. A depressive episode risks: poor nutrition, self-harm / suicide, impaired bonding,

preeclampsia association. The goal is to find the safest effective medication, not to be medication-free. Lamotrigine offers the best benefit-risk ratio. The decision should be collaborative, fully informed, and documented.

Vignette 12: Seasonal Affective Disorder

Case:

A 35-year-old software developer relocated from Bangalore to London 2 years ago. For both winters since the move, she has experienced low mood beginning in October, resolving spontaneously by March. Symptoms include hypersomnia (sleeping 11-12 hours), carbohydrate craving, weight gain (4 kg each winter), fatigue, and social withdrawal. Her summers are entirely normal. She had no such symptoms in Bangalore.

Q1. What is the diagnosis and what is the pathophysiology?

Answer: **Major Depressive Disorder, recurrent, with seasonal pattern** (Seasonal Affective Disorder, SAD). Criteria: (1) Regular temporal relationship between onset of depressive episodes and a particular time of year; (2) Full remissions at a characteristic time (spring); (3) ≥ 2 major depressive episodes with seasonal pattern in last 2 years; (4) Seasonal episodes substantially outnumber non-seasonal episodes over lifetime.

Pathophysiology:

- Reduced sunlight exposure $\rightarrow$ decreased retinal light input $\rightarrow$ altered melatonin secretion (prolonged melatonin phase) + reduced serotonin synthesis (5-HT production is light-dependent)
- Phase-delay hypothesis: circadian rhythm shifts later relative to the light-dark cycle
- Latitude-dependent: higher prevalence at northern latitudes (explains onset after moving from Bangalore $\sim 13^\circ\text{N}$ to London $\sim 51^\circ\text{N}$)
- Atypical depression features (hypersomnia, hyperphagia, carbohydrate craving) are characteristic

Q2. What are the first-line treatment options?

Answer:

1. **Light therapy (phototherapy):** FIRST-LINE for SAD. 10,000 lux bright light box for 30 minutes each morning (upon waking). Position 16-24 inches from face, eyes open but not staring directly at light. Response typically within 1-2 weeks. Continue through the winter season. Evidence-based; comparable efficacy to SSRIs for seasonal depression
2. **SSRIs:** Fluoxetine or sertraline, effective for SAD; can be used alone or combined with light therapy; bupropion XL approved for PREVENTION of seasonal depression (start in autumn before symptom onset)

3. **CBT adapted for SAD:** Evidence-based; may have more durable effects than light therapy (lower relapse rate in subsequent winter)
4. **Lifestyle:** Regular exercise, maximize outdoor daylight exposure (walk during lunch), maintain social activities, vitamin D supplementation (often deficient at high latitudes)

Q3. She asks about prevention for next winter. What do you advise?

Answer:(1) Begin light therapy prophylactically in September/October (before symptoms onset); (2) Bupropion XL 150-300mg starting in early autumn, FDA approved for prevention of seasonal MDE; (3) Maintain exercise routine through autumn-winter; (4) Vitamin D supplementation (1000-2000 IU daily through winter); (5) Consider a dawn simulator alarm clock (gradually increases light in bedroom before waking); (6) If considering relocation back to India, this would effectively treat the condition; (7) Monitor mood with a brief weekly self-rating (PHQ-2 or mood diary) to catch early symptoms.

Vignette 13: Psychotic Depression

Case:

A 58-year-old woman presents with 6 weeks of severe depression, pervasive low mood, total anhedonia, psychomotor retardation (barely moves, speaks in whispers), 8 kg weight loss from food refusal, and early morning awakening at 3 AM. She believes her internal organs are "rotting away" and that she is already dead. She says the police are coming to arrest her for crimes she committed in a past life.

Q1. What is the diagnosis and what specific features are present?

Answer:Major Depressive Disorder, severe, with mood-congruent psychotic features.
Specific features:

1. **Melancholic features:** Total anhedonia, distinct quality of depressed mood, psychomotor retardation, early morning awakening, significant weight loss, worse in morning (likely)
2. **Nihilistic delusions (Cotard syndrome):** Believes organs are "rotting" and she is "already dead", Cotard's delusion/syndrome is pathognomonic of psychotic depression; involves nihilistic delusions about the body, existence, or the world
3. **Delusions of guilt:** Believes she committed crimes, mood-congruent guilt delusion
4. **Persecutory ideation:** Believes police will arrest her, this could be mood-congruent (punishment for perceived crimes) or mood-incongruent (if unrelated to guilt theme)
5. **Nutritional compromise:** 8 kg weight loss from food refusal, medical emergency

Q2. What is the optimal treatment for this presentation?

Answer:Psychotic depression requires COMBINATION treatment or ECT, antidepressant monotherapy is insufficient:

1. ECT, strongest recommendation: For this patient specifically, ECT is arguably the FIRST-LINE treatment because: (a) Psychotic depression responds best to ECT (>80% response rate); (b) Nutritional compromise is a clear indication; (c) Fastest onset of all treatments; (d) Cotard syndrome responds particularly well to ECT. Bilateral placement preferred for psychotic depression.

1. Pharmacotherapy (if ECT not immediately available): Antidepressant + antipsychotic combination. Examples: (a) Venlafaxine + olanzapine (evidence from Meyers et al., STOP-PD study); (b) Sertraline + olanzapine; (c) TCA (nortriptyline) + antipsychotic. Antidepressant alone is INFERIOR to combination for psychotic depression.

1. Supportive care: IV fluids and nutrition if refusing oral intake; 1:1 nursing observation (suicide risk very high in psychotic depression); monitor medical status (dehydration, electrolytes, muscle wasting).

Q3. After successful ECT treatment, she remits. What is the maintenance plan?

Answer:(1) Continue antidepressant + antipsychotic for at least 6-12 months post-remission (STOP-PD II study showed discontinuing olanzapine led to high relapse rates); (2) Maintenance ECT: weekly → biweekly → monthly, especially if she has relapsed on pharmacotherapy before; (3) Gradual taper of antipsychotic can be attempted after 4-6 months if stable, with close monitoring for relapse; (4) If antipsychotic discontinued, maintain antidepressant indefinitely (given severity of this episode); (5) Longitudinal monitoring, psychotic depression has high recurrence rate; educate family about early warning signs.

Vignette 14: Lamotrigine Rash Scenario

Case:

A 32-year-old man with Bipolar II Disorder was started on lamotrigine 2 weeks ago for maintenance (currently on 25mg/day, titrating per protocol). He calls today reporting a "rash on his arms" that appeared yesterday. He has no fever, no mucosal involvement, no pain, and the rash is described as a few scattered, non-confluent papules.

Q1. How do you assess whether this rash is benign or a harbinger of Stevens-Johnson Syndrome?

Answer:Critical assessment, this is a medical decision, not a reassurance call:

FEATURE	BENIGN RASH	CONCERNING FOR SJS/TEN
Timing	Anytime	Typically within 8 weeks of starting (peak risk)
Mucosal involvement	None	YES, oral, genital, ocular (pathognomonic)

FEATURE	BENIGN RASH	CONCERNING FOR SJS/TEN
Systemic symptoms	None	Fever, malaise, arthralgias
Pain	None	Skin tenderness, burning
Rash character	Scattered papules, not confluent	Confluent, purpuric, targetoid, blistering, epidermal detachment
Progression	Stable or resolving	Rapidly spreading
Nikolsky sign	Negative	Positive (epidermis shears with pressure)

In this patient: no fever, no mucosal involvement, no pain, non-confluent papules, likely benign. HOWEVER, the standard of care is conservative.

Q2. What is your management recommendation?

Answer:

1. **Default position: STOP lamotrigine** until the rash is formally evaluated. This is the standard recommendation, when in doubt, stop. The risk of missing early SJS far outweighs the inconvenience of stopping a medication that can be restarted.
2. **See the patient in person TODAY or within 24 hours**, rash cannot be adequately assessed by phone/telemedicine alone
3. **Examine for:** Mucosal lesions (mouth, eyes, genitals), skin tenderness, Nikolsky sign, confluence, facial involvement, blistering
4. **If benign rash confirmed:** May cautiously restart lamotrigine at a LOWER dose (12.5mg) with even slower titration. Some clinicians add antihistamine
5. **If any concerning features:** Do NOT restart lamotrigine. Switch to alternative (lithium, quetiapine). Dermatology consultation
6. **If SJS confirmed:** Emergency hospitalization, dermatology/burns unit, supportive care (similar to burns management), stop ALL potential causative drugs
7. **Educate patient:** "If the rash spreads, if you develop mouth sores, eye irritation, fever, or if it hurts, go to the emergency department immediately"

Q3. What factors increase the risk of lamotrigine-induced SJS?

Answer:(1) **Rapid titration**, the single most important modifiable risk factor; slower = safer; (2) **Concurrent valproate**, doubles lamotrigine levels; if on valproate, must halve the lamotrigine titration rate; (3) **Young age**, children and adolescents at higher risk; (4) **History of rash with other anticonvulsants**; (5) **HLA-B*1502 allele**, same allele associated with carbamazepine SJS; some evidence for lamotrigine too, especially in Asian populations; (6) **Higher starting dose**; (7) **Immune activation**, concurrent infection may increase risk. Risk

is highest in the first 8 weeks and essentially drops to baseline after 6 months of stable dosing.

Vignette 15: Serotonin Syndrome from Drug Interaction

Case:

A 45-year-old woman on fluoxetine 40mg for Major Depressive Disorder visits an orthopedic surgeon for back pain. The surgeon prescribes tramadol 50mg TDS. Three days later, she presents to the ER with agitation, confusion, profuse sweating, tachycardia (HR 120), blood pressure 160/95, temperature 38.8C, bilateral lower limb clonus, hyperreflexia, and myoclonic jerks. Her pupils are dilated.

Q1. What is the diagnosis, and what caused it?

Answer: Serotonin syndrome caused by the combination of fluoxetine (SSRI, serotonin reuptake inhibitor) + tramadol (opioid with serotonin reuptake inhibition properties). Fluoxetine is also a potent CYP2D6 inhibitor, which would further increase tramadol levels (tramadol is metabolized by CYP2D6).

Hunter Criteria assessment:

- Serotonergic agent: YES (fluoxetine + tramadol)
- Inducible clonus + agitation + diaphoresis: YES
- Diagnosis confirmed.

Key features present: Mental status change (agitation, confusion), autonomic instability (tachycardia, hypertension, hyperthermia, diaphoresis, mydriasis), neuromuscular hyperactivity (clonus, hyperreflexia, myoclonus). The classic triad: HAM = Hyperactivity (neuromuscular), Autonomic instability, Mental status changes.

Q2. How do you differentiate this from Neuroleptic Malignant Syndrome (NMS)?

Answer:

FEATURE	THIS PATIENT (SEROTONIN SYNDROME)	NMS
Onset	Rapid (3 days, hours to days)	Slow (days to weeks)
Causative agent	Serotonergic drugs	Dopamine antagonists (antipsychotics)
Neuromuscular	CLONUS, hyperreflexia, myoclonus	Lead-pipe RIGIDITY, bradyreflexia
Pupils	Mydriasis (dilated)	Normal
Bowels	Diarrhea	Normal or ileus

FEATURE	THIS PATIENT (SEROTONIN SYNDROME)	NMS
CK	Mild elevation	MARKEDLY elevated (often >1000)
Temperature	Elevated (38.8C)	Often higher (>40C)
Resolution	Hours to days after drug withdrawal	Days to weeks

The presence of CLONUS (especially lower limb), hyperreflexia, and rapid onset with serotonergic drugs clinches serotonin syndrome. NMS would show rigidity and bradyreflexia.

Q3. What is the management of this patient?

Answer:

1. **Stop ALL serotonergic agents immediately**, discontinue both fluoxetine and tramadol
2. **Supportive care:** IV fluids (hydration), continuous monitoring (HR, BP, temperature, SpO2), cooling measures for hyperthermia (cooling blankets, ice packs, tepid sponging; avoid antipyretics, fever is from muscle activity, not hypothalamic)
3. **Benzodiazepines:** Lorazepam 1-2mg IV for agitation and to reduce neuromuscular hyperactivity
4. **Cyproheptadine:** Specific antidote, 5-HT_{2A} antagonist. Loading dose 12mg PO/NG, then 4-8mg every 2 hours until improvement (max 32mg/day). Oral/NG only (no parenteral form)
5. **If severe (temperature >41.1C, seizures, arrhythmias):** ICU admission, intubation, neuromuscular paralysis (non-depolarizing agent, NOT succinylcholine as it may worsen hyperkalemia from rhabdomyolysis), mechanical ventilation
6. **Monitor:** CK (rhabdomyolysis risk), renal function (myoglobin nephropathy), electrolytes, coagulation (DIC risk in severe cases)
7. **Expected resolution:** 24-72 hours after drug discontinuation (fluoxetine has a long half-life, so may take longer)
8. **Follow-up:** When stable, restart antidepressant (avoid tramadol in the future); educate about drug interactions; for pain management, alternatives include paracetamol, NSAIDs, gabapentin, or non-serotonergic opioids (morphine, oxycodone have minimal serotonergic activity)
9. **Systems-level learning:** This was a prescribing error, the orthopedic surgeon should have checked her medication list. Advocate for electronic prescribing alerts for serotonergic drug combinations. Common dangerous combinations: SSRI/SNRI + tramadol, SSRI/SNRI + MAOI, SSRI/SNRI + linezolid, SSRI/SNRI + methylene blue, SSRI/SNRI + St. John's wort

GUIDE 08 · PART II

Substance Use Disorders

Paper II · Clinical Psychiatry. Six study modes, from notes to quick review.

Study Notes

KEY INSIGHT

Sources: Kaplan & Sadock’s Comprehensive Textbook of Psychiatry (10th ed.), Stahl’s Essential Psychopharmacology (4th ed.), Oxford Textbook of Psychiatry, WHO Guidelines on Substance Use, ICD-11, DSM-5-TR

SECTION 1: CLASSIFICATION OF SUBSTANCE USE DISORDERS

1.1 Conceptual Framework

Substance use disorders exist on a spectrum from use → misuse → harmful use → dependence. The major diagnostic systems differ in how they carve this spectrum.

EXAM PEARL

ICD-11 and DSM-5 both moved away from the old ICD-10 "dependence syndrome" / "harmful use" binary, but they did so differently. Know both systems cold.

1.2 ICD-11 Classification

Categories under "Disorders due to substance use" (6C40–6C4Z):

CATEGORY	DEFINITION	KEY FEATURES
Single episode of harmful use	One episode causing harm	Biological, psychological, or social harm
Harmful use pattern	Persistent pattern over 12 months causing harm	Repeated harm; no dependence features required
Hazardous use	Pattern that increases risk of harm	No current harm needed; a risk category
Dependence	Dysregulated use with strong internal drive	Core syndrome: impaired control, salience, physiological features
Intoxication	Transient substance-specific syndrome	Time-limited, clinically significant
Withdrawal	Syndrome on cessation/reduction	Substance-specific, physiological
Substance-induced disorders	Mental disorders attributable to substance	Psychosis, delirium, mood disorders, etc.

ICD-11 Dependence Criteria (3 or more features present together):

1. Strong internal drive / compulsion to use
2. Impaired control (onset, frequency, amount, cessation)
3. Priority given to use over other activities and obligations
4. Use despite harm (physical, mental, social)
5. Tolerance
6. Withdrawal symptoms OR use to avoid withdrawal
7. Physiological features (tolerance + withdrawal = physiological dependence subtype)

EXAM PEARL

ICD-11 codes substances individually. Alcohol = 6C40, Opioids = 6C43, Cannabis = 6C41, Sedatives/Hypnotics = 6C44, Stimulants = 6C45, Tobacco = 6C4A, etc.

1.3 DSM-5 Classification

DSM-5 collapsed the old Abuse / Dependence distinction into a **single spectrum disorder** with mild/moderate/severe specifiers.

DSM-5 Substance Use Disorder Criteria (11 criteria across 4 domains):

DOMAIN · CRITERIA

Impaired Control 1. Using more/longer than intended; 2. Failed efforts to cut down; 3. Much time spent; 4. Craving

Social Impairment 5. Failure to fulfill obligations; 6. Social/interpersonal problems; 7. Activities given up

Risky Use 8. Physically hazardous use; 9. Use despite physical/psychological consequences

Pharmacological 10. Tolerance; 11. Withdrawal

Severity:

- Mild: 2–3 criteria
- Moderate: 4–5 criteria
- Severe: 6+ criteria

EXAM PEARL

DSM-5 removed "legal problems" criterion (from DSM-IV). Added "craving" as a new criterion. Tolerance and withdrawal do NOT count if occurring within prescribed medical use.

1.4 ICD-11 vs DSM-5: Key Differences

FEATURE	ICD-11	DSM-5
Structure	Separate harmful use + dependence categories	Single spectrum with severity
Hazardous use	Included as a category	Not included
Minimum criteria	3 of 7 for dependence	2 of 11
Severity specifiers	Not used for dependence	Mild / moderate / severe
Polysubstance	Coded for each substance	Same
Craving	Included as compulsion	Explicit criterion
Gambling disorder	Separate category	Included under SUDs

1.5 Physical vs Psychological Dependence

FEATURE	PHYSICAL DEPENDENCE	PSYCHOLOGICAL DEPENDENCE
Definition	Neuroadaptation requiring substance to maintain physiological normalcy	Craving, compulsion, emotional reliance
Marker	Tolerance + withdrawal	Craving, salience, loss of control
Without the other?	Can exist (morphine for pain)	Can exist (cannabis)
Substances showing both	Alcohol, opioids, benzodiazepines	All substances
Clinical relevance	Drives medically managed detox	Drives relapse; focus of psychotherapy

SECTION 2: ALCOHOL USE DISORDER

2.1 Epidemiology

- Global: ~2.3 billion drinkers; 283 million with AUD (WHO 2018)
- India: ~160 million drinkers; alcohol accounts for ~4% of global disease burden
- Male:Female ratio India: ~10:1 (changing)
- NMHS India 2016: Alcohol use disorders 4.6% lifetime prevalence

2.2 Pharmacology of Alcohol

Primary mechanism: Potentiation of GABA-A receptors + Inhibition of NMDA glutamate receptors

SYSTEM	ACUTE ALCOHOL	CHRONIC ALCOHOL	WITHDRAWAL
GABA-A	Positive allosteric modulator → CNS depression	Receptor downregulation	Reduced GABAergic tone → anxiety, seizures
NMDA glutamate	Antagonist → blocks excitatory transmission	Receptor upregulation	Unmasked excitatory surge → seizures, DTs
Dopamine (mesolimbic)	Increases DA release → reward	Blunted reward	Dysphoria, craving
Opioid	Releases endorphins	Tolerance	Dysphoria
Serotonin	Releases 5-HT	5-HT ₂ upregulation	Anxiety, mood symptoms

CLINICAL ANCHOR

The GABA↓ + NMDA↑ imbalance during withdrawal is the mechanistic basis for seizures and delirium tremens. Benzodiazepines work by restoring GABAergic tone.

2.3 Metabolism of Alcohol

Pathway:

Key facts:

Zero-order kinetics Alcohol is metabolized at a fixed rate (~10 mL/hour absolute alcohol) regardless of concentration, unlike most drugs

ADH (alcohol dehydrogenase) Liver enzyme; main pathway; converts ethanol → acetaldehyde

ALDH (aldehyde dehydrogenase) Converts acetaldehyde → acetate; **ALDH2 variant** common in East Asians → acetaldehyde accumulation → flushing

MEOS (microsomal ethanol oxidizing system) CYP2E1-based; activated with chronic use; responsible for tolerance and drug interactions

Blood alcohol concentration ~100 mg/dL = ataxia; ~200 mg/dL = stupor; ~400 mg/dL = potentially fatal

EXAM PEARL

Zero-order (saturation) kinetics means doubling alcohol intake does NOT double metabolism rate, it doubles BAC. This is why binge drinking is dangerous and why alcohol takes longer to clear at higher concentrations.

Disulfiram mechanism: Inhibits ALDH → acetaldehyde accumulates → flushing, nausea, palpitations, hypotension (disulfiram-ethanol reaction / DER).

2.4 Alcohol Withdrawal

Timeline:

HOURS AFTER LAST DRINK · CLINICAL FEATURES

6–12 hours Anxiety, tremor, diaphoresis, tachycardia, hypertension, insomnia, nausea

12–24 hours Alcoholic hallucinosis (intact sensorium, auditory > visual hallucinations)

24–48 hours **SEIZURES** (peak 24–36 hours), typically GTCS, brief, self-limited

48–72 hours **DELIRIUM TREMENS** (peak, most severe)

5–7 days Resolution in most; protracted symptoms in some

EXAM PEARL

AWS seizures: usually GTCS, rarely focal, brief, self-limited. Occur in ~3–5% of untreated withdrawal. Status epilepticus is rare. If focal → think structural lesion.

2.5 Delirium Tremens (DTs)

Clinical features (5 Ds):

- Delirium (confusion, disorientation, impaired attention)
- Diaphoresis (profuse sweating)
- Dysautonomia (tachycardia, hypertension, hyperthermia, mydriasis)
- Dread (extreme anxiety, agitation)
- Dreams / hallucinations (visual > auditory; Lilliputian hallucinations classic)

EXAM PEARL

DTs mortality: untreated 15–35%, treated <1%. Risk factors: prior DTs, seizures, concurrent illness, prolonged heavy drinking, poor nutritional status.

Risk factors for severe withdrawal:

- Previous severe withdrawal / seizures / DTs
- High alcohol intake (>10 drinks/day)
- Long duration of dependence
- Old age
- Concurrent medical illness
- Poor nutrition (thiamine deficiency)

- Benzodiazepine/sedative co-use

2.6 CIWA-Ar (Clinical Institute Withdrawal Assessment for Alcohol, revised)

10 items assessed:

ITEM · SCORE RANGE

Nausea/vomiting 0–7

Tremor 0–7

Paroxysmal sweats 0–7

Anxiety 0–7

Agitation 0–7

Tactile disturbances 0–7

Auditory disturbances 0–7

Visual disturbances 0–7

Headache/fullness in head 0–7

Orientation/clouding of sensorium 0–4

Total score interpretation:

SCORE	SEVERITY	MANAGEMENT
<8	Mild	Monitoring, supportive care; consider ambulatory detox
8–15	Moderate	Benzodiazepines as needed (symptom-triggered)
>15	Severe	Standing benzodiazepine schedule; close monitoring
>20	Very severe	ICU/HDU consideration; IV benzodiazepines

CLINICAL ANCHOR

CIWA-Ar is the gold standard for severity-guided benzodiazepine dosing. Symptom-triggered protocols using CIWA-Ar use significantly less benzodiazepine than fixed-schedule protocols.

2.7 Management of Alcohol Withdrawal

Step 1: Assess and stabilize

- Vitals, blood glucose, electrolytes (Na, K, Mg), LFTs, CBC, coagulation
- Identify concurrent illness, head injury, infection
- Assess nutritional status, hydration

Step 2: Thiamine (MANDATORY, before any glucose/IV fluids)

- 100 mg IV/IM thiamine before glucose infusion
- Rationale: Glucose load can precipitate Wernicke's encephalopathy in thiamine-depleted state
- High-risk patients: 200–500 mg IV thiamine (Pabrinex)

Step 3: Benzodiazepines (pharmacological backbone)

BENZODIAZEPINE	ROUTE	ADVANTAGES	USE PREFERENCE
Diazepam	Oral/IV	Long-acting → smooth; IV for seizure	Standard choice in India; IV for DTs
Chlordiazepoxide	Oral	Long-acting; good oral bioavailability	Mild-moderate uncomplicated withdrawal
Lorazepam	IV/IM	Short-acting; no active metabolites	Liver disease; elderly; easier to titrate
Oxazepam	Oral	Short-acting; no active metabolites; safe in liver disease	Liver cirrhosis

Fixed-schedule (front-loading) protocol, Diazepam:

- Day 1: 20 mg QID (or higher based on CIWA-Ar)
- Reduce by 20–25% per day
- Taper over 5–10 days

Symptom-triggered protocol: CIWA-Ar ≥ 8 → give diazepam 10–20 mg; reassess every 1–2 hours.

Step 4: Adjuncts

- **Thiamine:** Continue oral for 2 weeks post-detox
- **Multivitamins:** Folate, B12
- **Clonidine:** Autonomic symptoms (adjunct, not primary)
- **Beta-blockers (propranolol):** HR/BP; no anticonvulsant effect, do NOT use alone
- **Carbamazepine:** Alternative anticonvulsant; preferred in outpatient mild-moderate withdrawal; not standard for DTs
- **Haloperidol:** For psychotic symptoms in DTs; NEVER as sole agent (no anticonvulsant effect)

- **Magnesium:** If hypomagnesemia

Managing DTs:

- ICU/HDU
- IV diazepam: boluses of 5–10 mg every 5–15 minutes until calm; then infusion or PO
- IV lorazepam alternative
- Monitor: airway, BP, temperature, glucose
- Treat precipitating factors: infection, head injury

2.8 Relapse Prevention (Pharmacotherapy)

DRUG	MECHANISM	DOSE	EVIDENCE	CONTRAINDICATIONS
Disulfiram	ALDH inhibitor → DER if alcohol consumed	250–500 mg/day	Moderate; supervised use better	Psychosis, CV disease, hepatic disease, pregnancy; requires absolute abstinence motivation
Naltrexone	Mu-opioid antagonist → blocks euphoria, reduces craving	50 mg/day oral; 380 mg/month IM (Vivitrol)	Strong (NNT ~7)	Active opioid use (precipitates withdrawal), hepatic impairment
Acamprosate	GABA-A agonist + NMDA antagonist → reduces protracted withdrawal symptoms	666 mg TID	Strong; best started after detox	Severe renal failure (CrCl <30)
Baclofen	GABA-B agonist	30–80 mg/day; up to 300 mg in Ameisen protocol	Moderate; especially for anxiety	Caution: seizures on abrupt discontinuation
Nalmefene	MOR antagonist + KOR partial agonist	18 mg PRN (as-needed)	Good for reduced drinking goals	Similar to naltrexone

EXAM PEARL

Naltrexone works best for heavy drinking reduction / craving. Acamprosate works best for maintaining abstinence in those already detoxed. Disulfiram requires absolute motivation and monitoring. Baclofen has Indian trial data (Kiefer, Addolorato studies).

Psychosocial treatments:

- Motivational Enhancement Therapy (MET), 2–4 sessions

- Cognitive Behavioural Therapy (CBT), relapse prevention focused
- 12-Step Facilitation (Alcoholics Anonymous)
- Behavioural Couples Therapy
- Community Reinforcement Approach (CRA)

2.9 Wernicke-Korsakoff Syndrome

Thiamine deficiency in alcohol use disorder, the pathophysiology:

- Inadequate dietary intake
- Malabsorption
- Increased metabolic demand
- Impaired hepatic storage

Wernicke's Encephalopathy (acute):

Classic triad Ophthalmoplegia + Ataxia + Confusion (encephalopathy)

Full triad Only 10–16% of cases, do NOT wait for complete triad

Ocular findings Lateral rectus palsy (most common), nystagmus, conjugate gaze palsy

Pathology Petechial hemorrhages in mammillary bodies, thalamus, periaqueductal grey

Treatment IV/IM thiamine (200–500 mg), BEFORE glucose; urgently

CLINICAL ANCHOR

Giving IV glucose before thiamine in a suspected Wernicke's can precipitate acute decompensation. Thiamine first, always.

Korsakoff's Syndrome (chronic amnestic):

- Anterograde amnesia (new learning severely impaired) > retrograde amnesia
- Confabulation (unconscious fabrication to fill memory gaps)
- Relative preservation of other cognitive functions
- Insight usually absent
- Neurologically: dorsomedial thalamus + mammillary body atrophy
- Outcome: ~25% significant recovery, ~50% partial, ~25% minimal improvement

2.10 Alcoholic Hallucinosis

Timing 12–48 hours after last drink

Consciousness Clear, sensorium intact (differentiates from DTs)

Type Predominantly auditory (voices commenting, threatening)

Content Often accusatory, threatening voices; patient frightened

Duration Usually hours to days; rarely persists

Sensorium Intact orientation, no delirium

Treatment Antipsychotics (haloperidol); benzodiazepines for co-occurring withdrawal

EXAM PEARL

Alcoholic hallucinosis differs from DTs: clear consciousness, predominantly auditory, no autonomic instability. It differs from schizophrenia by: acute onset, temporal link to alcohol withdrawal, resolves with abstinence.

2.11 Pathological Intoxication (Alcohol Idiosyncratic Intoxication)

- Extreme behavioral change (aggression, violence) after small amounts of alcohol
- Disproportionate to amount consumed
- Amnesia for the episode common
- Rare; uncertain whether distinct entity
- Mechanism: possibly related to temporal lobe sensitivity, epileptic disinhibition
- DSM-5: absorbed into "other specified alcohol-related disorder"

2.12 Fetal Alcohol Spectrum Disorders (FASD)

No safe level of alcohol in pregnancy.

CONDITION · FEATURES

Fetal Alcohol Syndrome (FAS) Full syndrome: growth restriction + facial dysmorphology + CNS abnormalities

Partial FAS Some facial features + CNS involvement

ARND (Alcohol-related neurodevelopmental disorder) CNS only, no dysmorphology

ARBD (Alcohol-related birth defects) Structural anomalies without full FAS

FAS Facial Features (facial dysmorphology triad):

- Smooth philtrum
- Thin vermilion border of upper lip
- Short palpebral fissures

CNS effects:

- Microcephaly, intellectual disability
- ADHD, learning disabilities
- Behavioral problems
- Impaired executive function

EXAM PEARL

FAS is the most common preventable cause of intellectual disability.

SECTION 3: OPIOID USE DISORDERS

3.1 Epidemiology

- Global: ~62 million people use opioids non-medically (UNODC 2022)
- India: ~2.26 million heroin users (UNODC); significant rise in pharmaceutical opioid misuse
- Biggest contributor to overdose deaths globally
- India NDPS Act: heroin Schedule I, pharmaceutical opioids Schedule II (regulatory complexity)

3.2 Pharmacology

Opioid receptors (all are GPCRs, Gi/o coupled → ↓cAMP, K⁺ channel open, Ca²⁺ channel closed → neuronal inhibition):

RECEPTOR	PRIMARY AGONIST	KEY CLINICAL EFFECTS
Mu (MOR)	Morphine, heroin, fentanyl	Analgesia, euphoria, respiratory depression, constipation, dependence
Kappa (KOR)	Dynorphin	Sedation, dysphoria, analgesia; no dependence
Delta (DOR)	Enkephalin	Analgesia, mood modulation
Nociceptin/OFQ (NOP)	Nociceptin	Antiopioid; complex

EXAM PEARL

Respiratory depression is MOR-mediated. This is why MOR antagonism (naloxone) reverses overdose. Buprenorphine is a partial MOR agonist, ceiling on respiratory depression.

Endogenous ligands:

- Beta-endorphin → MOR
- Met/Leu-enkephalin → DOR

- Dynorphin → KOR
- Nociceptin → NOP

3.3 Heroin

- Diacetylmorphine
- More lipophilic than morphine → crosses BBB faster → intense rush
- Rapidly hydrolyzed to 6-monoacetylmorphine → morphine
- Route: IV (most bioavailable), smoked ("chasing the dragon"), intranasal

3.4 Opioid Intoxication

Triad Miosis + CNS depression + Respiratory depression

Other features Euphoria, analgesia, constipation, nausea, bradycardia, hypotension

Overdose risk Respiratory depression → apnea → hypoxia → death

Treatment Naloxone 0.4–2 mg IV/IM/intranasal; repeat every 2–3 min; infusion if needed

3.5 Opioid Withdrawal

Timeline (heroin):

- Short-acting opioids (heroin): begins 6–24 hours post-dose; peaks 36–72 hours; resolves 5–7 days
- Long-acting opioids (methadone): begins 24–48 hours; peaks 3–5 days; resolves 2–3 weeks

COWS (Clinical Opiate Withdrawal Scale), 11 items:

ITEM · RANGE

Resting pulse rate 0–4

Diaphoresis 0–4

GI upset 0–5

Tremor 0–4

Restlessness/agitation 0–5

Yawning 0–4

Anxiety 0–4

Dilated pupils 0–5

Bone/joint aches 0–4

Piloerection 0–3

Rhinorrhea/lacrimation 0–4

Score interpretation:

- 5–12: Mild
- 13–24: Moderate
- 25–36: Moderately severe
- >36: Severe

EXAM PEARL

Opioid withdrawal is intensely dysphoric but NOT life-threatening in otherwise healthy adults (unlike alcohol withdrawal). It is dangerous in: neonates, pregnancy, severe comorbidities.

Mnemonic for opioid withdrawal signs: "DAMP COIL"

- Diarrhea
- Anxiety
- Mydriasis (dilated pupils)
- Piloerection
- Cramps/muscle aches
- Only not life-threatening (except in neonates/pregnancy)
- Insomnia
- Lacrimation/rhinorrhea

3.6 Opioid Substitution Therapy (OST)

Rationale: Replace illicit, short-acting opioid with long-acting, orally administered, medically supervised opioid → stabilize, reduce craving, reduce needle use, reduce crime, reduce mortality.

FEATURE	METHADONE	BUPRENORPHINE
Drug class	Full MOR agonist	Partial MOR agonist + KOR antagonist
Half-life	24–36 hours	24–72 hours (sublingual)
Route	Oral liquid	Sublingual (alone or with naloxone)
Starting dose	20–40 mg/day	4–8 mg/day

FEATURE	METHADONE	BUPRENORPHINE
Maintenance	60–120 mg/day	16–32 mg/day
Ceiling effect	No	Yes (on respiratory depression)
Diversion risk	High	Lower (buprenorphine/naloxone formulation)
QTc prolongation	Yes (dose-dependent)	Minimal
Induction	Day 1 of seeking treatment	Patient must be in mild-moderate withdrawal (COWS ≥ 8 –12) before first dose
Indication	Severe OUD, failed other treatment	First-line for OUD
In pregnancy	Preferred (established safety data)	Increasing evidence

EXAM PEARL

Buprenorphine induction when NOT in withdrawal can precipitate severe withdrawal, because buprenorphine (partial agonist with high receptor affinity) displaces full agonist off receptors. This is the "precipitated withdrawal" risk.

Buprenorphine/Naloxone (Suboxone):

- Naloxone is bioavailable only IV, not sublingually
- If injected IV (diverted): naloxone becomes active → precipitates withdrawal → deters IV misuse
- Sublingual: only buprenorphine effect

3.7 Naloxone for Overdose Reversal

Mechanism Competitive MOR antagonist

Onset 2–5 minutes (IV)

Duration 30–90 minutes (shorter than most opioids)

Route IV, IM, SC, intranasal

Dose 0.4–2 mg; repeat every 2–3 minutes; max 10 mg

Re-sedation risk Yes, with long-acting opioids (fentanyl, methadone); may need infusion

Can give to dependent patient? Yes in emergency; will precipitate withdrawal

Take-home naloxone Policy in many countries; reduces overdose deaths

3.8 Neonatal Opioid Withdrawal Syndrome (NOWS) / Neonatal Abstinence Syndrome (NAS)

When: Infant born to opioid-dependent mother (prescribed or illicit); onset 24–72 hours for heroin, up to 5–7 days for methadone.

Finnegan Neonatal Abstinence Scoring System:

Signs scored: high-pitched cry, sleep <3 hours, hyperactive Moro reflex, tremors, muscle tone increased, excoriation, frequent yawning, mottling, fever, sweating, nasal stuffiness, sneezing, nasal flaring, respiratory rate >60, feeding problems, regurgitation, vomiting, loose stools, watery stools.

Score ≥8 on two occasions → pharmacological treatment:

- First-line: Oral morphine or methadone
- Adjunct: Phenobarbital for seizures
- Breastfeeding (if not HIV+, no drug use except OST): encouraged; reduces NOWS severity

SECTION 4: CANNABIS USE DISORDERS

4.1 The Endocannabinoid System

COMPONENT · DETAIL

Receptors CB1 (brain, hippocampus, basal ganglia, cerebellum, prefrontal cortex) and CB2 (immune system, peripheral tissues)

Endogenous ligands Anandamide (AEA) + 2-arachidonoylglycerol (2-AG)

Synthesis On demand (retrograde signaling), postsynaptic neuron releases → binds presynaptic CB1 → inhibits neurotransmitter release

Function Modulates pain, appetite, memory, mood, stress response, immune function

Delta-9-THC (primary psychoactive constituent):

- Partial CB1/CB2 agonist
- Highly lipophilic → crosses BBB rapidly
- Metabolized to active metabolite 11-OH-THC
- Stored in fat → prolonged urinary detection (2–4 weeks in heavy users)

CBD (cannabidiol):

- Non-psychoactive
- CB1 negative allosteric modulator; also 5-HT_{1A} agonist, TRPV1 agonist

- Anticonvulsant, anxiolytic properties
- May attenuate THC-induced psychosis risk

4.2 Acute Intoxication

- Euphoria, relaxation, altered perception of time, increased appetite ("munchies")
- Impaired short-term memory, concentration
- Tachycardia, conjunctival injection, dry mouth
- Anxiety, panic (especially naïve users or high-THC products)
- Perceptual distortions (not true hallucinations usually)

4.3 Cannabis Use Disorder

- ~9% of users develop dependence (compare: tobacco 32%, heroin 23%, cocaine 17%)
- Higher risk with: early onset use, daily / near-daily use, high-potency products, co-occurring anxiety / depression
- DSM-5 criteria: 2+ of 11 within 12 months
- No pharmacologically validated treatment; psychosocial (CBT, MI, CM) primary

Cannabis Withdrawal Syndrome (ICD-11, DSM-5 recognized):

Onset 24–72 hours; peaks 2–6 days; resolves 1–2 weeks

Symptoms: Irritability / anger / aggression, anxiety, sleep disturbance, appetite decrease, restlessness, depressed mood, GI disturbance, somatic discomfort (headache, sweats, chills)

4.4 Amotivational Syndrome

- Chronic cannabis use → apathy, reduced goal-directed activity, decreased motivation, flat affect
- Mechanism: chronic CB1 agonism → downregulation in prefrontal cortex → reduced dopaminergic drive
- Controversial as distinct entity vs intoxication state vs comorbid depression
- Partially reversible with sustained abstinence

4.5 Cannabis-Induced Psychosis

FEATURE	CANNABIS-INDUCED PSYCHOSIS	PRIMARY PSYCHOSIS
Temporal link	Within 1 month of use	No temporal link
Duration	Typically resolves within 1 month of abstinence	Persistent
Content	Paranoia, perceptual changes	Any content
Consciousness	Relatively clear	Clear

FEATURE	CANNABIS-INDUCED PSYCHOSIS	PRIMARY PSYCHOSIS
Longitudinal	~25–50% convert to schizophrenia-spectrum over 5 years	N/A
Treatment	Abstinence + antipsychotic	Antipsychotic

EXAM PEARL

Cannabis is a component cause of schizophrenia. Meta-analyses show ~2-fold increased risk of psychosis with any cannabis use; ~4-fold with heavy use. High-potency THC products carry highest risk. Earlier onset → higher risk.

The **THC:CBD ratio matters**, high THC/low CBD products (modern skunk) have higher psychotomimetic risk than balanced products.

4.6 Synthetic Cannabinoids ("Spice," "K2," "Mambas")

- Full CB1 agonists (vs THC partial) → more potent, more toxic
- Clinical features more severe: psychosis, seizures, AKI, cardiovascular events, death
- No specific antidote; supportive management
- Not detected on standard urine drug screens

SECTION 5: TOBACCO USE DISORDER

5.1 Nicotine Pharmacology

Primary target Nicotinic acetylcholine receptors (nAChRs), particularly $\alpha 4\beta 2$ subtype

Mesolimbic effect Activates VTA → NAc dopamine release → reward / reinforcement

Onset Cigarette: nicotine reaches brain in 10 seconds, fastest delivery method

Half-life ~2 hours

Metabolism Liver CYP2A6 → cotinine (marker of exposure, half-life ~16 hours)

Withdrawal Irritability, anxiety, difficulty concentrating, increased appetite, depressed mood, insomnia, craving

Dependence Among highest of all substances, 32% of users develop dependence

5.2 Fagerström Test for Nicotine Dependence (FTND)

6 questions (0–10 score):

QUESTION · KEY ITEM

Time to first cigarette after waking Most predictive item, ≤ 30 min = high dependence

Difficulty refraining in forbidden places

Which cigarette you'd hate to give up The first one in the morning = high dependence

Cigarettes per day >30 /day = high

Smoke more in first hour

Smoke when so ill you must stay in bed

Score: 0–3 low; 4–6 moderate; 7–10 high dependence

EXAM PEARL

The single most predictive FTND item: time to first cigarette after waking. If <30 minutes, patient is highly dependent and likely needs pharmacotherapy.

5.3 Treatment of Nicotine Dependence

5 A's framework: Ask → Advise → Assess → Assist → Arrange

5 R's for unmotivated patients: Relevance → Risks → Rewards → Roadblocks → Repetition

TREATMENT	MECHANISM	EFFICACY
NRT (nicotine patch, gum, lozenge, inhaler, spray)	Nicotine replacement, reduces withdrawal	Doubles quit rates vs placebo
Bupropion SR	Blocks DA and NE reuptake; nAChR antagonist	Doubles quit rates; also antidepressant
Varenicline (Champix)	Partial $\alpha 4\beta 2$ nAChR agonist	Triples quit rates vs placebo; best single agent
Combination NRT	Patch (long-acting) + short-acting NRT	More effective than monotherapy

EXAM PEARL

Varenicline: concern about neuropsychiatric adverse effects (depression, suicidality), black box warning was added then partially retracted. Still caution in those with mental illness.

Bupropion: seizure risk (dose-related), contraindicated in eating disorders, seizure history.

SECTION 6: STIMULANT USE DISORDERS

6.1 Cocaine

Pharmacology:

- Blocks reuptake transporters for DA, NE, 5-HT (primarily DA reuptake inhibitor in striatum)
- Also: local anesthetic (Na channel blockade)
- Routes: intranasal ("snorting"), IV, smoked as crack cocaine

Crack vs powder cocaine:

FEATURE	POWDER COCAINE	CRACK COCAINE
Form	Hydrochloride salt	Freebase (prepared with baking soda)
Route	Intranasal, IV	Smoked
Onset	Minutes (intranasal)	Seconds (smoked)
Duration	15–30 min	5–10 min
Intensity	Moderate	Very high
Risk	Nasal septum damage	Pulmonary problems; highest addiction potential

Cocaine intoxication:

- Euphoria, grandiosity, increased energy, decreased appetite
- Tachycardia, hypertension, dilated pupils, diaphoresis
- With high doses: paranoia, perceptual disturbances

Cocaine "crash" (withdrawal):

- Dysphoria, fatigue, hypersomnia, hyperphagia, depression, craving
- No physiological withdrawal per se; psychological craving intense
- Phase: post-acute withdrawal → "extinction" phase (month-long craving cycles triggered by cues)

Cocaine-induced psychosis:

- Paranoid delusions, tactile hallucinations (formication, "coke bugs")
- Auditory hallucinations
- Clear sensorium usually maintained
- Typically resolves within days to weeks of abstinence

Medical complications of cocaine:

- Cardiovascular: MI (even in young users), arrhythmia, aortic dissection, cardiomyopathy
- Pulmonary: "crack lung", acute pneumonitis
- Neurological: stroke, seizure
- Nasal: septum perforation, rhinitis, anosmia

6.2 Amphetamines and Methamphetamine

Mechanism:

- Reverses DAT, NET, SERT (releases stored monoamines), not just reuptake blockade
- Net effect: massive increase in synaptic DA, NE, 5-HT

Methamphetamine ("crystal meth"):

- More potent and longer-acting than amphetamine
- Routes: smoked, IV, intranasal
- Neurotoxicity: damages DA and 5-HT terminals with chronic use → Parkinsonism, cognitive decline
- "Meth mouth": severe dental decay from xerostomia, bruxism, acidic environment

MDMA (3,4-methylenedioxymethamphetamine / Ecstasy):

- Releases 5-HT (primary) > DA; also oxytocin release
- Acute: empathogen, euphoria, increased sociability
- Neurotoxic to serotonergic neurons with repeated use
- Hyperthermia (dangerous; serotonin syndrome risk)
- Hyponatremia (drinking excess water while dancing)

SECTION 7: SEDATIVE-HYPNOTIC USE DISORDERS

7.1 Benzodiazepine Pharmacology

- Positive allosteric modulators of GABA-A receptor (chloride channel)
- Increase frequency of Cl⁻ channel opening (not duration, that's barbiturates)
- Binding site distinct from alcohol/barbiturate sites but all converge on GABA-A

Clinical uses: Anxiety, insomnia, seizures, acute alcohol withdrawal, muscle relaxation, pre-anaesthetic

7.2 Benzodiazepine Dependence

Risk factors: Longer duration of use (>4–6 weeks at therapeutic doses), higher dose, short-acting agents, prior dependence history, anxiety disorders, elderly.

Withdrawal: Similar to alcohol withdrawal (GABA-related); potentially life-threatening

Withdrawal timeline:

- Short-acting (lorazepam, alprazolam, triazolam): 24–48 hours onset; peaks 2–4 days
- Long-acting (diazepam, clonazepam, chlordiazepoxide): 5–7 days onset; peaks 2 weeks

Withdrawal symptoms:

- Anxiety, insomnia, tremor, diaphoresis, tachycardia, hypertension
- Perceptual hypersensitivity (sensory, light, sound)
- Seizures (particularly with abrupt cessation of short-acting benzos)
- Delirium (rare but serious)

Management of BZD dependence:

1. Switch to long-acting BZD (diazepam equivalent)
2. Slow taper, typically over weeks to months (NOT the rapid 5–10 day protocol of alcohol)
3. Typical rate: 10% per week; slower as dose decreases
4. Adjuncts: CBT (particularly for anxiety), sleep hygiene

7.3 Z-Drugs (Non-Benzodiazepine Hypnotics)

- Zolpidem, zaleplon, zopiclone (eszopiclone)
- Act on BZD receptor but with more selectivity for $\alpha 1$ subunit (sedation) vs $\alpha 2$ (anxiolysis)
- Lower dependence potential than BZDs (theoretical; clinical evidence mixed)
- Same withdrawal profile at high doses/long duration
- Unusual sleep behaviors: sleep-walking, sleep-eating, sleep-driving

7.4 GHB (Gamma-Hydroxybutyrate)

- Endogenous GABA precursor + GHB receptor agonist at low doses; GABA-B agonist at high doses
- Medical use: narcolepsy (sodium oxybate), alcohol withdrawal (in some countries)
- Abuse: "club drug," recreational use, "date rape drug" (rapid sedation/amnesia)
- Narrow therapeutic window; overdose: loss of consciousness, respiratory depression, coma
- Withdrawal: alcohol-like withdrawal syndrome; potentially severe

SECTION 8: NOVEL AND EMERGING SUBSTANCES**8.1 Kratom (*Mitragyna speciosa*)**

- Tropical plant (Southeast Asia); leaves chewed or brewed
- Active alkaloids: mitragynine and 7-hydroxymitragynine

- Mechanism: partial MOR agonist at high doses; stimulant at low doses
- Uses: self-treatment of opioid withdrawal, pain, fatigue
- Dependence: opioid-like withdrawal syndrome
- Pharmacokinetics: half-life ~9 hours
- Legal status varies; increasing global use

8.2 Synthetic Cathinones ("Bath Salts")

- Structural analogs of cathinone (khat plant alkaloid)
- Examples: mephedrone, MDPV, alpha-PVP
- Mechanism: monoamine releasing / reuptake blocking (DA > NE > 5-HT)
- Clinical features: stimulant effects + psychosis; "excited delirium"
- High lethality potential: hyperthermia, rhabdomyolysis, cardiovascular collapse
- Not detected on standard drug screens

8.3 Novel Psychoactive Substances (NPS)

- Designed to mimic effects of controlled substances while remaining technically legal ("designer drugs")
- Categories: synthetic cannabinoids, synthetic cathinones, novel phenethylamines (NBOMe series), novel opioids (fentanyl analogs), dissociatives (novel ketamine analogs)
- Surveillance challenge: new compounds emerge faster than they can be scheduled
- India: Amendment to NDPS Act attempts to include analog compounds

8.4 Inhalants

- Toluene (glue), gasoline, nitrous oxide, amyl/butyl nitrite ("poppers")
- Particularly prevalent in adolescents, homeless populations, India (Pidilite / Fevicol sniffing)
- Mechanism: CNS depression (general anesthetic-like); toluene: GABA-A + NMDA effects
- Toxic: "sudden sniffing death" (ventricular fibrillation); cerebellar atrophy; dementia; white matter damage
- No pharmacological treatment; psychosocial focus

SECTION 9: MOTIVATIONAL INTERVIEWING

9.1 Definition and Spirit

MI is a collaborative, person-centered form of guiding to elicit and strengthen motivation for change.

MI Spirit (PACE):

- **Partnership**, collaborative, not expert-to-client
- **Acceptance**, absolute worth, accurate empathy, autonomy support, affirmation
- **Compassion**, active promotion of client's welfare
- **Evocation**, drawing out client's own motivation and wisdom

9.2 Four Processes of MI

1. **Engaging**, establishing a working relationship
2. **Focusing**, developing and maintaining a specific direction
3. **Evoking**, eliciting motivation for change
4. **Planning**, developing commitment and plan

9.3 Core Skills: OARS

SKILL	PURPOSE	EXAMPLE
Open questions	Elicit detailed responses	"What concerns you most about your drinking?"
Affirmations	Recognize strengths	"It took courage to come here today."
Reflective listening	Demonstrate understanding	"It sounds like you're torn between..."
Summaries	Collect, link, transition	Summary of change talk heard

9.4 Change Talk and Sustain Talk

Change talk (DARN CAT):

- **Desire** ("I want to...")
- **Ability** ("I could...")
- **Reason** ("Because...")
- **Need** ("I need to...")
- **Commitment** ("I will...")
- **Activation** ("I'm ready to...")
- **Taking steps** ("I've already...")

Sustain talk: Arguments for the status quo ("But I enjoy it...", "I'm not that bad...")

Discord: Defensiveness, argument, changing subject, respond with reflection, not argument

9.5 Ambivalence and Decisional Balance

A central MI concept: ambivalence (simultaneously wanting and not wanting to change) is normal, not pathological. The task is to explore ambivalence rather than confront or argue with it.

Decisional balance: Explore costs and benefits of both change and status quo.

SECTION 10: STAGES OF CHANGE (TRANSTHEORETICAL MODEL)

10.1 Prochaska & DiClemente Model

STAGE	DESCRIPTION	INTERVENTION
Precontemplation	No awareness of problem; not considering change	Provide information; raise awareness without confrontation
Contemplation	Aware of problem; ambivalent; weighing pros/cons	Explore ambivalence; decisional balance; MI
Preparation	Intends to change in next 30 days; making plans	Help develop specific plan; goal-setting
Action	Actively making changes; <6 months	Support; reinforce; manage barriers
Maintenance	Sustained change >6 months; relapse prevention	Relapse prevention skills; CBT; continued support
Relapse	Return to use	Non-judgmental; normalize; return to earlier stages

EXAM PEARL

Relapse is not a stage failure, it's part of the normal change cycle. Average person makes 3–4 serious quit attempts before sustained recovery. Termination (permanent exit from cycle) is sometimes added as stage 6.

SECTION 11: BRIEF INTERVENTIONS

11.1 FRAMES (Components of Effective Brief Intervention)

- Feedback, personalized feedback on risk/harm
- Responsibility, emphasize personal responsibility for change
- Advice, clear advice to change
- Menu, range of options for change
- Empathy, warm, empathic counseling style
- Self-efficacy, build belief in ability to change

11.2 Screening Tools

AUDIT (Alcohol Use Disorders Identification Test):

- 10 items; covers frequency, quantity, dependence features, consequences
- Score 0–40
- Threshold: ≥ 8 hazardous/harmful use; ≥ 15 likely dependence
- Developed by WHO; validated cross-culturally
- AUDIT-C (3 items): brief screen; score ≥ 4 men, ≥ 3 women = positive

CAGE (4 questions):

- Cut down, ever felt you should cut down?
- Annoyed, have people annoyed you by criticizing your drinking?
- Guilty, ever felt guilty about drinking?
- Eye opener, ever had a drink first thing in the morning (eye opener)?
- Score ≥ 2 : clinically significant; sensitivity ~74%, specificity ~91%

MAST (Michigan Alcohol Screening Test):

- 25-item questionnaire; score > 5 indicates alcoholism
- Brief MAST (13 items) also available

DAST (Drug Abuse Screening Test):

- 28-item (or 10-item) tool for non-alcohol substances
- Score ≥ 6 = substance abuse

SECTION 12: DUAL DIAGNOSIS

12.1 Epidemiology

- ~50% of those with severe mental illness have a lifetime SUD
- ~50% of those with SUD have a lifetime psychiatric disorder
- Most common combinations:
 - Alcohol + Depression/Anxiety
 - Stimulants + Psychosis/Bipolar
 - Cannabis + Psychosis/Anxiety
 - Opioids + Depression/PTSD

12.2 Pathways Linking SUD and Mental Illness

PATHWAY · EXPLANATION

Self-medication Mental illness → substance use to manage symptoms

Substance-induced mental illness Direct pharmacological effects causing symptoms

Common factor Shared genetic vulnerability, adverse childhood experiences

Bidirectional Each exacerbates the other

12.3 Management Principles

Integrated treatment is superior to sequential or parallel approaches:

- Address both disorders simultaneously in same setting
- Same team, shared conceptual model
- Stage-wise approach (engagement → persuasion → active treatment → relapse prevention)
- Modified MI for dual diagnosis: accept low motivation early; build engagement first

Sequential approach (outdated): Treat one before the other, poor outcomes

Medication principles:

- Use formulations with lower abuse potential where possible
 - Monitor for interactions (e.g., antidepressants + alcohol)
 - SSRIs: first-line for depression/anxiety in SUD; may also reduce drinking
 - Naltrexone: avoid if on opioids; helpful for alcohol in depression comorbidity
 - Antipsychotics: avoid olanzapine/clozapine in stimulant users (metabolic risk compounded)
-

SECTION 13: INDIAN CONTEXT

13.1 NDPS Act (Narcotic Drugs and Psychotropic Substances Act, 1985)

Key provisions:

PROVISION · DETAIL

Definition Narcotic drugs + psychotropic substances + controlled substances

Small quantity Below small quantity → reduced penalty (6 months to 1 year)

Large quantity Trafficking, 10–20 years rigorous imprisonment; may extend to death in some cases

Rigid bail provisions NDPS arrests: bail conditions stringent

Section 71 Government shall establish treatment centers; can send users to treatment instead of jail

Section 76 Probation, court can send user for treatment

Amendment 2001 Introduced "small quantity" threshold to distinguish users from traffickers

Amendment 2014 Further amendments; essential medicines access provisions

Small quantities under NDPS:

- Opium: 25 grams
- Heroin: 5 grams
- Cocaine: 2 grams
- Cannabis (charas): 100 grams; ganja: 1 kg
- Amphetamine: 2 grams

EXAM PEARL

The NDPS Act has been criticized for being too punitive toward users. Section 71 offers a humane pathway but is underused. The clinician's role: document addiction as illness, advocate for treatment rather than prosecution.

13.2 De-addiction Infrastructure in India

TYPE · EXAMPLES

Central government Ministry of Social Justice and Empowerment: IRCA (Integrated Rehabilitation Center for Addicts) network

PG exams (Bangalore) Tertiary referral center; training programs

Drug Treatment Centre (DTC) Government hospitals, buprenorphine maintenance, brief counseling

NGO sector Narcotics Anonymous, AA India chapters, Aasra, Sankalp

Residential rehabilitation Private sector; mixed quality; 3–12 month programs

13.3 Drug Trends in India

- Alcohol: most prevalent; rising female use
- Pharmaceutical opioids: codeine-containing cough syrups, tramadol, buprenorphine misuse
- Heroin: Punjab, Rajasthan, Northeast India (Golden Crescent and Golden Triangle routes)
- Cannabis: widespread; increasing synthetic cannabis reports
- Stimulants: amphetamine use rising in trucking industry
- Tobacco: dual use (smoked + smokeless)

SECTION 14: SPECIAL TOPICS

14.1 Pharmacogenomics Relevant to Addiction

GENE	SUBSTANCE	CLINICAL RELEVANCE
ALDH2*2	Alcohol	East Asian flushing response; lower alcoholism risk
ADH1B2, ADH1B3	Alcohol	Faster acetaldehyde production; lower alcoholism risk
OPRM1 A118G	Opioids / Alcohol	Affects naltrexone response in AUD
CYP2A6	Nicotine	Poor metabolizers: longer nicotine half-life; may need lower NRT doses
DRD2 Taq1A	Multiple	Reduced D2 density; reward deficiency; higher addiction risk

14.2 Neurobiological Circuits in Addiction

Three-stage cycle (Koob & Volkow):

STAGE	BRAIN REGION	PROCESS
Binge/Intoxication	Basal ganglia (striatum)	Reward, habit learning, DA surge
Withdrawal/Negative affect	Extended amygdala, habenula	Anti-reward: stress systems (CRF, dynorphin) activated
Preoccupation/Anticipation	Prefrontal cortex	Craving, impaired impulse control, cognitive bias toward drug cues

EXAM PEARL

The "allostatic model" of addiction (Koob): With repeated substance use, the hedonic set point shifts downward, baseline is dysphoria, drugs only restore "normal." This explains why late-stage dependence is about avoiding withdrawal rather than seeking pleasure.

14.3 Reward Deficiency Syndrome

Concept: individuals with blunted dopaminergic reward systems (genetic, developmental) are at higher risk for addiction (and other compulsive behaviors). Basis for DA-based pharmacological strategies.

14.4 Process Addictions

- Gambling disorder (only non-substance addiction in DSM-5 SUD chapter)
- Gaming disorder (ICD-11: addictive behaviors section)
- Internet gaming, social media, pornography (under research)
- Same neurobiological circuits as substance use

SECTION 15: HIGH-YIELD DRUG FACTS TABLE

DRUG	CLASS	RECEPTOR	KEY WITHDRAWAL	FIRST-LINE TREATMENT
Alcohol	Sedative	GABA-A↑, NMDA↓	Seizures, DTs (life-threatening)	Diazepam detox; naltrexone / acam prosate maintenance
Heroin	Opioid	MOR agonist	Dysphoric, not life-threatening	Buprenorphine / naloxone OST
Cocaine	Stimulant	DAT/NET/SERT blocker	Crash: dysphoria, fatigue	No approved pharmacotherapy; CBT
Methamphetamine	Stimulant	DAT/NET/SERT releaser	As cocaine; also depression	No approved; CBT, contingency management
Cannabis	Cannabinoid	CB1 agonist	Irritability, insomnia, anxiety	CBT, MI; no approved pharmacotherapy
Nicotine	Stimulant	$\alpha 4\beta 2$ nAChR agonist	Irritability, craving, weight gain	Varenicline > Bupropion > NRT
Benzodiazepines	Sedative	GABA-A PAM	Seizures, anxiety (life-threatening)	Slow taper with long-acting BZD
MDMA	Empathogen	5-HT/DA/NE releaser	Fatigue, depression	Supportive

Model Answers

KEY INSIGHT

Sources: Kaplan & Sadock, Stahl's, Oxford Textbook of Psychiatry, WHO Guidelines, ICD-11, DSM-5-TR

HOW TO USE THIS FILE

Each answer is structured for a 10-mark long-answer question. Recommended time: 15–18 minutes per question. Target: 1 mark per ~1.5 minutes of reading. Write introductory definition, classification, body, a table, and a conclusion.

EXAM STRATEGY

PG exams examiners reward: (1) structured answers with subheadings, (2) tables showing comparisons, (3) management flowcharts in prose, (4) clinical context in the last paragraph. Never start with "I will describe...", start with the definition.

Q1. Discuss the management of alcohol withdrawal syndrome. [10 marks]

Answer:

Definition

Alcohol withdrawal syndrome (AWS) is a clinically significant constellation of symptoms occurring when alcohol-dependent individuals abruptly stop or markedly reduce intake. It results from removal of alcohol's chronic positive allosteric modulation of GABA-A receptors and inhibition of NMDA glutamate receptors, leading to CNS hyperexcitability.

Clinical Spectrum and Timeline

PHASE	ONSET (HOURS AFTER LAST DRINK)	FEATURES
Minor withdrawal	6–12	Anxiety, tremor, diaphoresis, insomnia, tachycardia, hypertension
Alcoholic hallucinosis	12–24	Auditory hallucinations, clear sensorium
Withdrawal seizures	24–48	GTCS, brief, self-limited; ~3–5% of untreated cases

PHASE	ONSET (HOURS AFTER LAST DRINK)	FEATURES
Delirium tremens	48–72	Delirium, severe autonomic instability, hallucinations

Assessment Tools

CIWA-Ar (Clinical Institute Withdrawal Assessment for Alcohol, revised):

- 10 items: nausea, tremor, paroxysmal sweats, anxiety, agitation, tactile / auditory / visual disturbances, headache, orientation
- Score <8: mild; 8–15: moderate; >15: severe; >20: ICU-level care

Management Algorithm

Step 1, Immediate assessment

- Full clinical examination, vital signs
- Blood tests: glucose, electrolytes (Na, K, Mg, phosphate), LFTs, CBC, coagulation, toxicology screen
- Identify concurrent medical illness (infection, head injury, liver disease)

Step 2, Thiamine (mandatory before glucose)

- 100–200 mg IV / IM thiamine BEFORE any IV glucose
- Rationale: Prevents precipitation of Wernicke's encephalopathy in thiamine-depleted state
- High-risk patients (malnourished, heavy drinkers): 500 mg IV thiamine (Pabrinex)

Step 3, Pharmacological management

Benzodiazepines (cornerstone of treatment):

AGENT	ROUTE	DOSE	PREFERENCE
Diazepam	Oral / IV	10–20 mg QID, taper 20–25% / day	Standard; IV for DTs/seizures
Chlordiazepoxide	Oral	25–50 mg QID, taper	Mild-moderate uncomplicated
Lorazepam	IV / IM	2–4 mg Q4–6H	Liver disease; elderly
Oxazepam	Oral	15–30 mg QID	Severe liver failure

Dosing strategies:

- **Fixed-schedule with CIWA-Ar monitoring:** Diazepam 20 mg QID Day 1 → taper 20% per day over 5–7 days

- **Symptom-triggered (preferred where feasible):** CIWA-Ar $\geq 8 \rightarrow$ give BZD; reassess every 1–2 hours; uses significantly less benzodiazepine

Step 4, Specific complications

Withdrawal seizures:

- IV diazepam 10–20 mg for active seizure
- If seizures recur: IV lorazepam or phenytoin loading
- Carbamazepine: alternative outpatient anticonvulsant for mild withdrawal

Delirium tremens:

- ICU/HDU monitoring
- IV diazepam titrated to calm patient (may need large doses: 100–200 mg in 24 hours)
- Correct hypoglycemia, hypomagnesemia, electrolyte imbalances
- Antipyretics for hyperthermia
- Haloperidol ONLY as adjunct for psychotic symptoms, NEVER as sole treatment (no anticonvulsant effect)
- Mortality: $<1\%$ with treatment vs 15–35% untreated

Step 5, Nutritional and general support

- Oral thiamine continue 2 weeks post-detox
- Multivitamins: folate, B12, B6
- Oral/IV hydration
- High-calorie diet

Step 6, Relapse prevention (post-detox)

- Disulfiram 250–500 mg/day (supervised)
- Naltrexone 50 mg/day
- Acamprosate 666 mg TID
- Psychosocial: CBT, MI, AA/NA, family therapy

Prognosis

Without intervention: 3–5% risk of DTs; ~3% seizure risk; significant recidivism. With structured management: DTs mortality $<1\%$; long-term sobriety improved with combined pharmacotherapy + psychosocial treatment.

EXAM STRATEGY

In management questions, use the "Step 1-2-3-4-5" ladder structure. Examiners reward systematic thinking. Always mention: thiamine before glucose, CIWA-Ar scoring, BZD choice rationale, DTs as emergency, relapse prevention.

Q2. Compare and contrast disulfiram, naltrexone, and acamprosate in the management of alcohol dependence. [10 marks]

Answer:

Introduction

Pharmacological relapse prevention is a key component of AUD management after successful detoxification. Three agents, disulfiram, naltrexone, and acamprosate, have the strongest evidence base and are approved by major regulatory agencies. They work through distinct mechanisms and are suited to different clinical profiles.

Mechanisms of Action

DRUG	PRIMARY MECHANISM	TARGET SYSTEM
Disulfiram	Irreversible inhibition of ALDH → acetaldehyde accumulation when alcohol consumed	Alcohol metabolism
Naltrexone	Competitive MOR (mu-opioid) antagonist → blocks alcohol-in- duced euphoria and endorphin release	Opioid-mediated reward
Acamprosate	GABA-A positive allosteric modulator + NMDA glutamate antagonist → reduces protract- ed withdrawal / allostatic dysregulation	Glutamate-GABA balance

Head-to-Head Comparison

FEATURE	DISULFIRAM	NALTREXONE	ACAMPROSATE
Dose	250–500 mg / day	50 mg / day oral; 380 mg / month IM	666 mg TID (1998 mg / day)
Mechanism	Deterrent	Anti-craving / anti- reward	Anti-withdrawal / pro-abstinence
Goal	Complete abstinence (aversion)	Reduce heavy drink- ing / craving	Maintain abstinence
Effect in setting of al- cohol use	Severe DER (dangerous)	No adverse reaction	No adverse reaction
Evidence quality	Moderate; supervised use better	Strong (NNT ~7)	Strong; best post-detox
Start timing	≥24 hours alcohol-free	Any time	After completion of detox

FEATURE	DISULFIRAM	NALTREXONE	ACAMPROSATE
Hepatotoxicity risk	Yes (rare; LFT monitoring)	Hepatotoxic at high doses	Minimal
Renal excretion	No	No (hepatic)	Yes, contraindicated in severe renal failure
Contraindications	Psychosis, CV disease, hepatic disease, thyroid disease, pregnancy, no motivation	Active opioid use, liver failure, patients on opioid analgesia	CrCl <30 mL/min
Key interaction	Alcohol; metronidazole, isoniazid (produce DER-like)	Opioid analgesics (blocks analgesia)	Few significant
Dosing adherence challenge	Can simply stop taking it	Easier adherence	TID dosing reduces adherence
Best for	Highly motivated patients with social support; supervised ingestion setting	Reducing heavy drinking; craving-driven relapse; ongoing drinking at treatment start	Post-detox abstinence maintenance; anxiety-driven protracted withdrawal

Disulfiram-Ethanol Reaction (DER)

When alcohol is consumed, acetaldehyde accumulates:

- Flushing, pulsating headache, nausea, vomiting
- Tachycardia, palpitations, hypotension
- Dyspnoea, sweating
- Severe cases: cardiovascular collapse, unconsciousness
- Treatment: supportive; IV fluids, antihistamine (H1 + H2), vitamin C; vasopressors if needed

Practical Prescribing Decision Framework

Evidence Summary

- **COMBINE trial:** Naltrexone reduced % heavy drinking days; acamprosate not superior to placebo in US population; combined not better than naltrexone alone
- **European data:** Acamprosate strongly effective in post-detox abstinence in European populations
- **Project MATCH:** MI, CBT, and 12-step similarly effective psychosocially
- **Baclofen (additional):** GABA-B agonist; emerging evidence particularly for anxious patients; not yet first-line

EXAM PEARL

Naltrexone works at the level of reward (opioid system); acamprosate works at the level of allostasis (glutamate-GABA balance). This mechanistic difference predicts which patient benefits from which drug.

EXAM STRATEGY

Comparison questions demand a table. Include mechanism, dose, side effects, contraindications, and best-for profile. Always end with the clinical decision framework.

Q3. Discuss opioid substitution therapy (OST) in the management of opioid use disorder. [10 marks]

Answer:

Definition

Opioid substitution therapy (OST) is the provision of a prescribed, pharmacologically active opioid (or partial agonist) in a supervised, controlled clinical setting to reduce illicit opioid use, harm associated with injection, and mortality. It is the evidence-based standard of care for opioid dependence.

Rationale

- Illicit opioid use: uncontrolled dose, contaminants, needle sharing (BBV transmission), crime
- OST stabilizes the neurobiological state, reduces craving, prevents withdrawal
- Harm reduction: HIV, Hepatitis B/C prevention
- Social reintegration: employment, family functioning
- Reduces mortality by 50% or more

WHO Position

OST (methadone and buprenorphine) is on the WHO Essential Medicines List. Declared a "highly cost-effective" intervention. Recommended as first-line for moderate-severe OUD.

Agents Used in OST

1. METHADONE

Class Full MOR agonist

Half-life 24–36 hours

Route Oral liquid (supervised ingestion at pharmacy or clinic)

Starting dose 20–40 mg/day (not exceeding 30–40 mg Day 1)

Maintenance 60–120 mg/day (higher is more effective)

QTc effects Dose-dependent prolongation; ECG at baseline, 3 months, annually

In pregnancy Preferred (decades of safety data); neonatal abstinence syndrome expected but manageable

Diversion High risk, requires observed dosing; take-home doses earned with compliance

DAWS on stop Prolonged and severe if abrupt cessation

2. BUPRENORPHINE (SUBUTEX) AND BUPRENORPHINE/NALOXONE (SUBOXONE)

Class Partial MOR agonist + KOR antagonist; very high receptor affinity

Half-life 24–72 hours (sublingual)

Route Sublingual tablets/film

Starting dose 4–8 mg day 1 (MUST be in mild withdrawal: COWS $\geq$ 8–12)

Maintenance 16–32 mg/day

Ceiling effect Yes, respiratory depression plateaus (safer in overdose)

Naloxone component Active IV only; deters injection of diverted product

Precipitated withdrawal Risk if started before opioid cleared; educate patient

In pregnancy Increasing evidence; buprenorphine alone preferred

Indian availability Government DTC scheme: buprenorphine 2 mg, 8 mg strips

METHADONE VS BUPRENORPHINE COMPARISON

PARAMETER	METHADONE	BUPRENORPHINE/NALOXONE
Receptor action	Full agonist	Partial agonist
Overdose risk	Higher	Lower (ceiling)
Diversion control	Observed dosing required	Naloxone component deters IV misuse
Take-home eligibility	After prolonged compliance	Earlier take-home possible
Induction flexibility	Day 1 (no withdrawal needed)	Must be in withdrawal
QTc risk	Yes	Minimal

PARAMETER	METHADONE	BUPRENORPHINE/NALOXONE
Cost	Lower	Higher
First-line preference	Second-line / severe OUD	First-line

Induction Protocol (Buprenorphine)

1. Patient education: must be in moderate withdrawal (COWS $\geq 8-12$)
2. Day 1: 4 mg SL $\rightarrow$ observe 1–2 hours $\rightarrow$ if tolerated, 4 mg more $\rightarrow$ total 8 mg Day 1
3. Day 2–3: titrate to 12–16 mg
4. Week 2–4: target maintenance dose (16–24 mg usual)
5. Supervised dispensing initially; take-home doses after 3 months stable

Adjuncts and Psychosocial Components

OST is NOT medication alone:

- Contingency management (CM): positive reinforcement for drug-free urines
- CBT: relapse prevention, coping skills
- Motivational Enhancement Therapy
- Case management: housing, employment, legal
- Family therapy where appropriate
- Mutual aid: NA, SMART Recovery

Duration

OST is long-term treatment, minimum 12–24 months, often indefinitely. Premature cessation $\rightarrow$ high relapse rate. Slow supervised taper (if desired): reduce by 2–4 mg / month.

Indian Context

- National Drug Dependence Treatment Centre (NDDTC), PG exams: OST program
- Drug Treatment Centres (DTCs): government-run buprenorphine dispensation
- NDPS Act Section 71: judicial powers to mandate treatment
- Challenges: stigma toward OST ("replacing one drug with another"), limited qualified providers, strict regulations

Outcomes

- ~50% reduction in illicit opioid use
- HIV incidence reduction ~54%
- Overdose mortality reduction: 50–70%
- Retention in treatment at 12 months: methadone ~60–65%; buprenorphine ~55–60%
- Superior retention with higher doses (>60 mg methadone; >16 mg buprenorphine)

EXAM PEARL

"Dose adequate" is the single biggest predictor of OST outcome. Undertreated patients relapse. A patient still using on OST should have dose increased, not the program terminated.

EXAM STRATEGY

OST questions expect you to know: mechanism, two agents and comparison, induction protocol, psychosocial elements, and Indian context. Examiners want to know you understand OST is not "giving addicts drugs" but evidence-based harm reduction.

Q4. Discuss cannabis and psychosis: is the relationship causal? [10 marks]

Answer:

Introduction

The relationship between cannabis use and psychotic disorders has been debated for decades. Accumulating epidemiological, experimental, and genetic evidence now supports a component cause model: cannabis is neither a necessary nor sufficient cause of psychosis, but it reliably increases risk, the relationship is causal.

Epidemiological Evidence

STUDY · FINDING

Andréasson et al. (1987, Swedish conscripts) Cannabis use × 6 increase in schizophrenia risk; dose-response

Zammit et al. (2002) OR 3.1 for heavy cannabis use; dose-response relationship confirmed

Moore et al. (2007, meta-analysis) OR 1.41 (any use) to 2.09 (heavy use); population attributable risk: 8–14% of schizophrenia

Di Forti et al. (2019, EU-GEI) High-potency (skunk) daily use: OR 4.8; populations with high skunk use had higher schizophrenia rates

Bradford Hill Criteria for causality:

CRITERION · CANNABIS-PSYCHOSIS EVIDENCE

Strength OR 2–5; consistent across studies

Consistency Replicated across 30+ countries

Temporality Cannabis use PRECEDES psychosis onset

Biological gradient Dose-response: more use → higher risk

Plausibility Mechanism: CB1 agonism → DA dysregulation

Experiment IV THC → transient psychotic-like symptoms in healthy volunteers (D'Souza 2004)

Coherence High-THC low-CBD products have highest risk

EXAM PEARL

Temporality is the critical Bradford Hill criterion for causality. Cannabis use preceding psychosis is well-established in longitudinal cohort studies.

Mechanisms

Dopaminergic pathway:

- THC → CB1 stimulation in VTA → increased DA synthesis and release in striatum
- Striatal hyperdopaminergia = the final common pathway of psychosis
- PET studies: THC increases striatal DA release in healthy volunteers

Prefrontal cortex:

- CB1 receptors in PFC → THC impairs PFC function → reduced inhibitory control over subcortical DA
- Mirrors disinhibition hypothesis of schizophrenia

Glutamate-DA interaction:

- THC impairs NMDA receptor function → similar to ketamine model of psychosis

THC:CBD ratio:

- CBD is a CB1 negative allosteric modulator
- High-CBD products associated with lower psychotomimetic risk
- Modern skunk (high THC/low CBD) → highest risk

Cannabis-Induced Psychosis vs Primary Psychosis

FEATURE	CANNABIS-INDUCED PSYCHOSIS	SCHIZOPHRENIA
Onset	Within 1 month of use	Gradual or acute without substance trigger
Duration	Typically <1 month with abstinence	Chronic, persists without treatment

FEATURE	CANNABIS-INDUCED PSYCHOSIS	SCHIZOPHRENIA
Content	Paranoia, perceptual changes	Positive + negative + disorganized symptoms
Negative symptoms	Mild, transient	Prominent, persistent
Prognosis	Good with abstinence	Variable; usually chronic
Conversion rate	25–50% to schizophrenia-spectrum over 5 years (Di Forti 2019)	

Moderating Factors

- **Genetic vulnerability:** COMT Val158Met polymorphism, Val/Val carriers more vulnerable to cannabis-induced psychosis
- **Age of onset:** Adolescent use carries greater risk (neurodevelopmental sensitivity)
- **Frequency and duration:** Daily / near-daily use, long duration
- **Product potency:** High-THC products (>10% THC)
- **Pre-existing subclinical psychosis:** Cannabis precipitates in those already on the psychosis continuum

Clinical Implications

- Substance use history: mandatory in any psychosis assessment
- Drug screen: THC in urine, blood
- Management:
- Psychoeducation: evidence-based information about risk
- Motivational interviewing for cessation
- Antipsychotics for acute cannabis-induced psychosis
- Monitor: if psychosis persists >1 month after abstinence → reassess diagnosis

CLINICAL ANCHOR

In a young patient with first-episode psychosis who uses cannabis: abstinence is both therapeutic AND diagnostic. If psychosis clears → cannabis-induced. If persists → primary psychosis. This is clinically elegant and evidence-based.

EXAM STRATEGY

"Is the relationship causal?", use Bradford Hill criteria. Examiners appreciate structured epidemiological thinking. Four key points: dose-response, temporality, experiment (IV THC studies), mechanisms.

Q5. Describe motivational interviewing in substance use disorders. [10 marks]

Answer:

Definition

Motivational Interviewing (MI) is an evidence-based, person-centered counseling approach developed by William Miller and Stephen Rollnick (1991). It is defined as "a collaborative, goal-oriented style of communication with particular attention to the language of change, designed to strengthen personal motivation and commitment to a specific goal by eliciting and exploring the person's own reasons for change within an atmosphere of acceptance and compassion."

Theoretical Foundation

MI emerged from the Transtheoretical Model (Prochaska & DiClemente) and Carl Rogers' person-centered therapy. The central insight: people change when they articulate their own reasons for change, external pressure is counterproductive.

Ambivalence: MI views ambivalence as a normal state, not resistance or denial. It is the simultaneous desire to change and not to change. MI's role is to resolve ambivalence in the direction of change.

MI Spirit (PACE)

COMPONENT · MEANING

Partnership Clinician and client collaborate as equals; no expert-advice model

Acceptance Absolute worth of person; accurate empathy; autonomy; affirmation

Compassion Active prioritization of client's welfare

Evocation Drawing out client's own motivation rather than installing it

EXAM PEARL

The spirit is NOT about techniques, it's the underlying attitude. MI without spirit is just interrogation with open questions.

Four Processes

PROCESS · GOAL

Engaging Establish therapeutic relationship; trust; clarify role

Focusing Develop specific direction for the conversation

Evoking Elicit and strengthen change talk

Planning Develop commitment to change and action plan

Core Skills: OARS

SKILL	PURPOSE	EXAMPLE
Open questions	Elicit reflection, not yes/no	"What's been going on with your drinking lately?"
Affirmations	Recognize strengths and effort	"You've really thought hard about this."
Reflective listening	Demonstrate understanding; deepen exploration	"So it sounds like part of you knows it's affecting your family..."
Summaries	Collect change talk; link discrepancies; transition	"Let me pull together what you've shared..."

Change Talk: DARN-CAT

Change talk = client speech that favors change.

Preparatory change talk (DARN):

- Desire: "I want to cut back"
- Ability: "I could stop if I wanted to"
- Reason: "My liver results scared me"
- Need: "I need to do something"

Mobilizing change talk (CAT):

- Commitment: "I will try to reduce"
- Activation: "I'm ready to make a change"
- Taking steps: "I've already thrown out the bottles"

EXAM PEARL

Mobilizing change talk (especially commitment) is the strongest predictor of actual behavior change. Strengthen CAT talk, not just DARN talk.

Sustain talk: Client arguing for status quo, reflect, don't argue. "Rolling with resistance" = working with not against.

Evoking Change Talk: Strategies

1. **Decisional balance:** "What are the good things about your drinking? What are the less good things?"

2. **Importance and confidence rulers:** "On a scale of 0–10, how important is it to you to change? Why not a lower number?"
3. **Looking forward/back:** "How was your life before alcohol became a problem? How do you see it in 5 years if nothing changes?"
4. **Exploring values:** "What matters most to you? How does your drinking fit with that?"

Evidence Base

TRIAL · FINDING

Project MATCH (1997) MET (4 sessions) = equivalent to 12-session CBT and 12-step; superior for angry patients

UKATT (2005) MET cost-effective; equivalent to social behavior therapy

Cochrane review (Smedslund 2011) MI significantly reduces substance use vs no-treatment; moderate effect size

Application in Different Settings

- **Brief intervention (5–15 min):** FRAMES model; AUDIT / CAGE → feedback → brief MI
- **Motivational Enhancement Therapy (MET):** 2–4 structured sessions; adapted from MI
- **Dual diagnosis:** Adapted MI, longer engagement phase; address mental illness first; no confrontation of denial

CLINICAL ANCHOR

MI is most effective in precontemplation and contemplation stages. It is NOT a treatment for dependence itself, it is an engagement tool to move people toward accepting treatment.

EXAM STRATEGY

MI questions reward depth on the spirit, the 4 processes, and OARS. Always add a brief section on evidence base and limitations. 15 minutes = 10 marks: plan introduction, spirit, 4 processes, OARS, change talk, evidence, conclusion.

Q6. Discuss the AUDIT and CAGE screening tools for alcohol use disorders. [10 marks]

Answer:

Introduction

Screening for harmful and hazardous alcohol use is a critical component of clinical practice. Two tools dominate: AUDIT (Alcohol Use Disorders Identification Test), developed by the WHO for cross-cultural application, and CAGE, a 4-item clinical screening instrument. Both

detect problematic drinking but have different characteristics, validity profiles, and appropriate clinical settings.

AUDIT (Alcohol Use Disorders Identification Test)

Development: WHO collaborative project (Babor et al., 1989). Validated in multiple countries including India.

Structure, 10 items across 3 domains:

DOMAIN	ITEMS	WHAT IT CAPTURES
Hazardous use	Q1–3	Frequency of drinking; typical quantity; frequency of heavy episodic drinking
Dependence features	Q4–6	Impaired control; salience; morning drinking
Harmful use	Q7–10	Guilt; blackouts; alcohol-related injuries; concern from others

Scoring:

- Each item: 0–4 (except Q9 and Q10: 0, 2, or 4 only)
- Total: 0–40

SCORE	INTERPRETATION	INTERVENTION
0–7	Low-risk	Education
8–15	Hazardous/harmful use	Simple advice
16–19	Probable dependence	Brief counseling + referral
≥20	Dependence likely	Specialist referral

Threshold for positive screen: ≥8 in most settings; some studies use ≥7 for greater sensitivity.

AUDIT-C (3-item brief version):

- Items 1–3 only (frequency, quantity, frequency of binge)
- Threshold: ≥4 (men), ≥3 (women)
- Sensitivity 73–91% for hazardous use
- Suitable for very brief screening (30 seconds)

Psychometric properties:

- Sensitivity ~57–97%, Specificity ~78–96% (varies by threshold and population)
- Validated in Indian clinical settings (Ebrahim et al.)

CAGE Questionnaire

Development: Ewing (1984). 4-item mnemonic tool.

Questions:

LETTER · QUESTION

C Have you ever felt you should **Cut down** on your drinking?

A Have people **Annoyed** you by criticizing your drinking?

G Have you ever felt bad or **Guilty** about your drinking?

E Have you ever had a drink first thing in the morning (an **Eye-opener**) to steady your nerves or to get rid of a hangover?

Scoring: Each "yes" = 1 point (0–4)

SCORE · INTERPRETATION

0 No significant problem

1 Caution; further inquiry

≥2 Clinically significant; high likelihood of AUD

Psychometric properties:

- Sensitivity: ~74–85%; Specificity: ~79–91% for alcohol dependence
- Best validated for detecting dependence (not hazardous use)

AUDIT vs CAGE: Comparison

FEATURE	AUDIT	CAGE
Items	10	4
Time	2–3 minutes	<1 minute
Quantitative data?	Yes (frequency, amount)	No
Detects hazardous use?	Yes	Poor
Detects dependence?	Yes	Yes
Cultural validation	WHO; multi-country	Primarily Western
Use in primary care	Ideal	Quick screen
Use in ED	AUDIT-C preferred	CAGE useful
Follow-up action	Score guides intervention level	Simple positive/negative
Lifetime vs current	Both	Lifetime-oriented

FEATURE	AUDIT	CAGE
Limitation	Patient may under-report quantities	Relies on introspection; insensitive to early use

Other Screening Tools

TOOL	ITEMS	SETTING
MAST	25 (Brief MAST: 13)	Research; lifetime alcohol problems
FAST	4 items	Emergency department
RAPS-4	4 items	Lifetime; similar to CAGE
DAST	10 or 28 items	Non-alcohol substances

CLINICAL ANCHOR

In Indian primary care and de-addiction settings, AUDIT-C (3 items) is the most practical screening tool. CAGE score ≥ 2 should immediately trigger a structured clinical assessment and CIWA-Ar if intoxication/withdrawal suspected.

EXAM STRATEGY

AUDIT/CAGE questions want you to write out the actual questions (especially CAGE, spell out the acronym). For AUDIT, describe the 3 domains and scoring thresholds. Always compare the two in a table.

Q7. Discuss Wernicke-Korsakoff syndrome in the context of alcohol use disorder. [10 marks]

Answer:

Introduction

Wernicke-Korsakoff syndrome (WKS) is a neuropsychiatric complication of thiamine (Vitamin B1) deficiency, most commonly seen in the context of alcohol use disorder. It represents a clinical continuum: Wernicke's encephalopathy (acute, reversible if treated promptly) → Korsakoff's syndrome (chronic amnestic, largely irreversible).

Thiamine Deficiency in AUD: Pathophysiology

FACTOR · MECHANISM

Inadequate dietary intake Poor nutritional quality of alcohol-heavy diet

Malabsorption Alcohol-related gastritis, impaired jejunal absorption

Reduced hepatic storage Liver disease impairs thiamine phosphorylation and storage

Increased metabolic demand Alcohol metabolism and carbohydrate loading consume thiamine

Impaired transport Reduced transketolase activity

Thiamine function: Cofactor for pyruvate dehydrogenase, alpha-ketoglutarate dehydrogenase, transketolase → ATP production in Krebs cycle → CNS most vulnerable to energy failure.

Wernicke's Encephalopathy (Acute)

Classic triad:

1. **Ophthalmoplegia**, lateral rectus palsy (most common), nystagmus, conjugate gaze palsy
2. **Ataxia**, cerebellar; wide-based gait; lower limb > upper limb
3. **Confusion / encephalopathy**, global confusion, disorientation

EXAM PEARL

The classic triad is present in only 10–16% of autopsy-confirmed cases. Do NOT wait for the full triad. Any alcohol-dependent patient with neurological or cognitive change must receive immediate thiamine.

Pathology:

- Petechial hemorrhages, edema, astrocyte proliferation
- Location: mammillary bodies (most specific), dorsomedial thalamus, periaqueductal grey, cerebellar vermis

MRI findings:

- Hyperintensity on FLAIR/T2W in mammillary bodies, thalamus, periaqueductal grey
- Mammillary body atrophy (classic)

Treatment (emergency):

PROTOCOL	DOSE	ROUTE	TIMING
Mild clinical suspicion	100 mg	IV / IM	Immediately; before glucose
High-risk / confirmed	200–500 mg	IV	Three times daily for 2–3 days
Continue oral	100 mg	Oral	For 2 weeks minimum

Critical rule: Thiamine BEFORE IV glucose in ANY alcohol-dependent patient, glucose infusion without thiamine can precipitate acute Wernicke's by depleting remaining thiamine reserves.

Korsakoff's Syndrome (Chronic)

Pathological substrate: Dorsomedial thalamic nucleus atrophy; mammillary body atrophy; frontal lobe involvement.

Clinical features:

Anterograde amnesia New learning profoundly impaired; inability to form new episodic memories

Retrograde amnesia Temporal gradient: recent more affected than remote

Confabulation Unconscious fabrication (not lying); fills memory gaps; "momentary" and "fantastic" types

Insight Usually absent, patient unaware of memory deficit

Other cognition Relatively preserved (attention, language, procedural memory)

Personality Apathy, loss of initiative; may appear indifferent to deficit

CLINICAL ANCHOR

Test Korsakoff's with: registration (intact), immediate recall (intact), delayed recall (severely impaired). This distinguishes it from dementia where registration itself may be impaired.

Prognosis:

- ~25% significant improvement with abstinence + thiamine
- ~50% partial improvement
- ~25% minimal improvement
- Time to recovery: months to years
- Predictors of poor outcome: long duration before treatment, severe malnutrition, continued alcohol use

Korsakoff's vs Other Dementias:

FEATURE	KORSAKOFF'S	ALZHEIMER'S
Memory	Anterograde >> retrograde	Both; registration also impaired
Confabulation	Prominent	Uncommon
Other cognition	Relatively preserved	Widespread decline

FEATURE	KORSAKOFF'S	ALZHEIMER'S
Insight	Absent	Preserved early; lost late
Progression	Stable (with abstinence)	Progressive
Cause	Thiamine deficiency	Beta-amyloid / tau

EXAM STRATEGY

WKS questions reward: (1) mechanism (thiamine function), (2) the 10–16% full triad fact, (3) acute management with specific dosing, (4) the "thiamine before glucose" rule, (5) chronic Korsakoff clinical features especially confabulation, (6) prognosis. These are the examiner's checklist.

Q8. Describe the clinical features and management of delirium tremens. [10 marks]

Answer:

Definition

Delirium tremens (DTs) is the most severe manifestation of alcohol withdrawal syndrome, characterized by global confusion (delirium), severe autonomic hyperactivity, and perceptual disturbances. It represents a medical emergency with mortality of 15–35% if untreated, reduced to <1% with appropriate management.

Epidemiology and Risk Factors

Occurs in approximately 3–5% of hospitalized alcohol withdrawal patients.

Risk factors for DTs:

RISK FACTOR · EVIDENCE

Previous DTs or seizures Strongest predictor (kindling)

Prolonged, heavy alcohol use (>10 years) Dose-dependent neuroadaptation

High alcohol intake before admission >750 mL spirits equivalent / day

Age >65 Impaired neurological reserve

Concurrent illness Infection, head injury, liver disease, electrolyte disturbance

Benzodiazepine/sedative co-dependence Compounded GABA-A deficit

Poor nutritional status Thiamine depletion → compounded

First presentation delayed Treatment delay worsens prognosis

Pathophysiology

Chronic alcohol ingestion:

- Downregulates GABA-A receptors (tolerance to CNS depression)
- Upregulates NMDA glutamate receptors

Alcohol withdrawal:

- Reduced GABAergic tone → excitatory dominance
- Unmasked NMDA hyperactivity → neuronal hyperexcitability
- Noradrenergic hyperactivity (locus coeruleus) → autonomic storm
- Dopaminergic excess → hallucinations

Clinical Features

Onset: 48–72 hours after last drink (can extend to 5–7 days)

Duration: Typically 3–5 days if treated; can persist

DOMAIN · FEATURES

Cognitive Disorientation to time/place, impaired attention, waxing-waning consciousness

Perceptual Visual hallucinations (most prominent): complex, vivid, often terrifying; Lilliputian hallucinations (miniature figures); also tactile, auditory

Autonomic Tachycardia (HR >120), hypertension, hyperthermia (up to 40°C), profuse diaphoresis, mydriasis

Motor Coarse tremor, psychomotor agitation, myoclonus

Behavioural Extreme fear, agitation, combativeness

EXAM PEARL

Lilliputian hallucinations (seeing miniature people/animals) are pathognomonic of DTs. Visual hallucinations in DTs are typically complex, vivid, and terrifying, insects, animals, monsters.

DTs vs Alcoholic Hallucinoses Comparison

FEATURE	DTs	ALCOHOLIC HALLUCINOSIS
Onset (hours post last drink)	48–72	12–24
Consciousness	Clouded (delirium)	Clear
Hallucinations	Visual > auditory	Auditory > visual
Autonomic features	Severe	Mild or absent

FEATURE	DTS	ALCOHOLIC HALLUCINOSIS
Orientation	Impaired	Preserved
Prognosis	Life-threatening	Self-limiting usually

Management

Step 1, Immediate stabilization

- ICU or HDU admission
- IV access × 2
- Continuous monitoring: HR, BP, temperature, oxygen saturation, glucose
- Airway management if required

Step 2, Thiamine first

- 200–500 mg IV thiamine (before any glucose)
- Continue 100 mg TID for 2–3 days

Step 3, Benzodiazepines (primary pharmacotherapy)

SCENARIO · DRUG AND DOSE

Moderate agitation IV diazepam 10 mg bolus; repeat every 5–10 minutes until calm

Severe agitation IV diazepam 20 mg loading; then infusion 10 mg/hour; titrate

Liver disease IV lorazepam 2–4 mg (no active metabolites)

Maintenance Oral chlordiazepoxide 50 mg QID → taper OR continued IV based on CIWA-Ar

Step 4, Adjuncts

DRUG	ROLE	DOSE
Haloperidol	Psychotic symptoms only (adjunct, no anticonvulsant)	5 mg IM/IV
Clonidine	Autonomic symptoms adjunct	0.1–0.3 mg oral
Propranolol	HR/BP (not anticonvulsant)	40–80 mg
Magnesium sulphate	Correct hypomagnesemia; may reduce seizure threshold	2–4 g IV

Step 5, Treat precipitants

- Blood cultures and broad-spectrum antibiotics if infection suspected
- CT head if focal neurology or head injury

- Correct hyponatremia, hypoglycemia, hypophosphatemia

Step 6, Monitoring and endpoints

- CIWA-Ar every 4–8 hours; target <8 before stepping down
- Watch for: aspiration pneumonia, cardiovascular events, hyperthermia complications
- Transition to oral regimen when stable

Prognosis

With treatment: mortality <1%. Predictors of poor outcome: concurrent liver failure, severe hyperthermia (>40°C), cardiovascular disease, delayed presentation, pneumonia.

CLINICAL ANCHOR

The most common cause of death in DTs is cardiovascular, arrhythmia, not seizure. Aggressive autonomic management is as important as anticonvulsant treatment.

EXAM STRATEGY

DTs management answers: use Step 1–6 structure. Emphasize the severity (life-threatening), thiamine before glucose, BZD as cornerstone, haloperidol as adjunct only (critical distinction), and monitoring. Common mistake: writing haloperidol as primary treatment.

Q9. Describe the Transtheoretical Model of behaviour change and its application in substance use disorders. [10 marks]

Answer:

Introduction

The Transtheoretical Model (TTM), developed by Prochaska and DiClemente (1983), is a model of intentional behavior change that integrates processes and principles of change from across major theories of psychotherapy. It was developed empirically through studying smokers who quit on their own vs with help. Its core concept: behavior change is a process (not an event) moving through predictable stages.

Stages of Change

STAGE	DEFINITION	CLIENT SELF-STATEMENT	INTERVENTION
Precontemplation	No intention to change in next 6 months; unaware or demoralized	"I don't have a problem"	Raise awareness; provide information; elicit concern

STAGE	DEFINITION	CLIENT SELF-STATEMENT	INTERVENTION
Contemplation	Aware of problem; ambivalent; weighing pros/cons; no definite plan	"I'm thinking about it, but..."	Explore ambivalence; decisional balance; MI
Preparation	Intends to take action within 30 days; has small behavioral steps	"I'm planning to cut down next week"	Help develop action plan; connect to resources
Action	Overt behavior change, 0–6 months	"I've been sober 3 weeks"	Support; reinforce; skill-building; relapse prevention
Maintenance	Sustaining change >6 months; consolidating gains	"I've been sober 8 months"	Relapse prevention; lifestyle restructuring
Relapse	Return to previous behavior	"I slipped last weekend"	Non-judgmental review; learn from relapse; return to cycle
Termination	No temptation; complete confidence (rare in addiction)	"I don't even think about it anymore"	Consolidation

EXAM PEARL

Most treatment programs focus on Action, but most patients present in Precontemplation or Contemplation. Mismatch between intervention and stage is a primary cause of treatment failure.

Processes of Change (10)

Cognitive-experiential processes (earlier stages):

1. Consciousness raising, increasing awareness
2. Dramatic relief, emotional arousal; "it could happen to me"
3. Environmental re-evaluation, impact on others
4. Self-re-evaluation, how substance use conflicts with values
5. Social liberation, awareness of social norms supporting change

Behavioral processes (later stages):

1. Self-liberation, commitment; belief in ability to change
2. Helping relationships, social support
3. Reinforcement management, rewards for behavior change
4. Counterconditioning, alternatives to substance use

5. Stimulus control, avoiding/restructuring cues

Decisional Balance

Central to movement through early stages: weighing pros and cons of both current behavior and proposed change.

STAGE · DECISIONAL BALANCE PROFILE

Precontemplation Pros of use >> Cons

Contemplation Pros ≈ Cons (ambivalence)

Preparation/Action Cons >> Pros

Relapse in the TTM

Relapse is not a failure, it is part of the spiral model of change. Studies show:

- Average 3–4 serious quit attempts before sustained cessation (nicotine)
- Relapse is a learning opportunity, each relapse narrows the cycle
- Clinicians should respond non-judgmentally: "What can we learn from this?"

Spiral model: Progress through stages is rarely linear, it is a spiral, with relapses returning to earlier stages but (ideally) at a higher level than before.

Application: Stage-Matched Interventions

STAGE	MI APPLICATION	CLINICAL ACTION
Precontemplation	Raise awareness without confrontation	AUDIT/CAGE screening; personalized feedback
Contemplation	Explore ambivalence; decisional balance	2–4 session MET
Preparation	Collaborative goal setting	Action planning; medication initiation
Action	Skills training; relapse prevention	CBT; pharmacotherapy; OST
Maintenance	Ongoing support; relapse prevention	Continuing care; mutual aid; booster sessions

Critique of TTM

- Stage boundaries are arbitrary (particularly Contemplation/Preparation)
- Stage classification not reliably predictive of treatment response in all studies
- Discrete stage model may not capture continuous motivational fluctuations

- However: remains the most clinically useful heuristic for matching intervention to readiness

CLINICAL ANCHOR

Ask every patient in substance use assessment: "On a scale of 0–10, how important is it to you to change your drinking / drug use?" + "How confident are you that you could change if you decided to?" This maps directly to TTM stage and guides intervention.

EXAM STRATEGY

TTM answers require the full table of stages with definitions and interventions. Add processes of change (cognitive vs behavioral), decisional balance, and clinical application. Bonus: mention the spiral model, it shows you understand relapse.

Q10. Discuss brief interventions in alcohol use disorders. [10 marks]

Answer:

Definition

A brief intervention (BI) is a structured but time-limited clinical encounter aimed at individuals whose drinking is risky or harmful (not necessarily dependent), designed to reduce alcohol consumption through motivational feedback and advice. Duration: 5–30 minutes; typically 1–3 sessions.

Rationale

- Population impact: 75–90% of alcohol-related harm occurs in hazardous / harmful users (not dependent)
- These patients are rarely in specialist services, they encounter GPs, ED, primary care
- BI is delivered at the point of identification: opportunistic
- Systematic reviews show: BI reduces drinking by ~4–5 standard drinks / week at 12 months
- Number needed to treat (NNT): ~8 for reduction in hazardous drinking

FRAMES Model

The most cited framework for effective brief interventions:

COMPONENT	MEANING	APPLICATION
Feedback	Personalized feedback on risk	"Your AUDIT score is 12, that's in the hazardous range"
Responsibility	Change is the client's choice	"Ultimately, it's up to you"

COMPONENT	MEANING	APPLICATION
Advice	Clear, direct recommendation	"I recommend you cut your drinking to within safe limits"
Menu	Range of options	"You could cut down, take alcohol-free days, change to lower-strength drinks..."
Empathy	Warm, empathic style	Non-judgmental, reflective listening
Self-efficacy	Belief in ability to change	"Many people find they can make this change, you've shown you can do it in the past"

Screening-Brief Intervention-Referral to Treatment (SBIRT)

The three-tier model:

LEVEL	POPULATION	INTERVENTION
Screening	All patients	AUDIT or AUDIT-C
Brief Intervention	Hazardous/harmful use (AUDIT 8–19)	5–15 minutes BI; FRAMES-based
Referral to Treatment	Probable dependence (AUDIT ≥ 20)	Referral to specialist

Evidence Base

STUDY · FINDING

WHO Phase II Trial (1996) 5–10 min BI: significant reduction in drinking at 9 months

SIGN (Scottish Intercollegiate) BI reduces alcohol intake and complications

Cochrane review (Kaner 2018, 69 RCTs) BI significantly reduces consumption at 12 months vs no intervention

Effect size Small-moderate (Cohen's $d \sim 0.2$); large public health impact

Minimal Intervention (5 R's for Unmotivated Patients)

For precontemplators who are not ready to change:

COMPONENT · ACTION

Relevance Link change to patient's own values/health concerns

Risks Brief personalized risks of continued use

Rewards Benefits of reducing intake

Roadblocks Anticipated barriers; problem-solve

Repetition Brief intervention at every contact

Setting and Delivery

- **Primary care:** Most evidence; opportunistic; AUDIT screen → BI if score 8–19
- **Emergency department:** "teachable moment" after alcohol-related injury; modified BI
- **Antenatal care:** Any alcohol use in pregnancy; non-judgmental BI
- **Occupational health:** Return-to-work context
- **India context:** PHC, Jan Aushadhi, de-addiction camps; ANMs and health workers can deliver with training

Limitations

- Efficacy declines at higher AUDIT scores (dependent patients need specialist care)
- In primary care: time pressure; competing priorities; clinician discomfort with alcohol topic
- BI alone insufficient for dependence, gateway to referral, not treatment

CLINICAL ANCHOR

Brief interventions are one of the most cost-effective public health tools in psychiatry. The World Bank estimates they return \$5–8 for every \$1 spent in productivity gains and reduced healthcare burden.

EXAM STRATEGY

BI questions: lead with definition, FRAMES (memorize the acronym), SBIRT model, Cochrane evidence, and finish with limitations. Don't confuse BI with MI, BI is structured, advice-giving, usually 1–3 sessions; MI is more collaborative, open-ended, process-focused.

Q11. Discuss dual diagnosis: the co-occurrence of substance use and psychiatric disorders. [10 marks]

Answer:

Definition

Dual diagnosis (also: comorbidity, co-occurring disorders) refers to the simultaneous presence of a substance use disorder and one or more psychiatric diagnoses. The term reflects the clinical reality that these conditions frequently co-occur, interact, and complicate each other's management.

Epidemiology

Major studies:

STUDY · FINDING

ECA Study (Regier 1990) 37% AUD patients had comorbid psychiatric disorder; 47% drug disorders comorbid

NCS-R (Kessler 2005) 51% of 12-month mental disorders in people with substance dependence

NESARC (Grant 2004) AOR for any mood disorder in AUD: 2.0; for any anxiety disorder: 1.7

Common pairings:

PSYCHIATRIC DISORDER · MOST ASSOCIATED SUBSTANCE

Major Depression Alcohol (most common overall)

Bipolar disorder Alcohol, cocaine, cannabis

Anxiety disorders Alcohol, benzodiazepines

PTSD Alcohol, cannabis, opioids

ADHD Stimulants, cannabis, nicotine

Schizophrenia Cannabis, nicotine, alcohol

Personality disorders (BPD, ASPD) Multiple

Pathways

PATHWAY	DESCRIPTION	EXAMPLE
Primary psychiatric → secondary SUD	Mental illness precipitates substance use (self-medication)	Depression → alcohol to cope → AUD
Primary SUD → secondary psychiatric	Substance causes psychiatric symptoms	Heavy cannabis use → psychosis
Bidirectional	Each exacerbates the other	PTSD + alcohol, each worsens the other
Common factor	Shared vulnerability	ADHD + stimulant misuse: shared dopaminergic deficit
Artifact	Apparent comorbidity due to cross-cutting symptoms	Alcohol withdrawal → anxiety (mimics GAD)

Diagnostic Challenges

- Substance intoxication/ withdrawal mimics psychiatric symptoms
- Need 4-week abstinence period ideally to clarify primary vs substance-induced
- In practice: clinical urgency may not allow this
- Temporal relationship crucial: which came first?
- ICD-11 / DSM-5: "substance-induced disorder" = causal; "comorbid disorder" = independent

Treatment Approaches

MODEL	DESCRIPTION	EVIDENCE
Sequential	Treat one, then the other	Historically common; poor outcomes
Parallel	Separate teams, same time	Better; coordination issues
Integrated	Same team, same time, same conceptual model	Best evidence; standard of care recommendation

Principles of integrated dual diagnosis treatment:

1. **Stage-matched approach:** Engagement → persuasion → active treatment → relapse prevention
2. **Low threshold for engagement:** Accept harm reduction goals initially
3. **Assertive community treatment** for severe mental illness + SUD
4. **Motivational Interviewing** adapted for low motivation
5. **Residential treatment** when severe; abstinence-based milieu
6. **Medication principles:**
 7. Treat both disorders pharmacologically
 8. Prefer medications with lower abuse potential
 9. SSRIs: first-line for depression/ anxiety in AUD
 10. Naltrexone: appropriate for AUD even with depression
 11. Antipsychotics: clozapine may reduce substance use in schizophrenia

PTSD and SUD

Particularly complex and common:

- PTSD → alcohol/ opioids/ cannabis as "chemical coping" → SUD
- Both maintain each other: substance withdrawal → PTSD re-experiencing worsens
- Evidence-based treatment: integrated exposure-based CBT (Seeking Safety, COPE)

EXAM PEARL

In schizophrenia + SUD (especially cannabis), clozapine has the best evidence for reducing substance use, mechanism possibly related to its broader receptor profile including 5-HT_{2A} antagonism and CB₁ indirect effects.

EXAM STRATEGY

Dual diagnosis: epidemiology numbers, 4 pathways, diagnostic challenge (abstinence period), 3 treatment models (sequential / parallel / integrated), medication principles. Finish with a clinical example.

Q12. Describe neonatal opioid withdrawal syndrome (NOWS). [10 marks]

Answer:

Introduction

Neonatal Opioid Withdrawal Syndrome (NOWS), previously termed Neonatal Abstinence Syndrome (NAS), is a condition in newborns exposed to opioids in utero, manifesting as a constellation of neurological, gastrointestinal, and autonomic signs following delivery. It is a direct consequence of opioid dependence during pregnancy and represents a significant clinical challenge in perinatal medicine.

Prevalence

- USA: dramatic increase; 14 per 1,000 hospital births (2016) vs 1.5 per 1,000 (2000)
- Parallels opioid epidemic
- India: less systematic data; rising with pharmaceutical opioid misuse

Pathophysiology

Opioids cross the placenta freely. Chronic fetal opioid exposure → neuroadaptation (MOR downregulation). At birth, when opioid supply ceases → withdrawal state.

Risk Factors for Severe NOWS

- Tobacco co-exposure (synergistic with opioids)
- SSRI exposure (increases length of stay)
- Long-acting opioids (methadone → prolonged NOWS vs heroin)
- Polypharmacy
- Preterm birth
- Male sex

Clinical Features and Assessment: Finnegan Neonatal Abstinence Scoring System

Onset:

- Short-acting opioids (heroin): 24–72 hours
- Methadone: 48 hours to 5–7 days
- Buprenorphine: 12–48 hours (shorter, milder)

Finnegan categories:

CATEGORY · SIGNS

CNS High-pitched cry, sleep <3 hours, sleep <2 hours, hyperactive Moro, tremors, increased muscle tone, excoriation, seizures

Metabolic/Vasomotor Sweating, fever, mottling, nasal stuffiness, frequent yawning, sneezing

GI Poor feeding, regurgitation, projectile vomiting, loose stools, watery stools

Respiratory Nasal flaring, respiratory rate >60

Scoring: Each sign scored 1–3 depending on severity.

- Score ≥ 8 on two consecutive assessments 4 hours apart → pharmacological treatment indicated

Management

Non-pharmacological (first-line for mild NOWS):

- Skin-to-skin care (kangaroo care)
- Breastfeeding (if mother on OST, not HIV+, no other drug use): decreases NOWS severity and length of stay
- Quiet, low-stimulation environment
- Swaddling, rocking
- Small, frequent feeds (caloric needs increased)

Pharmacological (Finnegan score $\geq 8 \times 2$ consecutive):

DRUG	ROUTE	DETAILS
Oral morphine	PO	0.03–0.1 mg/kg/dose Q3–4H; titrate to Finnegan <8; first-line in USA
Methadone	PO	0.05–0.1 mg/kg Q12H; longer half-life; less frequent dosing
Buprenorphine (sublingual)	SL	Emerging evidence; shorter treatment courses

DRUG	ROUTE	DETAILS
Phenobarbital	IV/PO	Adjunct; for seizures or inadequate response to opioids
Clonidine	PO/IV	Alpha-2 agonist; reduces autonomic symptoms; adjunct

Weaning: Once stable (Finnegan <8), reduce morphine by 10–20% every 24–48 hours.

Prognosis and Long-Term Outcomes

- NOWS itself: usually resolves within weeks; longer with long-acting opioids
- Long-term: cognitive, behavioral, and developmental concerns
- ADHD, learning difficulties in opioid-exposed children
- Confounded by: socioeconomic factors, maternal mental illness, adverse childhood experiences

Maternal OST and NOWS

- NOWS is expected and manageable in methadone/buprenorphine-exposed neonates
- NOWS from OST is preferred over risks of untreated maternal addiction (preterm birth, fetal growth restriction, overdose)
- Buprenorphine-exposed neonates: shorter, milder NOWS than methadone-exposed (clinical advantage)
- Mothers on OST should NOT be encouraged to taper or stop during pregnancy

CLINICAL ANCHOR

Telling a pregnant woman on buprenorphine to stop her medication to protect the baby is wrong and dangerous. Undertreated maternal opioid dependence is far more harmful to the fetus than NOWS.

EXAM STRATEGY

NOWS questions: Finnegan scale (categories), pharmacological treatment (oral morphine first-line), buprenorphine vs methadone NOWS comparison, non-pharmacological care, and the maternal OST point. These are the 5 examiner checkpoints.

Q13. Discuss the NDPS Act provisions relevant to clinical practice. [10 marks]

Answer:

Introduction

The Narcotic Drugs and Psychotropic Substances (NDPS) Act, 1985 is India's primary legislation governing narcotic and psychotropic substances. For the psychiatrist, knowledge of the Act is clinically relevant: it shapes how we prescribe controlled substances, advise patients in legal trouble, advocate for treatment over punishment, and access essential medicines.

Background and Purpose

- Enacted to fulfil India's obligations under UN Conventions on Narcotic Drugs (1961), Psychotropic Substances (1971), and Illicit Traffic (1988)
- Replaced Opium Act 1857, Dangerous Drugs Act 1930, Prevention of Illicit Traffic in Narcotic Drugs Act
- Regulates manufacture, sale, purchase, transport, storage, and use of narcotic drugs and psychotropic substances

Key Definitions

TERM · DEFINITION

Narcotic drug Coca leaf and cocaine; cannabis (hemp) and resin; opium, poppy straw, and their derivatives

Psychotropic substance Substances listed in Schedule I–IV of the Convention on Psychotropic Substances 1971

Illicit traffic Cultivation, production, manufacture, transport without legal authorization

Schedules Relevant to Psychiatry

SCHEDULE · CATEGORY

Schedule I Completely prohibited narcotics: heroin, LSD, MDMA, methamphetamine

Schedule II (Essential Medicines Amendment 2015) Opioids for medical use: morphine, methadone, fentanyl, buprenorphine, codeine; accessible under specific conditions

Psychotropic substances Benzodiazepines, barbiturates, amphetamines (Schedule II–IV)

Quantity Thresholds

SUBSTANCE	SMALL QUANTITY	LARGE QUANTITY
Heroin	5 grams	250 grams
Cocaine	2 grams	100 grams
Morphine	5 grams	250 grams

SUBSTANCE	SMALL QUANTITY	LARGE QUANTITY
Cannabis (charas)	100 grams	1 kg
Cannabis (ganja)	1 kg	20 kg
Opium	25 grams	2.5 kg
Methamphetamine	2 grams	50 grams

Penalties:

- Small quantity: rigorous imprisonment up to 1 year + fine, or both
- Intermediate quantity: 1–10 years + fine
- Large quantity: 10–20 years + fine; repeat offence up to 30 years / death in exceptional cases

Provisions Relevant to Treatment

Section 71, Power to establish centres:

"The Government may, in consultation with local authority, establish centres for identification, treatment, education, and rehabilitation of addicts."

This section authorizes the government to refer drug users to treatment instead of the criminal justice pathway. Courts have wide discretion.

Section 76, Probation:

Court may release a convicted person on probation with condition of undergoing treatment for drug dependence.

Section 64-A (Amendment 2001), Immunity from prosecution:

A person who voluntarily seeks treatment for drug dependence is immune from prosecution for offences relating to personal use (small quantity). This is the most important provision for psychiatrists: encourage patients to seek treatment; they are protected.

EXAM PEARL

Section 64-A is the key provision protecting patients: voluntary treatment-seeking = immunity from prosecution. This removes a significant barrier to help-seeking.

The 2014 Amendment: Essential Medicines Access

Major reform: removed barriers to prescribing opioids for pain and OST by:

- Simplifying procurement procedures for state governments
- Allowing hospitals to stock morphine without repeated licensing
- Enabling palliative care access

- Supporting OST programs

Challenges in Clinical Practice

CHALLENGE · IMPACT

Overly punitive approach to users Criminalization discourages help-seeking

Bail restrictions (Section 37) Pre-trial detention for NDPS arrests; persons with SUD imprisoned before treatment

Stigma from criminalization Patients fear disclosing drug use to medical professionals

Opioid phobia Even after 2014 amendment, many states have barriers to essential opioid medicines

Unregulated rehabilitation Private de-addiction centres often unregulated; human rights concerns

Psychiatrist's Role

- Maintain accurate medical documentation (substance use as illness, not moral failure)
- Advocate for treatment diversion via Section 64-A / Section 71
- Support OST programs, prescribing within legal framework
- Report suspected child abuse when NOWS/ parental addiction presents
- Participate in medico-legal process: provide clinical reports on addiction as disease

EXAM STRATEGY

NDPS Act questions reward: Section numbers (64-A, 71, 76), quantity thresholds for key drugs, the 2014 Essential Medicines amendment, and the clinical advocacy angle. Do NOT simply list penalties, examiners want clinical and public health relevance.

Mnemonics & Memory Tricks

KEY INSIGHT

Sources: Kaplan & Sadock, Stahl's, Oxford Textbook of Psychiatry, WHO Guidelines

EXAM STRATEGY

Mnemonics are retrieval hooks, they only work if you've attached meaning to each letter. Read each mnemonic alongside its full expansion. Then test yourself by covering the expansion and reconstructing it cold. Do this 3 times over 3 days.

1. Delirium Tremens Features

MNEMONIC

DREAD V

- **D**, Diaphoresis (profuse sweating) - **R**, Restlessness / agitation - **E**, Encephalopathy (confusion, disorientation) - **A**, Autonomic instability (tachycardia, hypertension, hyperthermia) - **D**, Delusions / hallucinations (visual > auditory; Lilliputian) - **V**, Vivid tremor (coarse, persistent)

EXAM PEARL

Lilliputian hallucinations (tiny people/ animals) = pathognomonic of DTs. Visual hallucinations in DTs are complex and terrifying, microanimals, insects, attacking figures.

2. CAGE Questionnaire

MNEMONIC

MNEMONIC: "CAGE", self-explanatory but expand it fully - **C**, Have you ever felt you should **Cut down** on your drinking? - **A**, Have people **Annoyed** you by criticizing your drinking? - **G**, Have you ever felt bad or **Guilty** about your drinking? - **E**, Have you ever had a drink first thing in the morning (**Eye-opener**) to steady your nerves or get rid of a hangover?

EXAM PEARL

Score ≥ 2 is clinically significant. Sensitivity ~74–85%, specificity ~79–91% for alcohol dependence. Best for detecting established dependence; poor for hazardous use. Use AUDIT for broader screening.

3. Wernicke's Encephalopathy Triad

MNEMONIC

MNEMONIC: "AOC", "Altered O Clock" - **A**, Ataxia (cerebellar; gait disturbance) - **O**, Ophthalmoplegia (lateral rectus palsy most common; nystagmus) - **C**, Confusion / encephalopathy (global; clouded sensorium)

EXAM PEARL

Only 10–16% of autopsy-confirmed cases show the full triad. Never wait for all three. Thiamine first, before IV glucose, in ANY alcohol-dependent patient with neurological change.

Alternative: "WOW", Wernicke's = Ophthalmoplegia + Wobbling (ataxia) + Woozy (confusion)

4. Alcohol Withdrawal Timeline: "6-12-24-48-72"

MNEMONIC

Six Angry Seizures Drinking Tremens

- **6** hours, Symptoms start (anxiety, tremor, tachycardia, diaphoresis) - **12** hours, Alcoholic hallucinosis (auditory hallucinations, clear sensorium) - **24** hours, Seizures peak (GTCS, self-limited, ~3–5%) - **48** hours, Delirium Tremens onset - **72** hours, DTs peak / most dangerous

CLINICAL ANCHOR

"6-12-24-48-72" is the most important timeline in addiction psychiatry. In a GTCS patient with alcohol use, immediately think: was their last drink ~24 hours ago?

5. Opioid Withdrawal Signs

MNEMONIC

DAMPS COIL

- **D**, Diarrhoea - **A**, Anxiety / agitation - **M**, Mydriasis (dilated pupils) - **P**, Piloerection ("gooseflesh") - **S**, Sweating - **C**, Cramps (abdominal; muscle aches) - **O**, Only dysphoric, not life-threatening (in healthy adults) - **I**, Insomnia - **L**, Lacrimation / rhinorrhoea (runny nose, watery eyes)

EXAM PEARL

Opioid withdrawal = miosis in intoxication, MYDRIASIS in withdrawal. This reversal is a high-yield exam fact. COWS (Clinical Opiate Withdrawal Scale) scores mydriasis as a key item.

6. Stages of Change: Prochaska & DiClemente

MNEMONIC

Please Come Prepare Actions, Maintaining Recovery

- Pre-contemplation - Contemplation - Preparation - Action - Maintenance - Relapse (part of the cycle)

KEY INSIGHT

Alternative, "PPCAMR": "People Can Prepare And Maintain Recovery"

EXAM PEARL

Most patients present for treatment in Precontemplation or Contemplation, NOT action. Matching the intervention to the stage is the core clinical skill. Confronting a precontemplator about their "denial" is explicitly contraindicated in MI.

7. FRAMES: Brief Intervention Components

MNEMONIC

MNEMONIC: "FRAMES", the frame that holds brief interventions together - **F**, Feedback (personalised, based on assessment results) - **R**, Responsibility (emphasise personal responsibility for change) - **A**, Advice (clear, direct, non-judgmental) - **M**, Menu (range of options offered) - **E**, Empathy (warm, reflective counselling style) - **S**, Self-efficacy (build confidence that change is possible)

EXAM PEARL

FRAMES is not a step-by-step protocol, it is a description of the active ingredients of effective brief interventions. Not all components need to appear in fixed sequence.

8. Disulfiram Reaction Features

MNEMONIC

FLUSH PH

- **F**, Flushing (facial, neck) - **L**, Light-headedness / dizziness - **U**, Urge to vomit / nausea - **S**, Sweating - **H**, Headache (pulsating, throbbing) - **P**, Palpitations / tachycardia - **H**, Hypotension (severe cases → cardiovascular collapse)

CLINICAL ANCHOR

Disulfiram-ethanol reaction (DER) is caused by acetaldehyde accumulation (ALDH inhibition). Can also occur with metronidazole, isoniazid, cephalosporins. Severity correlates with acetaldehyde level. Treatment: supportive, IV fluids, H1 + H2 antihistamines, vitamin C.

9. Fetal Alcohol Spectrum: Facial Features

MNEMONIC

3 Ps of FAS Face

- **P**hiltrum, smooth (no groove) - **P**alpebral fissures, short - **P**out (upper lip), thin vermilion border

KEY INSIGHT

Alternative, "STS": - Smooth philtrum - Thin upper lip - Short palpebral fissures

EXAM PEARL

FAS is the most common preventable cause of intellectual disability. No safe level of alcohol in pregnancy. Full FAS = dysmorphic facies + growth restriction + CNS abnormalities.

10. Opioid Receptors: "MKD"

MNEMONIC

Morphine Kills Delta

(Mu, Kappa, Delta) - **Mu** (μ), Morphine's primary receptor; euphoria, analgesia, respiratory depression, dependence - **Kappa** (κ), Kappa = "K"raziness: dysphoria, sedation, analgesia (no dependence) - **Delta** (δ), Delta = mood modulation, analgesia; minor role in dependence

EXAM PEARL

All opioid receptors are Gi/o-coupled GPCRs $\rightarrow$ $\downarrow$ cAMP, $\uparrow$ K⁺ conductance, $\downarrow$ Ca²⁺ conductance $\rightarrow$ neuronal inhibition. Respiratory depression is MOR-mediated $\rightarrow$ naloxone reverses by MOR antagonism.

11. Naltrexone: Key Clinical Facts

MNEMONIC

NO MORE OUD

- Naltrexone - Opioid (μ) antagonist, competitive - **M**ust ensure opioid-free $\geq 7-10$ days before starting (precipitates withdrawal otherwise) - **O**ral (50 mg/day) or monthly injection (380 mg IM = Vivitrol) - **R**educes craving and alcohol euphoria (for AUD too) - **E**xcluded if: active opioid use, hepatic failure

12. Acamprosate: Quick Memory

MNEMONIC

GABA-A-CAMP

(acamprosate = GABA-A enhancer + NMDA antagonist) - **A**, Acamprosate - **C**, Controls protracted withdrawal symptoms - **A**, After detox: start post-detox (not during active drinking) - **M**, Mechanism: GABA-A agonism + NMDA antagonism - **P**, Post-detox abstinence is its best indication - "CAMP" sounds like "acamP", reinforces the name

EXAM PEARL

Acamprosate is renally excreted, contraindicated in severe renal failure ($\text{CrCl} < 30$). Dose: 666 mg TID (=1998 mg/day). Safe in liver disease (advantage over disulfiram and naltrexone).

13. Buprenorphine Pharmacology: "PARTIAL CEILING"

MNEMONIC

PARTIAL CEILING

, describes buprenorphine's key properties - **Partial MOR agonist** - **Affinity**: very high (displaces full agonists) - **Respiratory depression**: ceiling effect (safer in overdose) - **Taper** must be slow; long half-life (24–72 hours) - **Induction**: patient must be in withdrawal (COWS ≥ 8), otherwise precipitated withdrawal - **Antagonism at KOR** (κ) $\rightarrow$ reduces dysphoria - **L**, Long duration allows once-daily or alternate-day dosing

EXAM PEARL

Precipitated withdrawal happens because buprenorphine has **HIGHER** receptor affinity than most full agonists. If full agonist still occupying receptors, buprenorphine kicks it off $\rightarrow$ withdrawal. Wait for COWS ≥ 8 –12 before first dose.

14. Korsakoff's Syndrome Features

MNEMONIC

CART

- **C**, Confabulation (unconscious memory fabrication) - **A**, Anterograde amnesia (new learning severely impaired, primary deficit) - **R**, Retrograde amnesia (recent > remote; temporal gradient) - **T**, Thiamine deficiency (and therefore: dorsomedial thalamus + mammillary bodies)

CLINICAL ANCHOR

Korsakoff's patients **CAN** register new information briefly (unlike Alzheimer's) but cannot consolidate it. Ask the patient who just told you their name, in 5 minutes, they cannot recall meeting you. This is anterograde amnesia.

15. Nicotine Dependence Treatment Ladder

MNEMONIC

V > B > NRT

(Varenicline > Bupropion > NRT) - **Varenicline** (Champix), triples quit rates vs placebo; best single agent; partial $\alpha 4\beta 2$ nAChR agonist - **Bupropion SR**, doubles quit rates; DA/NE reuptake blocker + nAChR antagonist; also antidepressant - **Nicotine Replacement Therapy**, doubles quit rates; patch + short-acting NRT = best NRT

EXAM PEARL

Combination NRT (long-acting patch + short-acting PRN gum/lozenge) is more effective than single NRT formulation. Varenicline + NRT shows additive benefit in some trials.

16. Change Talk: DARN CAT

MNEMONIC

DARN CAT

, change talk types - **D**esire: "I want to..." - **A**bility: "I could..." - **R**eason: "Because..." - **N**eed: "I have to..." - **C**ommitment: "I will..." - **A**ctivation: "I am ready to..." - **T**aking steps: "I've already started..."

EXAM PEARL

DARN = preparatory change talk. CAT = mobilising change talk. Commitment language (CAT) is the strongest predictor of actual behaviour change. The MI therapist's job is to selectively reinforce and elaborate change talk, especially CAT.

17. CIWA-Ar Items: "TATPHAAVO"

MNEMONIC

TATPHAAVO

(10 items) - **T**, Tremor - **A**, Anxiety - **T**, Tactile disturbances - **P**, Paroxysmal sweats - **H**, Headache / fullness in head - **A**, Auditory disturbances - **A**, Agitation - **V**, Visual disturbances - **O**, Orientation / sensorium clouding (*plus Nausea/Vomiting = 10th item, "N" can precede TATPHAAVO*)

EXAM PEARL

CIWA-Ar maximum score = 67. DTs-level severity = >15–20. Orientation item has maximum score of 4 (not 7 like others), ask for date, day, month, year. Only sensorium item distinguishes mild withdrawal from DTs.

18. DSM-5 SUD Criteria: 4 Domains

MNEMONIC

MNEMONIC: "ICSRP", "I Can't Stop Risky Pharmacology" - Impaired Control (4 criteria: more/longer than intended, failed efforts to cut down, much time, craving) - Continued use despite Social impairment (3 criteria: failed obligations, interpersonal problems, activities given up) - Risky use (2 criteria: physically hazardous use; use despite consequences) - Pharmacological (2 criteria: tolerance; withdrawal)

EXAM PEARL

11 criteria total. 2–3 = mild; 4–5 = moderate; 6+ = severe. DSM-5 removed "legal problems" and ADDED "craving" vs DSM-IV. Tolerance and withdrawal do NOT count if occurring in the context of prescribed use.

19. Alcohol Metabolism: Sequential Steps

MNEMONIC

MNEMONIC: "EAA", Ethanol → Acetaldehyde → Acetate - Ethanol + ADH (alcohol dehydrogenase) → Acetaldehyde - Acetaldehyde + ALDH (aldehyde dehydrogenase) → Acetate - Acetate → CO₂ + H₂O Disulfiram blocks the second arrow (ALDH) → acetaldehyde accumulates → DER

EXAM PEARL

Zero-order kinetics: alcohol is metabolised at ~10 mL/hour (absolute alcohol) regardless of concentration. This is why doubling alcohol intake doubles BAC, the metabolism enzyme is saturated.

20. Opioid Overdose Triad

MNEMONIC

MRS OD

, the three signs of opioid overdose - **M**iosis (pinpoint pupils) - **R**espiratory depression (the killer) - **S**ensorium depressed (unconscious/stupor) - **OD**, overdose → **Naloxone** (0.4–2 mg IV/IM/intranasal; repeat every 2–3 min)

CLINICAL ANCHOR

In the street or emergency setting: unconscious person + pinpoint pupils + slow breathing = opioid overdose until proven otherwise. Give naloxone, it does not harm non-opioid OD patients significantly. This is the empirical treatment rule.

High-Yield Comparisons

KEY INSIGHT

Sources: Kaplan & Sadock, Stahl’s, Oxford Textbook of Psychiatry, WHO Guidelines, ICD-11, DSM-5-TR

EXAM STRATEGY

Comparison tables are the highest-yield format for long-answer endings and short-answer cores. Know at minimum: mechanism, onset, key features, and management for every comparison pair. When writing answers, always include at least one comparison table, examiners reward structured differentiation.

TABLE 1: Alcohol Withdrawal vs Delirium Tremens vs Alcoholic Hallucinosi

FEATURE	UNCOMPLICATED ALCOHOL WITHDRAWAL	DELIRIUM TREMENS	ALCOHOLIC HALLUCINOSIS
Onset after last drink	6–12 hours	48–72 hours	12–24 hours
Peak	24–48 hours	48–72 hours	24–48 hours
Consciousness	Clear	Clouded (delirium)	Clear
Hallucinations	Absent or mild	Visual > auditory; complex, vivid	Auditory (voices) >> visual
Orientation	Intact	Disoriented	Intact
Autonomic instability	Mild–moderate (tremor, tachycardia, diaphoresis)	Severe (hyperthermia, profuse diaphoresis, tachycardia >120, hypertension)	Minimal or absent
Tremor	Present	Coarse, severe	Variable
Mortality	Very low	15–35% untreated; <1% treated	Very low
Seizures	May precede (24–36h)	May co-occur	Rare
Hallucination content		Miniature figures (Lilliputian), insects, terrifying animals	Threatening voices, accusatory voices, commentary

FEATURE	UNCOMPLICATED ALCOHOL WITHDRAWAL	DELIRIUM TREMENS	ALCOHOLIC HALLUCINOSIS
Patient insight	Preserved	Absent	Preserved (patient frightened but knows hallucinations are abnormal)
Primary treatment	Oral benzodiazepines (CIWA-Ar guided)	IV diazepam/lorazepam; ICU; thiamine	Antipsychotics (haloperidol); BZD for co-occurring withdrawal
ICD-11 code	6C40.5 (withdrawal)	6C40.6 (withdrawal with delirium)	6C40.70 (substance-induced psychosis)

EXAM PEARL

Alcoholic hallucinosis = auditory hallucinations + clear consciousness + no autonomic storm.
DTs = all three. This distinction is the most common exam trap in alcohol disorders.

CLINICAL ANCHOR

A patient with vivid visual hallucinations of insects crawling on the walls, confused and disoriented with HR 130 and temperature 38.5°C = DTs. Same patient with clear consciousness, hearing threatening voices, oriented × 3 = alcoholic hallucinosis. Treatment differs: ICU-level BZD titration for DTs vs antipsychotic for hallucinosis.

TABLE 2: Disulfiram vs Naltrexone vs Acamprosate

FEATURE	DISULFIRAM	NALTREXONE	ACAMPROSATE
Drug class	Aldehyde dehydrogenase inhibitor	Opioid mu-receptor antagonist	GABA-A agonist / NMDA antagonist
Mechanism	Blocks alcohol metabolism → acetaldehyde accumulation → aversive reaction	Blocks opioid-mediated euphoria of alcohol; reduces craving	Reduces glutamatergic hyperexcitability of protracted withdrawal
Standard dose	250–500 mg/day oral	50 mg/day oral OR 380 mg IM monthly	666 mg TID (1998 mg/day)
Start timing	≥24 hours abstinent; some say ≥48 hours	Can start while still drinking	After completion of detox
Goal	Complete abstinence (deterrence)	Reduce heavy drinking / craving	Maintain abstinence; anti-craving

FEATURE	DISULFIRAM	NALTREXONE	ACAMPROSATE
Effect if alcohol consumed	Disulfiram-ethanol reaction (DER), dangerous	No adverse effect	No adverse effect
Primary evidence	Moderate; RCTs favour supervised disulfiram	Strong (NNT ~7); COMBINE trial	Strong; best in European abstinence-focused trials
Hepatotoxicity	Yes (rare; check LFTs at baseline + 3 months)	Yes at supratherapeutic doses; check LFTs	Minimal
Renal considerations	Hepatic metabolism; renal excretion	Hepatic; no dose adjustment needed	Renally excreted; contraindicated CrCl <30
Contraindications	Psychosis, CV disease, hepatic failure, pregnancy, severe pulmonary disease, peripheral neuropathy	Active opioid use or need, liver failure	Severe renal failure
Key drug interaction	Metronidazole, isoniazid, cephalosporins (also cause DER); warfarin (↑ INR)	Opioid analgesics (blocks effect); precipitates opioid withdrawal if not opioid-free	Few significant
Adherence challenge	Patient can simply stop taking it	Injection formulation improves adherence	TID dosing reduces adherence
Best clinical profile	Highly motivated; supervised ingestion; strong social support	Craving-driven relapse; continued heavy drinking at treatment start	Post-detox abstinence; anxiety / protracted withdrawal; liver disease
Available in India	Yes (generic)	Yes (oral; injectable less available)	Yes (generic 333 mg tablets)

EXAM PEARL

The key conceptual contrast: disulfiram = deterrence (what happens IF you drink); naltrexone = reward blockade (what you DON'T get when you drink); acamprosate = allostatic reset (reduces the internal drive to drink).

CLINICAL ANCHOR

Patient presents for relapse prevention after detox, liver cirrhosis, high anxiety, strong motivation. Choice: acamprosate (safe in liver disease, targets anxiety-driven craving). Patient with ongoing heavy drinking and cue-triggered craving: naltrexone. Patient with poor compliance who agrees to supervised ingestion daily: disulfiram.

TABLE 3: Methadone vs Buprenorphine in OST

FEATURE	METHADONE	BUPRENORPHINE (± NALOXONE)
Receptor profile	Full MOR agonist	Partial MOR agonist + KOR antagonist
Half-life	24–36 hours	24–72 hours (sublingual)
Bioavailability	Oral ~80%	Sublingual ~30–50%; IV / intranasal blocked by naloxone component
Starting dose	20–40 mg / day (never >30–40 mg day 1 without tolerance confirmation)	4–8 mg day 1 (MUST be in withdrawal: COWS ≥8–12)
Maintenance dose	60–120 mg / day (higher = bet- ter retention)	16–32 mg / day
Ceiling effect on respiratory depression	No, overdose risk proportional to dose	Yes, respiratory depression plateaus at ~24–32 mg
QTc prolongation	Yes, dose-dependent; ECG monitoring required	Minimal
Overdose risk	Significant; narrow therapeutic window at induction	Lower; preferred in high-risk patients
Diversion potential	High, liquid form, fully active orally	Lower, naloxone component deters injection misuse
Supervised consumption requirement	Daily (initially); take-home earned over months	Less restrictive; earlier take- home
Induction requirement	No withdrawal needed, start any time	Patient MUST be in mild-mod- erate withdrawal
Precipitated withdrawal risk	None	Yes, if started when opioid still present on receptors
In pregnancy	Gold standard (decades of safe- ty data); NOWS expected	Increasing evidence; NOWS typically shorter and milder
NOWS severity	Severe, prolonged	Milder, shorter
Appropriate for	Severe OUD; failed buprenor- phine; high tolerance	First-line for moderate-severe OUD
Regulatory access in India	NDPS Act Schedule II; clinic- based dispensation	DTC-based dispensation; NDPS Amendment 2014

EXAM PEARL

Buprenorphine precipitated withdrawal mechanism: its very HIGH receptor affinity (higher than most full agonists) + partial agonism = it displaces the full agonist but activates the receptor less → net withdrawal. This is why COWS ≥ 8 before induction is mandatory.

CLINICAL ANCHOR

A young patient starting OST for the first time, no prior failed treatment, using heroin daily, buprenorphine/naloxone is first-line. An older patient with >20 years heroin dependence, multiple failed buprenorphine attempts, methadone.

TABLE 4: ICD-11 vs DSM-5 Substance Use Classification

FEATURE	ICD-11	DSM-5
Overall structure	Separate categories: hazardous use, harmful use (single episode / pattern), dependence, intoxication, withdrawal, substance-induced disorders	Single spectrum disorder (SUD) with mild / moderate / severe specifiers; no separate harmful use category
Hazardous use	Included as a distinct ICD-11 category (risk without current harm)	Not included as a diagnosis
Harmful use	Two separate categories: single episode + pattern	Incorporated into the SUD spectrum
Dependence	Requires 3 of 7 features over unspecified period	6+ criteria of 11 = severe SUD (no separate dependence label)
Minimum diagnostic threshold	1 feature (harmful use) or 3 features (dependence)	2 of 11 criteria for any SUD diagnosis
Severity specifiers	Not used for dependence	Mild (2–3), moderate (4–5), severe (6+)
"Abuse" category	Abolished	Abolished, merged into SUD spectrum
Craving	Included (compulsion)	Explicit criterion (added in DSM-5)
Legal problems	Not a criterion	Removed in DSM-5 (was in DSM-IV abuse)
Gambling disorder	Separate category (disorders due to addictive behaviours)	Included in chapter on substance-related and addictive disorders
Gaming disorder	Included (ICD-11)	Under further study (DSM-5)

FEATURE	ICD-11	DSM-5
Coding	Substance-specific codes (6C40–6C4Z)	Substance-specific codes (F10–F19 equivalent)
Polysubstance	Code each substance separately	Code each substance separately
Physiological subtype	"With physiological features" specifier for dependence	Tolerance + withdrawal criteria included but no separate specifier

EXAM PEARL

ICD-11 is the system used in India clinically and medico-legally. DSM-5 is used in research and increasingly in clinical practice. Key ICD-11 advantage: includes hazardous use, allows intervention before harm occurs.

TABLE 5: Opioid Intoxication vs Opioid Withdrawal

FEATURE	OPIOID INTOXICATION	OPIOID WITHDRAWAL
Pupil size	Miosis (pinpoint)	Mydriasis (dilated)
Consciousness	Drowsy → coma	Anxious, alert, agitated
Respiratory	Depressed, slow	Increased rate; yawning
GI	Constipation; reduced motility	Diarrhoea, vomiting, cramps
Skin	Warm, flushed	Piloerection ("gooseflesh"); diaphoresis
Pulse	Bradycardia	Tachycardia
BP	Hypotension	Hypertension
Pain	Analgesia	Hyperalgesia, myalgias, bone pain
Mood	Euphoria → sedation	Dysphoria, anxiety, irritability
Temperature	Normal / mildly hypothermic	Low-grade fever
Nose/Eyes	Dry	Rhinorrhoea, lacrimation
Life threat	Yes, respiratory depression → death	Not life-threatening in healthy adults (exception: neonates)
Assessment scale		COWS (Clinical Opiate Withdrawal Scale)
Treatment	Naloxone (0.4–2 mg IV / IM / IN); supportive	Buprenorphine; clonidine; symptomatic

EXAM PEARL

The most important axis: pupils. Intoxication = miosis. Withdrawal = mydriasis. In the same patient, these signs REVERSE across the intoxication-withdrawal cycle, this is diagnostically reliable.

CLINICAL ANCHOR

COWS is a 11-item scale scoring withdrawal severity. Score 5–12 = mild; 13–24 = moderate; 25–36 = moderately severe; >36 = severe. A COWS ≥ 8 –12 is the threshold for safe buprenorphine induction.

TABLE 6: Cannabis-Induced Psychosis vs Primary Psychosis (Schizophrenia)

FEATURE	CANNABIS-INDUCED PSYCHOSIS	SCHIZOPHRENIA
Temporal link to cannabis	Within 1 month of use/intoxication	No temporal link
Onset	Acute, often rapid	Typically insidious (prodrome months-years)
Duration	Usually resolves within 1 month of abstinence	Chronic; persists without treatment
Positive symptoms	Paranoia, perceptual distortions, ideas of reference	Hallucinations (auditory > visual), delusions
Negative symptoms	Mild, transient	Prominent; flat affect, avolition, alogia
Disorganisation	Mild	Prominent
Consciousness	Relatively clear	Clear
Drug screen	THC positive	Often negative (unless comorbid SUD)
Insight	Variable; often retained	Often absent
Response to abstinence	Significant symptom resolution	No resolution with abstinence alone
Long-term conversion	25–50% convert to schizophrenia-spectrum disorder (5 years)	
Risk factors for conversion	Family history of psychosis, COMT Val/Val genotype, adolescent onset, daily high-potency THC use	Complex multifactorial

FEATURE	CANNABIS-INDUCED PSYCHOSIS	SCHIZOPHRENIA
Treatment	Abstinence + short-term antipsychotic	Long-term antipsychotic; psychosocial
Prognosis	Good with abstinence (if no conversion)	Variable; often chronic

EXAM PEARL

The 25–50% conversion rate from cannabis-induced psychosis to schizophrenia-spectrum disorders means it is NOT a benign condition. Long-term follow-up is mandatory.

CLINICAL ANCHOR

Diagnostic approach in first-episode psychosis with cannabis use: (1) screen for cannabis with urine drug screen; (2) enforce abstinence; (3) reassess at 4 weeks. If psychosis resolves → cannabis-induced. If persists → primary psychosis or comorbid. Document timeline meticulously, this is both clinical and medico-legal.

TABLE 7: Physical Dependence vs Psychological Dependence

FEATURE	PHYSICAL DEPENDENCE	PSYCHOLOGICAL DEPENDENCE
Definition	Neuroadaptation requiring continued substance use to maintain physiological normalcy	Emotional and cognitive reliance on a substance for pleasure, relief, or functioning
Markers	Tolerance + withdrawal syndrome	Craving, compulsive use, salience, loss of control
Mechanism	Receptor downregulation/ up-regulation; homeostatic adaptation	Mesolimbic dopamine system changes; cue-conditioned responses
Without the other?	Yes, opioids for pain (physical only)	Yes, cannabis (mainly psychological, mild physical)
Onset	Develops with sustained use	Develops quickly with rewarding substances
On cessation	Withdrawal symptoms, can be managed medically	Craving, emotional distress, requires psychosocial treatment
Clinical priority	Medically supervised detox	Psychotherapy, relapse prevention, MI
Substances showing both	Alcohol, opioids, benzodiazepines	All addictive substances

FEATURE	PHYSICAL DEPENDENCE	PSYCHOLOGICAL DEPENDENCE
ICD-11 / DSM-5 relevance	Tolerance + withdrawal = physiological features specifier (ICD-11) / criteria 10 & 11 (DSM-5)	Impaired control + craving + salience = criteria 1–4 (DSM-5)

EXAM PEARL

Physical dependence $\neq$ addiction. A patient taking morphine for cancer pain who develops tolerance and withdrawal is physically dependent but NOT addicted. Addiction = compulsive use despite harm + loss of control. This distinction has important medico-legal and prescribing implications.

TABLE 8: Benzodiazepine Withdrawal vs Alcohol Withdrawal

FEATURE	BENZODIAZEPINE WITHDRAWAL	ALCOHOL WITHDRAWAL
Mechanism	GABA-A downregulation + NMDA upregulation	Same pathophysiology
Life-threatening	Yes, seizures, delirium	Yes, seizures, DTs
Onset	Short-acting BZD: 24–48 hours; long-acting: 5–7 days	6–12 hours (minor); 48–72 hours (DTs)
Duration	Weeks to months (protracted in some)	Usually 5–7 days (acute)
Seizure risk	Yes, particularly abrupt cessation of short-acting BZD	Yes, peak at 24–48 hours
Delirium	Rare; possible in severe dependence	DTs, 3–5% of untreated cases
Perceptual hypersensitivity	Hallmark of BZD withdrawal (light, sound, touch)	Less prominent
Autonomic features	Present	Prominent (diaphoresis, tachycardia, hypertension)
Protracted withdrawal	Very common, anxiety, insomnia for months	Less common; some "post-acute withdrawal"
Taper speed	SLOW, 10% / week; months	Faster, 5–10 day detox sufficient
Preferred detox agent	Switch to long-acting BZD (diazepam) + slow taper	Diazepam or chlordiazepoxide (short course)
Adjuncts	CBT for anxiety; sleep hygiene	Thiamine; multivitamins; clonidine

FEATURE	BENZODIAZEPINE WITHDRAWAL	ALCOHOL WITHDRAWAL
CIWA equivalent	CIWA-B (Clinical Institute Withdrawal Assessment, Benzodiazepines)	CIWA-Ar

EXAM PEARL

BZD withdrawal taper is much SLOWER than alcohol detox. Patients who have been on therapeutic doses of BZD for years may need 6–12 month tapers. Rushing = risk of protracted withdrawal and kindling.

CLINICAL ANCHOR

In a patient on long-term prescribed clonazepam presenting with anxiety after stopping, do NOT dismiss as "just anxiety." BZD withdrawal can cause life-threatening seizures up to 2 weeks after the last dose for long-acting agents. Restart and taper.

TABLE 9: Fetal Alcohol Syndrome vs Other Causes of Intellectual Disability

FEATURE	FETAL ALCOHOL SYNDROME	DOWN SYNDROME	FRAGILE X	ADHD (MILD ID)
Cause	Prenatal alcohol exposure	Trisomy 21	FMR1 gene mutation (CGG repeat)	Multifactorial
Facial features	Smooth philtrum, thin upper lip, short palpebral fissures	Epicanthal folds, flat face, Brushfield spots	Long face, large ears, macroorchidism	None specific
Growth	Pre- and post-natal growth restriction	Normal-slightly short	Normal	Normal
CNS	Microcephaly; frontal lobe hypoplasia; ADHD; EF deficits	Moderate ID; Alzheimer's risk	Moderate-severe ID; ADHD; autism features	Mild; EF deficits
Preventable	Yes, entirely	No	No	Partially
Diagnosis	Clinical (exposure + features); no biomarker	Chromosomal karyotype	Molecular genetic testing	Clinical
Key IQ range	Variable; 40–80	40–55 (moderate)	40–55 (males)	Variable

EXAM PEARL

FAS is the most common preventable cause of intellectual disability globally. There is NO safe level of alcohol in pregnancy. The facial features triad (smooth philtrum + thin upper lip + short palpebral fissures) is diagnostic but absent in milder FASD forms (ARND, ARBD).

TABLE 10: Stimulant Intoxication vs Stimulant Withdrawal ("Crash")

FEATURE	COCAINE / AMPHETAMINE INTOXICATION	COCAINE / AMPHETAMINE WITHDRAWAL (CRASH)
Timing	During use	Hours to days after last use
Mood	Euphoria, grandiosity	Dysphoria, depression, anhedonia
Energy	Increased	Profound fatigue, lethargy
Sleep	Insomnia	Hypersomnia (sleeping 12–18 hours)
Appetite	Decreased	Hyperphagia (increased appetite)
Pupils	Dilated	Normal
Vital signs	Tachycardia, hypertension, hyperthermia	Normal or bradycardia
Psychosis	Paranoia, hallucinations at high doses	Generally absent; severe depression possible
Craving	High during use	Intense craving in early withdrawal
Life-threatening	Yes, MI, stroke, arrhythmia	No (but depression may → suicidality)
Treatment	Supportive; BZD for agitation; cooling	Supportive; antidepressants for severe depression; no approved pharmacotherapy

EXAM PEARL

Cocaine does NOT cause physiological withdrawal in the traditional sense, there is no tremor, seizure, or autonomic instability. The "withdrawal" is a psychological crash: depression, fatigue, craving. This is sometimes tested as a differentiating fact.

TABLE 11: AUDIT vs CAGE: Screening Tool Comparison

FEATURE	AUDIT	CAGE
Developed by	WHO (Babor 1989)	Ewing (1984)
Items	10	4
Time to complete	2–3 minutes	<1 minute
Quantitative data	Yes (frequency, quantity)	No
Domains	Hazardous use + dependence features + harmful consequences	Dependence/emotional features only
Detects hazardous use	Yes	Poor
Detects dependence	Yes	Yes
Scoring range	0–40	0–4
Positive threshold	≥8 (hazardous use)	≥2
Cultural validation	WHO; multi-country (including India)	Primarily Western
Best use	Systematic screening + intervention level guidance	Rapid clinical screen in any setting
Lifetime vs current	Current (last year)	Lifetime orientation
Sensitivity for AUD	~57–97% (threshold-dependent)	~74–85%
Specificity for AUD	~78–96%	~79–91%
AUDIT-C brief version	3 items; ≥4 (men) / ≥3 (women)	No abbreviated version
Appropriate for primary care	Yes, AUDIT-C especially	Yes

EXAM PEARL

AUDIT detects the full spectrum from hazardous use to dependence. CAGE is specifically designed for dependence detection. In systematic public health screening, AUDIT is preferred. In a quick clinical encounter, CAGE is sufficient.

PYQ Frequency Analysis

KEY INSIGHT

Analysis based on recurring patterns in Indian PG Psychiatry exit examination question banks (PG exams, TN-MGR, Exam pattern). No university-specific references included, applicable to all Indian PG psychiatry examinations.

SECTION 1: OVERALL TOPIC FREQUENCY MAP

The following analysis categorises SUD subtopics by their historical appearance frequency across Indian PG psychiatry written papers.

FREQUENCY TIER · TOPICS

Tier 1, Perennial (appears almost every exam cycle) Alcohol withdrawal management, Wernicke-Korsakoff syndrome, Motivational Interviewing, Stages of Change, AUDIT/CAGE

Tier 2, High frequency (1–2 per paper, rotating) Delirium Tremens (clinical features + management), Disulfiram vs Naltrexone vs Acamprosate, Opioid Substitution Therapy, Alcoholic Hallucinosis, CIWA-Ar

Tier 3, Rising (increasing frequency last 5 years) Cannabis and psychosis, NDPS Act provisions, Novel Psychoactive Substances, Dual Diagnosis, Neonatal Abstinence Syndrome

Tier 4, Occasional (every 2–3 cycles) Cocaine pharmacology and complications, Fetal Alcohol Spectrum Disorders, Buprenorphine pharmacology, Methamphetamine, Inhalant use disorder

Tier 5, Rare but targetable Pathological intoxication, Synthetic cannabinoids, Kratom, Pharmacogenomics in addiction, Reward deficiency syndrome

EXAM STRATEGY

If you have limited preparation time, Tier 1 and Tier 2 topics cover approximately 70–80% of SUD marks in any given paper. Master these first. Tier 3 topics are rising sharply, examiners are updating with current literature and policy.

SECTION 2: PERENNIAL FAVOURITES: DEEP ANALYSIS

2.1 Alcohol Withdrawal Management

Why it appears every cycle: Core clinical competency. The single topic where a psychiatry postgraduate is expected to demonstrate immediate practical skills.

What examiners test:

- Timeline of withdrawal (6-12-24-48-72 hours framework)
- CIWA-Ar: what it scores, thresholds, symptom-triggered vs fixed-dose
- Benzodiazepine choice and rationale (diazepam vs lorazepam vs oxazepam)
- Thiamine before glucose, rule must be explicitly stated
- DTs: recognition, ICU management
- Relapse prevention pharmacotherapy

Common mistakes that cost marks:

- Mentioning haloperidol as primary treatment for DTs (it is adjunct only, no anticonvulsant effect)
- Not mentioning CIWA-Ar
- Omitting thiamine supplementation protocol
- Confusing the withdrawal seizure timing with DTs onset

Likely question formats:

- "Discuss management of alcohol withdrawal syndrome" [10 marks]
- "Describe delirium tremens, features and management" [10 marks]
- "A 45-year-old male presents with tremors and confusion 48 hours after his last drink. Discuss diagnosis and management" [10 marks]

EXAM PEARL

In the management of DTs, explicitly state: (1) ICU/HDU admission, (2) IV diazepam titrated to sedation, (3) thiamine before glucose, (4) correct electrolytes, (5) treat precipitating cause. These five points appear in every model answer.

2.2 Wernicke-Korsakoff Syndrome

Why it appears every cycle: Bridging neurology + psychiatry + addiction. High clinical consequence if missed. Tested in both short and long answers.

What examiners test:

- Wernicke's triad (but note only 10–16% have full triad)
- Pathology (mammillary bodies, dorsomedial thalamus, periaqueductal grey)

- Korsakoff's features: anterograde amnesia, confabulation, insight
- The "thiamine before glucose" rule
- Treatment dosing (200–500 mg IV thiamine)
- Prognosis (25% good, 50% partial, 25% minimal)

Common mistakes:

- Stating the full triad is always present
- Confusing anterograde and retrograde amnesia directionality
- Not distinguishing Korsakoff's from Alzheimer's dementia

Likely question formats:

- "Discuss Wernicke-Korsakoff syndrome" [10 marks]
- "Describe the neuropsychiatric complications of alcohol use disorder" [10 marks, where WKS is expected as a major section]
- Short note: "Korsakoff's psychosis" [5 marks]

EXAM PEARL

Three facts that always earn marks: (1) full triad in only 10–16%, (2) thiamine before glucose, (3) mammillary bodies as the key pathological site.

2.3 Motivational Interviewing

Why it appears every cycle: MI is now part of standard psychiatry competency frameworks. Examiners see it as testable theory with direct clinical application.

What examiners test:

- MI Spirit (PACE acronym)
- Four processes (Engaging, Focusing, Evoking, Planning)
- OARS (Open questions, Affirmations, Reflective listening, Summaries)
- Change talk: DARN-CAT
- Ambivalence as central concept
- Evidence base (Project MATCH, Cochrane)

Common mistakes:

- Describing MI as "advice giving" (it is explicitly NOT directive advice-giving)
- Confusing MI with Brief Intervention (BI is advice-focused; MI is evocative)
- Not mentioning the Spirit, examiners specifically look for this

Likely question formats:

- "Describe motivational interviewing in the management of substance use disorders" [10 marks]
 - "Discuss psychosocial interventions in alcohol dependence" [10 marks, MI expected as main section]
 - Short note: "Stages of change and motivational interviewing" [5 marks]
-

2.4 Stages of Change (Transtheoretical Model)

Why it appears every cycle: Conceptually elegant, well-researched, directly applicable to every clinical encounter with a person who uses substances.

What examiners test:

- 6 stages (PC, C, Prep, Action, Maintenance, Relapse) with definitions
- Stage-matched interventions
- Decisional balance concept
- Spiral model of relapse
- Integration with MI

Likely question formats:

- "Describe the transtheoretical model of behaviour change" [10 marks]
 - "Discuss stages of change and their relevance in managing substance use disorders" [10 marks]
 - Short note: "Prochaska and DiClemente's model" [5 marks]
-

2.5 AUDIT and CAGE

Why it appears every cycle: Screening is a core public health and clinical competency. Examiners test both the tool content and the clinical decision-making that follows.

What examiners test:

- CAGE: all 4 questions verbatim (must be written out)
- AUDIT: 3 domains, scoring thresholds (8/15/20)
- AUDIT-C: 3 items, threshold
- Comparison of the two tools
- SBIRT framework (Screening-Brief Intervention-Referral to Treatment)

Likely question formats:

- "Describe screening instruments used in alcohol use disorder" [10 marks]
 - "Compare AUDIT and CAGE" [10 marks]
 - Short note: "CAGE questionnaire" [5 marks]
-

SECTION 3: HIGH-FREQUENCY ROTATING TOPICS

3.1 Disulfiram vs Naltrexone vs Acamprosate

Pattern: Appears as either a 10-mark long answer or a 5-mark "compare and contrast."

What to include:

- Mechanism (ALDH inhibition / MOR antagonism / GABA-NMDA modulation)
- Dose for each
- DER features for disulfiram
- Clinical profile (best for whom)
- Contraindications (especially: naltrexone contraindicated in opioid users)

EXAM PEARL

The comparison table format is almost always expected for this question. Draw it in your answer, examiners reward visual organisation.

3.2 Delirium Tremens

Pattern: Either as a standalone question or as part of "complications of alcohol withdrawal."

Must-include:

- Clinical features (5 Ds: Delirium, Diaphoresis, Dysautonomia, Dread, Dreams/hallucinations)
- Timing (48–72 hours)
- Mortality (untreated 15–35%; treated <1%)
- Management (IV diazepam, ICU, thiamine, treat precipitants)
- DTs vs alcoholic hallucinosis differentiation

3.3 Opioid Substitution Therapy

Pattern: Appears regularly as either "discuss OST" or "compare methadone and buprenorphine."

Must-include:

- Rationale (harm reduction framework)
- WHO essential medicines status
- Methadone (full agonist) vs buprenorphine (partial agonist + ceiling effect)
- Induction protocol for buprenorphine (COWS $\geq 8-12$)
- Psychosocial components
- Indian DTC context

3.4 Alcoholic Hallucinosi

Pattern: Frequently tested as a short note or as part of "differentials in psychosis."

Key differentiating features from DTs: Clear consciousness, auditory > visual, no autonomic instability, must be stated explicitly.

SECTION 4: RISING TOPICS: INCREASING EXAM FREQUENCY

4.1 Cannabis and Psychosis

Trend: Significant increase in frequency over last 3–5 years, driven by:

- Public policy debates around cannabis decriminalisation
- High-profile international research (Di Forti, EU-GEI study)
- Rising prevalence of cannabis use among Indian youth

What to master:

- Bradford Hill criteria for causality (especially temporality, dose-response, experiment)
- THC:CBD ratio and psychosis risk
- Cannabis-induced psychosis vs schizophrenia differentiation table
- 25–50% conversion to schizophrenia-spectrum figure
- Management: abstinence + antipsychotic + psychoeducation

EXAM PEARL

The EU-GEI study (Di Forti 2019) found daily high-potency cannabis use associated with OR 4.8 for psychosis. This specific figure is cited in recent exam answers.

4.2 NDPS Act Provisions

Trend: Rising sharply, reflects curriculum updates emphasising law, ethics, and policy in psychiatry.

What to master:

- Section 64-A (immunity for voluntary treatment seeking), most clinically relevant
- Section 71 (government treatment centres)
- Section 76 (probation to treatment)
- Small vs large quantity thresholds for heroin, cocaine, cannabis
- 2014 Amendment (essential medicines access)

EXAM PEARL

Section 64-A is the provision that directly affects your clinical practice, patients seeking treatment voluntarily are protected from prosecution. This is the key point examiners look for.

4.3 Dual Diagnosis

Trend: Increasing, reflects integration of addiction psychiatry with general psychiatry in practice.

What to master:

- Epidemiology (ECA study: 37% AUD with comorbid psychiatric disorder)
- Four pathways (self-medication, substance-induced, common factor, bidirectional)
- Sequential vs parallel vs integrated treatment models
- Integrated treatment as gold standard
- Clozapine in schizophrenia + cannabis use

4.4 Novel Psychoactive Substances

Trend: Included in recent curriculum updates. Examiners testing awareness of emerging substances.

What to master:

- Definition (designed to mimic controlled substances while evading scheduling)
- Categories: synthetic cannabinoids, synthetic cathinones, novel phenethylamines, novel opioids
- Synthetic cannabinoids: full CB1 agonists, more toxic than THC, not detected on standard screens
- Synthetic cathinones ("bath salts"): stimulant + psychosis
- Clinical approach: supportive management, no specific antidotes

4.5 Neonatal Abstinence Syndrome / NOWS

Trend: Rising with increased emphasis on perinatal psychiatry.

What to master:

- Finnegan Scale (categories: CNS, metabolic, GI, respiratory)
- Pharmacological treatment threshold (Finnegan ≥ 8 on two consecutive assessments)
- First-line treatment: oral morphine
- Buprenorphine-exposed NOWS vs methadone-exposed NOWS (buprenorphine = milder, shorter)

- Non-pharmacological: kangaroo care, breastfeeding, low stimulation

SECTION 5: QUESTION TYPE FREQUENCY BY FORMAT

QUESTION FORMAT · TOPICS MOST COMMONLY TESTED THIS WAY

10-mark long answer Alcohol withdrawal management, DTs, Wernicke-Korsakoff, Motivational Interviewing, OST, Disulfiram / Naltrexone / Acamprosate comparison

5-mark short note CAGE questionnaire, Korsakoff's psychosis, Alcoholic hallucinosis, CIWA-Ar, Stages of change, Naloxone, AUDIT

Clinical vignette + management Alcohol withdrawal, Opioid overdose, Cannabis-induced psychosis, Benzodiazepine dependence, Dual diagnosis

Compare and contrast DTs vs alcoholic hallucinosis, Disulfiram vs naltrexone vs acamprosate, Methadone vs buprenorphine, Physical vs psychological dependence

Describe + discuss Motivational Interviewing, NDPS Act, Brief interventions, Stages of change

SECTION 6: TOPIC PRIORITY MATRIX

PRIORITY	TOPIC	MARKS AVAILABLE PER PAPER	PREPARATION TIME RECOMMENDED
MUST KNOW, Tier 1	Alcohol withdrawal management	10–20	3 hours
MUST KNOW, Tier 1	Wernicke-Korsakoff	10	2 hours
MUST KNOW, Tier 1	Motivational Interviewing	10	2 hours
MUST KNOW, Tier 1	Stages of change	10	1 hour
MUST KNOW, Tier 1	AUDIT / CAGE	5–10	1 hour
HIGH, Tier 2	DTs clinical features + management	10	2 hours
HIGH, Tier 2	Disulfiram vs naltrexone vs acamprosate	10	1.5 hours
HIGH, Tier 2	Opioid substitution therapy	10	2 hours
HIGH, Tier 2	Alcoholic hallucinosis	5	45 min
HIGH, Tier 2	CIWA-Ar	5	30 min

PRIORITY	TOPIC	MARKS AVAILABLE PER PAPER	PREPARATION TIME RECOMMENDED
RISING, Tier 3	Cannabis and psychosis	10	1.5 hours
RISING, Tier 3	NDPS Act	10	1 hour
RISING, Tier 3	Dual diagnosis	10	1.5 hours
RISING, Tier 3	NOWS / NAS	5–10	1 hour
OCCASIONAL, Tier 4	FAS / FASD	5	30 min
OCCASIONAL, Tier 4	Cocaine complications	5	30 min
OCCASIONAL, Tier 4	Buprenorphine pharmacology	5	30 min

EXAM STRATEGY

Total recommended preparation time for SUD chapter: 24–28 hours. Spread across 3 weeks at 1.5 hours/session = 16–18 sessions. First 4 sessions: Tier 1. Sessions 5–10: Tier 2. Sessions 11–14: Tier 3. Sessions 15–16: Tiers 4–5 rapid review.

SECTION 7: HIGH-YIELD ANSWER-WRITING FRAMEWORK

The "DEMC" Answer Structure for Management Questions

Every management question should follow:

- Define, 2–3 sentences on the condition being managed
- Evaluate, assessment tools, severity grading, examination findings
- Manage, step-by-step pharmacological + non-pharmacological, complications
- Consolidate, relapse prevention, prognosis, follow-up

The "MAPS" Answer Structure for Comparison Questions

- Mechanism, compare mechanisms
- Application, clinical use, dosing, indication
- Problems, contraindications, side effects
- Summary table, always end with or include a comparison table

Writing Tables Under Exam Conditions

- Draw the table first (skeleton: 3 columns × 6–8 rows)
- Fill rows from memory (mechanism, dose, side effects, contraindications)
- Label rows and columns clearly

- Add a pearl box after the table

EXAM STRATEGY

Under time pressure, a well-drawn table counts as much as 3–4 paragraphs of prose. If you can draw the Methadone vs Buprenorphine table from memory in 3 minutes, you have secured 3–4 marks in a 10-mark answer.

SECTION 8: PREDICTED HIGH-PROBABILITY QUESTIONS: NEXT EXAM CYCLE

Based on topic rotation patterns and recent literature citations in Indian psychiatry curricula:

PREDICTED QUESTION · BASIS

"Discuss the role of cannabis in the aetiology of schizophrenia" Rising topic; EU-GEI data now in curricula

"Discuss integrated treatment for dual diagnosis" Increased emphasis in NMC curriculum

"Describe motivational interviewing in addiction management" Perennial; MI is now a clinical competency standard

"Discuss opioid substitution therapy with reference to buprenorphine and methadone" Perennial; Indian DTC context increasingly examinable

"Describe the NDPS Act provisions relevant to clinical practice" Policy emphasis rising

"A 28-year-old develops acute psychosis after cannabis use, discuss diagnosis and management" Clinical vignette format increasing

"Compare pharmacological agents used in alcohol relapse prevention" Disulfiram / naltrexone / acamprosate perennial comparison

"Discuss neonatal opioid withdrawal syndrome" Rising with perinatal psychiatry emphasis

SECTION 9: EXAMINER CHECKLIST: WHAT EARNS MARKS

Reconstructed from answer key patterns across multiple exam cycles:

Alcohol Withdrawal Management (10 marks)

- [] Timeline stated (6-12-24-48-72 framework)
- [] CIWA-Ar mentioned with scoring threshold
- [] Thiamine before glucose stated explicitly

- [] Benzodiazepine named with dose and route
- [] DTs differentiated from uncomplicated withdrawal
- [] Relapse prevention agents mentioned
- [] Psychosocial component mentioned

Motivational Interviewing (10 marks)

- [] MI Spirit (PACE) defined
- [] Four processes named
- [] OARS skills described
- [] Change talk (DARN-CAT) mentioned
- [] Ambivalence as central concept
- [] Evidence base (Project MATCH or Cochrane)
- [] Limitation or appropriate context

Wernicke-Korsakoff (10 marks)

- [] Thiamine deficiency mechanism explained
 - [] Wernicke's triad stated
 - [] "Only 10–16% have full triad" stated
 - [] Pathological sites: mammillary bodies, dorsomedial thalamus
 - [] Treatment: IV thiamine before glucose, dose stated
 - [] Korsakoff's: anterograde amnesia, confabulation, insight absent
 - [] Prognosis percentages given
-

Quick Review

KEY INSIGHT

Sources: Kaplan & Sadock, Stahl's, Oxford Textbook of Psychiatry, WHO Guidelines, ICD-11, DSM-5-TR All patient names are fictitious. All clinical details are for educational purposes only.

EXAM STRATEGY

Clinical vignettes test applied knowledge, diagnosis + justification + management in one package. Practice answering the questions before reading the answers. For each vignette: (1) identify the diagnostic clues in the case, (2) apply the relevant classification criteria, (3) structure management using the step framework from D1 and D2.

Vignette 1: The Morning Tremor

Case: Ramesh, a 48-year-old accountant, is brought to the emergency department by his wife at 8 AM. She reports he had his last drink at 10 PM the previous night. He appears sweaty, tremulous, and agitated. His heart rate is 112 bpm, BP 158/96 mmHg, temperature 37.6°C. He is oriented but anxious. He says he has been drinking 12–15 units of alcohol daily for the past 8 years. His CIWA-Ar score is assessed as 14.

Q1. What is the diagnosis? Justify using ICD-11 criteria.

Answer:

Ramesh has **Alcohol Withdrawal Syndrome** (ICD-11: 6C40.5, Alcohol withdrawal, uncomplicated).

Justification:

- Established pattern of heavy alcohol use (8 years, 12–15 units/day = physiological dependence)
- Recent cessation/reduction (last drink 10 hours ago)
- Classic withdrawal features within the expected 6–12 hour window: tremor, tachycardia (112), hypertension (158/96), diaphoresis, anxiety, agitation
- CIWA-Ar 14 = moderate severity (threshold 8–15)
- Consciousness clear, excludes delirium tremens at this stage

EXAM PEARL

ICD-11 alcohol dependence requires 3 of 7 features including tolerance (likely given 8 years, 12–15 units/day) and withdrawal (now presenting). The ICD-11 code for withdrawal is 6C40.5.

Q2. What is the CIWA-Ar score of 14 telling you, and how does it guide management?

Answer:

CIWA-Ar 14 = **moderate withdrawal severity**. This score indicates:

- Pharmacological treatment is required (threshold for treatment ≥ 8)
- Symptom-triggered benzodiazepine protocol appropriate
- Does not yet meet threshold for ICU/HDU (>20)
- Oral route likely adequate if patient can cooperate

Management based on CIWA-Ar 14:

1. Oral diazepam 20 mg now → reassess in 1 hour
2. If CIWA-Ar still ≥ 8 at 1 hour → repeat 10–20 mg
3. Thiamine 100–200 mg IV/IM before any IV glucose
4. Monitor vitals 2-hourly; CIWA-Ar 4-hourly
5. Target CIWA-Ar <8 before stepping down

Q3. What are the risk factors in this case for progression to delirium tremens?

Answer:

Risk factors in Ramesh's case:

- Long duration and high quantity of use (8 years, 12–15 units/day): chronic neuroadaptation
- Hypertension already at presentation: suggests significant autonomic activation
- Moderate CIWA-Ar at 10 hours: may worsen over next 24–48 hours

General DTs risk factors to mention in exam: prior DTs/seizures, age >65 , concurrent illness, benzodiazepine co-dependence, severe malnutrition, prolonged use.

CLINICAL ANCHOR

The most important predictor of DTs is a prior episode of DTs or withdrawal seizures. Always ask about previous withdrawal history. Ramesh's 8-year history without stated prior DTs is partially reassuring, but 48–72 hours is the danger window.

Vignette 2: The Terrifying Insects

Case: Priya, a 52-year-old retired teacher with a 20-year history of alcohol dependence, is admitted on Day 3 after her last drink. She is severely agitated, diaphoretic, and disoriented to time and place. She is screaming that insects are crawling over her body and that small men are trying to harm her. Her temperature is 39.2°C, HR 134 bpm, BP 172/104 mmHg. Her husband reports she had a witnessed seizure at home 12 hours ago before admission.

Q1. Identify the syndrome and list its diagnostic features.

Answer:

Priya has **Delirium Tremens** (ICD-11: 6C40.6, Alcohol withdrawal with delirium).

Diagnostic features present:

- **Delirium:** disorientation to time and place, grossly disturbed consciousness
- **Visual hallucinations:** insects on body (tactile + visual), Lilliputian figures ("small men")
- **Severe autonomic instability:** temperature 39.2°C (hyperthermia), HR 134 (tachycardia), BP 172/104 (hypertension), diaphoresis
- **Timing:** 72 hours post-last drink (DTs peak: 48–72 hours)
- **Prior seizure:** withdrawal seizure preceded DTs (as expected, seizures peak at 24–48h, DTs at 48–72h)

EXAM PEARL

Lilliputian hallucinations (miniature people / animals) are considered pathognomonic of DTs. Tactile + visual hallucinations together are characteristic. Auditory hallucinations are LESS prominent than in alcoholic hallucinosis.

Q2. Outline the immediate management priorities.

Answer:

PRIORITY · ACTION

- 1. Airway/Breathing** Assess airway, oxygen if SpO₂ <94%, crash trolley available
- 2. Thiamine FIRST** 200–500 mg IV thiamine before any IV glucose (prevents Wernicke's)
- 3. IV access + bloods** Glucose, Na, K, Mg, phosphate, LFTs, CBC, cultures if sepsis suspected
- 4. Benzodiazepines** IV diazepam 10–20 mg bolus; repeat every 5–15 minutes until calm; may need 100–200 mg in 24h
- 5. Treat hyperthermia** Paracetamol; cooling measures; blood cultures; broad-spectrum antibiotics if infection

6. Correct electrolytes IV magnesium sulphate 2–4 g (low Mg common; lowers seizure threshold)

7. Monitor Continuous cardiac monitoring; CIWA-Ar 2-hourly; glycaemic control

8. Haloperidol 5 mg IM for psychotic agitation, adjunct only, NOT primary treatment

9. ICU/HDU Transfer for ventilatory support if oversedated; continuous monitoring required

Q3. What is the prognosis and what determines it?

Answer:

- Untreated DTs mortality: 15–35%
- With treatment: <1%
- Most common cause of death: cardiovascular (arrhythmia, not seizure)

Poor prognostic factors:

- Concurrent infection / sepsis
- Severe hyperthermia >40°C
- Underlying liver failure (hepatic encephalopathy may compound)
- Delayed presentation (>72 hours of untreated DTs)
- Aspiration pneumonia
- Prior DTs (kindling)

CLINICAL ANCHOR

Priya's temperature of 39.2°C is a red flag. Hyperthermia in DTs indicates severe autonomic storm, possible concurrent infection, and significantly worsens prognosis. Aggressive cooling and infection workup are not optional.

Vignette 3: The Man Who Hears Voices but Knows It

Case: Suresh, a 41-year-old businessman, presents with his wife at 11 PM, 18 hours after his last drink. He is agitated but answers questions correctly, knows the date and where he is. He says he has been hearing two men's voices for the past 4 hours talking about him, calling him a thief and saying they will hurt him. He is visibly frightened. Vitals: HR 88, BP 130/82, afebrile. He has been drinking 8–10 units daily for the past 6 years.

Q1. What is the diagnosis? Distinguish it from delirium tremens.

Answer:

Suresh has **Alcoholic Hallucinosis** (ICD-11: 6C40.70, Substance-induced psychotic disorder).

Key diagnostic features:

- Onset 12–24 hours after last drink (hallucinosis window)
- **Clear consciousness:** oriented, answering correctly, knows date and place
- **Auditory hallucinations** (voices): threatening, accusatory, characteristic content
- **Intact sensorium:** no delirium
- **Mild/absent autonomic features:** HR 88, BP 130/82, afebrile

Distinction from DTs:

FEATURE	SURESH (HALLUCINOSIS)	DTs
Consciousness	Clear	Clouded
Hallucinations	Auditory >> visual	Visual > auditory
Orientation	Intact	Impaired
Autonomic storm	Absent	Severe
Timing	12–24 hours	48–72 hours
Mortality	Very low	Up to 35% untreated

Q2. What is the treatment and expected course?

Answer:

Treatment:

1. **Benzodiazepines:** for co-occurring withdrawal features (even mild), diazepam 10 mg oral + monitor
2. **Antipsychotics:** haloperidol 5 mg oral/IM → for psychotic symptoms (voices)
3. **Monitor:** CIWA-Ar to detect escalating withdrawal
4. **Safe environment:** low stimulation, orientation cues, family presence
5. **Thiamine:** oral thiamine 100 mg (prophylactic against Wernicke's)

Expected course:

- Most cases resolve within hours to days with abstinence and treatment
- Rare persistence beyond 2 weeks
- If persists >1 month after abstinence → reassess diagnosis → primary psychotic disorder?

Q3. What is the risk of progression to DTs in this patient?

Answer:

Moderate risk:

- He is only 18 hours post-last drink, DTs peak is 48–72 hours, still 30–54 hours away
- Current mild autonomic state is reassuring but can change
- Risk factors present: 6-year heavy use
- Monitor CIWA-Ar every 4 hours over the next 48 hours
- Escalation protocol: if CIWA-Ar rises >15, upgrade to IV route and consider HDU

Vignette 4: The Unconscious Young Man

Case: Vikram, a 24-year-old, is brought by friends to the emergency department unconscious. Friends report he "injected something" 30 minutes ago. On examination: deeply unconscious (GCS 6), respiratory rate 6 breaths/minute, pupils 2 mm bilaterally (pinpoint), cyanotic, HR 52, BP 90/60.

Q1. What is the most likely diagnosis and the diagnostic triad?

Answer:

Opioid overdose (ICD-11: 6C43.10, Opioid intoxication with overdose).

The diagnostic triad of opioid overdose:

1. **Miosis** (pinpoint pupils, 2 mm), pathognomonic when bilateral
2. **CNS depression** (GCS 6, stupor/coma)
3. **Respiratory depression** (RR 6, life-threatening; normal is 12–20)

Additional findings consistent: bradycardia, hypotension, cyanosis (from hypoxia).

EXAM PEARL

Any one of the triad may be absent, but the combination of unconsciousness + slow breathing + pinpoint pupils = opioid overdose until proven otherwise. IV naloxone should not be delayed pending laboratory confirmation.

Q2. Outline immediate emergency management.

Answer:

STEP · ACTION

A, Airway Position, jaw thrust; suction secretions; consider airway adjunct

B, Breathing Bag-valve-mask ventilation; oxygen 15 L/min; target SpO₂ >94%

C, Circulation IV access × 2; IV fluids for hypotension; cardiac monitoring

D, Naloxone 0.4–2 mg IV (or IM/intranasal if no IV access); repeat every 2–3 minutes

E, Evaluate Response to naloxone? Pupil dilation, increased RR, responsiveness

F, Further naloxone If partial response: 0.4–2 mg repeat; if opioid suspected but no response at 10 mg → reconsider diagnosis

G, Infusion If long-acting opioid suspected (fentanyl, methadone): naloxone infusion 2/3 of effective bolus dose per hour

H, Hospital admission All opioid OD patients admitted minimum 4 hours; longer if long-acting agent

CLINICAL ANCHOR

Naloxone half-life is 30–90 minutes, shorter than most opioids. Re-sedation ("renarcotisation") is a genuine risk, especially with long-acting opioids like methadone (half-life 24–36 hours) or fentanyl. Never discharge after one naloxone dose without extended monitoring.

Q3. Vikram recovers and is willing to talk. What is the next step in management?

Answer:

This is a "teachable moment", the overdose has increased his readiness to engage.

Immediate next steps:

1. **Motivational interview:** brief MI while the experience is fresh; assess stage of change
 2. **Psychoeducation on overdose risk:** tolerance reduction after any abstinence period (even hours); risks of resuming at previous dose
 3. **Take-home naloxone:** prescribe and train Vikram AND his friends on administration
 4. **Harm reduction:** if not ready for abstinence, sterile needles, wound care, BBV testing (HIV, HBsAg, HCV)
 5. **OST referral:** offer buprenorphine / naloxone induction, if COWS ≥ 8 –12, can begin same day
 6. **Social assessment:** housing, support, NDPS legal status
-

Vignette 5: The Student Who Stopped Making Sense

Case: Kaveri, a 19-year-old engineering student, is brought by her parents after a 3-week history of increasingly disorganised behaviour, social withdrawal, and talking about "being monitored by satellites." Her parents report she began smoking cannabis regularly 8 months ago and was using it daily for the last 3 months. Urine drug screen: THC positive. She denies alcohol or other drug use. Family history: paternal uncle has schizophrenia.

Q1. What are the two differential diagnoses and how would you approach them?

Answer:

The two primary differential diagnoses:

1. **Cannabis-induced psychotic disorder** (ICD-11: 6C41.70)
2. **Schizophrenia, first episode** (ICD-11: 6A20.0)

Diagnostic approach:

STEP · ACTION

Full psychiatric assessment Timeline: cannabis onset (8 months ago) vs psychosis onset (3 weeks ago); which came first?

Urine drug screen Confirm THC (done); screen for other substances

Physical examination + bloods FBC, TFTs, glucose, metabolic screen; exclude medical causes

Enforce abstinence Admit to drug-free environment

Reassess at 4 weeks If psychosis resolves → cannabis-induced. If persists → primary psychosis

EXAM PEARL

ICD-11 requires symptoms to "develop during or within 1 month after substance use" AND "resolve within 1 month of abstinence" for a substance-induced disorder diagnosis. If psychosis persists beyond 1 month of confirmed abstinence → reassess for schizophrenia.

Q2. What features increase the probability of conversion to a primary psychotic disorder?

Answer:

High-risk features for conversion to schizophrenia-spectrum disorder:

FACTOR · KAVERI'S PROFILE

Family history of schizophrenia YES, paternal uncle

Early age of cannabis onset YES, presumably adolescent

Duration before psychosis 8 months, significant cumulative exposure

High-frequency use YES, daily × 3 months

COMT Val158Met genotype Unknown (genetic testing not routine)

Subclinical psychosis features before cannabis Uncertain, requires longitudinal history

Kaveri has 3–4 identified risk factors → conversion probability elevated (~40–50% at 5 years).

Q3. What is the initial pharmacological and psychosocial management?

Answer:

Pharmacological:

- **Antipsychotic:** low-dose risperidone 1–2 mg/day or olanzapine 5 mg/day
- Duration: minimum 3–6 months; reassess at 6 months with confirmed abstinence
- If psychosis persists beyond 6 months of abstinence: long-term antipsychotic per first-episode schizophrenia guidelines

Psychosocial:

- **Psychoeducation:** cannabis-psychosis link, evidence-based, non-blaming communication to Kaveri and family
- **Motivational Interviewing:** she is likely in contemplation or preparation stage (the episode has created ambivalence)
- **CBT-p:** cognitive behavioural therapy for psychosis, addresses both residual symptoms and cannabis use
- **Family education:** high expressed emotion management; avoid blame; support abstinence without coercion

CLINICAL ANCHOR

Telling a young patient "cannabis definitely caused your psychosis" is both scientifically imprecise and therapeutically counterproductive. Better MI-aligned framing: "Cannabis significantly increases the risk, and the evidence suggests your brain is particularly sensitive to it. What do you make of that?"

Vignette 6: The Executive on Benzodiazepines

Case: Meena, a 44-year-old corporate executive, presents requesting a refill of clonazepam 2 mg at night, which she has been prescribed by her GP for insomnia for the past 4 years. She reports that if she misses even one dose, she gets "unbearable anxiety, heart racing, and can't sleep at all." She has tried stopping twice and once had a brief convulsion. She denies alcohol use. She wants to stop but says she "can't function without it."

Q1. What is the diagnosis? Apply ICD-11 criteria.

Answer:

Benzodiazepine dependence (ICD-11: 6C44.2, Dependence on sedative, hypnotic, or anxiolytic, clonazepam).

ICD-11 criteria met (3 of 7 required):

1. **Impaired control:** has tried to stop twice unsuccessfully ("can't function without it")
2. **Salience:** dependence on clonazepam for sleep and anxiety management
3. **Tolerance:** likely, 4 years of regular use; may need dose increases over time

4. **Withdrawal:** definite, anxiety, tachycardia, insomnia on missing dose; **convulsion on previous cessation** (severe withdrawal)
5. **Continued use despite harm:** psychological and likely occupational impairment

EXAM PEARL

Benzodiazepine dependence can develop at therapeutic doses within 4–6 weeks of regular use. This is iatrogenic dependence, the prescriber bears responsibility. Short-acting BZDs (alprazolam, lorazepam, triazolam) carry higher dependence risk than long-acting agents (clonazepam is intermediate; diazepam is long-acting).

Q2. Outline a safe detoxification plan.

Answer:

Clonazepam 2 mg / night = approximately diazepam 20 mg equivalent (clonazepam 0.5 mg $\approx$ diazepam 5 mg).

Step 1, Switch to diazepam equivalent:

- Convert to diazepam 20 mg nocte (or split 10 mg AM / 10 mg PM for comfort)
- Diazepam: long-acting, smooth plasma levels, fewer interdose symptoms

Step 2, Slow taper:

- Reduce by 10% every 1–2 weeks
- At 10 mg diazepam: slow to 5% per week
- Total duration: 6–12 months (this is NOT a 10-day alcohol detox equivalent)

Step 3, Monitor closely:

- Weekly review initially; fortnightly when stable
- CIWA-B (benzodiazepine withdrawal scale) or symptom diary
- Educate on withdrawal seizure risk, do NOT miss doses; do NOT abrupt stop

Step 4, Address underlying anxiety / insomnia:

- SSRI / SNRI for anxiety disorder (start during taper)
- Cognitive Behavioural Therapy for Insomnia (CBT-I)
- Sleep hygiene

Step 5, Given prior seizure:

- Slower taper rate; consider neurological consultation
- Seizure precautions during taper (no driving, no heights, supervision)

Q3. What psychosocial interventions support BZD discontinuation?

Answer:

- **CBT:** addresses the anxiety and catastrophic thinking that drives BZD use; most evidence-based
 - **CBT-I** (specifically for insomnia): stimulus control, sleep restriction, relaxation, as effective as BZD for insomnia long-term
 - **Psychoeducation:** explain physiological dependence $\neq$ addiction; reduce shame; explain the taper rationale
 - **Motivational Interviewing:** Meena is in preparation stage (she WANTS to stop but feels she can't), appropriate for planning intervention
 - **Mindfulness-based stress reduction (MBSR):** supports anxiety management without medication
-

Vignette 7: The Truck Driver

Case: Raju, a 35-year-old long-distance truck driver, is referred by occupational health after a routine health check. His AUDIT score is 18. He drinks every evening and on long trips uses 1–2 units during driving "to stay calm." He has never attempted to stop. He becomes defensive when the occupational health nurse mentions his drinking. His wife has complained about his drinking but he says "she's overreacting."

Q1. What stage of change is Raju in, and what intervention is appropriate?

Answer:

Raju is in **Precontemplation** (possibly early Contemplation).

Evidence for precontemplation:

- Denies problem ("she's overreacting")
- Never attempted to stop
- Defensive when drinking mentioned
- No expressed desire to change

However, his AUDIT of 18 (probable dependence range), occupational health referral, and wife's complaints suggest he is receiving multiple external signals, he may be early Contemplation.

Appropriate intervention: **Brief Motivational Interviewing** (not advice-giving; not confronting denial)

MI strategy for precontemplation:

1. **Avoid argument:** rolling with resistance, "It sounds like this is an area where you and your wife see things differently."
2. **Raise doubt gently:** "I'm wondering if it's okay to share with you what I'm noticing in your health results?"

3. **Decisional balance:** "What are the good things about drinking for you? What are the not-so-good things?"
4. **Importance ruler:** "On a scale of 0–10, how important is it to you right now to make a change? ... What would need to happen for that number to go up?"
5. **Affirm:** "It takes something to come in and have this conversation."

EXAM PEARL

The worst response to a precontemplator is confrontation ("You have a serious drinking problem and need to stop NOW"). Research consistently shows confrontation increases resistance, not motivation. MI is the evidence-based alternative.

Q2. Raju's AUDIT score is 18. What does this indicate and what screening + intervention pathway should follow?

Answer:

AUDIT 18 = Probable alcohol dependence (threshold ≥ 16 suggests probable dependence; ≥ 20 definite).

SBIRT pathway:

- **Screening:** done (AUDIT 18) → positive
- **Brief Intervention:** 5–15 minute structured session; FRAMES components; offered today
- **Referral to Treatment:** AUDIT > 15 = referral to specialist (addiction psychiatrist / de-addiction centre) recommended; offer actively today

Occupational context, additional concerns:

- Driving under influence (1–2 units while driving): legal and safety issue
- Fitness to drive: occupational health must formally assess
- Mandatory notification: occupational physician may have duty to notify licensing authority depending on jurisdiction
- NDPS Act: alcohol not under NDPS but road safety law applies

Q3. What brief intervention content is most appropriate for Raju?

Answer:

FRAMES-structured brief intervention (15 minutes):

COMPONENT · RAJU-SPECIFIC APPLICATION

Feedback "Your AUDIT score of 18 places you in the range where significant health problems are common. Your liver function tests show early elevation."

Responsibility "Only you can decide what to do with this information. I'm here to help if you want it."

Advice "Based on what I'm seeing, I'd recommend reducing your drinking to within safe limits, or stopping altogether. That's my medical recommendation."

Menu "There are several ways to tackle this: cutting down yourself, getting some support from a counsellor, or medications that reduce craving. What sounds interesting to you?"

Empathy "It sounds like things at home are under a lot of pressure. That's really hard."

Self-efficacy "You've clearly managed demanding situations as a driver, that requires real discipline. Those same skills work for this."

Vignette 8: The New Mother on Methadone

Case: Ananya, a 29-year-old, presents at 14 weeks of pregnancy. She discloses she has been using heroin IV daily for 3 years and is currently using 0.5 g/day. She is also HIV negative. She is frightened about what her drug use means for her baby. She asks if she should stop immediately to protect the foetus.

Q1. What is the appropriate response to her question about stopping?

Answer:

Ananya should **NOT** be advised to stop abruptly or rapidly. This is a critical clinical error.

Rationale:

- Abrupt opioid cessation in pregnancy → fetal withdrawal → fetal distress, preterm labour, spontaneous abortion risk (especially in first and second trimester)
- The foetus has developed opioid dependence in utero; abrupt withdrawal is hazardous to the foetus
- Maternal abstinence syndrome → maternal relapse → illicit opioid use → uncontrolled dosing, contaminants, needle sharing, all far more dangerous
- OST (methadone or buprenorphine) is the standard of care in pregnancy

CLINICAL ANCHOR

The right answer: "The safest thing for your baby is to start opioid substitution therapy (methadone or buprenorphine) now. Abrupt stopping is actually more dangerous. Your baby will be monitored for neonatal abstinence syndrome after delivery, which is manageable."

Q2. Which OST agent is preferred in pregnancy and why?

Answer:

FACTOR	METHADONE	BUPRENORPHINE
Evidence in pregnancy	Decades; gold standard	Increasing; MOTHER trial evidence
NOWS severity	Prolonged, moderate-severe	Shorter, milder (advantage)
Regulatory status	Standard of care	Increasingly accepted
Supervised consumption	Required	More flexible
Preferred in India	Most programmes offer methadone	Buprenorphine available at some DTCs

Both are acceptable. Methadone historically preferred; buprenorphine increasingly supported based on MOTHER trial (Jones 2010): buprenorphine-exposed neonates had significantly shorter NOWS duration and needed less morphine.

EXAM PEARL

The MOTHER trial (2010) is the landmark RCT comparing methadone vs buprenorphine in pregnancy. Key finding: buprenorphine = significantly milder NOWS. This trial is frequently cited in exam answers on perinatal opioid use.

Q3. Ananya is started on buprenorphine. Her baby is born at 38 weeks. The neonatologist calls you asking about neonatal management. What are the key points?

Answer:

Key neonatal management points:

1. **Expected NOWS:** buprenorphine-exposed neonates, onset 12–48 hours, typically milder than methadone
2. **Assessment:** Finnegan Neonatal Abstinence Scoring System (FNASS) every 4 hours
3. **Pharmacological threshold:** Finnegan ≥ 8 on two consecutive assessments → pharmacological treatment
4. **First-line treatment:** Oral morphine (0.03–0.1 mg/kg/dose every 3–4 hours, titrated to Finnegan < 8)
5. **Non-pharmacological:** Skin-to-skin, breastfeeding (Ananya is HIV negative, on stable OST, no other drug use → breastfeeding encouraged), low stimulation, swaddling
6. **Breastfeeding:** strongly encouraged, buprenorphine passes into breast milk in minimal amounts; breastfeeding significantly reduces NOWS severity and duration
7. **Discharge planning:** ensure Ananya has robust social support; postnatal depression screening; continued OST; community health worker link

Vignette 9: The Reluctant Abstainer

Case: Mohan, a 58-year-old retired civil servant, has completed alcohol detoxification 2 weeks ago. He is now in the relapse prevention phase. He is abstinent but does not feel well, he is anxious, not sleeping, and reports persistent low-grade craving. He says "I feel worse sober than I ever did drunk." He is asking for something to help.

Q1. What syndrome is he experiencing and what is its mechanism?

Answer:

Mohan is experiencing **Protracted Alcohol Withdrawal Syndrome** (also: Post-Acute Withdrawal Syndrome, PAWS).

Features consistent:

- Persistent anxiety, insomnia, and craving beyond the acute 5–7 day withdrawal window
- Anhedonia/dysphoria ("feel worse sober")
- Onset 2 weeks post-detox, consistent with protracted course

Mechanism: **Allostatic dysregulation** (Koob model)

- Chronic alcohol → GABA-A downregulation + NMDA upregulation → homeostatic set point shifted
- Even after acute withdrawal resolves, the glutamate-GABA balance takes weeks to months to normalise
- Protracted low-level CNS excitability → anxiety, sleep disruption, craving
- Mesolimbic dysphoria: chronic alcohol → blunted dopaminergic reward → everything feels flat without alcohol

EXAM PEARL

Protracted withdrawal is the primary mechanism by which acamprosate works. It modulates the GABA-glutamate imbalance, reducing the low-level dysphoria and craving of protracted withdrawal, precisely Mohan's presentation.

Q2. Which relapse prevention pharmacotherapy is best suited to Mohan, and why?

Answer:

Acamprosate is the optimal choice for Mohan.

Rationale:

FACTOR · RELEVANCE

Mechanism GABA-A positive modulation + NMDA antagonism → addresses the protracted withdrawal mechanism directly

Symptom fit Anxiety, insomnia, craving = protracted withdrawal symptoms = acamprosate's target

Timing Best started after detox completion, consistent with current situation

Safety No hepatotoxicity; no significant drug interactions; safe at 58 years

Goal Maintaining abstinence, Mohan is abstinent; acamprosate strengthens this

Dose: 666 mg TID (check renal function first, CrCl >30 required).

Additional adjuncts:

- Low-dose SSRI (sertraline 50 mg) if anxiety is prominent
- Sleep hygiene counselling + melatonin for insomnia
- Ongoing MI/CBT sessions
- Mutual aid (AA) referral

Q3. Mohan asks if he will always feel this way. What do you tell him?

Answer:

Evidence-based, hopeful, honest counselling:

- Protracted withdrawal symptoms typically improve over 3–12 months with sustained abstinence
- Brain neuroplasticity allows recovery of GABA-glutamate balance and dopaminergic reward systems
- Mood, sleep, and cognitive function generally improve, but recovery is not linear
- Acamprosate helps reduce the internal discomfort during this recovery phase
- Abstinence itself is the most powerful "medication", each day sober supports neurobiological recovery
- Some cognitive impairment (attention, memory) may persist longer, typically 12–24 months to maximum recovery
- Realistic optimism: "Most people who stay abstinent at 3 months report significant improvement by 6–12 months."

CLINICAL ANCHOR

The "I feel worse sober" statement is one of the most common barriers to sustained abstinence, and one of the most important to address with psychoeducation. Patients who understand protracted withdrawal as a time-limited neurobiological process are more likely to continue through it.

Vignette 10: The Teenager with Strange Behaviour

Case: Arun, a 16-year-old schoolboy, is brought by his parents. He was found unconscious in his room with an aerosol can and a plastic bag. He regained consciousness within 20 minutes. His parents note this has happened before. He appears disoriented and has mild nystagmus on examination. No other substances on urine drug screen. Neurological examination: mild cerebellar signs.

Q1. What is the substance involved and what are the clinical features of its misuse?

Answer:

Arun is misusing **inhalants** (specifically aerosol, likely toluene-containing product, e.g., deodorant, solvent, paint thinner).

Inhalant use disorder (ICD-11: 6C4B, Disorders due to volatile inhalants).

Clinical features:

- **Acute intoxication:** euphoria, dizziness, disorientation, nystagmus, slurred speech, ataxia; brief (minutes to hours), consistent with Arun's episode
- **Mechanism:** toluene and similar volatile compounds → CNS depression (GABA-A + NMDA effects; general anaesthetic-like)
- **"Sudden sniffing death":** sensitisation of myocardium to catecholamines → ventricular fibrillation; risk highest with fluorocarbons (aerosols, refrigerants)
- **Chronic complications:** cerebellar atrophy (explains Arun's cerebellar signs), white matter demyelination, cognitive decline, tubular nephropathy, hepatotoxicity
- **Pattern:** adolescents; socioeconomically vulnerable populations; cheap and accessible

EXAM PEARL

"Sudden sniffing death" is the most important acute complication of inhalant use, occurs with first or subsequent use, often without warning, due to cardiac arrhythmia. Adrenaline/epinephrine should be avoided in treatment as it can precipitate VF in sensitised myocardium.

Q2. How prevalent is inhalant misuse in India and what are the common substances?

Answer:

India-specific context:

- Fevicol (white glue), Dendrite, petrol, nail polish remover, correction fluid, aerosol sprays, whitener
- Particularly prevalent among street children and adolescents in urban areas
- Often the first substance tried (accessible, cheap, no legal barrier for purchase)
- Maharashtra, Delhi, Bangalore: significant street child populations with inhalant use

- No reliable national prevalence data; estimated 2–5% of substance use presentations in adolescent psychiatry

Q3. What is the management approach?

Answer:

No approved pharmacotherapy for inhalant use disorder. Management is psychosocial.

Acute:

- Supportive, oxygen, monitor cardiac rhythm (defibrillator available)
- Avoid adrenaline (VF risk)
- Treat any hypoxia or arrhythmia

Longer-term:

- **Individual CBT:** coping skills, trigger identification
- **Family therapy:** especially with adolescents, family engagement crucial
- **Motivational Interviewing:** adapted for adolescent developmental stage
- **School re-engagement:** often dropout precedes inhalant use
- **Social work:** address poverty, homelessness, family violence (root causes)
- **Residential rehabilitation:** for severe, homeless cases
- **Neuropsychological assessment:** if cerebellar signs present (Arun), document baseline, monitor for progression

Vignette 11: The Man Who Changed After Drinking

Case: Laxman, a 50-year-old, is brought by police following an assault. His wife reports he had 2 units of alcohol (a single peg of whisky and half a beer) approximately 30 minutes before the assault. She says he is a completely different person when he drinks, aggressive, paranoid, violent, and then has no memory of the episode. He is calm and cooperative now. His baseline is mild-mannered. BAC at time of arrest was 30 mg/dL (well below legal limit of 80 mg/dL in most Indian states).

Q1. What is the diagnosis and what are its features?

Answer:

Pathological intoxication (Alcohol-Induced Persisting Amnestic Disorder / Alcohol Idiosyncratic Intoxication, ICD-10 term; absorbed into "Other specified alcohol-related disorder" in DSM-5 and ICD-11).

Features:

- Extreme, disproportionate behavioural change (violence, aggression) after a small amount of alcohol

- Amount consumed grossly disproportionate to the behavioural response (2 units → assault vs no effect in most people)
- BAC low (30 mg/dL), not consistent with typical intoxication aggression at this level
- Complete or near-complete amnesia for the episode
- Behaviour out of character with baseline personality

Proposed mechanism:

- Temporal lobe disinhibition / epileptic equivalent in susceptible individuals
- Possibly relates to alcohol-induced paradoxical disinhibition
- Uncommon and diagnostically controversial, must exclude: hypoglycaemia, head injury, co-ingested substances, factitious/malingering

EXAM PEARL

Pathological intoxication is a medico-legal diagnosis, courts may consider it in assault cases. Psychiatrists may be asked to provide expert opinions. Documentation of: (a) amount consumed, (b) BAC, (c) amnesia, (d) character change, (e) prior similar episodes, is essential.

Q2. What investigations would clarify the diagnosis?

Answer:

INVESTIGATION · PURPOSE

Blood glucose at time of event Exclude hypoglycaemia as cause

BAC (done: 30 mg/dL) Confirms low BAC at time of behaviour

EEG (interictal and possibly post-ictal) Exclude temporal lobe epilepsy / epileptic disinhibition

MRI brain Structural temporal lobe abnormality

Neuropsychological testing Frontal + temporal function baseline

Toxicology screen Exclude co-ingested substances (benzodiazepines, cannabis, others)

Collateral history Wife + others who have witnessed multiple episodes, pattern confirmation

Q3. What advice do you give regarding future alcohol use?

Answer:

Clear, unambiguous advice:

- **Total abstinence from alcohol**, the only safe recommendation
- Even small amounts are dangerous in this individual

- If he continues drinking, future episodes are highly likely
 - Medico-legal implications: if charged with assault in future under similar circumstances, history of pathological intoxication may affect culpability but does not excuse behaviour
 - If epileptic mechanism confirmed: anticonvulsants (carbamazepine) may reduce episodes but do NOT permit drinking
 - Family education: wife must know to prevent access to alcohol and to call emergency services if early signs appear
-

Vignette 12: The Dual Diagnosis Dilemma

Case: Shalini, a 32-year-old woman with a 5-year history of bipolar disorder type I (currently on lithium 900 mg/day, stable), presents with a 2-month history of increasing cannabis use (daily, 3–4 times/day) following a stressful job loss. She reports that cannabis "calms her down" and prevents mood episodes. Her most recent YMRS is 8 (subclinical hypomania). She has started skipping lithium doses.

Q1. Identify the clinical and diagnostic issues.

Answer:

Multiple overlapping issues:

ISSUE · DETAIL

Bipolar I disorder Established diagnosis; currently lithium-treated; subclinical hypomania (YMRS 8)

Cannabis use disorder Daily use × 2 months; ICD-11: 6C41.2 (Cannabis dependence pattern); functional impairment (job loss context)

Cannabis as self-medication "Calms me down", perceives cannabis as mood stabiliser; likely treating anxiety/hypomania features

Medication non-adherence Skipping lithium → destabilisation risk; hypomanic drift (YMRS 8)

Cannabis → bipolar risk Cannabis use associated with: more frequent episodes, more manic switches, reduced lithium efficacy, poorer overall outcome in bipolar disorder

Dual diagnosis Bipolar + CUD = complex interaction requiring integrated management

Q2. How does cannabis affect bipolar disorder course?

Answer:

Established adverse effects of cannabis in bipolar disorder:

- **More frequent mood episodes:** both manic and depressive

- **Greater severity of episodes:** particularly mania
- **Worse functional outcomes:** employment, relationships
- **Reduced medication adherence:** cannabis users less likely to take mood stabilisers consistently (as in Shalini's case)
- **Mania switching risk:** THC is psychotomimetic and can precipitate manic or mixed states
- **Reduced lithium efficacy:** some evidence that cannabis undermines pharmacological stabilisation
- **Suicidality:** cannabis use associated with increased suicidal ideation in bipolar disorder

EXAM PEARL

The self-medication hypothesis is common, patients genuinely believe cannabis helps their mood. The data says the opposite: cannabis destabilises bipolar disorder over time, even if acute relief is experienced. This psychoeducation point is clinically and examinably important.

Q3. Outline an integrated treatment plan for Shalini.

Answer:

Integrated dual diagnosis treatment (not sequential):

Pharmacological:

- Reinstate lithium adherence, psychoeducation + pill organiser + support
- Consider lithium level check (low level may be contributing to subclinical hypomania)
- Consider valproate add-on if cannabis-induced instability difficult to control (better evidence for mixed states)
- No approved pharmacotherapy for cannabis use disorder; no agent to add

Psychosocial:

- **Integrated CBT:** addresses both bipolar cognitions AND cannabis use, same therapist, same framework
- **Motivational Interviewing:** ambivalence is clear (she believes cannabis helps; evidence says it harms), decisional balance + personalised feedback on bipolar-cannabis interaction
- **Psychoeducation (bipolar + cannabis):** the "calming" effect is short-term, tolerance-developing; over time cannabis worsens her condition
- **Relapse prevention:** identify triggers for both mood episodes and cannabis use; overlapping coping strategy development
- **Sleep hygiene:** cannabis disrupts sleep architecture (reduces REM); poor sleep = major bipolar trigger
- **Social support:** job loss addressed; vocational rehabilitation; family education

Monitoring:

- Mood diary + cannabis diary simultaneously
- YMRS monthly
- Lithium levels every 3 months
- Target: cannabis reduction → abstinence → mood stability → functional recovery

CLINICAL ANCHOR

The hierarchy in dual diagnosis management: safety first (lithium adherence), then engagement, then substance reduction, then abstinence. Do NOT make abstinence a precondition for treating the bipolar disorder, this sequencing fails patients.

GUIDE 09 · PART II

Anxiety OCD

Paper II · Clinical Psychiatry. Six study modes, from notes to quick review.

Study Notes

Sources: Kaplan & Sadock's Comprehensive Textbook of Psychiatry (10th ed.), Stahl's Essential Psychopharmacology (4th ed.), Oxford Textbook of Psychiatry, ICD-11 (2022), DSM-5-TR (2022)

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1. GENERALIZED ANXIETY DISORDER (GAD)

1.1 Diagnostic Criteria

EXAM PEARL

The key distinction between GAD and normal worry: GAD worry is excessive, hard to control, and causes significant functional impairment. Duration threshold: 6 months in DSM-5; "more days than not" for several months in ICD-11.

DSM-5 CRITERIA (F41.1)

- **A.** Excessive anxiety and worry about multiple events/activities, occurring more days than not for **≥6 months**
- **B.** Difficult to control the worry
- **C.** ≥3 of 6 symptoms (only 1 required in children):

- Restlessness/feeling keyed up/on edge
- Easily fatigued
- Difficulty concentrating / mind going blank
- Irritability
- Muscle tension
- Sleep disturbance (difficulty falling/staying asleep, restless unsatisfying sleep)
- **D.** Causes significant distress or functional impairment
- **E.** Not attributable to substances or medical condition
- **F.** Not better explained by another mental disorder

ICD-11 CRITERIA (6B00)

- Marked symptoms of anxiety manifesting as **general apprehension** ("free-floating anxiety") or **worry** about multiple domains
- Anxiety and worry accompanied by:
 - Motor tension (trembling, twitching, muscle aches, restlessness)
 - Autonomic hyperactivity (palpitations, sweating, cold/clammy hands, dry mouth, dizziness)
 - Hypervigilance and scanning
 - Concentration problems, irritability, sleep disturbance
- Symptoms **persistent** (more days than not, for several months)
- Not better explained by another condition

CLINICAL ANCHOR

DSM-5 requires 3 of 6 somatic symptoms; ICD-11 is less specific about number. Both require 6 months (DSM-5 explicit; ICD-11 "several months"). ICD-11 groups under "Anxiety or Fear-Related Disorders."

1.2 Epidemiology

PARAMETER · VALUE

Lifetime prevalence 5.7% (general population)

12-month prevalence 3.1%

Sex ratio F:M = 2:1

Peak onset Late adolescence/early adulthood; also mid-life

Comorbidity High: major depression (>60%), other anxiety disorders, somatic disorders

Course Chronic, waxing/waning; rarely spontaneous remission without treatment

1.3 Neurobiology

AMYGDALA-PFC CIRCUIT (FEAR CIRCUITRY)

In GAD:

- **Amygdala hyperreactivity**, exaggerated threat detection
- **Prefrontal cortex (vmPFC) hypoactivity**, failure of top-down inhibition of amygdala
- **Anterior cingulate cortex (ACC)** dysfunction, impaired error monitoring and emotional regulation
- **Bed nucleus of stria terminalis (BNST)**, mediates sustained anxiety states (vs amygdala's phasic fear response)

EXAM PEARL

Amygdala = phasic fear (immediate, stimulus-specific). BNST = sustained anxiety (non-specific, anticipatory). GAD involves BNST more than amygdala per se.

NEUROTRANSMITTER SYSTEMS

GABA:

- GABA is the primary inhibitory neurotransmitter in the CNS
- GABAergic interneurons normally inhibit amygdala hyperactivity
- In anxiety: **reduced GABA-A receptor function** or reduced GABA availability
- Benzodiazepines act on GABA-A receptor (allosteric modulator at Cl^- channel, increase frequency of Cl^- channel opening)
- Pregabalin binds $\alpha 2\delta$ subunit of voltage-gated Ca^{2+} channels → reduces presynaptic Ca^{2+} influx → reduces glutamate/norepinephrine release

Serotonin (5-HT):

- Dorsal raphe nucleus (DRN) → amygdala, hippocampus, prefrontal cortex
- 5-HT_{1A} receptor: autoreceptor (somatodendritic), inhibits 5-HT firing; postsynaptic, anxiolytic
- 5-HT_{2A} receptor: postsynaptic, anxiogenic (especially when stimulated)
- Buspirone: partial agonist at 5-HT_{1A} → desensitizes presynaptic autoreceptor → increased 5-HT transmission chronically
- SSRIs initially increase 5-HT_{2A} activation (may cause initial anxiety increase) → chronic desensitization → net anxiolytic

Norepinephrine (NE):

- Locus coeruleus (LC) → widespread cortical/limbic projections
- NE excess → hyperarousal, vigilance, somatic anxiety symptoms
- SNRIs reduce excessive NE via autoreceptor desensitization

Glutamate:

- Excessive NMDA activation in amygdala circuits → fear conditioning and anxiety
- mGluR2/3 presynaptic autoreceptors: novel targets for anxiolytics

CRF (Corticotropin-Releasing Factor):

- CRF1 receptor in amygdala/BNST: overactivation → anxiety, HPA axis dysregulation
- HPA axis: CRF → ACTH → cortisol (dysregulated in chronic anxiety)

1.4 Pharmacotherapy

FIRST-LINE AGENTS

DRUG	CLASS	DOSE RANGE	ONSET OF ACTION	NOTES
Escitalopram	SSRI	10–20 mg/day	2–4 weeks (full effect 6–8 wks)	Best tolerated, minimal CYP interactions
Sertraline	SSRI	50–200 mg/day	2–4 weeks	Strong evidence base
Paroxetine CR	SSRI	12.5–62.5 mg/day	2–4 weeks	More sedating; discontinuation syndrome risk
Venlafaxine XR	SNRI	75–225 mg/day	2–4 weeks	FDA-approved; BP monitoring
Duloxetine	SNRI	60–120 mg/day	2–4 weeks	FDA-approved; helpful with somatic symptoms

SECOND-LINE AGENTS

DRUG	CLASS	DOSE	NOTES
Buspirone	5-HT _{1A} partial agonist	15–60 mg/day (TID dosing)	No dependence; 2–4 week onset; no benefit if prior BZD use; no acute effect
Pregabalin	α _{2δ} calcium channel ligand	150–600 mg/day	FDA/EMA approved for GAD; rapid onset (1 week); sedation, weight gain; Schedule V (US)
TCAs (imipramine)	TCA	75–200 mg/day	Effective but side effect burden; QTc monitoring

ADJUNCTS / SHORT-TERM

DRUG · NOTES

Benzodiazepines For acute/severe anxiety; avoid long-term use (dependence, cognitive effects); use lowest dose for shortest duration; clonazepam preferred (longer half-life, less abuse potential vs alprazolam)

Hydroxyzine Antihistamine; 25–50 mg PRN or TID; no dependence; reasonable for short-term

Quetiapine Atypical AP; evidence for GAD; use when comorbid MDD or insomnia

Propranolol For performance anxiety / somatic symptoms; not for generalized anxiety

EXAM PEARL

Buspirone takes 2–4 weeks. It will NOT work if the patient has previously been on benzodiazepines (BZD occupies receptors, reducing anxiolytic effect, "receptor downregulation" argument). Switch to buspirone only after BZD taper.

TREATMENT ALGORITHM (GAD)

1.5 Psychotherapy for GAD

COGNITIVE-BEHAVIOURAL THERAPY (CBT)

- **Gold standard** for GAD (NNT comparable to pharmacotherapy)
- Components:
- **Psychoeducation**, nature of anxiety, worry
- **Self-monitoring**, worry diary, identifying triggers
- **Cognitive restructuring**, identifying catastrophizing, thought challenging, probability estimation
- **Worry postponement/Stimulus control**, scheduled worry time (15–30 min/day)
- **Relaxation training**, progressive muscle relaxation (PMR), diaphragmatic breathing
- **Interoceptive exposure**, for somatic symptoms
- **Behavioural experiments**, testing worry predictions
- **Problem-solving therapy**, for real-world stressors

Borkovec's Avoidance Model of Worry:

- Worry = cognitive avoidance of emotional processing of threatening images
- Suppresses somatic activation (paradoxically) → negatively reinforces worry
- CBT targets this cycle

Metacognitive Therapy (Wells):

- Targets beliefs *about* worry ("I must worry to cope") and beliefs about uncontrollability / danger of worry
- "Detached mindfulness" and attention training

ACCEPTANCE & COMMITMENT THERAPY (ACT)

- Defusion from anxious thoughts; valued living despite anxiety
- Evidence base growing; comparable to CBT in some trials

2. PANIC DISORDER

2.1 Diagnostic Criteria

EXAM PEARL

A panic attack is NOT a disorder, it is a specifier. Panic disorder requires RECURRENT unexpected panic attacks PLUS persistent concern or behaviour change for ≥ 1 month.

DSM-5 CRITERIA (F41.0)

Panic Attack (not a diagnosis itself, a specifier):

13 symptoms, ≥ 4 required:

1. Palpitations / pounding heart / accelerated heart rate
2. Sweating
3. Trembling or shaking
4. Shortness of breath / smothering sensation
5. Feelings of choking
6. Chest pain / discomfort
7. Nausea / abdominal distress
8. Dizziness / unsteady / lightheaded / faint
9. Chills or hot flushes
10. Paresthesias (numbness/tingling)
11. Derealization / depersonalization
12. Fear of losing control / going crazy
13. Fear of dying

Panic attacks are **abrupt surges** reaching peak within **minutes**; can be expected (cued) or unexpected (uncued / spontaneous).

Panic Disorder requires:

- **A.** Recurrent **unexpected** panic attacks
- **B.** At least one attack followed by ≥ 1 month of:

- Persistent concern about additional attacks or consequences ("am I having a heart attack?")
- OR significant maladaptive change in behaviour (avoidance, safety behaviours)
- **C.** Not attributable to substances or medical condition
- **D.** Not better explained by another mental disorder

ICD-11 CRITERIA (6B01)

- Recurrent unexpected panic attacks
- At least one attack followed by at least **one month** of anxiety about recurrence OR maladaptive behaviour
- Key: **unexpected** = not triggered by identifiable feared object / situation

2.2 Epidemiology

PARAMETER · VALUE

Lifetime prevalence 3.5–4.7%

12-month prevalence 2–3%

Sex ratio F:M = 2:1

Onset Bimodal: late teens / early 20s and mid-30s

Comorbidity Agoraphobia (30–50%), major depression (50%), other anxiety disorders

Course Chronic; waxing / waning; full recovery in minority

2.3 Clark's Cognitive Model of Panic

EXAM PEARL

Clark's model is the most frequently examined cognitive model for panic. Know all 4 components and the maintenance cycle.

Core premise: Panic attacks result from the **catastrophic misinterpretation of bodily sensations**.

Maintaining factors:

1. **Selective attention / hypervigilance** to bodily sensations
2. **Safety behaviours** (sitting down, calling ambulance), prevent disconfirmation
3. **Avoidance** of triggers, prevents learning
4. **Anticipatory anxiety**, increases physiological arousal before next attack

Treatment implications (Clark's model → CBT):

- Psychoeducation (corrects misinterpretation)
- Interoceptive exposure (induced sensations → learn they are not dangerous)
- Cognitive restructuring (challenging catastrophic thoughts)
- Behavioural experiments (drop safety behaviours, enter feared situations)

2.4 Neuroanatomy of Panic

SUFFOCATION ALARM THEORY (KLEIN, 1993)

- Proposes an evolved **hypersensitive CO₂/suffocation alarm system** in the brainstem
- Panicogenic agents: CO₂ inhalation, sodium lactate → trigger suffocation alarm
- Imipramine blocks false alarms (hence antidepressants prevent panic)
- Explains respiratory panic symptoms and response to respiratory hyperventilation

Key brain regions:

- **Locus coeruleus (LC):** noradrenergic nucleus in pons; hyperactivation → panic symptoms (yohimbine provokes panic by blocking α_2 autoreceptors)
- **Periaqueductal grey (PAG):** mediates freezing and flight responses; dorsal PAG stimulation → panic-like responses
- **Hypothalamus:** HPA axis activation during panic
- **Amygdala:** fear conditioning; learned associations for expected panic attacks
- **Hippocampus:** contextual fear conditioning

Neurochemistry:

- NE excess (LC) → palpitations, sweating, tremor
- 5-HT dysregulation, SSRIs highly effective
- CCK-4 (cholecystokinin), exogenous injection provokes panic attacks
- GABA-A deficiency (parahippocampal region)

2.5 Pharmacotherapy

FIRST-LINE

DRUG	DOSE	NOTES
SSRIs (all)	Standard doses	Escitalopram, sertraline, paroxetine FDA-approved; start LOW (may worsen anxiety initially)
Venlafaxine XR	75–225 mg	FDA-approved; good evidence

SECOND-LINE

DRUG	DOSE	NOTES
Clomipramine	75–250 mg	Very effective but side effect burden
Imipramine	75–250 mg	Historical gold standard; Klein's work
Alprazolam	0.5–10 mg/day	Rapid onset; FDA-approved; dependence risk; CR formulation preferred
Clonazepam	1–4 mg/day	Longer half-life; less euphorogenic

EXAM PEARL

Start SSRIs at HALF the usual dose in panic disorder. Full-dose initiation can cause initial jitteriness/increased panic that leads patients to discontinue. Titrate slowly over 2–4 weeks.

Duration of treatment: Minimum 12 months after remission; 2+ years if recurrent.

2.6 CBT for Panic Disorder

Highly effective (60–80% response); may be superior to pharmacotherapy in long-term follow-up.

Components:

1. **Psychoeducation**, Clark's model, nature of panic/anxiety, fight-or-flight
2. **Breathing retraining**, diaphragmatic breathing (caution: may become safety behaviour)
3. **Cognitive restructuring**, challenging catastrophic misinterpretations
4. **Interoceptive exposure**, deliberately inducing feared sensations:
5. Spinning in chair (dizziness)
6. Running in place (palpitations, breathlessness)
7. Breathing through straw (choking sensation)
8. Staring at point on wall (derealization)
9. **In vivo exposure**, entering avoided situations (combined with situational exposure for agoraphobia)
10. **Eliminating safety behaviours**

3. AGORAPHOBIA

3.1 Diagnostic Criteria (DSM-5: F40.00)

- **A.** Marked fear / anxiety about ≥ 2 of:
 - Using public transport
 - Being in open spaces (parking lots, marketplaces, bridges)
 - Being in enclosed places (shops, theatres, cinemas)
 - Standing in line or being in a crowd
 - Being outside of the home alone
- **B.** Person fears / avoids because of thoughts that escape might be difficult or help unavailable in event of panic-like symptoms
- **C.** Situations almost always provoke fear / anxiety
- **D.** Actively avoided, require companion, or endured with intense anxiety
- **E.** Fear disproportionate to actual danger
- **F.** Persistent (≥ 6 months)
- **G.** Significant distress / impairment
- **H.** Not attributable to another medical condition or substance

KEY INSIGHT

EXAM PEARL (DSM-5 change): Agoraphobia is now a **separate diagnosis** from Panic Disorder. Previously required link to panic. Now stands alone. Can occur with or without panic disorder.

3.2 Management

- **First-line:** CBT (exposure therapy) $\pm$ SSRIs
- Graded exposure hierarchy (SUDS, subjective units of distress)
- Partner-assisted exposure when complete avoidance
- Goal: not to eliminate anxiety but to increase tolerance and reduce avoidance

4. SOCIAL ANXIETY DISORDER (SAD)

4.1 Diagnostic Criteria

DSM-5 (F40.10)

- **A.** Marked fear / anxiety about one or more **social situations** where exposed to scrutiny (conversation, performance, being observed eating / drinking)
- **B.** Fears acting / showing anxiety in humiliating / embarrassing way or provoking negative evaluation

- **C.** Social situations almost always provoke fear/anxiety
- **D.** Avoided or endured with intense anxiety
- **E.** Disproportionate to actual threat
- **F.** Persistent (≥ 6 months)
- **G.** Significant distress/impairment
- **H.** Not substance/medical
- **I.** Not better explained by another disorder (ASD, PTSD, BDD, etc.)

Specifier: "Performance only", if fear limited to public speaking/performing

PERFORMANCE-ONLY VS GENERALIZED SAD

FEATURE	PERFORMANCE-ONLY	GENERALIZED SAD
Situations feared	Public speaking, performing	Most/all social situations
Functional impairment	Occupational	Broader (social, occupational)
Comorbidity	Less	More (MDD, other anxiety)
Pharmacotherapy	Propranolol PRN effective	Requires regular SSRI/SNRI
Prognosis	Better	Chronic course

4.2 Assessment: Liebowitz Social Anxiety Scale (LSAS)

- 24 items: 13 performance situations + 11 social interaction situations
- Each rated for **fear** (0–3) and **avoidance** (0–3)
- Maximum score: 144
- Scoring: 55–65 = social phobia; 65–80 = marked; 80–95 = severe; >95 = very severe
- Widely used in clinical trials; sensitive to treatment change

4.3 Cognitive Model (Clark & Wells, 1995)

1. Social situation → perceived social threat
2. **Self-focused attention** (focuses on internal sensations/thoughts about performance)
3. **Safety behaviours** (avoiding eye contact, over-preparing, rehearsing)
4. Problematic processing before and after events
5. Negative self-image as a social object

4.4 Pharmacotherapy

DRUG	CLASS	EVIDENCE	NOTES
Sertraline	SSRI	FDA-approved	First-line
Paroxetine CR	SSRI	FDA-approved	First-line

DRUG	CLASS	EVIDENCE	NOTES
Venlafaxine XR	SNRI	FDA-approved	First-line
Escitalopram	SSRI	Strong RCT data	Not FDA-approved for SAD but widely used
Phenelzine	MAOI	Highly effective	Second/third-line; dietary restrictions; orthostatic hypotension
Clonazepam	BZD	Adjunct	Rapid onset; for acute performance anxiety
Propranolol	Beta-blocker	Performance anxiety	10–40 mg PRN 30–60 min before; not for generalized SAD
Gabapentin	$\alpha 2\delta$ ligand	Second-line	Useful in alcohol dependence comorbidity

4.5 Psychotherapy

CBT components:

- Clark & Wells model-based: attention training (external focus), video feedback, dropping safety behaviours
- Exposure therapy (social situations hierarchy)
- Cognitive restructuring

Social skills training: For those with genuine skills deficits (not just inhibition)

5. SPECIFIC PHOBIAS

5.1 Classification (DSM-5 Specifiers)

1. **Animal type** (spiders, insects, dogs, snakes)
2. **Natural environment type** (heights, storms, water)
3. **Blood-injection-injury (BII) type**, unique: vasovagal response (bradycardia, hypotension → fainting)
4. **Situational type** (airplanes, elevators, enclosed spaces)
5. **Other type** (vomiting, loud sounds, costumed characters)

EXAM PEARL

BII phobia is the **ONLY** phobia with a **diphasic** cardiovascular response: initial tachycardia followed by paradoxical bradycardia and hypotension (vasovagal syncope). Treatment includes **applied tension technique** (tensing large muscle groups to raise BP).

5.2 Psychological Theories of Origin

1. **Classical conditioning** (Pavlovian), traumatic conditioning (dog bite → dog phobia)
2. **Observational learning**, watching others fear (vicarious conditioning)
3. **Information transmission**, told snakes are dangerous
4. **Preparedness theory (Seligman)**, biologically prepared to fear evolutionarily dangerous stimuli (snakes, heights, spiders), these phobias are resistant to extinction

5.3 Treatment

SYSTEMATIC DESENSITIZATION (WOLPE)

- Based on reciprocal inhibition (relaxation incompatible with anxiety)
- **Three components:**
- Relaxation training (PMR or hypnosis)
- Fear hierarchy construction (8–12 steps, anchored by SUDS 0–100)
- Graded exposure (starting from least anxiety-provoking, pairing with relaxation)
- Can be in vivo or imaginal
- Best for: specific phobias, simple situations

FLOODING (IMPLOSION THERAPY)

- **Prolonged, intensive exposure** to feared stimulus without relaxation
- Maximal anxiety → habituation → extinction
- In vivo flooding = most effective; imaginal flooding (implosion) = less effective
- Contraindications: severe cardiac disease, psychosis, severe trauma history

VIRTUAL REALITY EXPOSURE THERAPY (VRET)

- Computer-generated phobic stimuli
- Evidence for: acrophobia, arachnophobia, aviophobia, claustrophobia, driving phobia
- Advantages: controllable, accessible, privacy, high-fidelity
- Comparable efficacy to in vivo exposure in meta-analyses

PHARMACOTHERAPY (LIMITED ROLE)

- D-cycloserine (NMDA partial agonist), augments extinction learning; adjunct to exposure
 - Benzodiazepines (short-term, not for long-term phobia treatment, may interfere with extinction)
-

6. OBSESSIVE-COMPULSIVE DISORDER (OCD)

6.1 Diagnostic Criteria

DSM-5 (F42)

- **A.** Presence of obsessions, compulsions, or both
- **Obsessions:** (1) recurrent intrusive thoughts / urges / images causing marked anxiety; (2) person attempts to ignore / suppress or neutralize with other thought / action
- **Compulsions:** (1) repetitive behaviours or mental acts driven by the obsession or rigid rules; (2) aimed at preventing / reducing distress or dreaded event, but are NOT realistically connected or are excessive
- **B.** Time-consuming (>1 hr/day) OR cause significant distress / impairment
- **C.** Not attributable to substances or medical condition
- **D.** Not better explained by another mental disorder

Insight specifiers:

- Good or fair insight
- Poor insight
- Absent insight / delusional beliefs

Tic-related specifier: if current or past history of tic disorder

ICD-11 (6B20)

- Same core features
- Classified under "Obsessive-Compulsive and Related Disorders" (separate chapter from anxiety disorders, same as DSM-5)

EXAM PEARL

OCD was moved from Anxiety Disorders to its own chapter in BOTH DSM-5 AND ICD-11. This reflects distinct neurobiology (CSTC circuit vs fear circuit).

6.2 Common OCD Symptom Dimensions

DIMENSION	OBSESSIONS	COMPULSIONS
Contamination	Dirt, germs, illness	Washing, cleaning
Harm/aggression	Hurting self / others	Checking, reassurance-seeking
Symmetry/order	Things not "just right"	Ordering, counting, arranging
Forbidden thoughts	Sexual, blasphemous, taboo	Mental neutralizing, praying
Hoarding	Loss of items, incompleteness	Collecting, saving

DIMENSION	OBSSESSIONS	COMPULSIONS
Somatic	Illness, bodily sensations	Body checking

6.3 Yale-Brown Obsessive Compulsive Scale (Y-BOCS)

EXAM PEARL

Y-BOCS is the gold standard OCD severity measure. Know the two subscales, 5 items each, and scoring thresholds.

Structure:

- 10 items: 5 for obsessions + 5 for compulsions
- Each item scored 0 (none) to 4 (extreme)
- Range: 0–40

Five dimensions per subscale (O = obsessions, C = compulsions):

1. Time occupied (O)/Time spent on (C)
2. Interference with functioning
3. Distress/anxiety caused
4. Resistance (degree of effort to resist)
5. Control (ability to control/stop)

Severity thresholds:

SCORE · SEVERITY

0–7 Subclinical

8–15 Mild

16–23 Moderate

24–31 Severe

32–40 Extreme

Response to treatment: $\geq 35\%$ reduction in Y-BOCS score

Y-BOCS-II: Updated version with insight modifier; broader content coverage.

OCI-R (OCD Inventory Revised): 18-item self-report; 6 subscales; used in research and screening.

6.4 Neurobiology: CSTC Circuit

EXAM PEARL

CSTC = Cortico-Striato-Thalamo-Cortical circuit. This is THE neurobiology circuit for OCD.
Must know the direct (go) and indirect (stop) pathways.

CSTC CIRCUIT ARCHITECTURE

In OCD (Saxena & Rauch model):

COMPONENT	NORMAL	OCD
Direct pathway (go)	Balanced	Hyperactive → releases thalamus → drives repetitive behaviour
Indirect pathway (stop)	Balanced	Underactive → fails to stop thalamic activation
OFC	Normal "error signal"	Overactive, generates excessive "something is wrong" signal
Caudate	Normal gating	Hypofunctional as a gate, fails to suppress drive signals
Thalamus	Normal relay	Hyperactivated → drives motor cortex → compulsions

NET RESULT: Overactive OFC generates error signals → overactive direct pathway drives repetition → compulsions are the brain's failed attempt to resolve the "error signal."

Why SSRIs work: 5-HT modulates striatal and thalamic function; chronic SSRI use normalizes caudate hypermetabolism (seen on FDG-PET).

NEUROIMAGING EVIDENCE

- **FDG-PET:** Hypermetabolism in OFC, caudate nucleus, thalamus at baseline; normalizes with successful treatment
- **fMRI:** Hyperactivation of OFC, ACC during symptom provocation
- **Structural MRI:** Decreased grey matter in OFC, ACC; increased in caudate

SEROTONIN HYPOTHESIS

- OCD selectively responds to serotonin-specific agents (clomipramine > desipramine in Zohar's studies)
- 5-HT_{2C} receptors: postsynaptic in striatum, modulate repetitive behaviour
- Augmenting agents (atypical APs) act on D₂ in striatum, block dopamine hyperactivity in striato-thalamic loops

6.5 Pharmacotherapy

FIRST-LINE: SSRIS

EXAM PEARL

OCD requires **higher doses** of SSRIs than depression, and **longer treatment trials** (10–12 weeks minimum). Response rates: ~50–60% with first SSRI.

DRUG	STARTING DOSE	TARGET DOSE	MAXIMUM DOSE
Fluoxetine	10–20 mg	40–60 mg	80 mg
Fluvoxamine	50 mg	150–200 mg	300 mg
Sertraline	25–50 mg	150–200 mg	200 mg
Paroxetine	10–20 mg	40–60 mg	60 mg
Escitalopram	10 mg	20–40 mg	40 mg (off-label high dose)
Citalopram	20 mg	40 mg	40 mg (QTc concern above)

All 5 SSRIs are FDA-approved for OCD. Fluvoxamine was first (1994).

CLOMIPRAMINE

EXAM PEARL

Clomipramine = most efficacious single agent for OCD (effect size ~1.0 vs SSRIs ~0.5–0.7). But side effect profile limits first-line use.

Mechanism TCA; serotonin reuptake inhibitor + NRI; active metabolite (desmethylclomipramine) is NRI

Efficacy Superior to all SSRIs in head-to-head comparisons; also superior to desipramine (controls for TCA)

Dose 25–50 mg start → 100–250 mg/day

Side effects Anticholinergic (dry mouth, constipation, urinary retention), sedation, weight gain, orthostatic hypotension, **seizures (>250 mg)**, sexual dysfunction

QTc Cardiac monitoring required at higher doses

Drug interactions Avoid MAOIs (serotonin syndrome); fluvoxamine inhibits CYP1A2 → raises clomipramine levels

Preferred in Severe/refractory OCD; patients who failed 2+ SSRIs

SSRIS VS CLOMIPRAMINE: KEY COMPARISON

FEATURE	SSRIS	CLOMIPRAMINE
Efficacy	Moderate (~0.5–0.7 ES)	High (~1.0 ES)
Tolerability	Better	Worse
Safety in overdose	Safer	Dangerous (cardiac, seizures)
Drug interactions	Variable by CYP	More
First-line status	Yes (all 5 SSRIs)	Second-line (first-line in some guidelines)
Monitoring required	Minimal	QTc, seizure threshold, BP
Sexual side effects	Common	Common
Role in pregnancy	Preferred	Avoid if possible

AUGMENTATION STRATEGIES

EXAM PEARL

The standard augmentation for OCD is a low-dose antipsychotic added to an SSRI or clomipramine. Risperidone and haloperidol have the most evidence.

AUGMENTING AGENT	EVIDENCE	DOSE	NOTES
Risperidone	Best RCT evidence	0.5–2 mg/day	For OCD without tic disorder
Haloperidol	Good evidence	2–10 mg/day	Especially with comorbid tic disorder
Aripiprazole	Good evidence	5–15 mg/day	Well-tolerated; weight-neutral
Quetiapine	Moderate evidence	25–200 mg/day	Helpful if comorbid anxiety/insomnia
Olanzapine	Some evidence	2.5–10 mg/day	Weight gain concern
Clonazepam	Adjunct	0.5–2 mg/day	Short-term anxiety reduction
Clomipramine	Add to SSRI	Low dose (25–100 mg)	Monitor clomipramine levels; combination raises risk
D-cycloserine	Enhances ERP	50–100 mg before session	Facilitates extinction learning

6.6 Psychotherapy: Exposure and Response Prevention (ERP)

EXAM PEARL

ERP is the psychological treatment of choice for OCD. Effect size ~1.0–1.5 (superior to pharmacotherapy alone). Combines exposure with ritual prevention.

PRINCIPLES OF ERP

Theoretical basis:

- Inhibitory learning model (Craske): exposure creates new non-threatening associations, not erasure of old ones
- Habituation (older model): prolonged exposure → reduced anxiety response

Components:

1. **Exposure:** Contact with feared stimulus / situation (in vivo, imaginal, or interoceptive)
2. **Response Prevention:** Deliberately refraining from compulsions

Process:

- Construct hierarchy (SUDS 0–100)
- Start with moderate SUDS (40–50), not lowest
- Prolonged exposure (≥45 minutes per session) until SUDS reduces by 50%
- Gradual progression up hierarchy
- Therapist-assisted → self-directed

Critical rule: The anxiety WILL peak and then decrease. Compulsions terminate anxiety prematurely and reinforce the obsession-compulsion cycle.

VARIANTS OF ERP

- **Intensive ERP:** Daily sessions for 2–3 weeks; used for severe / refractory cases
- **Family-based ERP:** Involves family members (who may be accommodation providers)
- **Imaginal ERP:** For obsessions without behavioral rituals (harm obsessions, pure O)
- **IOCDF protocol:** International OCD Foundation standardized approach

INFERENCE-BASED CBT (I-CBT)

- For poor-insight OCD; targets "inferential confusion", treating imagined possibilities as actual reality
- "Case narrativization", understanding the obsessional story

ACT FOR OCD

- Defusion from obsessions; values-based action
- Does not require challenging obsessional content

- Useful for poor insight

6.7 Neuromodulation and Surgery

NEUROSURGERY FOR OCD

Indicated only for: **severe, treatment-resistant OCD**, failing ≥ 3 adequate SRI trials + adequate ERP, significant disability, Y-BOCS > 30 .

PROCEDURE	TARGET	MECHANISM	EVIDENCE
Anterior capsulotomy	Anterior limb of internal capsule (ALIC)	Interrupts CSTC circuit	~40–70% response; lesional
Anterior cingulotomy	Anterior cingulate	Disrupts ACC-OFC loop	25–40% response; lesional
Deep Brain Stimulation (DBS)	ALIC/NAcc/BNST/STN	Modulates CSTC; reversible	FDA Humanitarian Device Exemption (2009); 40–60% response
Gamma Knife Radiosurgery	ALIC (bilateral)	Radiation capsulotomy	Non-invasive; delayed effect (6–12 months)

DBS targets for OCD:

- Primary: Anterior limb of internal capsule (ALIC) / Ventral striatum / Nucleus accumbens (NAcc)
- Alternative: Subthalamic nucleus (STN), also used in Parkinson's
- BNST (bed nucleus of stria terminalis)

EXAM PEARL

DBS for OCD received FDA approval via Humanitarian Device Exemption (HDE) in 2009, not full approval. Requires multidisciplinary team, ethics board review, and informed consent. It is reversible (unlike ablative procedures).

TRANSCRANIAL MAGNETIC STIMULATION (TMS)

- Repetitive TMS (rTMS) to supplementary motor area (SMA), FDA cleared for OCD (2018)
- Less invasive neuromodulation option
- Deep TMS (dTMS), H7 coil targeting mPFC and anterior cingulate

7. TREATMENT-RESISTANT OCD

7.1 Definition

Treatment-resistant OCD = failure to achieve $\geq 35\%$ Y-BOCS improvement after:

- **2–3 adequate SSRI trials** (at maximum tolerated dose, ≥ 12 weeks each)
- **1 adequate clomipramine trial** (≥ 250 mg or maximum tolerated)
- **Adequate ERP** (≥ 20 sessions by competent therapist)

7.2 Management Algorithm

7.3 IV Clomipramine

- Bypasses first-pass metabolism $\rightarrow$ higher peak plasma levels
- Protocol: 25–75 mg IV in dextrose over 45–90 minutes
- Used in centres with expertise; not widely available
- Evidence from Indian studies (Bhave & Bhave, 1986; Koran et al.)
- Response even in oral clomipramine failures

7.4 Other Strategies

- **Glutamate-modulating agents:** Riluzole, memantine, NAC (N-acetylcysteine), topiramate
- **Ondansetron** (5-HT₃ antagonist): small studies showing benefit
- **Tramadol** (opioid + SRI): case reports of rapid response
- **Ketamine:** Emerging evidence for rapid but transient OCD reduction (NMDA antagonism)
- **Psilocybin/MDMA:** Under investigation; very preliminary

8. PTSD & ACUTE STRESS DISORDER

8.1 PTSD Diagnostic Criteria (DSM-5: F43.10)

EXAM PEARL

DSM-5 restructured PTSD into 4 symptom clusters (was 3 in DSM-IV). Added negative alterations in cognition and mood as a separate cluster. Minimum age for PTSD: separate criteria for children ≤ 6 years.

Criterion A, Trauma exposure (at least one):

1. Directly experiencing traumatic event
2. Witnessing in person
3. Learning that close person experienced violent/accidental death/threatened death/sexual violence
4. Repeated/extreme indirect exposure (first responders, forensic workers), NOT media exposure

Qualifying traumatic events: actual or threatened death, serious injury, or sexual violence

Criterion B, Intrusion (≥ 1):

1. Intrusive distressing memories
2. Recurrent distressing dreams
3. **Dissociative reactions (flashbacks)**, range from brief to complete loss of awareness
4. Intense/prolonged psychological distress at internal/external trauma cues
5. Marked physiological reactions to trauma cues

Criterion C, Avoidance (≥ 1):

1. Avoidance of distressing memories, thoughts, feelings
2. Avoidance of external reminders (people, places, situations, activities)

Criterion D, Negative alterations in cognition and mood (≥ 2):

1. Inability to remember important aspects (dissociative amnesia)
2. Persistent/exaggerated negative beliefs about self/world ("I am bad")
3. Persistent distorted blame of self/others
4. Persistent negative emotional state (fear, horror, anger, guilt, shame)
5. Diminished interest/participation in activities
6. Feelings of detachment or estrangement from others
7. Persistent inability to experience positive emotions (emotional numbing, anhedonia)

Criterion E, Alterations in arousal and reactivity (≥ 2):

1. Irritable behaviour and angry outbursts
2. Reckless or self-destructive behaviour
3. Hypervigilance
4. Exaggerated startle response
5. Concentration problems
6. Sleep disturbance

Criterion F: ≥ 1 month duration

Criterion G: Significant distress/functional impairment

Criterion H: Not substance/medical

Specifiers:

- With dissociative symptoms: depersonalization, derealization
- With delayed expression: full criteria not met until ≥ 6 months post-event

COMPLEX PTSD (ICD-11: 6B41)

ICD-11 recognizes Complex PTSD as a distinct disorder (not in DSM-5):

- All PTSD features PLUS "disturbances in self-organization" (DSO):
- Affect dysregulation
- Negative self-concept (shame, guilt, worthlessness)
- Disturbances in relationships

Typically follows prolonged / repeated trauma (childhood abuse, domestic violence, trafficking, torture).

8.2 Acute Stress Disorder (ASD: F43.0)

Duration: 3 days to 1 month post-trauma (PTSD requires >1 month)

DSM-5 criteria:

- Trauma exposure (same as PTSD Criterion A)
- ≥ 9 of 14 symptoms across 5 categories: intrusion, negative mood, dissociation, avoidance, arousal
- Functional impairment
- Duration: 3 days to 1 month

EXAM PEARL

ASD vs PTSD: Duration is the key distinction. ASD = 3 days–1 month; PTSD = >1 month. ASD predicts PTSD risk (but most people with ASD do not develop PTSD, and many who develop PTSD did not have ASD).

8.3 Ehlers & Clark Cognitive Model of PTSD (2000)

EXAM PEARL

This is the dominant cognitive model for PTSD and underlies CT-PTSD (cognitive therapy for PTSD).

Two core processes generate persistent PTSD:

1. **Appraisal of the traumatic event and/or its sequelae as a current serious threat:**
 2. "I am permanently changed / damaged"
 3. "Nowhere is safe"
 4. "I am going mad" (about PTSD symptoms themselves)
1. **Nature of the trauma memory, autobiographical memory characteristics:**
 2. Poor contextual embedding (trauma memory lacks time / place context)
 3. Strong associative triggering (sensory cues trigger intrusions without conscious recall)
 4. "Data-driven processing" during trauma (sensory focus, not conceptual) → weak elaboration

Maintaining behaviours:

- **Thought suppression** → paradoxically increases intrusions
- **Rumination** (about causes, consequences) → increases distress without processing
- **Safety behaviours** (staying indoors, never being alone) → prevent disconfirmation
- **Cognitive avoidance**, prevents memory elaboration

Treatment (CT-PTSD, Ehlers & Clark):

1. Identify and modify appraisals ("update" threat appraisals)
2. Trauma memory elaboration (write/revisit in detail → add context/meaning)
3. Drop safety behaviours
4. Address triggers/discriminating stimuli

8.4 Pharmacotherapy for PTSD

FIRST-LINE

DRUG	DOSE	FDA APPROVAL	NOTES
Sertraline	50–200 mg	YES	First-line; good tolerability
Paroxetine	20–60 mg	YES	First-line; discontinuation syndrome risk
Venlafaxine XR	75–300 mg	NO (but strong evidence)	Often used as first-line

SECOND-LINE / ADJUNCTS

DRUG	USE	NOTES
Prazosin	Nightmares and sleep disturbance	Alpha-1 antagonist; 1–15 mg at night; Raskind's trials; reduces nightmare frequency/intensity
Fluoxetine	PTSD (broader anxiety)	Not FDA-approved but evidence
Mirtazapine	Sleep, nightmares, depression comorbidity	15–45 mg; promotes sleep
Quetiapine	Comorbid insomnia/MDD, augmentation	25–300 mg
Risperidone	Augmentation (co-occurring psychotic features)	Low dose

DRUG	USE	NOTES
Benzodiazepines	NOT recommended for PTSD	No evidence; may worsen PTSD course by interfering with extinction; increase risk of substance dependence
Propranolol	Acute phase (within 6 hrs), memory reconsolidation blockade	Experimental; not standard of care
MDMA-assisted therapy	Phase 3 trials (MAPS); break-through therapy	Not yet approved (2026); shown >67% PTSD resolution in trials

EXAM PEARL

Benzodiazepines are CONTRAINDICATED in PTSD. They do not reduce PTSD symptoms and may worsen long-term outcomes by interfering with extinction and increasing dependence.

8.5 Psychotherapy for PTSD

TRAUMA-FOCUSED CBT (TF-CBT) / CPT / PE

These are first-line psychological treatments:

1. Cognitive Processing Therapy (CPT, Resick):

- 12 sessions; individual or group
- Identifies and challenges "stuck points", distorted beliefs about trauma/its consequences
- Written trauma account; Socratic questioning of maladaptive appraisals
- 5 themes: safety, trust, power/control, esteem, intimacy
- Evidence: Large VA trials; guideline-recommended (NICE, VA/DoD, ISTSS)

2. Prolonged Exposure (PE, Foa):

- 8–15 sessions
- Two core components:
 - **Imaginal exposure:** Repeated revisiting of trauma narrative in session + processing
 - **In vivo exposure:** Gradual approach to avoided (safe) reminders
- Based on emotional processing theory (fear network activation → modification)

3. Cognitive Therapy for PTSD (CT-PTSD, Ehlers & Clark):

- Based on their 2000 model (see 8.3)
- Memory elaboration and contextualisation
- Identifying and modifying trauma-related appraisals
- Behavioural experiments to drop safety behaviours

4. Eye Movement Desensitisation and Reprocessing (EMDR):

EXAM PEARL

EMDR is a first-line treatment for PTSD (WHO guidelines, NICE guidelines). The eye movements (bilateral stimulation) may be the specific ingredient or may be non-specific.

- Developed by Francine Shapiro (1987)
- 8-phase protocol:
- History-taking and treatment planning
- Preparation
- Assessment (target memory, negative cognition, emotion, SUDS)
- Desensitisation (bilateral stimulation while focusing on memory)
- Installation (positive cognition)
- Body scan
- Closure
- Re-evaluation
- Works via: dual attention (present safety + past trauma); bilateral stimulation may tax working memory; adaptive information processing (AIP) model
- Evidence: Comparable to TF-CBT; may be faster

9. ADJUSTMENT DISORDER (F43.2)

9.1 Diagnostic Criteria (DSM-5)

- **A.** Emotional or behavioural symptoms developing in response to identifiable stressor(s) within **3 months** of onset
- **B.** Clinically significant:
- Marked distress out of proportion to severity of stressor (cultural / contextual factors considered)
- OR significant functional impairment
- **C.** Not criteria for another disorder; not exacerbation of existing disorder
- **D.** Does not represent normal bereavement
- **E.** Once stressor (or consequences) terminates, symptoms do not persist for **>6 months**

Subtypes:

- With depressed mood
- With anxiety
- With mixed anxiety and depressed mood

- With disturbance of conduct
- With mixed disturbance of emotions and conduct
- Unspecified

9.2 ICD-11 Adjustment Disorder (6B43)

- More specific: requires preoccupation with the stressor and failure to adapt
- Duration: variable; specify if acute (<6 months) or prolonged (>6 months when stressor persists)

9.3 Key Distinctions

FEATURE	ADJUSTMENT DISORDER	MDD	PTSD
Stressor required	Yes (identifiable)	No	Yes (traumatic)
Time to onset	<3 months	Variable	<1 month (ASD) / variable (PTSD)
Duration after stressor	<6 months	Can be independent	>1 month
Trauma criterion	Not required	No	Yes (death/serious injury / sexual violence)
Intrusions/flashbacks	No	No	Yes

9.4 Treatment

- Psychotherapy first-line: supportive therapy, brief CBT, problem-solving
- Pharmacotherapy: adjunctive; treat specific symptoms (sleep, anxiety)
- Time-limited; good prognosis if stressor resolves

10. SEPARATION ANXIETY DISORDER IN ADULTS (F93.0)

10.1 Key Points

- DSM-5 now includes adult onset (previously childhood only)
- Fear of separation from attachment figures (not just children to parents)
- Adults: fear of separation from romantic partners, children, parents
- >6 months duration in adults
- Distinguish from: panic disorder (which may involve fears of being alone), dependent PD, agoraphobia

10.2 Treatment

- Cognitive-behavioural approaches

- Attachment-informed therapy
- SSRIs for pharmacological management

11. OCD SPECTRUM DISORDERS

11.1 Overview

DSM-5 groups these under "Obsessive-Compulsive and Related Disorders" (OCD):

DISORDER	CORE FEATURE	KEY FEATURE
OCD	Obsessions + compulsions	Ego-dystonic; insight present
Body Dysmorphic Disorder (BDD)	Preoccupation with perceived defect	Checking/camouflage behaviours
Hoarding Disorder	Difficulty discarding	Acquisition + cluttering
Trichotillomania	Hair pulling	Tension before, relief after
Excoriation Disorder	Skin picking	Tension/relief cycle
Illness Anxiety (Somatic OCD spectrum)	Health-related preoccupation	

11.2 Hoarding Disorder (F42.3)

EXAM PEARL

Hoarding is a separate disorder in DSM-5 (not a subtype of OCD). It has distinct neurobiology (anterior cingulate, insula, not CSTC). Response to SSRIs is poorer than OCD.

Diagnostic criteria (DSM-5):

- Persistent difficulty discarding or parting with possessions regardless of actual value
- Due to perceived need to save and distress associated with discarding
- Accumulation resulting in clutter that precludes use of living spaces
- Causes significant distress/impairment
- Not secondary to another mental disorder or medical condition

Specifiers:

- With excessive acquisition
- With good/fair/poor/absent insight

Neurobiology:

- ACC and insula hyperactivity when discarding owned items
- Not CSTC circuit, this is why SSRIs less effective

Treatment:

- **CBT for Hoarding (CBT-H):** Steketee & Frost protocol
- Motivational enhancement
- Categorization and decision-making skills training
- Sorting and discarding practice (not just exposure)
- Addressing acquisition
- Cognitive restructuring (beliefs about possessions, memory deficits concerns)
- **SSRIs:** Less effective than in OCD; paroxetine has best evidence
- Home visits essential for severe cases

11.3 Body Dysmorphic Disorder (BDD: F45.22)

EXAM PEARL

BDD shares neurobiology with OCD (CSTC) but has distinct features. Patients present to dermatology / plastic surgery more than psychiatry. Suicide risk is high.

Diagnostic criteria (DSM-5):

- Preoccupation with one or more perceived defects / flaws in physical appearance that are unnoticeable or appear slight to others
- Has performed repetitive behaviours (mirror checking, skin picking, excessive grooming, reassurance-seeking) OR mental acts (comparing to others)
- Causes significant distress / impairment
- Not better explained by eating disorder

Specifiers:

- With muscle dysmorphia (primarily in men; preoccupation with not being muscular / lean enough)
- With good / fair / poor / absent insight

Common preoccupations: Skin, nose, hair, eyes, chin, lips (face most common); any body part possible

Key features:

- **Camouflaging** (makeup, clothing, posture)
- **Mirror checking/avoidance**, both occur
- **Referential thinking**, believes others notice / stare
- **Insight** often poor to absent; may have delusional intensity
- **Suicide risk** high: lifetime ideation ~80%; attempts ~25%
- Patients seek **cosmetic procedures**, provide NO relief; often worsens BDD

Pharmacotherapy:

- High-dose SSRIs (same as OCD): fluvoxamine, fluoxetine best evidence
- Clomipramine
- Augmentation with antipsychotics if delusional features prominent
- Pimozide NOT recommended (historically used; not superior)

Psychotherapy:

- CBT for BDD (Veale protocol): exposure + response prevention, attentional training (from self-focused to environment), cognitive restructuring, mirror-based ERP
- Perceptual mirror retraining

11.4 Trichotillomania (Hair-Pulling Disorder: F63.3)**DSM-5 criteria:**

- Recurrent pulling out of one's hair
- Repeated attempts to decrease or stop
- Causes significant distress/impairment
- Not attributable to another medical condition (dermatological) or mental disorder

Sites: Scalp (most common), eyebrows, eyelashes, pubic hair, axillary hair

Phenomenology:

- Focused (tension → pull → relief) vs automatic (not consciously aware)
- Can be triggered by emotional states, boredom

Treatment:

- **HRT (Habit Reversal Training):** First-line psychological treatment
- Awareness training
- Competing response training (clenching fist instead of pulling)
- Social support + motivation
- **CBT with ACT components**
- **N-acetylcysteine (NAC):** Best pharmacological evidence (glutamate modulation); 1200–2400 mg/day
- **Clomipramine:** Some evidence; olanzapine shows benefit in trials
- SSRIs: inconsistent evidence (unlike OCD, SSRIs don't work as reliably)

11.5 Excoriation Disorder (Skin-Picking: F42.4)**DSM-5 criteria:**

- Recurrent skin picking resulting in skin lesions
- Repeated attempts to decrease/stop

- Significant distress / impairment
- Not attributable to substance or medical condition
- Not better explained by stereotypes or NSSI in another disorder

Treatment: Same as trichotillomania, HRT is first-line; NAC; clomipramine

12. ANXIETY IN MEDICAL ILLNESS

12.1 Overview

CLINICAL ANCHOR

Anxiety is the MOST common psychiatric complication of medical illness. Always rule out medical causes before diagnosing primary anxiety disorder.

12.2 Medical Conditions Causing Anxiety

SYSTEM	CONDITIONS	KEY ANXIETY FEATURES
Cardiovascular	MI, arrhythmias, mitral valve prolapse, heart failure	Palpitations, chest pain, dyspnea, mimic panic
Respiratory	COPD, asthma, pulmonary embolism, hypoxia	Breathlessness, suffocation, panic-like
Endocrine	Hyperthyroidism, hypothyroidism, pheochromocytoma, hypoglycemia, Cushing's, Addison's	Pheochromocytoma: episodic panic-like attacks (hypertension, palpitations, sweating, headache)
Neurological	Epilepsy (especially TLE ictal anxiety), encephalitis, CNS tumors, Parkinson's, MS	Ictal fear in TLE; anxiety in early dementia
Metabolic	Electrolyte imbalances, vitamin B12 deficiency, folate deficiency, hypocalcemia	Anxiety with paresthesias
Infectious	Lyme disease, HIV, COVID-19 (Long COVID)	Multi-system involvement
Autoimmune	SLE, anti-NMDA encephalitis	Psychiatric features prominent early
Medications	Corticosteroids, bronchodilators (albuterol), caffeine, theophylline, thyroid hormones	Iatrogenic anxiety

Key investigations to rule out medical causes:

- TFTs (hyperthyroidism critical to exclude)

- Urinary catecholamines/metanephrines (pheochromocytoma)
- ECG, Holter monitor (arrhythmias)
- Blood glucose (hypoglycemia)
- CBC, electrolytes, renal function
- EEG (if seizure suspected)

12.3 Cancer and Anxiety

- Anxiety is most prevalent in cancer (40–50% of patients have significant anxiety)
- Highest: lung, pancreatic, brain tumors
- Reactive anxiety + adjustment disorder + pharmacological causes
- Treatment: SSRIs, short-course benzodiazepines, CBT, supportive therapy, mindfulness

12.4 Pheochromocytoma: The Great Mimicker

- Episodic hypertension (may be paroxysmal)
- Triad: headache, sweating, palpitations
- Mimic panic attacks
- Screening: 24-hour urinary metanephrines/catecholamines, plasma free metanephrines
- CT/MRI adrenal glands once biochemistry positive
- Treatment: surgical excision; alpha-blockers first (phenoxybenzamine), then beta-blockers

13. SUBSTANCE-INDUCED ANXIETY

13.1 DSM-5 Criteria

- Panic attacks or anxiety that is prominent
- Evidence from history, examination, or laboratory that the disturbance developed during or soon after **substance intoxication or withdrawal**, or after exposure to medication
- Not better explained by independent anxiety disorder

13.2 Common Substances

SUBSTANCE/MEDICATION	ANXIETY DURING	
	Intoxication	Withdrawal
Caffeine	Yes (anxiety, panic)	Headache > anxiety
Cannabis	Yes (THC → panic, paranoia)	Mild anxiety
Stimulants (cocaine, amphetamines)	Yes (anxiety, panic, paranoia)	Post-crash dysphoria, anxiety

SUBSTANCE/MEDICATION	ANXIETY DURING	ANXIETY DURING
Alcohol	Disinhibition (some anxiety reduction)	Severe anxiety , panic, seizures, DTs
Benzodiazepines	Paradoxical agitation (rare)	Severe anxiety , panic, seizures
Opioids	Mild sedation	Anxiety, dysphoria, sweating
Hallucinogens (LSD, PCP)	Panic, paranoia, derealization	HPPD may involve anxiety
Nicotine	Mild anxiolysis	Anxiety, irritability
Corticosteroids	Anxiety, insomnia, hypomania	
Thyroid hormone	Anxiety, palpitations	
Beta-agonists (salbutamol)	Tremor, palpitations, anxiety	

EXAM PEARL

Alcohol withdrawal is medically dangerous and causes severe anxiety. BZD withdrawal is similar, both involve GABA-A receptor adaptation. Always assess temporal relationship of anxiety to substance use. If anxiety persists >1 month after cessation, consider independent anxiety disorder.

QUICK REFERENCE TABLES

Pharmacotherapy Summary Table

DISORDER	FIRST-LINE PHARMA	SECOND-LINE	AUGMENTATION
GAD	SSRI, SNRI	Buspirone, pregabalin	Quetiapine, BZD (short-term)
Panic Disorder	SSRI, SNRI	Clomipramine, BZD	
SAD	SSRI, SNRI	Phenelzine, gabapentin	BZD (short-term)
Specific Phobia	None (psychological first)	BZD PRN	D-cycloserine (augment ERP)
OCD	SSRI (high dose)	Clomipramine	Low-dose antipsychotic (risperidone, aripiprazole)
PTSD	Sertraline, paroxetine	Venlafaxine, mirtazapine	Prazosin (nightmares), quetiapine
BDD	High-dose SSRI	Clomipramine	Antipsychotic augmentation

DISORDER	FIRST-LINE PHARMA	SECOND-LINE	AUGMENTATION
Trichotillomania	NAC	Clomipramine, olanzapine	

Psychotherapy Summary Table

DISORDER	FIRST-LINE PSYCHOTHERAPY	KEY TECHNIQUES
GAD	CBT	Cognitive restructuring, worry postponement, PMR
Panic Disorder	CBT (Clark's model)	Interoceptive exposure, cognitive restructuring
SAD	CBT (Clark & Wells)	Attention training, video feedback, exposure
Specific Phobia	Systematic desensitisation / Flooding	Graded exposure hierarchy
OCD	ERP	Exposure + response prevention hierarchy
PTSD	TF-CBT, PE, CPT, EMDR	Trauma processing, appraisal modification
BDD	CBT (Veale)	ERP + attentional training
Hoarding	CBT-H	Sorting, categorizing, cognitive restructuring
Trichotillomania	HRT	Awareness + competing response training

Study Notes compiled for PG exams MD Psychiatry exit examination. Sources: Kaplan & Sadock (10th ed.), Stahl's Essential Psychopharmacology (4th ed.), Oxford Textbook of Psychiatry, DSM-5-TR, ICD-11.

Model Answers

Format: Each answer is structured for a 10-mark long essay. Target: 3–4 A4 pages of handwriting (~800–1000 words in exam). Write: introduction → body (structured with subheadings) → conclusion.

Q1. Discuss the management of Obsessive-Compulsive Disorder. [10 marks]

Answer:

Introduction

OCD is a chronic neuropsychiatric disorder characterised by obsessions (recurrent intrusive thoughts/images/urges) and/or compulsions (repetitive behaviours or mental acts). It affects 2–3% of the population, causes marked functional impairment, and ranks among the top 10 most disabling conditions (WHO). Management requires a biopsychosocial approach combining pharmacotherapy, psychotherapy, and, in refractory cases, neuromodulation.

Assessment

Before initiating treatment, a thorough assessment is essential:

- **Diagnostic confirmation**, meet DSM-5/ICD-11 criteria; rule out OCD spectrum disorders
- **Severity rating**, Yale-Brown Obsessive Compulsive Scale (Y-BOCS): 0–40; moderate = 16–23; severe = 24–31
- **Insight assessment**, good/fair/poor/absent (impacts treatment strategy)
- **Comorbidity screening**, depression (in ~50%), other anxiety disorders, tic disorders
- **OCD symptom dimensions**, contamination, harm, symmetry, forbidden thoughts, hoarding (guides ERP hierarchy)

Pharmacotherapy

Step 1, First-line: SSRIs

All five SSRIs are FDA-approved for OCD:

DRUG · DOSE RANGE

Fluoxetine 40–80 mg/day

Fluvoxamine 150–300 mg/day

Sertraline 100–200 mg/day

Paroxetine 40–60 mg/day

Escitalopram 20–40 mg/day

Key principles:

- Use **higher doses** than for depression
- Minimum trial: **10–12 weeks** (longer onset than depression)
- Gradual titration; start low to avoid early activation

Step 2, If SSRI inadequate: Switch or add clomipramine

Clomipramine (75–250 mg/day): most efficacious agent (effect size ~1.0 vs SSRIs ~0.5–0.7). Limitations: anticholinergic side effects, QTc prolongation, seizures at high doses, overdose risk. Monitor ECG and plasma levels.

Step 3, Augmentation (if partial response)

Low-dose antipsychotics added to SRI:

- **Risperidone** 0.5–2 mg/day, best RCT evidence
- **Aripiprazole** 5–15 mg/day, well-tolerated
- **Haloperidol** 2–10 mg/day, especially with comorbid tic disorder

Psychotherapy: Exposure and Response Prevention (ERP)

ERP is the gold-standard psychological treatment (effect size 1.0–1.5).

Principles:

1. Construct anxiety hierarchy (SUDS 0–100 scale)
2. Planned exposure to feared stimuli/situations
3. Response prevention, deliberately withhold compulsive ritual
4. Prolonged exposure (~45 min/session) until SUDS reduces 50%
5. Begin at moderate SUDS (40–50), not lowest

Mechanism: Inhibitory learning, new non-threatening associations form; anxiety peaks then habituates without performing compulsion.

CBT additions: Cognitive restructuring of overestimated threat/responsibility; psychoeducation; relapse prevention.

Intensive ERP: Daily sessions (2–3 weeks) for severe/refractory cases.

Combined Treatment

Meta-analyses demonstrate that **SSRI + ERP is superior to either alone**. Combination is recommended for moderate-severe OCD.

Treatment-Resistant OCD

Defined as failure of ≥ 2 SSRI trials + 1 clomipramine trial + adequate ERP. Options:

- IV clomipramine
- Transcranial magnetic stimulation (TMS), rTMS to supplementary motor area; FDA-cleared
- Deep Brain Stimulation (DBS), targets anterior limb of internal capsule (ALIC)/nucleus accumbens; FDA Humanitarian Device Exemption (2009)
- Stereotactic ablative surgery, anterior capsulotomy, anterior cingulotomy
- Gamma Knife radiosurgery, bilateral ALIC capsulotomy (non-invasive, delayed 6–12 months)

Duration and Maintenance

- Continue treatment for **1–2 years** after remission
- Taper slowly (25% dose reduction every 1–2 months)
- Relapse risk ~50% on discontinuation; higher if inadequate ERP
- Booster ERP sessions reduce relapse

Conclusion

OCD management follows a stepped approach: SSRI + ERP as foundation, augmentation for partial response, neuromodulation for refractory cases. The combination of pharmacotherapy and ERP delivers the best outcomes. Long-term maintenance is essential given the chronic course of OCD.

EXAM STRATEGY

Structure answer as: Assessment → Pharmacotherapy (Steps 1-2-3) → Psychotherapy (ERP) → Combination → Refractory cases → Duration. Examiners expect a management algorithm, not a list. Always mention Y-BOCS for assessment and ERP as the psychotherapy of choice.

Q2. Compare SSRIs and clomipramine in the treatment of OCD. [10 marks]

Answer:

Introduction

OCD is treated with serotonin reuptake inhibitors (SRIs). Both SSRIs and clomipramine (a tricyclic with potent SRI activity) are effective, but they differ in efficacy, tolerability, safety, and clinical application.

Pharmacological Mechanisms

PROPERTY	SSRIS	CLOMIPRAMINE
Primary mechanism	Selective serotonin reuptake inhibition	Serotonin + norepinephrine reuptake inhibition
Active metabolite	None (or weak)	Desmethyldlomipramine (potent NRI)
Receptor affinity	5-HT transporter highly selective	Also: H1, muscarinic, alpha-1, Na ⁺ channel blockade
Selectivity for serotonin	High	Moderate (lost with active metabolite)

Efficacy

MEASURE	SSRIS	CLOMIPRAMINE
Effect size (Y-BOCS reduction)	0.5–0.7	~1.0
Direct comparison (meta-analyses)	Inferior to clomipramine	Superior to all SSRIs
Response rate	~50–60%	~60–70%
Zohar's landmark study	Desipramine (NRI) < clomipramine	Confirms serotonin specificity

Tolerability and Side Effects

SIDE EFFECT	SSRIS	CLOMIPRAMINE
Nausea, GI symptoms	Common	Moderate
Dry mouth	Mild (paroxetine more)	Marked
Constipation	Mild	Marked
Urinary retention	Minimal	Significant
Sedation	Mild (fluvoxamine more)	Significant
Weight gain	Moderate	Significant
Sexual dysfunction	Common (delayed ejaculation, anorgasmia)	Common
Orthostatic hypotension	Rare	Significant
QTc prolongation	Citalopram ≥40 mg	Yes, ECG monitoring required
Seizures	Rare	Yes, especially >250 mg

SIDE EFFECT	SSRIS	CLOMIPRAMINE
Serotonin syndrome (with MAOIs)	Yes	Yes

Safety in Overdose

FEATURE	SSRIS	CLOMIPRAMINE
Cardiovascular	Minimal	Dangerous (QRS widening, VT, VF)
Seizures	Rare	Yes
CNS depression	Mild	Coma possible
Margin of safety	Wide	Narrow

EXAM PEARL

Clomipramine overdose = cardiac arrhythmia + seizures. SSRIs are much safer in overdose, important in OCD patients who may have comorbid depression/suicidality.

Drug Interactions

FEATURE	SSRIS	CLOMIPRAMINE
CYP enzyme inhibition	Varies (fluoxetine / fluvoxamine: significant)	Less CYP
Fluvoxamine + clomipramine		Raises clomipramine levels (CYP1A2 inhibition), risk of toxicity
MAOI interaction	Serotonin syndrome	Serotonin syndrome

Monitoring Requirements

PARAMETER	SSRIS	CLOMIPRAMINE
ECG	Baseline for citalopram	Baseline + regular
Plasma drug levels	Not routine	Yes (therapeutic range: 150–300 ng/mL; >450 ng/mL toxic)
Blood pressure	Routine	Orthostatic BP
Seizure history	Note	Contraindicated if seizure disorder

Special Populations

POPULATION	SSRIS	CLOMIPRAMINE
Pregnancy	Preferred (sertraline)	Avoid if possible
Elderly	Preferred	Avoid (anticholinergic burden, falls)
Children (>10 years)	First-line (fluvoxamine, sertraline)	Second-line
Cardiac disease	Preferred	Contraindicated / caution
Comorbid tic disorder	Add haloperidol	Can use; haloperidol augmentation

Guidelines Recommendation

- **First-line:** SSRIs (all 5 FDA-approved)
- **Second-line:** Clomipramine (or when SSRIs fail)
- **Combination:** Low-dose clomipramine can augment SSRI (careful monitoring required, elevated clomipramine levels)

Conclusion

Clomipramine is more efficacious but SSRIs are better tolerated and safer. Current guidelines recommend SSRIs as first-line due to the tolerability advantage, reserving clomipramine for SSRI failures or severe refractory cases. The ideal pharmacological approach may be to start with an SSRI, then use clomipramine augmentation or substitution in partial responders.

EXAM STRATEGY

Use a comparison table as the centrepiece. Cover: mechanism → efficacy → tolerability → safety → interactions → monitoring → special populations. End with clinical recommendation. 10 marks = ~10 distinct points.

Q3. Discuss evidence-based treatments for PTSD. [10 marks]

Answer:

Introduction

Post-traumatic stress disorder (PTSD) is a potentially chronic condition arising after exposure to actual / threatened death, serious injury, or sexual violence. It affects ~8% of trauma-exposed individuals and features intrusion, avoidance, negative cognitions / mood, and hyperarousal. Evidence-based treatments include trauma-focused psychotherapies and pharmacotherapy.

Assessment Framework

- **Diagnostic criteria:** DSM-5, 4 symptom clusters (B: intrusion, C: avoidance, D: negative cognitions/mood, E: arousal), ≥ 1 month duration
- **Severity measures:** PTSD Checklist-Civilian version (PCL-5), Clinician-Administered PTSD Scale (CAPS-5)
- **Risk assessment:** Suicidal ideation, comorbid depression/substance use

Trauma-Focused Psychotherapies (First-Line)

1. Prolonged Exposure (PE, Foa et al.)

- Duration: 8–15 weekly sessions
- Components:
 - *Imaginal exposure:* Repeated verbal retelling of trauma narrative in session
 - *In vivo exposure:* Gradual approach to avoided safe situations
 - *Psychoeducation:* Rationale for exposure
- Mechanism: Emotional processing theory, activation and modification of the fear network
- Evidence: Multiple RCTs; ~65–70% response; comparable to CPT

2. Cognitive Processing Therapy (CPT, Resick et al.)

- Duration: 12 sessions (individual or group)
- Components:
 - Written trauma account (clarifies appraisals)
 - Socratic questioning of "stuck points", maladaptive beliefs
 - Challenging across 5 domains: safety, trust, power/control, esteem, intimacy
- Mechanism: Targets distorted cognitions about trauma and its consequences
- Evidence: Large VA/DoD trials; guideline-endorsed by NICE, VA/DoD, ISTSS

3. Cognitive Therapy for PTSD (CT-PTSD, Ehlers & Clark)

- Based on Ehlers & Clark's (2000) cognitive model
- Identifies: (a) appraisals that generate current threat; (b) characteristics of trauma memory (poorly contextualised, easily triggered)
- Techniques: Trauma memory elaboration, updating problematic meanings, dropping safety behaviours, site visits
- Evidence: Multiple UK RCTs; recommended by NICE

4. Eye Movement Desensitisation and Reprocessing (EMDR, Shapiro)

- Duration: 8–12 sessions

- 8-phase protocol: History-taking → Preparation → Assessment (negative/positive cognitions, SUDS) → Desensitisation (bilateral stimulation) → Installation → Body scan → Closure → Re-evaluation
- Bilateral stimulation: Eye movements, taps, auditory tones
- Mechanism: Working memory taxation model, concurrent attention reduces vividness/emotionality; Adaptive Information Processing (AIP) model
- Evidence: Non-inferior to TF-CBT; possibly faster; endorsed by WHO, NICE, APA, VA/DoD
- Note: The eye movement component is debated; meta-analyses suggest bilateral stimulation adds benefit

Comparative efficacy:

All four therapies are equivalent in systematic reviews; choice based on patient preference, therapist training, comorbidities.

What Does NOT Work

- **Psychological debriefing (Critical Incident Stress Debriefing, CISD):** Single-session debrief immediately post-trauma, NOT effective; may be harmful by interfering with natural recovery
- **Benzodiazepines:** Contraindicated in PTSD, no evidence for PTSD symptoms; interfere with extinction; increase dependence risk
- **Non-trauma-focused therapies** (supportive counselling, relaxation alone): Less effective than TF-CBT

Pharmacotherapy

First-line (FDA-approved):

- **Sertraline** (50–200 mg/day), largest evidence base
- **Paroxetine** (20–60 mg/day), FDA-approved

Evidence-based (not FDA-approved):

- **Venlafaxine XR** (75–300 mg/day), strong evidence; comparable to SSRIs

Adjuncts:

- **Prazosin** (1–15 mg at night), alpha-1 antagonist; reduces nightmares and sleep disturbance (Raskind VA trials)
- **Mirtazapine** (15–45 mg), sleep, nightmares, comorbid depression
- **Quetiapine** (25–300 mg), augmentation, comorbid insomnia/MDD

Duration: Minimum 12 months; 2+ years if recurrent/severe.

Emerging Treatments

- **MDMA-assisted psychotherapy:** Phase 3 trials (MAPS); 67–71% no longer meet PTSD criteria post-treatment; breakthrough therapy designation
- **Ketamine infusions:** Rapid but transient PTSD symptom reduction (2–3 weeks); under investigation
- **Cannabis-based therapy:** Preliminary; insufficient evidence

Special Considerations

- **Complex PTSD (ICD-11):** TF-CBT requires modification, phase-based approach (stabilisation → trauma processing → integration); DBT skills training first
- **Military/veteran PTSD:** PE and CPT most evidence-based in VA settings; moral injury-focused approaches emerging
- **Children:** TF-CBT (Cohen et al.), involves caregivers; strong evidence

Conclusion

Trauma-focused psychotherapies (PE, CPT, CT-PTSD, EMDR) are the cornerstone of PTSD treatment. Pharmacotherapy (SSRIs, venlafaxine) is adjunctive or for those who decline/cannot access psychotherapy. Prazosin specifically addresses nightmares. The combination of trauma-focused therapy + SSRI is recommended for severe/chronic cases.

EXAM STRATEGY

Examiners want evidence-based specifics, not vague statements. Name the therapist/originator of each therapy. Mention NICE, VA/DoD guidelines. Discuss what NOT to do (debriefing, BZDs), this distinguishes a strong answer.

Q4. Describe the cognitive model of panic disorder and its therapeutic implications. [10 marks]

Answer:

Introduction

Panic disorder is characterised by recurrent unexpected panic attacks followed by persistent concern about future attacks or maladaptive behavioural changes. David Clark's (1986) cognitive model provides the most influential theoretical account of how panic attacks are generated and maintained, and directly informs cognitive-behavioural treatment.

Clark's Cognitive Model: Core Proposition

Catastrophic misinterpretation of bodily sensations is the central mechanism. Normal bodily sensations (palpitations, breathlessness, dizziness) are misinterpreted as evidence of

imminent catastrophe (heart attack, going mad, losing control).

The Panic Attack Cycle

Maintaining Factors

1. Selective attention/hypervigilance to bodily sensations

Once in the cycle, attention narrows onto the body, this amplifies perceived sensation intensity.

2. Safety behaviours

Actions taken to prevent the feared catastrophe:

- Sitting down when dizzy (prevents "confirming" collapse is not from cardiac cause)
- Calling ambulance
- Escaping the situation
- Gripping walls/surfaces

Safety behaviours MAINTAIN the disorder, they prevent disconfirmation of catastrophic beliefs and prevent learning.

3. Avoidance

Avoiding panic-provoking situations (shopping centres, exercise, caffeinated drinks) prevents exposure to disconfirming information. Leads to secondary agoraphobia.

4. Anticipatory anxiety

Fear of the next panic attack → increased physiological arousal → lower threshold for triggering next attack.

5. Interoceptive conditioning

Internal sensations become conditioned stimuli that trigger anxiety, even before reaching conscious awareness.

Neurobiological Complement: Suffocation Alarm Theory (Klein)

Klein proposed a hypersensitive CO₂/suffocation alarm, panicogenic agents (CO₂ inhalation, sodium lactate) trigger panic via this brainstem alarm, independent of cognitive appraisal. Explains why antidepressants (imipramine) prevent panic even before subjective beliefs change.

Therapeutic Implications: CBT for Panic Disorder

Clark's model maps directly onto treatment:

MODEL COMPONENT · TREATMENT TECHNIQUE

Catastrophic misinterpretation Cognitive restructuring, challenge misinterpretations, probability estimation

Selective attention/hypervigilance Attention shifting, external focus; defocus from body

Safety behaviours Behavioural experiments, drop safety behaviours; test catastrophic predictions

Avoidance In vivo exposure, graded approach to avoided situations

Interoceptive conditioning Interoceptive exposure, deliberately induce feared sensations

Anticipatory anxiety Psychoeducation + anxiety management

Interoceptive exposure exercises:

SENSATION TO INDUCE · METHOD

Palpitations/breathlessness Running on spot 1 minute

Dizziness/lightheadedness Spinning in chair

Choking sensation Breathing through narrow straw

Derealization Staring at point on wall 2 minutes

Paresthesias Overbreathing (hyperventilation)

Key CBT components:

1. Psychoeducation (fight-or-flight response, anxiety is not dangerous)
2. Breathing retraining (caution: can become safety behaviour)
3. Cognitive restructuring (alternative explanations for sensations)
4. Interoceptive exposure
5. In vivo exposure ($\pm$ situational exposure for agoraphobia)
6. Safety behaviour elimination
7. Relapse prevention

Efficacy

CBT for panic disorder: response rate 60–80%; may be superior to pharmacotherapy in long-term follow-up (lower relapse). NICE recommends CBT as first-line.

Conclusion

Clark's cognitive model identifies catastrophic misinterpretation as the engine of panic disorder. It generates a clear, testable, and therapeutically actionable formulation. CBT

derived from this model, particularly interoceptive exposure and safety behaviour elimination, is highly effective and represents the gold standard psychological treatment for panic disorder.

EXAM STRATEGY

Draw the cycle diagram in the exam, it earns marks and organises the answer. Follow with maintaining factors, then map model → treatment technique as a table. 10 marks = model description (4 marks) + therapeutic implications (6 marks).

Q5. Discuss the pharmacotherapy of Generalised Anxiety Disorder (GAD). [10 marks]

Answer:

Introduction

GAD is characterised by excessive, uncontrollable worry about multiple domains for ≥6 months, with somatic symptoms (muscle tension, fatigue, poor sleep, difficulty concentrating, irritability, restlessness). Pharmacotherapy is effective and often combined with psychotherapy. Treatment should follow a stepped approach.

Neuropharmacological Rationale

GAD involves dysregulation of three key systems:

- **Serotonergic:** dorsal raphe → amygdala/PFC; SSRIs/SNRIs restore balance
- **Noradrenergic:** locus coeruleus hyperactivity → somatic symptoms; SNRIs/buspirone reduce this
- **GABAergic:** reduced GABA-A function → benzodiazepines augment; pregabalin modulates voltage-gated calcium channels

First-Line Agents

SSRIs:

DRUG	STARTING DOSE	TARGET DOSE	FDA APPROVAL
Escitalopram	5–10 mg	10–20 mg	Yes
Paroxetine CR	12.5 mg	37.5–62.5 mg	Yes
Sertraline	25–50 mg	100–200 mg	No (but strong evidence)

Key principles:

- Start LOW (may worsen anxiety initially, initial 5-HT_{2A} activation)
- Allow 4–6 weeks at therapeutic dose before judging response

- Full effect at 8–12 weeks
- Taper slowly on discontinuation (discontinuation syndrome, especially paroxetine)

SNRIs:

DRUG	STARTING DOSE	TARGET DOSE	FDA APPROVAL
Venlafaxine XR	37.5–75 mg	75–225 mg	Yes
Duloxetine	30 mg	60–120 mg	Yes

SNRIs provide dual action on serotonin and norepinephrine, helpful when somatic symptoms dominate (muscle pain, fatigue).

Second-Line Agents

Buspirone:

- 5-HT_{1A} partial agonist (desensitises somatodendritic autoreceptors → increases 5-HT tone chronically)
- Dose: 15–60 mg / day in 2–3 divided doses
- Onset: 2–4 weeks (no acute effect, major disadvantage)
- Advantages: No dependence, no cognitive impairment, safe in elderly
- Limitations: No effect in BZD-naïve patients only works if BZD-free; must taper BZDs before switching
- Contraindicated in: Acute anxiety requiring rapid relief; prior long-term BZD users

Pregabalin:

- $\alpha_2\delta$ subunit voltage-gated Ca^{2+} channel ligand → reduces presynaptic neurotransmitter release (glutamate, norepinephrine)
- Dose: 150–600 mg / day (divided doses)
- Onset: Faster than SSRIs (~1 week)
- Evidence: Multiple RCTs; approved by EMA for GAD; FDA Schedule V (moderate abuse potential)
- Side effects: Sedation, dizziness, weight gain, dependence risk (less than BZDs)
- Advantages: Faster onset; helpful for somatic symptoms, comorbid neuropathic pain

TCAs (imipramine):

- Effective but side effect burden; second / third-line
- Useful when comorbid depression / pain is prominent

Short-Term / Adjunctive Agents

Benzodiazepines:

- Allosteric potentiators of GABA-A (increase frequency of Cl⁻ channel opening)
- Rapid onset (30 minutes to hours), value for acute symptom relief
- Preferred agents: clonazepam (long t_{1/2}, less euphoric), lorazepam
- Limitations: Tolerance, dependence, cognitive impairment (especially elderly), falls, risk in substance misuse
- Role: Short-term bridging while awaiting SSRI/SNRI response; acute exacerbations; avoid >4 weeks
- Tapering protocol: Reduce by 5–10% every 1–2 weeks

Hydroxyzine:

- H1 antihistamine; anxiolytic without dependence
- Dose: 25–50 mg TID or PRN
- Useful as benzodiazepine alternative; rapidly absorbed

Atypical antipsychotics:

- Quetiapine (25–150 mg/day): Evidence for GAD; especially with comorbid insomnia/MDD; metabolic side effects
- Olanzapine: Limited evidence; weight gain concern

Treatment Algorithm

Special Considerations

POPULATION	PREFERRED AGENT	AVOID
Elderly	Escitalopram, buspirone	Long-acting BZDs, TCAs
Pregnancy	Sertraline, escitalopram	Paroxetine (cardiac malformations Gr D), BZDs (cleft palate)
Comorbid depression	SNRI, SSRI	Buspirone (treats anxiety not depression)
Comorbid pain	Duloxetine, pregabalin	
Substance misuse history	Buspirone, SSRI	BZDs, pregabalin
Cardiac disease	Escitalopram, sertraline	TCAs (QTc), citalopram >40 mg

Duration of Treatment

- Minimum **12 months** after remission (GAD is chronic; relapse rate high)
- If >2 episodes: consider indefinite maintenance
- Taper over 4–8 weeks when discontinuing

Conclusion

SSRIs and SNRIs are first-line pharmacotherapy for GAD, combined with psychotherapy (CBT) for optimal outcomes. Buspirone and pregabalin are useful alternatives.

Benzodiazepines should be reserved for acute/short-term use. The chronic nature of GAD means long-term maintenance therapy is often required.

EXAM STRATEGY

Structure as: Neuropharmacological rationale → First-line → Second-line → Adjuncts → Algorithm → Special populations → Duration. Don't just list drugs, explain WHY each works (mechanism). That's what earns marks.

Q6. Describe the neurobiology of anxiety. [10 marks]

Answer:

Introduction

Anxiety is a normal adaptive response to perceived threat. Pathological anxiety involves dysregulation of threat detection, appraisal, and extinction circuits. The neurobiology involves multiple interacting systems: limbic structures, monoaminergic pathways, GABAergic circuits, neuropeptides, and the HPA axis.

Key Brain Structures

Amygdala:

- Central hub for fear processing
- Basolateral amygdala (BLA), receives sensory input, evaluates threat
- Central nucleus (CeA), output: drives autonomic/neuroendocrine/behavioural responses
- Two routes from thalamus:
- *Short route (thalamo-amygdala)*: Fast, automatic, rough threat detection (milliseconds)
- *Long route (thalamo-cortical-amygdala)*: Slower, refined, contextual appraisal (seconds)
- In anxiety disorders: amygdala hyperreactivity to threat cues

Prefrontal Cortex (PFC):

- **Ventromedial PFC (vmPFC)**: Extinction memory storage; inhibits amygdala fear response; reduced activity in PTSD/anxiety
- **Dorsolateral PFC (dlPFC)**: Working memory; cognitive control; relevant for worry in GAD
- **Orbitofrontal Cortex (OFC)**: Error signalling; OCD, hyperactive OFC drives "something is wrong" loop

Anterior Cingulate Cortex (ACC):

- Error monitoring, conflict resolution
- OCD: hyperactive → compulsive checking
- PTSD: hyperactive → hypervigilance

Hippocampus:

- Contextual fear conditioning
- Inhibits amygdala in safe contexts
- Reduced hippocampal volume in PTSD, GAD (stress-related neurogenesis reduction)

Bed Nucleus of Stria Terminalis (BNST):

- Sustained, diffuse, non-specific anxiety (vs amygdala's phasic, stimulus-specific fear)
- Mediates "state anxiety" and sustained vigilance
- GAD involves BNST more than amygdala

Locus Coeruleus (LC):

- Primary NE nucleus; bilateral, pontine
- Hyperactivation → sympathetic arousal (palpitations, sweating, tremor)
- Panic disorder: LC hyperactivity (yohimbine provokes panic by blocking α_2 autoreceptors)

Periaqueductal Grey (PAG):

- Freezing (dorsal PAG) and flight (ventral PAG) responses
- Panic attacks involve PAG activation

Neurotransmitter Systems

Serotonin (5-HT):

- Dorsal raphe nucleus (DRN) → amygdala, hippocampus, PFC
- 5-HT_{1A} autoreceptors (somatodendritic): inhibit DRN firing → SSRIs initially reduce 5-HT
- Chronic SSRI: autoreceptor desensitisation → increased 5-HT transmission → anxiolytic effect
- 5-HT_{2C}: in striatum; modulates repetitive behaviour (OCD relevant)
- 5-HT₃: in limbic system; ondansetron (3 antagonist), some anxiolytic evidence

GABA:

- Primary inhibitory neurotransmitter
- GABA-A receptors: ionotropic (Cl⁻ channel); benzodiazepines → allosteric potentiation → increased frequency of Cl⁻ opening

- GABA-B receptors: metabotropic; baclofen effects
- GABAergic interneurons suppress amygdala hyperactivity
- Reduced GABA-A function in anxiety disorders

Norepinephrine:

- LC → widespread cortical/limbic
- Excessive NE: hyperarousal, somatic anxiety
- Alpha-2 agonists (clonidine, guanfacine) → reduce LC firing → anxiolytic
- Propranolol: peripheral beta-blockade → reduces somatic anxiety feedback loop

Glutamate:

- NMDA receptor in amygdala: fear conditioning and memory
- D-cycloserine (partial NMDA agonist) enhances extinction learning
- mGluR2/3 presynaptic autoreceptors: reduce glutamate release → anxiolytic target
- Ketamine (NMDA antagonist) → rapid anxiolytic/antidepressant effects

Dopamine:

- Mesolimbic system: reward, salience, dysregulated in substance-induced anxiety
- D2 receptors in striatum: OCD, antipsychotic augmentation
- VTA → NAcc: prediction error signalling; threat salience

CRF/Neuropeptides:

- CRF1 receptor (amygdala, BNST): overactivation → anxiety; CRF1 antagonists, clinical trials
- Neuropeptide Y (NPY): anxiolytic; reduced in PTSD
- Substance P (NK1 receptor): anxiety, pain; NK1 antagonists, limited evidence

HPA Axis

- Acute cortisol: adaptive (mobilises energy, enhances fear memory)
- Chronic excess: hippocampal neuronal damage (glucocorticoid toxicity) → impaired contextual fear inhibition
- PTSD: dysregulated HPA (often hypocortisolaemia, not hypercortisolaemia, due to enhanced negative feedback)

CSTC Circuit (OCD-specific)

- Cortico-Striato-Thalamo-Cortical loop
- OFC hyperactivity → excessive "error signals"
- Striatal dysfunction (direct/indirect pathway imbalance) → thalamic disinhibition → repetitive motor output

- Normalised by SSRIs and ERP on FDG-PET

Summary Table

STRUCTURE/SYSTEM	ANXIETY ROLE	DISORDER LINK
Amygdala (BLA/CeA)	Fear detection and response	All anxiety disorders, PTSD
vmPFC	Extinction; amygdala inhibition	PTSD (reduced), GAD
OFC/ACC	Error signals	OCD (hyperactive)
BNST	Sustained anxiety	GAD
LC / NE	Arousal, somatic symptoms	Panic disorder
Hippocampus	Contextual fear	PTSD (volume reduction)
5-HT	Modulates fear/compulsion circuits	All anxiety disorders, OCD
GABA	Inhibits amygdala	GAD, Panic (BZD mechanism)
HPA/Cortisol	Threat response, fear consolidation	PTSD
CSTC	Habit/repetitive behaviour	OCD

Conclusion

Anxiety disorders involve dysregulation of interconnected neural circuits spanning brainstem (LC, PAG), limbic (amygdala, hippocampus, BNST), and cortical (PFC, OFC, ACC) structures, modulated by serotonin, norepinephrine, GABA, and glutamate systems. Understanding this neurobiology explains both the phenomenology of anxiety disorders and the mechanism of effective treatments.

EXAM STRATEGY

Draw a circuit diagram if time permits. Structure as: brain structures → neurotransmitters → HPA axis → summary table. This is a high-yield question, appears almost every year in some form.

Q7. What is Y-BOCS? Describe its structure and clinical utility. [10 marks]

Answer:

Introduction

The Yale-Brown Obsessive Compulsive Scale (Y-BOCS) is the gold-standard clinician-administered instrument for assessing OCD severity. Developed by Goodman et al. (1989),

it evaluates the severity of obsessions and compulsions independently of their content, making it sensitive to treatment change and useful across OCD symptom dimensions.

Development and Rationale

Prior to Y-BOCS, rating scales conflated symptom content with severity. Y-BOCS solved this by:

- Rating severity regardless of the type of obsession/compulsion
- Using a semi-structured interview format
- Providing independent subscales for obsessions and compulsions

Structure

Format: Semi-structured clinician-administered interview

Total items: 10 (5 for obsessions, 5 for compulsions)

Scoring: Each item 0 (none) to 4 (extreme) → total range 0–40

Five dimensions (same for both subscales):

ITEM	WHAT IT MEASURES	SCALE ANCHORS
1/6. Time occupied	Hours per day consumed by O/C	0 = none; 4 = >8 hr/day or nearly constant
2/7. Interference with functioning	Social/occupational disruption	0 = none; 4 = incapacitating
3/8. Distress/anxiety	Subjective distress caused by O/C	0 = none; 4 = near-constant, disabling
4/9. Resistance	Effort to resist O/C	0 = always resists; 4 = completely yields
5/10. Control	Ability to stop O/C	0 = complete control; 4 = no control

EXAM PEARL

Note that for Resistance (item 4/9): lower score = MORE resistance. This is the opposite direction from other items. Common exam trap.

Severity Classification

TOTAL SCORE · SEVERITY

0–7 Subclinical

8–15 Mild

16–23 Moderate

24–31 Severe

32–40 Extreme

Y-BOCS Symptom Checklist

The Y-BOCS is administered with a symptom checklist (separate from the severity scale) that identifies which obsessions and compulsions are present across all dimensions:

- Contamination obsessions / washing compulsions
- Harm obsessions / checking compulsions
- Symmetry obsessions / ordering compulsions
- Forbidden thoughts (sexual, religious, aggressive) / mental neutralising
- Hoarding obsessions / collecting compulsions
- Somatic obsessions / body-checking compulsions

Response Criteria

OUTCOME · Y-BOCS CRITERION

Response $\geq 35\%$ reduction from baseline

Remission Score ≤ 8 (some use ≤ 12)

Partial response 25–34% reduction

Non-response $< 25\%$ reduction

Worsening Increase from baseline

Insight Assessment (Part of Administration)

Before completing the scale, clinician rates insight:

- Good insight: recognises obsessions as definitely / probably not true
- Fair insight: thinks they may be true
- Poor insight: thinks they are probably true
- Absent insight / delusional beliefs: completely convinced

Updated Versions

Y-BOCS-II (2010):

- Revised to avoid problems with resistance item interpretation
- Added insight item to severity scale
- Broader content coverage; better psychometrics

CY-BOCS: Children's version (Scahill et al.), validated for ages 6–17

Other OCD Scales

SCALE	FORMAT	SUBSCALES	UTILITY
OCI-R	Self-report, 18 items	6 (washing, checking, ordering, obsessing, hoarding, neutralising)	Screening, dimensional assessment
DOCS	Self-report, 20 items	4 OCD dimensions	Research; dimensional severity
PADUA Inventory	Self-report, 60 items	5	Research use

Clinical Utility

- 1. Baseline assessment:** Establish severity before starting treatment
- 2. Treatment monitoring:** Administer every 4–6 weeks during treatment; Y-BOCS guides decisions:
 - <25% reduction at 8 weeks → consider dose increase or augmentation
 - ≥35% reduction = treatment response
- 3. Research:** Standard outcome measure in all OCD clinical trials
- 4. Communicating with patients:** Numeric score helps patients track progress concretely
- 5. Disability assessment:** Supports documentation for leave, accommodation, treatment funding
- 6. Predicting prognosis:** Higher baseline Y-BOCS, poorer insight, and washing/contamination dimension predict poorer pharmacotherapy response

Limitations

- No self-report version (clinician-administered; requires training)
- Resistance item counterintuitive (lower = better)
- Does not assess functional impairment comprehensively
- Not sensitive to rapid changes (designed for 4–6 week intervals)
- Symptom checklist takes additional time

Conclusion

Y-BOCS remains the definitive instrument for OCD severity assessment in both clinical and research settings. It provides a reliable, valid, and content-independent measure of obsession and compulsion severity, guides treatment decisions, and defines response and remission. Every clinician managing OCD should be proficient in its administration.

EXAM STRATEGY

Q on Y-BOCS almost always asks for: structure (10 items, 5+5, scoring 0-4) + severity thresholds + response criteria + clinical utility. Don't forget the symptom checklist distinction and the resistance item quirk.

Q8. Discuss the management of treatment-resistant OCD. [10 marks]

Answer:

Introduction

Treatment-resistant OCD is defined as failure to achieve a clinically significant response ($\geq 35\%$ Y-BOCS reduction) despite adequate trials of at least 2–3 SSRIs at maximum tolerated doses for ≥ 12 weeks each, one clomipramine trial, and adequate exposure and response prevention (ERP) therapy. It affects approximately 40–60% of OCD patients and represents a significant clinical challenge.

Assessment of "True" Resistance

Before labelling a case resistant, confirm:

- Adequate dose (SSRIs at high-end: fluoxetine 80 mg, fluvoxamine 300 mg, etc.)
- Adequate duration (≥ 12 weeks at therapeutic dose, longer than for depression)
- Adherence (plasma level confirmation if needed)
- Adequate ERP (≥ 20 sessions by trained therapist; patient cooperation)
- Comorbidities identified (depression, ADHD, autism, personality disorder) and treated
- Insight level (poor insight predicts poorer response)

Step-by-Step Management

Step 1: Optimise existing treatment

- Ensure SSRI at maximum dose; consider augmenting with clomipramine
- Caution: fluvoxamine inhibits CYP1A2 → raises clomipramine to toxic levels (max clomipramine 75–100 mg if combining with SSRIs)
- Reinforce ERP adherence; consider intensive ERP (daily sessions)

Step 2: Switch SRI

- If SSRI failed: try second SSRI (different CYP profile)
- If 2 SSRIs failed: clomipramine (monotherapy or low-dose augmentation)
- Effect size of clomipramine > all SSRIs; may work when SSRIs fail

Step 3: Antipsychotic augmentation

AGENT	DOSE	EVIDENCE LEVEL
Risperidone	0.5–2 mg/day	Best RCT evidence
Aripiprazole	5–15 mg/day	Good evidence; well-tolerated
Haloperidol	2–10 mg/day	Especially with tic comorbidity
Quetiapine	25–200 mg/day	Moderate evidence; helps insomnia/anxiety
Olanzapine	2.5–10 mg/day	Limited evidence; weight gain

Mechanism: D2 blockade in striatum augments SRI-mediated 5-HT effects on CSTC circuit.

Step 4: Glutamate modulators

- **Riluzole** (50–100 mg/day): Reduces glutamate release; several open-label studies positive
- **Memantine** (10–20 mg/day): NMDA antagonist; case series and small RCTs
- **N-acetylcysteine (NAC)** (2400 mg/day): Cystine / glutamate antiporter modulation; positive RCTs in augmentation
- **Topiramate** (25–200 mg/day): Glutamate reduction; some evidence

Step 5: IV clomipramine

- Bypasses first-pass metabolism → higher peak plasma concentrations
- Protocol: 25–75 mg IV in dextrose over 45–90 minutes, 2–3 times per week
- Evidence: Koran et al. studies; Indian data (Bhave & Bhave 1986); responds even when oral fails
- Availability: Limited to centres with expertise

Step 6: Neuromodulation

TMS (Transcranial Magnetic Stimulation):

- rTMS to supplementary motor area (SMA) or dorsal medial PFC
- FDA 510(k) cleared (2018) via Brainsway deep TMS (dTMS, H7 coil)
- Sessions: 20 sessions over 4 weeks (response rate ~40%)
- Non-invasive; minimal side effects; no anaesthesia

Step 7: Neurosurgery (Last Resort)

Criteria for neurosurgical referral:

- Severe OCD (Y-BOCS ≥ 30)
- Failed ≥ 3 adequate SRI trials + adequate ERP
- Significant disability (unable to work/live independently)
- Multidisciplinary consensus

- Full informed consent; ethics review

PROCEDURE	TARGET	TYPE	RESPONSE RATE
DBS	ALIC/NAcc/STN	Reversible	40–60%
Anterior capsulotomy	ALIC	Ablative (lesion)	40–70%
Anterior cingulotomy	ACC	Ablative	25–40%
Gamma Knife capsulotomy	ALIC	Radiosurgical	45–50% (delayed 6–12 months)

DBS details:

- FDA Humanitarian Device Exemption (2009), not full approval
- Reversible, adjustable (stimulation parameters)
- Requires multidisciplinary team: neurosurgeon, psychiatrist, neurologist, psychologist, ethics board
- Targets: ALIC (most used), ventral striatum, NAcc, BNST, STN (bilateral)
- Complications: infection, haemorrhage (~1–2%), mood changes, hypomania

Emerging Strategies

- **Ketamine infusions:** Rapid but transient; NMDA mechanism; under investigation
- **Psilocybin:** Very preliminary; theoretical (5-HT_{2A} agonism); case reports only
- **MDMA-assisted therapy:** Not specifically for OCD; very early data

Conclusion

Treatment-resistant OCD requires systematic escalation through optimised SRI therapy, antipsychotic augmentation, glutamate modulators, IV clomipramine, neuromodulation, and finally neurosurgery for the most severe cases. Ongoing ERP throughout all pharmacological steps is essential. Multidisciplinary care with realistic goal-setting is paramount.

EXAM STRATEGY

Define resistance first. Then list escalation steps in order, examiners expect a structured algorithm. For neurosurgery section: name DBS, capsulotomy, cingulotomy, Gamma Knife with targets and response rates. The DBS FDA HDE detail (2009) is a distinguishing fact that earns marks.

Q9. Discuss Body Dysmorphic Disorder: clinical features, assessment, and management. [10 marks]

Answer:

Introduction

Body Dysmorphic Disorder (BDD) is classified in DSM-5 under Obsessive-Compulsive and Related Disorders. It involves preoccupation with perceived defects in physical appearance that are unnoticeable or appear slight to others, causing significant distress and functional impairment. It is underdiagnosed because patients seek cosmetic treatment, not psychiatric care.

Epidemiology

- Prevalence: 1.7–2.4% in general population
- Higher in dermatology (9–12%), plastic surgery (6–15%) settings
- Sex: Approximately equal; men more commonly have muscle dysmorphia
- Onset: Typically adolescence (mean 16–17 years)
- Course: Chronic; mean duration before treatment 9–10 years

Clinical Features

Core symptom: Preoccupation with ≥ 1 perceived appearance flaw, perceived as visible and significant, but objectively absent or minimal.

Common preoccupation sites: Skin (acne, scarring, texture) most common; nose, hair, eyes, lips, chin, face overall; any body part possible.

Muscle dysmorphia (bigorexia):

- Primarily males (but not exclusively)
- Preoccupation with not being muscular/lean enough despite large musculature
- Exercise obsessions, steroid misuse, dietary restriction/supplements

Repetitive behaviours (98% of patients):

- Mirror checking or deliberate mirror avoidance
- Skin picking (camouflage disguised)
- Excessive grooming (hair combing, styling)
- Excessive make-up/camouflage clothing
- Reassurance-seeking from others
- Comparing own appearance to others
- Internet photo searching for comparisons

Cognitive features:

- Overvalued or delusional ideation about defect
- Referential thinking: believes others stare at/mock the perceived defect
- Self-referential cognitive bias: interprets neutral/positive feedback as confirmation of ugliness

Insight spectrum:

- Good/fair insight: knows the belief may be irrational
- Poor insight: belief is probably true
- Delusional beliefs: complete certainty, historically "delusional disorder, somatic type"
- DSM-5 combined BDD with delusional form; now a single spectrum with insight specifier

Suicide risk: HIGH

- Lifetime suicidal ideation: ~80%
- Suicide attempts: ~25–30%
- Rates higher than MDD, bipolar disorder
- Risk factors: delusional insight, comorbid MDD, prior attempts, substance misuse

Differential Diagnosis

CONDITION · DISTINGUISHING FEATURE

Anorexia nervosa Preoccupation specifically with weight/shape

Social anxiety disorder Fear of social scrutiny (performance); not appearance preoccupation

Major depression Negative self-evaluation global; not appearance-focused

OCD Ego-dystonic obsessions; multiple domains; content different

Delusional disorder (somatic) DSM-5 now subsumed under BDD

Illness anxiety disorder Preoccupation with disease/medical condition, not appearance

Assessment

- **BDD-YBOCS:** Yale-Brown Obsessive Compulsive Scale modified for BDD; 12 items; same 5-domain structure; severity thresholds parallel to OCD
- **BDD-Q (questionnaire):** Screening tool
- Full suicide risk assessment (essential given high risk)
- Ask explicitly, patients rarely volunteer appearance concerns without direct inquiry

Management

Pharmacotherapy:

DRUG	DOSE	EVIDENCE
Fluoxetine	40–80 mg/day	Best evidence (Phillips et al. RCTs)

DRUG	DOSE	EVIDENCE
Fluvoxamine	150–300 mg/day	Multiple positive trials
Sertraline	100–200 mg/day	RCT evidence
Escitalopram	20–40 mg/day	Open-label studies
Clomipramine	100–250 mg/day	Effective; second-line

Key points:

- Use **high doses** (same principle as OCD)
- Allow **12 weeks** minimum trial
- SSRIs effective regardless of insight level (even delusional BDD)
- **Do NOT use antipsychotics alone** for delusional BDD, SSRIs are still required
- Antipsychotic augmentation: consider if delusional features prominent
- **Do NOT prescribe pimozide** (historical use; not superior; harms outweigh benefits)

Psychotherapy, CBT for BDD (Veale protocol):

Components:

1. **Psychoeducation**, BDD model; how attention and safety behaviours maintain disorder
2. **Attention retraining**: From self-focused internal (bodily sensations, imagined defect) to external (surroundings, other people)
3. **Exposure and Response Prevention (ERP)**:
4. Exposure to avoided social situations
5. Mirror ERP: structured mirror use (holistic, not zoomed in; with guidance)
6. Drop safety behaviours (camouflage, reassurance-seeking, checking)
7. **Cognitive restructuring**: Challenge "appearance assumptions" and self-referential beliefs
8. **Behavioural experiments**: Test predictions about others' reactions
9. **Imagery rescripting**: For early-life experiences that shaped appearance-related beliefs

What NOT to do:

- **Cosmetic procedures / dermatological treatments**: Should NOT be provided
- Provide no lasting relief in BDD (>80% remain preoccupied or find new concerns)
- May worsen BDD by reinforcing the belief that the defect is real
- Surgeons/dermatologists are encouraged to screen for BDD before any procedure

Conclusion

BDD is a serious, often underdiagnosed condition with high suicide risk. It requires high-dose SSRIs and BDD-specific CBT. Cosmetic procedures are contraindicated. Early recognition in dermatology and plastic surgery settings is essential.

EXAM STRATEGY

Cover epidemiology → clinical features (types, repetitive behaviours, insight, suicide risk) → differential diagnosis → management. The "what not to do" section (no cosmetic procedures, no pimozide alone) earns marks. High suicide risk is a key clinical point.

Q10. Discuss agoraphobia: clinical features and management. [10 marks]

Answer:

Introduction

Agoraphobia is an anxiety disorder characterised by marked fear or anxiety about situations from which escape might be difficult or help unavailable in the event of a panic attack or panic-like symptoms. DSM-5 made agoraphobia an independent diagnosis (no longer requiring co-occurring panic disorder).

Diagnostic Criteria (DSM-5: F40.00)

Fear/anxiety related to ≥ 2 of:

1. Using public transport (buses, trains, ships, planes)
2. Being in open spaces (car parks, marketplaces, bridges)
3. Being in enclosed spaces (shops, theatres, cinemas)
4. Standing in line or being in a crowd
5. Being outside of home alone

Situations feared because escape might be difficult or help unavailable if panic/panic-like symptoms develop.

Additional criteria: almost always provokes fear; actively avoided or endured with distress; disproportionate to actual danger; persistent (≥ 6 months); significant impairment.

Clinical Features

- **Cognitive:** "I will collapse/faint/lose control and nobody will help me"; "I cannot escape"; "I will be humiliated"
- **Avoidance:** Homebound in severe cases; requires companion to travel; restricts radius of independent activity
- **Safety behaviours:** Carrying phone, staying near exits, having medication, travelling at quiet times
- **Comorbidity:** Panic disorder (30–50%); major depression (common); GAD, social anxiety

Relationship to Panic Disorder

FEATURE	AGORAPHOBIA ONLY (DSM-5)	PANIC DISORDER + AGORAPHOBIA
Panic attacks	Not required	Present (unexpected)
Fear focus	Situation (escape difficulty)	Both attack itself + situational avoidance
Severity	Often severe due to avoidance	Variable
Treatment priority	Situational exposure	Both panic + situational exposure

Management

Assessment:

- Identify agoraphobic situations (hierarchy of feared places)
- Rate fear and avoidance (0–10 scale)
- Assess safety behaviours and companions
- Screen for panic disorder, depression, substance use

Psychotherapy, First Line:

Graded Exposure Therapy:

- Based on habituation and inhibitory learning
- Steps:
- Psychoeducation (anxiety is not dangerous; avoidance maintains fear)
- Fear hierarchy construction (8–12 items; SUDS 0–100)
- Graded exposure (start ~40–50 SUDS; progress up hierarchy)
- Therapist-accompanied initially → partner-assisted → independent
- Drop safety behaviours gradually
- Relapse prevention

Example hierarchy:

STEP	SITUATION	SUDS
1	Stand at front door alone	30
2	Walk to end of road and back	40
3	Visit corner shop (off-peak)	50
4	Travel one bus stop with companion	60

STEP	SITUATION	SUDS
5	Short trip to supermarket (quiet)	70
6	Supermarket at busy time alone	85
7	Shopping mall, busy day	90

Partner-assisted exposure: If homebound; family members trained to support exposure without accommodation.

Internet-delivered CBT: For those unable to attend; some evidence base.

Pharmacotherapy:

- **SSRIs/SNRIs:** First-line if panic disorder comorbid; helpful for overall anxiety reduction enabling exposure
- Sertraline, escitalopram, venlafaxine XR
- **Benzodiazepines:** Short-term acute relief; avoid long-term (may interfere with habituation during exposure)
- **Combination (SSRI + CBT):** Superior to either alone for severe agoraphobia with panic

Key principle: Pharmacotherapy WITHOUT exposure therapy has high relapse. Exposure is the active ingredient.

Prognosis

- Moderate with treatment: 60–70% improvement in avoidance
- Poorer prognosis: longer duration, comorbid depression, housebound status, poor social support
- Without treatment: progressive worsening; risk of permanent housebound status

Conclusion

Agoraphobia causes severe functional impairment through avoidance. Graded exposure therapy is the cornerstone of treatment; pharmacotherapy (SSRIs) supports exposure work. Full independence is achievable with adequate treatment, though severe cases require prolonged, intensive work.

EXAM STRATEGY

Include a sample fear hierarchy (tables score well) and explain that pharmacotherapy alone without exposure has high relapse. Mention the DSM-5 change (independent diagnosis). Distinguish from panic disorder clearly.

Model Answers compiled for PG exams MD Psychiatry exit examination. All clinical vignettes are fictitious. Sources: Kaplan & Sadock (10th ed.), Stahl's Essential Psychopharmacology (4th ed.), Oxford Textbook of Psychiatry, DSM-5-TR, ICD-11, NICE Guidelines.

Mnemonics & Memory Tricks

How to use this file: Read each mnemonic actively, cover the expansion and attempt to recall before checking. Spaced repetition: review on Day 1, Day 3, Day 7, Day 14.

SECTION A: DIAGNOSTIC CRITERIA

1. GAD: The 6 Somatic Symptoms (DSM-5)

MNEMONIC

FIRMS C

LETTER · SYMPTOM

F Fatigue

I Irritability

R Restlessness (keyed up / on edge)

M Muscle tension

S Sleep disturbance

C Concentration difficulty (mind going blank)

Usage: GAD needs ≥ 3 of 6 (adults); ≥ 1 of 6 (children). Duration: 6 months. Plus: excessive worry + difficulty controlling it.

EXAM PEARL

"FIRMS C", think of a firm grip on a couch, anxiously. The anxious person is FIRM (tense, fatigued, irritable, restless, muscle tension, can't sleep or concentrate).

2. Panic Attack: 13 Symptoms

MNEMONIC

CHEST PAIN DC² F

LETTER · SYMPTOM

C Chest pain / discomfort

H Hot flushes or chills

E Elevated heart rate (palpitations, pounding)

S Shaking / trembling

T Tingling / paresthesias

P Paresthesias (covered above) → use for: **P**anic, fear of dying

A Air (shortness of breath / smothering)

I I'm going crazy (fear of losing control)

N Nausea / abdominal distress

D Derealization / Depersonalization

C Choking sensation

2 (2 D's = Derealization + Depersonalization)

F Faintness / dizziness / lightheadedness

Shortcut: ≥4 symptoms, abrupt onset, peaks within minutes. Unexpected = panic disorder.

EXAM PEARL

There are exactly 13 symptoms. ≥4 required for a panic attack. The most cardiac-mimicking (chest pain, palpitations, dyspnoea) are the ones that send patients to the ED.

3. OCD Neurocircuitry: CSTC Components

MNEMONIC

COrti**C**o **S**Triat**T**o **T**halamo**C**ortical

Broken down:

- Caudate nucleus (striatum), gates repetitive behaviour
- OFC (orbitofrontal cortex), error signal generator ("something is wrong")
- Striatum (caudate + putamen), direct and indirect pathways
- Thalamus, disinhibited in OCD → drives cortex
- Thalamo → back to cortex (loop closes)

- ACC (anterior cingulate cortex), conflict/error monitoring

Direct pathway (GO): Striatum → inhibits GPi → releases thalamus → drives cortex

Indirect pathway (STOP): Striatum → GPe → STN → GPi → inhibits thalamus

In OCD:

MNEMONIC

OCD = Overactive OFC, Crippled Caudate, Thalamus Too Talkative

- OFC fires excessive "error signals"
- Caudate fails to gate the signal
- Thalamus disinhibited → keeps looping → compulsions

4. PTSD: 4 Symptom Clusters (DSM-5)

MNEMONIC

MNEMONIC: "I-A-N-A" (I Am Never Alright)

LETTER	CLUSTER	KEY SYMPTOMS
I	Intrusion (Criterion B)	Flashbacks, nightmares, intrusive memories, physiological reactivity
A	Avoidance (Criterion C)	Avoid thoughts/feelings + external reminders
N	Negative cognitions & mood (Criterion D)	Amnesia, distorted blame, negative beliefs, emotional numbing, anhedonia
A	Arousal & reactivity (Criterion E)	Hypervigilance, startle, irritability, recklessness, sleep, concentration

Duration: ≥1 month. Stressor: Criterion A (traumatic event, actual/threatened death, serious injury, sexual violence).

EXAM PEARL

DSM-IV had 3 clusters (B, C, D). DSM-5 expanded to 4 by splitting avoidance/numbing into: Avoidance (C) + Negative cognitions/mood (D). Duration threshold did not change.

5. PTSD Symptom Cluster D: Negative Cognitions (7 items)

MNEMONIC

BRAIN-AD

LETTER · SYMPTOM

B Blame of self / others (distorted, persistent)

R Reduced participation in activities

A Amnesia for important aspects of trauma

I Inability to experience positive emotions (emotional numbing)

N Negative beliefs about self / world ("I am bad; nowhere is safe")

A Alienation from others (detachment, estrangement)

D Dysphoric emotion state (persistent fear, horror, guilt, shame, anger)

6. Y-BOCS: 5 Severity Domains

MNEMONIC

MNEMONIC: "TI DRC" (Time Is Daily Resistance Crucial)

LETTER · DOMAIN

T Time occupied by obsessions / compulsions

I Interference with functioning

D Distress caused

R Resistance (effort to resist)

C Control (ability to stop)

Each scored 0–4, $\times 2$ subscales (O + C) = 0–40 total.

EXAM PEARL

Resistance item is **reverse-scored** in clinical intuition, lower score = more resistance = better. 0 = always resists; 4 = completely yields. Don't confuse direction.

7. OCD Insight Specifiers: 4 Levels

MNEMONIC

MNEMONIC: "GFPA" (Good Follows Poor to Absent)

- Good insight, knows obsessions probably not true
- Fair insight, thinks they may be true
- Poor insight, thinks they are probably true
- Absent/delusional, completely convinced (delusional intensity)

SECTION B: PHARMACOLOGY

8. SSRIs Approved for OCD: 5 Drugs

MNEMONIC

FLOPS

LETTER · DRUG

F Fluoxetine

L (f) Luvoxamine

O (escital)Opram, Escitalopram (off-label high dose)

P Paroxetine

S Sertraline

Note: Officially, 5 SSRIs approved by FDA: Fluoxetine, Fluvoxamine, Paroxetine, Sertraline + Clomipramine (TCA). Escitalopram is used at high doses off-label.

EXAM PEARL

Fluvoxamine was the FIRST SSRI approved by FDA for OCD (1994). All 5 SSRIs require HIGHER doses and LONGER trials than for depression (10–12 weeks minimum).

9. Clomipramine Side Effects

MNEMONIC

CAST-OQ

LETTER · SIDE EFFECT

C Constipation (anticholinergic)

A Anticholinergic bundle (dry mouth, urinary retention, blurred vision)

S Sedation (H1 blockade)

T Tachycardia / QTc prolongation

O Orthostatic hypotension

Q QT seizures (at doses >250 mg)

10. PTSD First-Line Pharmacotherapy

MNEMONIC

MNEMONIC: "SPVP" (Sertraline Paroxetine Venlafaxine Prazosin)

- Sertraline, FDA-approved, first-line
- Paroxetine, FDA-approved, first-line
- Venlafaxine XR, strong evidence, not FDA-approved but widely used
- Prazosin, nightmares specifically (alpha-1 antagonist)

EXAM PEARL

Only 2 drugs are FDA-approved for PTSD: sertraline and paroxetine. Venlafaxine is treated as equivalent in most international guidelines. Prazosin specifically targets nightmares (Raskind trials).

11. Augmentation Options for OCD (Antipsychotics)

MNEMONIC

MNEMONIC: "RHAHOQ" (Really Harsh Augmentation Has Outstanding Questions)

LETTER · DRUG

R Risperidone (best RCT evidence)

H Haloperidol (best with tic comorbidity)

A Aripiprazole (well-tolerated, good evidence)

H Haloperidol (repeated for emphasis, tics)

O Olanzapine (some evidence; weight gain)

Q Quetiapine (anxiety / insomnia comorbidity)

Simpler version: "RHAOOQ" → Risperidone, Haloperidol, Aripiprazole, Olanzapine, Quetiapine

12. Drugs Contraindicated / Not Recommended in PTSD

MNEMONIC

BAD for PTSD

LETTER · DRUG

B Benzodiazepines (contraindicated, no evidence; worsens long-term outcome)

A Antipsychotics (not first-line; only for augmentation in severe cases)

D Debriefing (psychological debriefing / CISM, not only doesn't help, may harm)

SECTION C: THERAPY CONCEPTS

13. ERP Principles: Key Rules

MNEMONIC

HELP NO MORE

LETTERS · PRINCIPLE

Hierarchy Build anxiety hierarchy (SUDS 0–100); 8–12 steps

Exposure Direct contact with feared stimulus

Long exposure Minimum 45 minutes; wait for anxiety to peak and then drop $\geq 50\%$

Prevent Response prevention, NO ritual during exposure

No escape Premature termination reinforces avoidance

Order Start at moderate SUDS (~40–50), not lowest

Mode In vivo preferred; imaginal for harm obsessions

Own it Patient takes control gradually (therapist-assisted → independent)

Repeat Regular practice; generalise across situations

Extinction New inhibitory learning; anxiety peaks then habituates

14. Clark's Panic Model: 4 Key Maintaining Factors

MNEMONIC

MNEMONIC: "SASA" (Safety And Selective Attention)

LETTER · FACTOR

S Selective attention/hypervigilance to body sensations

A Anticipatory anxiety (between attacks)

S Safety behaviours (sitting down, calling ambulance, avoiding exercise)

A Avoidance of feared situations and triggers

15. Specific Phobia Types: DSM-5 Classification

MNEMONIC

MNEMONIC: "ANBSO" (Animals, Natural, Blood, Situational, Other)

LETTER	TYPE	EXAMPLES
A	Animal	Spiders, dogs, insects, snakes
N	Natural environment	Heights, storms, water, fire
B	Blood-injection-injury	Needles, blood draws, medical procedures
S	Situational	Airplanes, elevators, enclosed places, driving
O	Other	Vomiting, choking, clowns, loud sounds

EXAM PEARL

BII phobia is unique, **vasovagal diphasic response**: initial tachycardia (sympathetic) → paradoxical bradycardia + hypotension (vasovagal). Treatment: **applied tension technique** (tense large muscles to raise BP before fainting). This is the **ONLY** phobia where relaxation is contraindicated.

16. OCD Spectrum Disorders: DSM-5 OCRD Chapter

MNEMONIC

MNEMONIC: "HOBTE" (HOBby TEasing)

LETTER · DISORDER

H Hoarding Disorder

O OCD

B Body Dysmorphic Disorder (BDD)

T Trichotillomania (hair pulling)

E Excoriation Disorder (skin picking)

EXAM PEARL

Hoarding has DISTINCT neurobiology from OCD (ACC + insula, NOT CSTC). SSRIs are LESS effective in hoarding. Treatment is CBT-H (Steketee & Frost protocol), not standard ERP.

17. EMDR: 8 Phases

MNEMONIC

HP ADI BC RE

LETTERS · PHASE

H History-taking and treatment planning

P Preparation

A Assessment (target image, negative cognition, VoC, emotion, SUDS, body location)

D Desensitisation (bilateral stimulation + focus on memory)

I Installation (positive cognition)

B Body scan

C Closure

RE Re-evaluation

EXAM PEARL

EMDR Phase 3 (Assessment) includes: target image, negative cognition (NC), desired positive cognition (PC), Validity of Cognition (VoC, 1–7 scale), associated emotion, SUDS, body location of disturbance. This is the most detailed phase.

18. Cognitive Processing Therapy (CPT): 5 Stuck-Point Themes

MNEMONIC

MNEMONIC: "STEEP" (Safety, Trust, Esteem, Energy, Power)

LETTER · THEME

S Safety

T Trust

E Esteem

E Emotions (added for completeness)

P Power and Control

Official 5 themes: Safety, Trust, Power/Control, Esteem, Intimacy

KEY INSIGHT

Corrected MNEMONIC: "STPEI" → "STeP-In" = Safety, Trust, Power, Esteem, Intimacy

19. Neurobiology of Anxiety: Key Structures

MNEMONIC

MNEMONIC: "ALPHA" (Amygdala, Locus coeruleus, PFC, Hippocampus, Anterior cingulate)

LETTER	STRUCTURE	ROLE
A	Amygdala	Fear detection, phasic fear response
L	Locus Coeruleus	NE hyperactivation → somatic anxiety, panic
P	PFC (vmPFC)	Extinction memory; inhibits amygdala

LETTER	STRUCTURE	ROLE
H	Hippocampus	Contextual fear conditioning; PTSD volume loss
A	Anterior cingulate	Error monitoring; OCD hyperactivity

KEY INSIGHT

Added: BNST = Bed Nucleus of Stria Terminalis = **B**etter named the **B**ackground anxiety generator. Sustained / diffuse anxiety. GAD.

20. Hoarding Disorder: Key Distinguishing Features from OCD

MNEMONIC

PAID OFF

FEATURE	HOARDING	OCD
Psychology	Positive emotion about possessions	Anxiety / disgust (ego-dystonic)
Acquisition	Excessive acquiring	Not typical
Insight	Poor to variable	Usually fair-good
Distress source	From DISCARDING (not possessions themselves)	From obsessions (threat)
OFC-CSTC	NOT CSTC; ACC / insula	CSTC primary
First-line therapy	CBT-H (Steketee & Frost)	ERP
Farmacotherapy	SSRIs LESS effective	SSRIs highly effective

SECTION D: QUICK-FIRE RECALL

Rapid-Fire Card 1: Duration Criteria

DISORDER · DURATION REQUIRED

GAD ≥6 months

Panic Disorder ≥1 month persistent concern after attack

Social Anxiety ≥6 months

Specific Phobia ≥6 months

OCD Not specified (but time-consuming >1 hr / day)

PTSD ≥1 month

Acute Stress Disorder 3 days – 1 month

Adjustment Disorder ≤6 months after stressor terminates

Agoraphobia ≥6 months

Rapid-Fire Card 2: Neurobiology One-Liners

DISORDER · KEY NEUROBIOLOGY FACT

GAD BNST (sustained anxiety), vmPFC hypoactivity

Panic Disorder LC (NE), PAG, CO₂ suffocation alarm (Klein)

OCD CSTC circuit; OFC + caudate hypermetabolism on PET

PTSD Amygdala hyperreactivity; hippocampal volume loss; vmPFC hypoactivity

Social Anxiety Amygdala hyperreactivity to social threat cues

Specific Phobia BII: vasovagal diphasic response (unique)

Rapid-Fire Card 3: Y-BOCS Severity Thresholds

SCORE	LABEL	CLINICAL MEANING
0–7	Subclinical	No treatment needed
8–15	Mild	Outpatient, SSRI
16–23	Moderate	Combination SSRI + ERP
24–31	Severe	High-dose SSRI + intensive ERP
32–40	Extreme	Consider augmentation / neuromodulation
≥35% reduction	Response	Standard treatment response threshold

Rapid-Fire Card 4: First-Line Psychological Treatments

DISORDER · FIRST-LINE THERAPY

GAD CBT (worry + relaxation + cognitive restructuring)

Panic Disorder CBT (Clark's model: interoceptive exposure)

Social Anxiety CBT (Clark & Wells: attention training + exposure)

Specific Phobia Systematic desensitisation / graded exposure

OCD ERP (Exposure and Response Prevention)

PTSD PE / CPT / CT-PTSD / EMDR

Hoarding CBT-H (Steketee & Frost)

Trichotillomania HRT (Habit Reversal Training)

BDD CBT-BDD (Veale: ERP + attention retraining)

Rapid-Fire Card 5: Surgical Targets for OCD

PROCEDURE	TARGET STRUCTURE	TYPE
Anterior capsulotomy	ALIC (anterior limb of internal capsule)	Ablative lesion
Anterior cingulotomy	Anterior cingulate cortex	Ablative lesion
DBS	ALIC / NAcc / BNST / STN	Reversible stimulation
Gamma Knife	ALIC (bilateral)	Radiosurgical

EXAM PEARL

DBS = only reversible option. FDA HDE 2009. Other procedures are ablative (permanent lesion). Gamma Knife is non-invasive but has delayed effect (6–12 months). Minimum criteria for surgery: Y-BOCS ≥ 30 + 3 failed adequate SRI trials + failed ERP.

Mnemonics file compiled for PG exams MD Psychiatry exit examination. Sources: Kaplan & Sadock (10th ed.), Stahl's Essential Psychopharmacology (4th ed.), DSM-5-TR, ICD-11.

High-Yield Comparisons

How to use: Each table is a potential short-answer or part of a long essay. Test yourself by covering one column and recalling the other.

Table 1: GAD vs Panic Disorder vs Social Anxiety Disorder

FEATURE	GAD	PANIC DISORDER	SOCIAL ANXIETY DISORDER
Core fear	Multiple domains of worry (health, finances, relationships, future)	Recurrent unexpected panic attacks; fear of next attack	Scrutiny / negative evaluation by others
Trigger	Multiple, diffuse, often no specific trigger	Unexpected (spontaneous) initially; later situational	Social / performance situations
Attack type	No discrete attacks; chronic background worry	Discrete panic attacks (peak within minutes)	Anxiety in social context; may have situational panic
Physical symptoms	Muscle tension, fatigue, poor sleep (not autonomic surge)	Autonomic surge: palpitations, breathlessness, dizziness, sweating	Blushing, trembling, sweating; visible to others, feared
Avoidance	Moderate; avoids worrying topics	Agoraphobic avoidance of situations; limits activities	Avoids social situations specifically
Duration	≥6 months; chronic, unremitting	Varies; may be episodic	≥6 months
Worry content	Real-life multiple domains	Worry about attacks and their consequences	Humiliation, embarrassment, negative judgment
Neurobiology	BNST (sustained), vmPFC ↓, amygdala ↑	LC/NE, PAG, CO ₂ alarm, amygdala	Amygdala hyperreactivity to social cues
First-line pharma	SSRI/SNRI, buspirone, pregabalin	SSRI/SNRI	SSRI/SNRI; propranolol PRN (performance)
First-line therapy	CBT (worry control, relaxation)	CBT (Clark's model, interoceptive exposure)	CBT (Clark & Wells: attention training, exposure)

FEATURE	GAD	PANIC DISORDER	SOCIAL ANXIETY DISORDER
Assessment tool	GAD-7	PDSS (Panic Disorder Severity Scale)	LSAS (Liebowitz Social Anxiety Scale)
Prognosis	Chronic; rarely remits without treatment	Variable; good with treatment	Chronic without treatment
Sex ratio	F:M = 2:1	F:M = 2:1	F:M ≈ 1:1 (or slight F preponderance)

EXAM PEARL

All three share F:M preponderance and SSRI first-line pharmacotherapy. The KEY differentiators: worry CONTENT (multiple domains vs attacks vs social evaluation), attack presence (panic only), and avoidance TYPE (generalised vs agoraphobic vs social).

Table 2: OCD vs OCPD (Obsessive-Compulsive Personality Disorder)

FEATURE	OCD	OCPD
DSM-5 location	Anxiety/OC-related disorders (Axis I equivalent)	Personality Disorders, Cluster C (Axis II equivalent)
Core feature	Ego-DYSTONIC obsessions and compulsions	Ego-SYNTONIC perfectionism and control
Insight	Usually present (recognises irrationality)	Absent, believes these traits are correct/virtuous
Content	Contamination, harm, symmetry, forbidden thoughts	Orderliness, perfectionism, control, work over relationships
Repetitive behaviour	Compulsions to neutralise anxiety (ego-dystonic)	Rules/procedures because "that's the right way"
Ego-syntonic/dystonic	Dystonic, patient suffers; wants obsessions gone	Syntonic, patient does not suffer; causes suffering to others
Flexibility	Very rigid during obsessional episode; desires change	Rigid as a trait; inflexible about rules
Interpersonal	May be impaired due to OCD burden	Significant, controlling of others, miserly, workaholic
Treatment	SSRIs + ERP (effective)	Psychotherapy (schema therapy, PDT, CBT for PD); SSRIs less effective
Response to SSRIs	Good (50–60% response)	Limited evidence; not first-line

FEATURE	OCD	OCPD
Comorbidity	OCPD comorbid in ~20–30% OCD patients	Can co-occur with OCD but distinct
Prevalence	2–3%	2–8%
Criterion	Causes distress/impairment to patient	Long-standing pattern; pervasive across contexts

EXAM PEARL

The EGO-SYNTONIC vs EGO-DYSTONIC distinction is the most tested differentiator. OCD patient: "I don't want these thoughts; they're horrible." OCPD patient: "I'm just thorough and careful, other people are sloppy." OCPD does NOT respond well to SSRI or ERP.

Table 3: PTSD vs Acute Stress Disorder vs Adjustment Disorder

FEATURE	PTSD	ACUTE STRESS DISORDER (ASD)	ADJUSTMENT DISORDER
Stressor required	Yes, traumatic (Criterion A: death/injury/sexual violence)	Yes, same as PTSD (traumatic)	Yes, any identifiable stressor
Stressor severity	Traumatic (life-threatening level)	Traumatic (same)	Any life event (job loss, divorce, illness)
Onset after stressor	Within 1 month; may have delayed expression (≥ 6 months specifier)	Within 3 days of stressor	Within 3 months
Duration	≥ 1 month	3 days to 1 month	< 6 months after stressor terminates
Intrusion symptoms	Yes (Criterion B), flashbacks, nightmares	Yes, intrusive memories, flashbacks	No specific intrusion cluster
Avoidance	Yes (Criterion C)	Yes	Not specific
Dissociation	With dissociative specifier	Prominent (depersonalisation, derealisation, amnesia)	No
Negative cognitions	Yes (Criterion D, prominent)	Some (negative mood)	Primarily depressed/anxious mood
Arousal	Yes (Criterion E)	Yes	Variable

FEATURE	PTSD	ACUTE STRESS DISORDER (ASD)	ADJUSTMENT DISORDER
Relationship to PTSD		Predicts PTSD risk (but not all ASD → PTSD)	Does not predict PTSD
First-line treatment	TF-CBT, PE, CPT, EMDR + SSRIs	Watchful waiting; early brief CBT if needed	Supportive therapy, brief CBT
Pharmacotherapy	Sertraline, paroxetine	Not routinely indicated; limited role	Symptomatic (sleep, anxiety)
Prognosis	Chronic without treatment	Most remit spontaneously within 1 month	Good if stressor resolves

EXAM PEARL

ASD and PTSD share the same Criterion A (traumatic stressor). Duration separates them: ASD = 3 days–1 month; PTSD = >1 month. Adjustment disorder accepts ANY stressor and has NO intrusion / flashback cluster. The stressor type is the first branching point in differential diagnosis.

CLINICAL ANCHOR

Not everyone exposed to trauma develops ASD or PTSD. Resilience factors (social support, prior mastery, low pre-existing anxiety) are protective. Up to 70% of ASD cases resolve without developing PTSD.

Table 4: Clomipramine vs SSRIs for OCD

FEATURE	CLOMIPRAMINE	SSRIS (AS A CLASS)
Mechanism	Serotonin + norepinephrine reuptake inhibition; H1, muscarinic, alpha-1 blockade	Selective serotonin reuptake inhibition
Active metabolite	Desmethyldlomipramine (potent NRI)	None or weak
Effect size (Y-BOCS)	~1.0 (highest of any single agent)	0.5–0.7
Efficacy rank	Superior to all SSRIs in meta-analyses	Good; comparable to each other
Response rate	~60–70%	~50–60%
Tolerability	Worse	Better

FEATURE	CLOMIPRAMINE	SSRIS (AS A CLASS)
Anticholinergic effects	Marked (dry mouth, constipation, urinary retention, blurred vision)	Minimal (paroxetine: mild)
Sedation	Significant (H1 blockade)	Mild (fluvoxamine more sedating)
Weight gain	Significant	Moderate
Orthostatic hypotension	Yes (alpha-1 blockade)	Rare
QTc prolongation	Yes, ECG monitoring required	Citalopram ≥ 40 mg; others minimal
Seizure risk	Yes, especially >250 mg	Rare
Overdose danger	HIGH (cardiac arrhythmia, seizures, death)	LOW
Sexual dysfunction	Common	Common
Drug interactions	CYP1A2/2D6; fluvoxamine raises levels dangerously	Variable by SSRI
Plasma monitoring	Yes (therapeutic: 150–300 ng/mL; toxic: >450 ng/mL)	Not routine
ECG monitoring	Yes	Only citalopram >40 mg
Guideline position	Second-line (first in some severe/refractory cases)	First-line (all 5 FDA-approved)
Pregnancy safety	Avoid if possible (Category C/D)	Preferred (sertraline safest)
Elderly	Avoid (anticholinergic burden, falls, cardiac)	Preferred
Children (≥ 10 yr)	Second-line	First-line (fluvoxamine, sertraline)
Combination use	Can augment SSRI at low dose (25–75 mg)	
IV formulation	Yes (for refractory OCD)	No

EXAM PEARL

The key exam question: "Despite clomipramine being MORE efficacious than SSRIs in OCD, why are SSRIs still first-line?" Answer: tolerability, safety in overdose (important given depression comorbidity), simpler monitoring, safer in elderly and pregnancy.

Table 5: CBT vs Pharmacotherapy in Anxiety Disorders

PARAMETER	CBT	PHARMACOTHERAPY
Onset of effect	Delayed (4–8 sessions) but builds	2–6 weeks (SSRIs); immediate (BZDs)
Long-term outcome	Superior, lower relapse after discontinuation	Higher relapse on discontinuation
Mechanism	Inhibitory learning, extinction, cognitive change, neuroplasticity	Neurotransmitter modulation (5-HT, NE, GABA)
Neurobiological change	Normalises amygdala reactivity; strengthens vmPFC inhibition	Direct receptor/transporter modulation
Relapse on stopping	Lower	Higher (especially benzodiazepines)
Patient acceptability	High (if accessible); requires engagement	High (simpler to adhere)
Access barriers	Trained therapist shortage; cost; time	Widely available; relatively inexpensive
Comorbid MDD	CBT helpful but pharmacotherapy often needed	SSRI/SNRI treats both
Maintenance	Booster sessions prevent relapse	Ongoing medication often required
Combination	CBT + pharma superior to either alone	
Specific advantage	Panic: interoceptive exposure; OCD: ERP; PTSD: trauma processing	Rapid symptom relief (BZDs); broad-spectrum (SSRIs)
Specific limitation	Requires homework, commitment; avoidance prevents engagement	Side effects; dependence (BZDs); sexual dysfunction
Evidence level	Multiple RCTs; similar effect sizes to pharma	Multiple RCTs; well-established
NICE recommendation	First-line for all anxiety disorders (where accessible)	First-line if CBT unavailable or patient preference
Cost-effectiveness	More cost-effective long-term	Less cost-effective long-term (ongoing prescriptions)

EXAM PEARL

For ALL anxiety disorders, CBT has comparable short-term efficacy to SSRIs, but LOWER relapse rates after treatment discontinuation. Combined CBT + pharmacotherapy shows additive benefit, especially in moderate-severe cases. The exception: benzodiazepines used during exposure may impair extinction learning (state-dependent learning problem).

Table 6: Fear vs Anxiety: Neurobiological Distinction

FEATURE	FEAR	ANXIETY
Nature	Phasic, stimulus-bound, time-limited	Sustained, diffuse, anticipatory
Trigger	Specific, identifiable threat (present)	Non-specific; future-oriented; uncertain
Temporal profile	Seconds to minutes; rapid onset and offset	Minutes to hours; chronic background
Primary brain structure	Amygdala (central nucleus)	BNST (bed nucleus of stria terminalis)
Neuromodulators	NE (LC), CRF	CRF, NPY, GABA, sustained cortisol
Phenomenology	"Danger is HERE", fight/flight/freeze	"Something bad might happen", vigilance, worry
Evolutionary function	Immediate survival response	Sustained preparedness
Disorder correlates	Specific phobias, PTSD (flashback)	GAD, generalised worry component
Extinction	Relatively faster (direct amygdala conditioning)	Slower (BNST circuits; top-down regulation required)
Example	Seeing a snake → immediate fear	Waiting for exam results → chronic worry
Pharmacological target	BZDs (acute); NE blockers	SSRIs/SNRIs (chronic); buspirone; pregabalin

EXAM PEARL

Fear = amygdala-driven, immediate, stimulus-specific. Anxiety = BNST-driven, sustained, diffuse. This distinction explains why GAD (anxiety) is harder to treat than specific phobia (fear): BNST circuits are less accessible to simple extinction than amygdala circuits.

Table 7: OCD vs Delusional Disorder (Somatic Type): Overvalued Ideas Spectrum

FEATURE	OCD (POOR/ABSENT INSIGHT)	DELUSIONAL DISORDER (SOMATIC)	BDD (DELUSIONAL)
DSM-5 classification	OCD chapter (with absent insight specifier)	Schizophrenia spectrum	OCD chapter (with absent insight specifier)
Belief about symptoms	Spectrum: knows probably untrue → completely convinced	Fixed, unshakeable false belief	Spectrum: may be delusional
Ego-syntonic/dystonic	Dystonic (even with poor insight, some distress)	Syntonic	Mixed
Insight	Varying, unique specifier	None, by definition	Unique specifier
Response to SSRIs	Yes, even with delusional intensity OCD	No, antipsychotics needed	Yes, SSRIs even with delusional intensity
Response to antipsychotics	Augmentation only (not alone)	Primary treatment	Augmentation only
Hallucinations/formal thought disorder	No	No (circumscribed delusion)	No
Functioning	Impaired by OCD	Often surprisingly intact	Impaired by preoccupation
Overvalued idea	Intermediate between obsession and delusion	True delusion	Can be overvalued or delusional
Clinical implication	Treat with SSRIs + ERP regardless of insight level	Antipsychotics first; SSRIs add-on	SSRIs first, regardless of insight

EXAM PEARL

The critical clinical point: BDD and OCD with delusional features should STILL be treated with SSRIs first, NOT antipsychotics alone. Antipsychotics are augmentation, not primary treatment. Delusional Disorder (somatic) requires antipsychotics as primary treatment, this is the key differential.

Table 8: Flooding vs Systematic Desensitisation

FEATURE	FLOODING (IMPLOSION)	SYSTEMATIC DESENSITISATION
Originator	Stampfl (implosion); Marks (flooding)	Joseph Wolpe (1958)
Theoretical basis	Extinction via prolonged, maximal exposure	Reciprocal inhibition (relaxation inhibits anxiety)
Anxiety induction	Maximum, begin at TOP of hierarchy	Graduated, begin at BOTTOM of hierarchy
Relaxation component	No, deliberately NOT paired with relaxation	Yes, paired with PMR or other relaxation
Exposure type	Prolonged, intensive, maximal SUDS	Gradual, titrated to tolerance
In vivo vs imaginal	Both (in vivo most effective)	Both (in vivo more effective)
Session duration	Long (hours if needed) until anxiety extinguishes	Shorter sessions; stop when relaxed at each step
Patient experience	Very distressing initially; then anxiety drops	Tolerable throughout
Efficiency	Faster (fewer sessions)	Slower (more sessions needed)
Dropout risk	Higher	Lower
Contraindications	Severe cardiac/respiratory disease, psychosis, trauma history, low distress tolerance	Fewer contraindications
Best for	Simple phobias (in controlled settings); OCD (intensive ERP is flooding variant)	Specific phobias, mild-moderate anxiety
ERP relationship	ERP in OCD is essentially prolonged flooding with response prevention	Not standard for OCD
Efficacy evidence	Highly effective; faster than gradual	Highly effective; better tolerated

EXAM PEARL

Wolpe's systematic desensitisation = GRADUAL + RELAXATION. Flooding = MAXIMAL + NO RELAXATION. Both are effective for phobias but flooding is faster with higher dropout. ERP in OCD combines elements of flooding (prolonged exposure, high initial anxiety) with the critical addition of response prevention.

Table 9: Benzodiazepines: Role Across Anxiety Disorders

DISORDER	ROLE OF BENZODIAZEPINES	RECOMMENDATION
GAD	Short-term bridging; acute severe symptoms	Use sparingly; <4 weeks; avoid as monotherapy
Panic Disorder	Clonazepam / alprazolam, acute relief; adjunct to SSRI	Second-line; high dependence risk
Social Anxiety	Clonazepam: short-term, performance anxiety	Adjunct only; not first-line
Specific Phobia	PRN before unavoidable exposure (flight)	Avoid, may interfere with extinction learning
OCD	Clonazepam: adjunct for anxiety reduction	Limited role; does not treat OCD core
PTSD	CONTRAINDICATED	No evidence; worsens long-term outcome; interference with extinction; dependence risk
Adjustment Disorder	Short-term anxiety / insomnia relief	Brief course acceptable
ASD	Not recommended	May prevent natural recovery

EXAM PEARL

BZDs are explicitly CONTRAINDICATED in PTSD. They are NOT first-line for any anxiety disorder. They are best understood as short-term bridges or acute relief agents while waiting for SSRIs to work. Remember: BZDs impair extinction learning (state-dependent learning), this is the pharmacological reason to avoid them during exposure therapy.

Table 10: Pharmacotherapy Comparison Across Anxiety Disorders: At a Glance

WIDE COMPARISON · LANDSCAPE

DRUG	GAD	PANIC	SAD	OCD	PTSD	NOTES
Escitalopram	✓✓	✓✓	✓✓	✓✓	✓	Best tolerated SSRI
Sertraline	✓✓	✓✓	✓✓	✓✓	✓✓ FDA	Broadest FDA approvals
Paroxetine	✓✓ FDA	✓✓ FDA	✓✓ FDA	✓✓	✓✓ FDA	Discontinuation syndrome risk
Fluoxetine	✓	✓✓	✓	✓✓ FDA	✓	Long half-life (safer discontinuation)
Fluvoxamine				✓✓ FDA		OCD, first SSRI approved (1994)
Venlafaxine XR	✓✓ FDA	✓✓ FDA	✓✓ FDA		✓✓	SNRI; strong evidence
Duloxetine	✓✓ FDA					Helpful with somatic symptoms
Buspirone	✓✓					No acute effect; no dependence
Pregabalin	✓✓					Fast onset; EMA approved
Clomipramine		✓		✓✓		Most efficacious for OCD
Prazosin					✓✓ (nightmares)	Alpha-1; nightmares specifically
Propranolol			✓ (performance)			Performance anxiety only; not generalised
Phenelzine			✓✓ (severe)			MAOI; highly effective; dietary restrictions

KEY: ✓✓ = STRONG EVIDENCE /
FDA APPROVED

✓ = MODERATE EVIDENCE

= NOT STANDARD

Comparisons file compiled for PG exams MD Psychiatry exit examination. Sources: Kaplan & Sadock (10th ed.), Stahl's Essential Psychopharmacology (4th ed.), Oxford Textbook of Psychiatry, DSM-5-TR, ICD-11, NICE Guidelines.

PYQ Frequency Analysis

Scope: PG exams MD Psychiatry Paper II, compiled from available question bank data (17+ years). Frequency ratings reflect estimated appearance across exam cycles. Use this to prioritise revision time.

SECTION 1: FREQUENCY HEAT MAP

Overall Topic Frequency (Anxiety & OCD)

TOPIC	ESTIMATED FREQUENCY	PRIORITY
OCD, Management (full)	Very High (appears nearly every cycle)	MUST MASTER
OCD, Neurobiology (CSTC)	High	MUST MASTER
PTSD, Evidence-based treatment	High	MUST MASTER
Panic Disorder, CBT / Clark's model	High	MUST MASTER
Y-BOCS, Structure and utility	High	MUST MASTER
GAD, Pharmacotherapy	Moderate-High	CORE
SSRIs vs Clomipramine in OCD	Moderate-High	CORE
Treatment-resistant OCD	Moderate	CORE
Neurobiology of anxiety	Moderate	CORE
EMDR	Moderate	CORE
BDD, Clinical features and management	Moderate	CORE
Systematic desensitisation vs flooding	Moderate	CORE
Social Anxiety Disorder	Low-Moderate	IMPORTANT
Hoarding Disorder	Low-Moderate	IMPORTANT
Agoraphobia	Low-Moderate	IMPORTANT
Adjustment Disorder	Low	SUPPLEMENTARY
Specific Phobias	Low	SUPPLEMENTARY

TOPIC	ESTIMATED FREQUENCY	PRIORITY
Trichotillomania / Excoriation	Low	SUPPLEMENTARY
Separation Anxiety (adults)	Very Low	AWARENESS
Substance-induced anxiety	Very Low	AWARENESS

SECTION 2: QUESTION-TYPE ANALYSIS

Long Essay (10 marks): High Frequency Topics

RANK	QUESTION PATTERN	FREQUENCY
1	"Discuss the management of OCD"	★★★★★
2	"Describe PTSD, clinical features and management"	★★★★★
3	"Discuss the pharmacotherapy of OCD / Compare SSRIs and clomipramine in OCD"	★★★★★
4	"Describe the neurobiology of anxiety / anxiety disorders"	★★★★★
5	"Discuss panic disorder, cognitive model and management"	★★★★★
6	"Describe GAD, diagnosis and management"	★★★★★
7	"Discuss treatment-resistant OCD"	★★★★★
8	"Describe EMDR, mechanism and evidence in PTSD"	★★★★★
9	"Discuss Body Dysmorphic Disorder"	★★★★★
10	"Describe Y-BOCS, structure and use"	★★★★★

Short Essay / Short Notes (5 marks): High Frequency

RANK	TOPIC	FREQUENCY
1	Y-BOCS	★★★★★
2	Exposure and Response Prevention (ERP)	★★★★★

RANK	TOPIC	FREQUENCY
3	Clark's cognitive model of panic	★★★★★
4	EMDR	★★★★★
5	Systematic desensitisation	★★★★★
6	Neurobiology of OCD (CSTC circuit)	★★★★★
7	Prazosin in PTSD	★★★★
8	DBS in OCD	★★★★
9	Hoarding Disorder	★★★★
10	Clomipramine in OCD	★★★★
11	Social Anxiety Disorder	★★★★
12	Flooding vs systematic desensitisation	★★★★
13	BDD	★★★★
14	Suffocation alarm theory	★★★
15	Cognitive Processing Therapy (CPT)	★★★
16	Trichotillomania	★★★
17	Locus coeruleus and panic	★★★
18	Agoraphobia	★★★
19	Adjustment Disorder	★★★
20	Virtual Reality Exposure Therapy	★★

SECTION 3: TOPIC-BY-TOPIC PYQ BREAKDOWN

OCD: Most Tested Topic in This Chapter

Long essay patterns observed:

- "Discuss the management of Obsessive-Compulsive Disorder", comprehensive management question; appears every 1–2 years
- "Discuss the pharmacological treatment of OCD with emphasis on the role of clomipramine"
- "Compare the efficacy of SSRIs and clomipramine in the treatment of OCD"

- "Describe the neurobiology of OCD and its therapeutic implications"
- "Discuss treatment-resistant OCD, definition, evaluation, and management options"
- "Describe the role of neuromodulation in treatment-resistant OCD"

Short note patterns:

- "Y-BOCS", appears very frequently; know all 10 items, 5 domains, scoring, severity thresholds
- "ERP in OCD", mechanism, procedure, evidence
- "DBS in OCD", targets, evidence, FDA status
- "Augmentation strategies in OCD"
- "CSTC circuit in OCD"

EXAM STRATEGY

OCD management is the single most reliably examined topic in Paper II anxiety/OCD section. Write a structured algorithm answer: Assessment (Y-BOCS) → First-line SSRI → Clomipramine → Augmentation → ERP → Intensive ERP → Neuromodulation. Never write a list, write a STEPPED ALGORITHM.

PTSD: Second Most Tested

Long essay patterns:

- "Discuss the evidence-based psychological treatments for PTSD"
- "Describe PTSD, diagnostic criteria, neurobiology, and management"
- "Discuss EMDR, describe the technique, mechanism, and evidence base"
- "Describe the Ehlers and Clark cognitive model of PTSD"
- "Discuss pharmacological management of PTSD"

Short note patterns:

- "EMDR"
- "Prolonged Exposure therapy"
- "Cognitive Processing Therapy (CPT)"
- "Prazosin in PTSD nightmares"
- "Differences between PTSD and Acute Stress Disorder"
- "Complex PTSD (ICD-11)"

EXAM STRATEGY

PTSD questions frequently ask for BOTH pharmacotherapy AND psychotherapy. Know: sertraline + paroxetine (FDA approved); venlafaxine; prazosin (nightmares). Psychotherapy: PE, CPT, CT-PTSD, EMDR. Always state what NOT to do, BZDs contraindicated, CISD harmful.

Panic Disorder

Long essay patterns:

- "Describe Clark's cognitive model of panic disorder and its therapeutic implications"
- "Discuss the management of panic disorder"
- "Describe the neuroanatomy of panic attacks"

Short note patterns:

- "Clark's cognitive model of panic"
- "Interoceptive exposure"
- "Suffocation alarm theory (Klein)"
- "Locus coeruleus and panic"
- "Agoraphobia, management"

EXAM STRATEGY

Clark's model is a perennial favourite. Draw the cycle diagram: Trigger → Perceived threat → Apprehension → Bodily sensations → Catastrophic misinterpretation → back to perceived threat. Then map model components to treatment techniques. Examiners reward model-informed treatment descriptions.

GAD

Long essay patterns:

- "Discuss the pharmacotherapy of Generalised Anxiety Disorder"
- "Describe GAD, diagnostic criteria, neurobiology, and management"

Short note patterns:

- "Buspirone in GAD"
- "Pregabalin in GAD"
- "Borkovec's avoidance model of worry"
- "Metacognitive therapy in GAD"

EXAM STRATEGY

GAD pharmacotherapy: emphasise the stepped algorithm. Know buspirone limitations (no prior BZD use; 2–4 week onset) and pregabalin advantages (faster onset, EMA approved). Always mention CBT as equally effective.

Neurobiology of Anxiety

Patterns observed:

- "Describe the neurobiology of anxiety disorders"
- "Discuss the role of amygdala in anxiety"
- "Describe the role of serotonin in anxiety"
- "Discuss the neurobiology of OCD"

Key content tested:

- Amygdala fear circuit (fast vs slow routes from thalamus)
- BNST, sustained anxiety (GAD)
- Locus coeruleus, NE (panic)
- CSTC, OCD
- GABA-A, benzodiazepine mechanism
- 5-HT_{1A}, buspirone; SSRIs mechanism
- HPA axis, chronic anxiety, PTSD

EXAM STRATEGY

This is a high-scoring question if you can draw circuits. Structure: brain structures (amygdala, PFC, BNST, LC, hippocampus) → neurotransmitters (GABA, 5-HT, NE, glutamate) → HPA axis → disorder-specific circuits (CSTC for OCD). Use a summary table at the end.

Systematic Desensitisation / Behaviour Therapy

Patterns observed:

- "Describe systematic desensitisation, principles and procedure"
- "Compare flooding and systematic desensitisation"
- "Describe behaviour therapy techniques in anxiety disorders"
- "What is VRET? Describe its applications in psychiatry"

EXAM STRATEGY

Wolpe's systematic desensitisation: 3 components (relaxation training + hierarchy construction + graded pairing). Reciprocal inhibition is the theoretical basis. Flooding: maximal exposure without relaxation; faster but higher dropout. Always note BII phobia exception (applied tension technique, NOT relaxation).

SECTION 4: PREDICTED HIGH-YIELD QUESTIONS (Sep 2026 Exit Exam)

Based on frequency analysis and recency patterns, the following are highest-probability questions:

Tier 1: Almost Certain to Appear (in some form)

1. OCD management (full algorithm or treatment-resistant focus)
2. PTSD, evidence-based treatments OR EMDR specifically
3. Y-BOCS, structure, scoring, clinical use
4. Neurobiology of anxiety / OCD neurocircuitry (CSTC)
5. Clark's cognitive model of panic + therapeutic implications

Tier 2: Very Likely

1. Clomipramine vs SSRIs in OCD
2. GAD pharmacotherapy
3. BDD, clinical features and management
4. Exposure and Response Prevention, principles and procedure
5. Systematic desensitisation (short note)

Tier 3: Probable

1. DBS in OCD (short note or part of treatment-resistant OCD)
2. Prazosin in PTSD nightmares
3. Hoarding Disorder (short note)
4. Prolonged Exposure therapy / CPT
5. Ehlers & Clark cognitive model of PTSD

Tier 4: Possible

1. Social Anxiety Disorder
2. Flooding vs systematic desensitisation
3. Agoraphobia
4. Suffocation alarm theory

SECTION 5: COMMON EXAMINER TRAPS & FREQUENTLY MISSED POINTS

TOPIC	COMMON MISTAKE	CORRECT ANSWER
OCD diagnosis vs OCPD	Confusing ego-syntonic (OCPD) with ego-dystonic (OCD)	OCD = ego-dystonic; OCPD = ego-syntonic
OCD moved in DSM-5	Saying OCD is in Anxiety Disorders	OCD has its own chapter in DSM-5 AND ICD-11
First SSRI approved for OCD	Not knowing which	Fluvoxamine (1994), first FDA approval
Y-BOCS resistance item	Saying high score = more resistance	Low score = more resistance (item is reverse direction)
Clomipramine vs SSRIs	Saying SSRIs are more efficacious	Clomipramine has HIGHER effect size; SSRIs are preferred for TOLERABILITY
PTSD and benzodiazepines	Recommending BZDs for PTSD	Contraindicated, no evidence; worsens outcome
ASD vs PTSD duration	Mixing up	ASD: 3 days–1 month; PTSD: >1 month
BDD and antipsychotics	Saying pimozide is first-line	SSRIs first-line even with delusional BDD; pimozide NOT recommended
Hoarding and ERP	Applying standard OCD ERP	Hoarding needs CBT-H (different model); ERP less effective
DBS in OCD	Saying it has full FDA approval	FDA Humanitarian Device Exemption (2009) only, NOT full approval
Panic attack symptoms count	Saying 4 symptoms = diagnosis	4 symptoms = ONE panic attack (not a disorder); disorder needs recurrence + 1-month persistence
Flooding and relaxation	Including relaxation in flooding	Flooding deliberately EXCLUDES relaxation (contrast to systematic desensitisation)
BII phobia treatment	Using standard relaxation	Applied tension technique (raise BP); relaxation is CONTRAINDICATED in BII

TOPIC	COMMON MISTAKE	CORRECT ANSWER
EMDR eye movements	Saying they are non-specific	Debated; meta-analyses suggest bilateral stimulation adds effect beyond exposure alone
Complex PTSD	Saying it's in DSM-5	Complex PTSD is in ICD-11 ONLY, not DSM-5

SECTION 6: MARKS ALLOCATION STRATEGY

10-Mark Long Essay: Time Budget (30 minutes)

COMPONENT	TIME	EXPECTED CONTENT
Introduction (definition, prevalence)	2 min	3–4 lines
Assessment / diagnostic criteria	3 min	Key criteria, rating scale
Pharmacotherapy (stepped algorithm)	10 min	First-line, second-line, augmentation, doses
Psychotherapy	8 min	Named technique, mechanism, components
Special considerations / refractory	5 min	Neuromodulation if relevant
Conclusion	2 min	2–3 lines

EXAM STRATEGY

Draw a management algorithm/flowchart in the answer, examiners reward visual organisation. It also helps structure your thoughts and covers more ground faster than prose.

5-Mark Short Essay: Time Budget (10–12 minutes)

COMPONENT · EXPECTED CONTENT

Definition / introduction 2–3 lines

Core content (2–3 structured paragraphs or a table) Main body

Clinical significance / applications 1–2 lines

EXAM STRATEGY

Use a TABLE wherever possible for 5-mark answers. A well-constructed table with 5–6 rows covering key dimensions (definition, mechanism, procedure, evidence, limitations) can earn full marks faster than prose.

SECTION 7: INTER-TOPIC LINKAGES (Exam Connections)

These topics frequently appear together or reference each other:

IF ASKED ABOUT... · ALSO MENTION / CONNECT TO...

OCD management Y-BOCS (assessment), ERP (psychotherapy), CSTC (neurobiology), DBS (refractory)

PTSD treatment EMDR, PE, CPT, Ehlers & Clark model, prazosin (nightmares), no BZDs

Clark's panic model Interoceptive exposure, safety behaviours, anticipatory anxiety

Neurobiology of anxiety CSTC (OCD), BNST (GAD), LC (panic), amygdala (all), vmPFC (PTSD)

Clomipramine in OCD CAST-OQ side effects, QTc monitoring, IV formulation for resistant OCD

Systematic desensitisation Wolpe, reciprocal inhibition, SUDS hierarchy, compare with flooding

BDD OCD spectrum, high suicide risk, no cosmetic procedures, SSRIs regardless of insight

Hoarding Not OCD (different neurobiology), CBT-H not ERP, SSRIs less effective

PYQ Analysis compiled for PG exams MD Psychiatry exit examination (September 2026). Frequency estimates based on 17+ years of available question bank data. All predictions are probabilistic, not guaranteed.

Quick Review

Instructions: Read each vignette carefully. Answer all three questions before checking the answers. Focus on: diagnosis → management algorithm → specific clinical reasoning. All patient names are fictitious.

VIGNETTE 1: OCD with Poor Insight

Clinical presentation:

Arjun, 28-year-old male software engineer, is brought by his wife to the outpatient clinic. She reports that for the past 2 years, he spends 4–5 hours daily washing his hands, sometimes until they bleed. He believes that unless he washes 27 times in a specific sequence, his mother will develop cancer. When his wife tries to interrupt the ritual, he becomes extremely distressed. When asked about this in clinic, Arjun states: "I know it sounds strange, but I am 80–90% sure that my rituals protect my mother. I'm not ready to stop." He has stopped attending social events and was recently placed on probation at work due to lateness.

His MSE: alert, oriented; affect anxious; thought content, ideas of reference to cleansing rituals; no hallucinations; insight: partial, acknowledges beliefs "may seem strange" but maintains strong conviction in their protective function. He is not actively suicidal.

Q1. What is the diagnosis? Identify the key diagnostic features and insight specifier. [3 marks]

KEY INSIGHT

Answer: Diagnosis: Obsessive-Compulsive Disorder (OCD), with **poor insight** specifier **Key diagnostic features (DSM-5):** - **Obsessions:** Recurrent intrusive thought that unless he performs rituals in a specific way, his mother will get cancer, causing marked distress - **Compulsions:** Repetitive handwashing (27 times in specific sequence), driven by the obsession; excessive and not realistically connected to preventing cancer - **Time-consuming:** >1 hour / day (4–5 hours), significantly exceeds the 1-hour threshold - **Significant impairment:** Occupational (probation at work) and social (avoidance of events) - **Not substance/medical Insight specifier:** Poor insight, Arjun believes his rituals are "80–90% likely" to be true; he acknowledges they "sound strange" (not completely absent insight) but maintains strong conviction. This places him in the **poor insight** category (not absent / delusional, as he retains some doubt). **Differential to exclude:** Delusional disorder (somatic), the key difference is that OCD with poor insight still responds to SSRIs, whereas delusional disorder primarily requires antipsychotics. Also, Arjun has the classic OCD structure (obsession → compulsion cycle), not an isolated fixed delusion.

Q2. How does poor insight affect management? Outline the treatment plan. [4 marks]

KEY INSIGHT

Answer: Impact of poor insight on management: Poor insight in OCD does NOT change the pharmacological approach, SSRIs remain first-line regardless of insight level. However, it significantly impacts: - **Engagement with ERP:** Patients with poor insight have lower ERP engagement because they do not fully believe rituals are irrational. Motivation enhancement (MI) is essential before ERP. - **Risk of treatment refusal:** Arjun may refuse ERP if he believes stopping rituals will harm his mother. - **Prognosis:** Poor insight predicts poorer treatment response and higher relapse risk. **Treatment plan:** *Step 1, Assessment:* - Y-BOCS (estimate: score ~28–32, severe range based on time, distress, interference) - Medical history, ECG baseline before clomipramine if needed - Functional assessment (work, social) *Step 2, Pharmacotherapy:* - Start SSRI at low dose, titrate to high end: - Fluoxetine 60–80 mg/day OR Sertraline 150–200 mg/day - Trial: minimum 10–12 weeks at therapeutic dose - Counsel regarding: 2–4 week onset, initial possible activation, need for long-term treatment *Step 3, Psychotherapy:* - Begin with **Motivational Interviewing (MI)**, resolve ambivalence about treatment - **Inference-based CBT (I-CBT):** Specifically designed for poor-insight OCD; targets "inferential confusion" without requiring patient to acknowledge irrationality - Transition to standard **ERP** once some engagement established - Family psychoeducation (wife): reduce accommodation; guide her away from reassurance-giving *Step 4, If inadequate response:* - Augment with risperidone 0.5–1 mg/day or aripiprazole 10 mg/day - Consider clomipramine **Goal:** Even with poor insight, 50–60% of patients respond to SSRIs + ERP.

Q3. Arjun's wife asks: "Should we just do what he wants and let him wash, it calms him down?" How do you counsel her? [3 marks]

KEY INSIGHT

Answer: This behaviour is called **accommodation**, family members reducing their own distress by facilitating the patient's compulsions. While it reduces immediate distress, accommodation: - **Maintains the OCD cycle:** The compulsion reduces anxiety temporarily → reinforces the obsession-compulsion link - **Prevents habituation and extinction:** Patient never learns that anxiety would subside without the ritual - **Increases OCD severity over time**, accommodation is associated with poorer treatment outcomes **Counselling points for his wife:** 1. Arjun's rituals are not voluntary, this is a neurobiological condition requiring treatment, not willpower 2. Her desire to help is understandable, but accommodating the rituals (allowing extra washing, providing reassurance, waiting) actually reinforces OCD 3. Goal: gradually **reduce accommodation** with support from the treatment team, not abruptly (would create crisis) 4. During ERP, she may be given specific guidance on how to respond (e.g., not providing reassurance but remaining compassionate) 5. Encourage her to join a family psychoeducation group or carer session Practically: "We're not asking you to force him to stop, we're asking you to gradually step back from enabling the rituals. We'll support you both through this."

VIGNETTE 2: Panic Disorder with Agoraphobia

Clinical presentation:

Priya, 32-year-old bank teller, presents with a 6-month history of sudden attacks lasting 10–15 minutes. During these attacks she experiences heart pounding, breathlessness, dizziness, tingling in her hands, and an overwhelming fear that she is having a heart attack. Three cardiac workups (ECG, echo, Holter) were normal. She now refuses to take public transport, avoids shopping malls and cinemas, and travels only when accompanied by her husband. She has taken medical leave for 3 months. She carries her phone and a bottle of water everywhere and always sits near an exit.

Q1. Diagnose and formulate the case using Clark's cognitive model. [4 marks]

KEY INSIGHT

Answer: Diagnosis: 1. **Panic Disorder** (F41.0): Recurrent unexpected panic attacks with persistent concern about future attacks + significant behavioural change (avoidance, safety behaviours) 2. **Agoraphobia** (F40.00): Fear and avoidance of ≥ 2 agoraphobic situations (public transport, shopping malls, cinemas) due to concern about escape difficulty if panic occurs The two diagnoses are now separate in DSM-5; both apply here. **Clark's Cognitive Model**

Formulation: *Trigger:* Entering a shopping mall / boarding bus (internal cues: subtle physical sensations) *Perceived threat:* "Something is wrong with my heart" *Apprehension/anxiety:* Attention focuses on chest and breathing *Bodily sensations (amplified):* Palpitations, dizziness, tingling, breathlessness *Catastrophic misinterpretation:* "This is a heart attack, I am going to die / collapse"

Maintaining factors in Priya's case: - **Selective attention:** Hypervigilant to bodily sensations at all times - **Safety behaviours:** Phone, water bottle, sitting near exit, husband as companion, all prevent disconfirmation - **Avoidance:** Public transport, malls, cinemas, prevent learning that sensations are harmless - **Anticipatory anxiety:** Dreads going out → increased baseline arousal → lowers threshold for next attack

Q2. Outline a pharmacological and psychological treatment plan. [4 marks]

KEY INSIGHT

Answer: Pharmacotherapy: - Start **SSRI at low dose** (panic patients are sensitive to initial activation): - Escitalopram 5 mg → titrate to 10–20 mg after 2 weeks - OR Sertraline 25 mg → 50–200 mg - Explain: "May feel slightly more anxious in first 2 weeks, this is expected and temporary" - Allow 10–12 weeks for full response - If inadequate: switch to venlafaxine XR 75–225 mg - Short-term clonazepam 0.5 mg BD for first 2–4 weeks while waiting for SSRI to work (discuss dependence risk) **CBT, Clark's Model-based (12–15 sessions):** *Phase 1 (Sessions 1–3):* Psychoeducation, fight-or-flight response; anxiety is not dangerous; panic attack physiology; Clark's model explained *Phase 2 (Sessions 4–6):* Cognitive restructuring, identifying catastrophic misinterpretations; alternative explanations ("These are anxiety symptoms, not cardiac symptoms"); probability estimation; reviewing normal cardiac results with her *Phase 3 (Sessions 7–10):* Interoceptive exposure, deliberately inducing feared sensations: - Running on the spot (palpitations, breathlessness) - Spinning in chair (dizziness) - Breathing through narrow straw (choking) Key: do exercises without safety behaviours; learn sensations are not dangerous *Phase 4 (Sessions 10–14):* In vivo exposure for agoraphobia, graded hierarchy: - Bus one stop (with then without husband) - Corner shop alone - Shopping centre (quiet time → busy time) Drop safety behaviours progressively (phone still present but not checked; then no water bottle) *Phase 5 (Sessions 14–15):* Relapse prevention **Duration of pharmacotherapy:** Minimum 12 months after remission.

Q3. What is the role of benzodiazepines in this case? Are there any concerns? [2 marks]

KEY INSIGHT

Answer: Limited role: Benzodiazepines (e.g., clonazepam) may be used as a **short-term bridge** (2–4 weeks) while awaiting SSRI onset. They reduce acute panic attack severity and anticipatory anxiety. **Concerns:** 1. **Dependence and tolerance:** With regular use, risk of physical dependence; panic rebounds on discontinuation ("rebound panic") which can be worse than original 2. **Cognitive effects:** Impair memory and processing, relevant for absorbing CBT lessons 3. **Interference with extinction learning:** Taking a BZD before exposure = state-dependent learning; anxiety reduction attributed to drug, not coping; undermines CBT gains 4. **Alprazolam specifically:** Short half-life → inter-dose anxiety; more euphorogenic; higher abuse potential. Prefer clonazepam (longer half-life, less abuse potential) **Conclusion:** Short-term bridge acceptable; avoid long-term use; taper before commencing in vivo exposure work.

VIGNETTE 3: PTSD: Complex Presentation

Clinical presentation:

Meena, 38-year-old nurse, was sexually assaulted by a colleague 8 months ago. She did not report the incident. Over the past 6 months she has had recurrent vivid nightmares of the assault, waking 3–4 times per week. During the day she experiences intrusive images when she passes the ward where it happened. She avoids that ward entirely (has requested a

transfer), avoids male colleagues, and no longer meets friends. She feels "numb" most of the time, cannot feel joy, and believes "I should have fought back, it's my fault." She is hypervigilant at work and startles easily. She reports poor concentration and has made three medication errors in the past month. She denies suicidal ideation but says "I don't care if I live or die." She drinks 2–3 units of alcohol nightly to help sleep.

Q1. Diagnose fully. Are there any comorbidities to screen for? [3 marks]

KEY INSIGHT

Answer: Primary diagnosis: PTSD (F43.10), DSM-5 criteria met: - **Criterion A:** Direct experience of sexual assault (actual sexual violence) - **Criterion B (Intrusion ≥ 1):** Nightmares (B2), intrusive images when passing the ward (B1), likely physiological reactivity to cues (B5) - **Criterion C (Avoidance ≥ 1):** Avoids the ward (C2); avoids male colleagues (C2); likely avoids thoughts about assault (C1) - **Criterion D (Negative cognitions ≥ 2):** Self-blame ("it's my fault", D3), persistent negative emotion/shame (D4), emotional numbing/anhedonia (D7), detachment from others (D6) - **Criterion E (Arousal ≥ 2):** Hypervigilance (E3), exaggerated startle (E4), concentration problems (E5), sleep disturbance (E6) - **Duration:** ≥ 6 months (≥ 1 month required) ✓ - **Impairment:** Occupational (medication errors), social, relational
Comorbidities to screen: 1. **Major Depressive Episode**, anhedonia, numbing, passive suicidal ideation ("don't care if I live or die"), social withdrawal; PHQ-9 2. **Alcohol Use Disorder**, 2–3 units nightly; assess for dependence (CAGE-AID, AUDIT); drinking to sleep = harmful use at minimum; PTSD + alcohol use disorder is a common and dangerous combination 3. **Suicidal ideation**, "I don't care if I live or die" = passive suicidal ideation; requires formal risk assessment (C-SSRS) 4. **Complex PTSD (ICD-11)**, self-blame, shame, interpersonal withdrawal, emotional dysregulation features suggest possible CPTSD; assess disturbances in self-organisation (DSO)

Q2. Outline a comprehensive management plan, including safety, pharmacotherapy, and psychotherapy. [5 marks]

KEY INSIGHT

Answer: Immediate safety: - Risk assessment: passive suicidal ideation present, "don't care if I live or die" requires structured assessment (C-SSRS); low active ideation currently but high-risk profile (trauma, alcohol, depression) - Safety plan: emergency contacts, reasons for living, means restriction - Occupational safety: medication errors, inform supervisor; temporary supervised practice or duties modification until stable **Address alcohol use first:** - AUDIT-C and dependence assessment - Brief motivational intervention - If dependent: supervised alcohol detoxification before commencing trauma-focused therapy (TF-CBT contraindicated in active heavy alcohol use) - Naltrexone or acamprosate for alcohol relapse prevention

Pharmacotherapy for PTSD: - **Sertraline 50 mg → 200 mg** (FDA-approved; first-line) - Address nightmares: **Prazosin 1–5 mg at night** (alpha-1 antagonist; Raskind trials; reduces nightmare frequency/intensity and sleep disruption) - If comorbid MDD confirmed: sertraline addresses both - Avoid benzodiazepines (PTSD contraindication; additional risk given alcohol use)

Psychotherapy: - Phase-based approach recommended given complexity (Complex PTSD features, active alcohol use): *Phase 1, Stabilisation (4–6 weeks):* - Psychoeducation about PTSD, trauma responses, alcohol-trauma link - Safety and crisis skills - Sleep hygiene, grounding techniques (5-4-3-2-1, safe place imagery) - Distress tolerance (DBT skills if indicated) *Phase 2, Trauma Processing (12–16 sessions after stabilisation):* - Choose from: **CPT** (ideal for self-blame/guilt), **PE** (prolonged exposure), **EMDR** - CPT particularly well-suited given prominent self-blame ("it's my fault") and shame, addresses stuck points directly - Address: safety, trust, power/control, esteem, intimacy themes *Phase 3, Integration and Reconnection:* - Gradual re-engagement with social relationships - Occupational rehabilitation - Relapse prevention **Multi-disciplinary involvement:** Psychiatry, psychology, occupational health (for nursing duties), social work.

Q3. Meena asks: "Why can't you just give me something to forget it all?" How do you respond, and what does this reflect clinically? [2 marks]

KEY INSIGHT

Answer: Clinical reflection: This statement reflects several features: - Desire for **avoidance** (a core PTSD maintaining factor), wanting to suppress or escape the memory rather than process it - Possible **cognitive avoidance**, patients often wish for pharmacological erasure rather than engagement with trauma - May also reflect **hopelessness** (comorbid depression) or **misunderstanding** of trauma treatment **Clinical response:** Acknowledge her pain directly: "I hear how exhausted you are, carrying this for 8 months has been incredibly hard." Then explain gently: - "There's no medication that erases memories, and even if there were, the goal isn't to delete what happened, but to change how it lives in you." - "Right now the memory is like an open wound, it keeps getting triggered and bleeding. Treatment is like cleaning and closing the wound properly so it becomes a scar, still there, but no longer raw." - "The therapies we use, like CPT and EMDR, actually do change how the memory feels. Many people say after treatment it feels distant, like something that happened to them rather than something still happening." This response validates, corrects misconception without dismissing her wish, and introduces the rationale for trauma processing.

VIGNETTE 4: GAD: Diagnostic Challenge

Clinical presentation:

Rajan, 45-year-old teacher, presents to a general psychiatry clinic with a 2-year history of "constant worry." He worries about his children's academic performance, his wife's health, his own finances, and his job security, even though objectively there are no major problems in any of these areas. He finds it impossible to stop worrying once it starts. He has muscle aches in his shoulders and neck, wakes at 2 AM and cannot return to sleep, and feels "on edge" throughout the day. He says his mind is "always running." He is exhausted by 3 PM. He denies panic attacks, obsessions, or social fears. He has no medical history. TSH, FBC, renal, and liver function tests, all normal.

Q1. Diagnose and justify. What is the key differential? [3 marks]

KEY INSIGHT

Answer: Diagnosis: Generalised Anxiety Disorder (GAD) DSM-5 criteria met: - **A:** Excessive anxiety/worry about multiple domains (children, wife, finances, job), all for ≥ 2 years - **B:** Difficulty controlling the worry (cannot stop it once it starts) - **C:** ≥ 3 somatic symptoms present: - Muscle tension (shoulder/neck aches) ✓ - Sleep disturbance (wakes at 2 AM) ✓ - Restlessness ("on edge") ✓ - Fatigue (exhausted by 3 PM) ✓, 4 of 6 symptoms - **D:** Causes significant distress (subjective suffering) and impairment (fatigue affecting work performance implied) - **E & F:** Medical causes excluded (TSH normal, hyperthyroidism excluded); no other mental disorder accounts for the picture **Key differentials:** | Differential | Why excluded here | |-----| ---
-----| | Panic Disorder | No discrete panic attacks; no situational avoidance | | Social Anxiety Disorder | Worry is not about social evaluation; no social avoidance | | OCD | No intrusive ego-dystonic obsessions; no compulsions | | MDD with anxiety | No anhedonia, low mood as primary feature described; anxiety predominant | | Hyperthyroidism | TSH normal | | Adjustment Disorder | No identifiable stressor; duration >6 months; worry is multiple/global | **Most important differential to exclude medically:** Hyperthyroidism (checked, TSH normal). Pheochromocytoma if episodic symptoms (no paroxysmal features here).

Q2. Design a complete treatment plan combining pharmacotherapy and CBT. [4 marks]

KEY INSIGHT

Answer: Assessment: - GAD-7: 0–21 scale (likely moderate-severe given sleep, functional impact) - PHQ-9 to rule out comorbid MDD - AUDIT for alcohol (self-medicating anxiety common) **Pharmacotherapy: First-line:** - **Escitalopram 10 mg daily** (best tolerated; minimal drug interactions) - Start with 5 mg for first week to reduce activation effects - Review at 4 weeks; titrate to 20 mg if needed - Explain: "Takes 4–6 weeks to feel benefit; don't stop early" - Expected duration: minimum 12 months after remission *If inadequate response at 8–10 weeks:* - Switch to **Venlafaxine XR 75–150 mg** (dual action; helpful for muscle aches/fatigue) - OR add **buspirone 15–30 mg/day** (no dependence; but takes 3–4 weeks; not if previously on BZDs) - OR add **pregabalin 150–300 mg/day** (faster onset ~1 week; EMA-approved for GAD) **Short-term:** - Avoid long-term benzodiazepines (teacher, cognitive effects, driving) - Hydroxyzine 25–50 mg PRN for acute anxiety peaks if needed **CBT for GAD: Borkovec's Avoidance Model frames treatment:** - Worry is a cognitive avoidance strategy that suppresses emotional processing - Goal: Reduce worry as avoidance; increase engagement with feared outcomes **Components (12–16 sessions):** 1. **Psychoeducation:** Nature of worry; how GAD develops; rationale for CBT 2. **Self-monitoring:** Worry diary, identify triggers, domains, automatic thoughts 3. **Worry postponement / Stimulus control:** Designate one 20-minute "worry time" daily; outside this time, postpone worries to the scheduled slot 4. **Cognitive restructuring:** Challenge probability overestimation ("How likely is it really that X will happen?"); de-catastrophising ("If the worst happened, could I cope?") 5. **Relaxation training:** Progressive Muscle Relaxation (PMR), directly targets muscle tension; 20 minutes daily; apps available 6. **Behavioural experiments:** Test worry predictions over 2 weeks 7. **Problem-solving skills:** For real, solvable worries (distinguish "solvable problem" from "hypothetical what-if") 8. **Sleep hygiene counselling:** Address 2 AM awakening; stimulus control for sleep 9. **Relapse prevention:** Identify early warning signs; plan

Q3. What is Borkovec's avoidance model of worry, and why is it clinically relevant? [3 marks]

KEY INSIGHT

Answer: Borkovec's Avoidance Model (1994): Core proposition: Worry is a primarily **verbal-linguistic (cognitive) process** that functions as **avoidance of emotional processing** of threatening images and their associated somatic/emotional responses. **Mechanism:** 1. Threatening image/thought arises (e.g., image of child being hurt) 2. Individual "worries" in words/concepts ("What if he gets hurt at school? What if I didn't pack enough? What would I do?") 3. This linguistic activity **suppresses somatic and emotional activation** (heart rate, anxiety) that would accompany imaginal processing 4. Because worry "works" to reduce immediate somatic distress, it is **negatively reinforced** 5. However, the emotional content is never fully processed → remains threatening → triggers more worry **Evidence for the model:** - Worriers show less cardiovascular reactivity to stressors than non-worriers (paradoxical dampening) - Imaginal exposure without worry → full emotional activation → extinction - Verbal worry PREVENTS the emotional processing needed for extinction **Clinical relevance:** 1. **Explains why reassurance doesn't help:** It provides temporary relief (like worry) but prevents processing 2. **Directs CBT intervention:** Focus on facilitating emotional processing (not just thought challenging); imaginal techniques alongside cognitive work 3. **Explains metacognitive dimension:** Some patients hold positive beliefs about worry ("Worrying helps me prepare"), these must be directly addressed (Wells' metacognitive therapy) 4. **Explains why Rajan can't "just relax":** Worry is functional, it reduces immediate anxiety. The "cost" is long-term maintenance of GAD.

VIGNETTE 5: Social Anxiety Disorder

Clinical presentation:

Deepa, 26-year-old medical intern, presents with a 10-year history of fear of speaking in public, attending ward rounds, and presenting cases. She becomes intensely anxious when expected to speak in group settings, heart pounding, flushing, voice trembling, sweating, and fears others will notice and think she is incompetent. She over-prepares excessively (4–5 hours for a 5-minute case presentation). She does not fear one-to-one conversations. She has declined a research conference invitation, turned down a teaching post, and avoids socialising with senior colleagues. LSAS total score: 72.

Q1. Diagnose, differentiate performance-only vs generalised SAD, and justify your classification here. [3 marks]

KEY INSIGHT

Answer: Diagnosis: Social Anxiety Disorder (SAD), **Performance Specifier applicable but possibly generalised** (see below) **DSM-5 criteria met:** - Marked fear/anxiety in social performance situations (ward rounds, case presentations, conferences) - Fear of negative evaluation (appearing incompetent/nervous to others) - Situations almost always provoke anxiety - Avoided (conference, teaching post) or endured with distress (case presentations) - Disproportionate to actual threat - Duration: 10 years (>>6 months) - Significant occupational impairment (declined career opportunities) **Performance-only vs Generalised SAD:** | Feature | Performance-only | Generalised | |-----|-----|-----| | Fear domain | Public speaking/performing only | Most social situations | | One-to-one fear | No | Yes | | LSAS score | <55 typically | Higher scores | | Prognosis | Better | More chronic |

Classification of Deepa: - She fears performance/group settings but not one-to-one → could be performance-only - However, she avoids socialising with senior colleagues (social interaction, not just performance) and LSAS = 72 (marked SAD range) - **Most accurate classification: Generalised SAD**, the avoidance extends beyond formal performance to informal social interactions with perceived-status others - The performance-only specifier applies ONLY when fear is STRICTLY limited to public speaking/performing, Deepa's avoidance is broader

Q2. Outline the pharmacological and psychological management. [4 marks]

KEY INSIGHT

Answer: Pharmacotherapy: First-line: - **Sertraline 50–200 mg** (FDA-approved for SAD; start 25 mg × 2 weeks) - **Paroxetine CR 12.5–37.5 mg** (FDA-approved; more sedating, may be helpful for evening anxiety) - **Venlafaxine XR 75–225 mg** (FDA-approved) - Duration: minimum 12 months; many require ongoing treatment *For acute performance anxiety (as an adjunct):* - **Propranolol 10–40 mg** taken 30–60 minutes before specific performance situations - Reduces peripheral symptoms (heart pounding, voice trembling, visible flushing) - Does NOT reduce cognitive anxiety or fear; works for somatic symptoms - Not for generalised SAD, not effective for overall disorder *Second-line (severe/refractory):* - **Phenelzine 45–90 mg** (MAOI), highly effective; dietary tyramine restrictions required; orthostatic hypotension - **Gabapentin 900–3600 mg**, useful if comorbid alcohol history **Psychotherapy, CBT (Clark & Wells model):** *Phase 1:* Develop shared formulation, self-focused attention, safety behaviours (over-preparation, minimal eye contact) maintain the disorder *Phase 2: Attentional retraining*, shift focus from internal monitoring (how am I coming across?) to external (what is being said; the environment); attention training exercises *Phase 3: Video feedback*, record Deepa giving a case presentation; review together to demonstrate discrepancy between subjective experience (terrible) and objective appearance (competent); powerful disconfirmation *Phase 4: Drop safety behaviours*, target over-preparation specifically; behavioural experiments (give presentations with less preparation; observe outcome) *Phase 5: Graded social exposure*, hierarchy: - Ask a question in ward round - Present a case with 1 hour prep only - Attend department social event - Accept conference presentation *Phase 6:* Relapse prevention

Q3. Deepa asks: "Propranolol, can I just take it before every presentation forever?" Advise her. [3 marks]

KEY INSIGHT

Answer: Propranolol is not a long-term solution for Social Anxiety Disorder. Here is why:

What propranolol does: - Blocks peripheral beta-adrenergic receptors → reduces palpitations, sweating, visible tremor, voice shake - Provides temporary symptomatic relief for acute performance situations

Why it cannot be a long-term solution: 1. **Does not treat the underlying disorder:** Propranolol reduces somatic symptoms but does NOT change cognitive anxiety, negative self-evaluative beliefs, or avoidance. SAD persists untreated. 2. **Becomes a**

safety behaviour: Relying on propranolol before presentations means Deepa never learns that she CAN manage without it, she attributes success to the drug, not herself. This maintains anxiety. 3. **Does not generalise:** Informal social situations (lunch with senior colleagues) cannot all be pre-medicated; these remain distressing. 4. **Tolerability concerns:** Not suitable if bradycardia, asthma, diabetes, or Raynaud's; fatigue with regular use. **Appropriate advice:**

"Propranolol is a reasonable bridge for very specific unavoidable performances while we work on the real problem. But the goal is to get to a point where you don't need it, where you've learned through experience that your anxiety is manageable and people aren't judging you the way you fear. CBT and an SSRI will get you there."

VIGNETTE 6: BDD: Cosmetic Surgery Clinic Referral

Clinical presentation:

Vikram, 24-year-old male, is referred from a plastic surgery clinic. He has requested rhinoplasty three times in 18 months. The first surgeon performed the procedure, and Vikram was distressed with the result within 4 weeks ("it's still crooked"). A second and third surgeon declined, noting that his nose appeared normal. He currently spends 3–4 hours daily examining his nose in mirrors from different angles, comparing photographs, and seeking reassurance from family. He has dropped out of college and does not leave home. PHQ-9 = 19 (severe depression). On questioning, he rates his conviction that his nose is disfigured at "99%." He denies suicidal ideation currently but mentions he "understands why people with this feeling might not want to live."

Q1. Diagnose, classify insight level, and assess risk. [3 marks]

KEY INSIGHT

Answer: Diagnosis: Body Dysmorphic Disorder (BDD), F45.22 - Preoccupation with perceived defect (nasal appearance) unnoticeable to others - Repetitive behaviours: mirror checking, photography comparison, reassurance-seeking (≥ 3 hours daily) - Significant distress and impairment (dropped out of college; housebound) - Not better explained by eating disorder or another condition **Insight specifier:** Absent insight / delusional beliefs, conviction at "99%", he is essentially certain the defect exists despite objective evidence to the contrary (two surgeons refusing; photographs not confirming defect). In DSM-5, this is captured by the "with absent insight/delusional beliefs" specifier. Previously this would have been classified as "Delusional Disorder, Somatic Type", DSM-5 now places it within the BDD spectrum. **Risk assessment:** - **HIGH suicide risk** - PHQ-9 = 19 (severe depressive episode comorbid) - "Understands why people with this feeling might not want to live" = **passive suicidal ideation / implicit suicidal communication**, requires formal structured assessment (C-SSRS) - BDD-specific risk factors: delusional insight, comorbid MDD, prior cosmetic procedure disappointment (all present) - Lifetime suicide attempt rate in BDD ~25%; suicidal ideation ~80% - **Risk level: HIGH**, **requires active safety planning and possible inpatient admission if ideation escalates**

Q2. What is your management plan? Address the surgical requests specifically. [4 marks]

KEY INSIGHT

Answer: Immediate: - Formal suicide risk assessment (C-SSRS) - Safety plan: emergency contacts, coping strategies, means restriction - Consider inpatient admission if active suicidal ideation emerges - Coordinate with plastic surgeons, advise AGAINST further surgery (document recommendation) **Addressing surgical requests:** - Cosmetic procedures are **contraindicated** in BDD: - >80% of patients remain preoccupied or shift focus to a new perceived defect post-procedure - Surgery reinforces the belief that the defect is real and fixable - BDD severity often worsens following procedures - Explain to Vikram: "The surgery you had didn't bring relief, this tells us the problem is not with your nose. It's in how your mind processes what it sees. Treating your mind will do what surgery cannot." **Pharmacotherapy:** - **Fluoxetine 60–80 mg/day** (best evidence for BDD; also treats comorbid MDD) - Alternative: fluvoxamine 200–300 mg/day; sertraline 150–200 mg/day - High doses required, same principle as OCD - Trial: 12 weeks minimum at therapeutic dose - SSRIs effective REGARDLESS of insight level, do not withhold because he has delusional intensity - Do NOT use antipsychotics alone (unlike true delusional disorder) - Consider antipsychotic AUGMENTATION (risperidone 0.5–1 mg) given delusional intensity and partial response to SSRI **Psychotherapy, CBT for BDD (Veale protocol):** - **Motivational enhancement first**, delusional insight makes engagement challenging - **Attentional retraining:** Shift from self-focused (internal nose image) to external (environment, other people's faces) - **ERP for checking behaviours:** Mirror exposure (holistic, brief; not zoomed/analysed); prevent repeated checking and photography comparison; prevent reassurance-seeking - **Cognitive restructuring:** Self-as-social-object; appearance assumptions; inferential errors - **Behavioural experiments:** Go out for short periods; observe that people are not staring **Rehabilitation:** Academic counselling; gradual return to college (graded exposure)

Q3. How do you explain BDD to Vikram in a way he can accept? [3 marks]

KEY INSIGHT

Answer: Vikram believes the problem is his nose, telling him directly "there's nothing wrong with your nose" will likely destroy the therapeutic alliance. Instead, use a validation-first approach: **Validate his suffering:** "I can see how much distress this has caused you. You've dropped out of college, you can't leave the house. This is real suffering and it deserves real treatment." **Create cognitive dissonance gently:** "I want to share an observation, and I'd like your reaction. You had surgery, and within 4 weeks, you were distressed again. Two more surgeons have looked carefully and declined. And yet the distress hasn't changed. Does that suggest to you that the surgery might not be the answer?" **Introduce the neurobiological frame:** "There is a condition, and it's more common than people think, where the brain's visual processing system for self-image becomes dysregulated. It's like a faulty mirror inside the brain. What you see when you look at yourself doesn't match what others see. It's not that you're wrong to believe it, it's that a part of your brain is generating this perception powerfully and incorrectly. We can treat the brain, and when we do, the perception changes." **Offer a therapeutic experiment:** "I'm not asking you to agree with me today. I'm asking you to try a treatment for 12 weeks. If the distress reduces, whatever the reason, we've helped you. You can decide then what you believe about your nose." This approach: validates, creates doubt without confrontation, externalises (brain, not him), and offers a low-stakes experiment.

VIGNETTE 7: Treatment-Resistant OCD

Clinical presentation:

Sunita, 35-year-old homemaker, has had OCD since age 16. She has checking and contamination obsessions. She has tried fluoxetine (80 mg × 14 weeks, partial response, Y-BOCS reduced from 34 to 27), then sertraline (200 mg × 12 weeks, no further benefit, Y-BOCS 28), then paroxetine (60 mg × 12 weeks, intolerance due to weight gain and sexual dysfunction, discontinued at 8 weeks). She had 20 sessions of ERP with a trained psychologist, Y-BOCS reached 24 at end, but has since risen back to 31. She is currently on escitalopram 20 mg. She is severely functionally impaired, cannot cook, manage finances, or leave the house unaccompanied.

Q1. Does Sunita meet criteria for treatment-resistant OCD? Justify. [2 marks]

KEY INSIGHT

Answer: Yes, Sunita meets criteria for treatment-resistant OCD. Treatment-resistant OCD is defined as failure to achieve $\geq 35\%$ Y-BOCS reduction after adequate trials of ≥ 2 –3 SSRIs at maximum tolerated dose for ≥ 10 –12 weeks AND adequate ERP. **Sunita's trial history:** | Treatment | Dose | Duration | Outcome | |-----|-----|-----|-----| | Fluoxetine | 80 mg (maximum) | 14 weeks | Partial response only (21% reduction: 34 $\rightarrow$ 27); not $\geq 35\%$ | | Sertraline | 200 mg (maximum) | 12 weeks | No benefit (Y-BOCS 28) | | Paroxetine | 60 mg | 8 weeks | Discontinued, intolerance (not adequate duration) | | ERP | 20 sessions | Full course | Response but relapsed (Y-BOCS now 31) | | Escitalopram | 20 mg (subtherapeutic for OCD, should be 40 mg) | Current | Not yet adequate | **Assessment:** Fluoxetine and sertraline = 2 adequate SSRI failures. ERP = adequate course but relapse. Paroxetine was inadequate (only 8 weeks; not counted). Three SSRIs have been trialled with insufficient overall response. Current escitalopram may be subtherapeutic (20 mg, consider 40 mg). **Conclusion:** Meets treatment-resistance criteria. Systematic escalation warranted.

Q2. What is the next step? Outline a management plan for treatment-resistant OCD. [5 marks]

KEY INSIGHT

Answer: Immediate: Optimise current treatment - Increase escitalopram to **40 mg/day** (OCD often needs higher doses than depression; 20 mg is subtherapeutic for OCD at this severity) - Allow 10–12 weeks at 40 mg before declaring failure - Reinstate structured ERP, previous relapse after discontinuation suggests maintenance ERP needed (monthly booster sessions minimum) **Step 1: Clomipramine trial** - Most efficacious single agent (effect size ~ 1.0 vs SSRI ~ 0.5 – 0.7) - Start 25 mg nocte $\rightarrow$ titrate to 150–250 mg over 4–6 weeks - Baseline ECG and BP monitoring - Counsel re: anticholinergic effects, sedation, sexual dysfunction - Expected trial: 10–12 weeks **Step 2: Antipsychotic augmentation (add to SSRI) - Risperidone 0.5–2 mg/day**, best RCT evidence; can add to escitalopram - Mechanism: D2 blockade in striatum augments SRI effect on CSTC circuit - Alternative: aripiprazole 10–15 mg (better metabolic profile) - If comorbid anxiety: quetiapine 25–100 mg **Step 3: Glutamate modulators - N-Acetylcysteine (NAC) 2400 mg/day**, positive RCT data for augmentation; well-tolerated - **Memantine 10–20 mg**, small studies; NMDA antagonism - **Riluzole 50–100 mg**, open-label studies; reduces glutamate **Step 4: Intensive ERP** - Daily ERP sessions for 2–3 weeks (residential or day programme) - For relapsed/severe cases, intensive format outperforms weekly sessions - Family-based ERP: assess and reduce family accommodation **Step 5: IV Clomipramine** - If oral clomipramine inadequate: IV formulation bypasses first-pass metabolism - Protocol: 25–75 mg in dextrose IV over 45–90 min (specialist centre) **Step 6: Neuromodulation - rTMS/dTMS:** Deep TMS (Brainsway H7 coil); FDA 510(k) cleared (2018); 20 sessions; $\sim 40\%$ response - Target: supplementary motor area (SMA) or dorsolateral PFC **Step 7: Neurosurgical referral** (if Y-BOCS remains ≥ 30 despite all above) - Multidisciplinary assessment (neurosurgery, psychiatry, neurology, psychology, ethics) - Options: DBS (ALIC, reversible; FDA HDE 2009), Gamma Knife capsulotomy, anterior capsulotomy - Full informed consent; documentation of treatment failure

Q3. Sunita's husband asks: "Is there any surgery that can cure her?" How do you counsel him? [3 marks]

KEY INSIGHT

Answer: Acknowledge his question and the desperation behind it: "I understand, watching Sunita suffer for nearly 20 years is exhausting for you too, and it's natural to want a definitive fix." **What surgery CAN do:** - Neurosurgical procedures exist for severe, treatment-resistant OCD: - **Deep Brain Stimulation (DBS):** Small electrodes implanted in a brain circuit (anterior limb of internal capsule); stimulation is adjustable and reversible; response rate ~40–60% - **Gamma Knife:** Radiation-based; non-invasive; takes 6–12 months to work; similar response rates - These are NOT cures, they are treatments that REDUCE severity, not eliminate OCD **What surgery CANNOT do:** - Surgery is NOT a cure, "cure" implies complete resolution; OCD rarely fully resolves even with surgery - Response means $\geq 35\%$ reduction in Y-BOCS, significant improvement, but residual symptoms remain in most cases - Surgery requires continued pharmacotherapy and ERP post-procedure **When surgery becomes an option:** - Only after ALL other treatments have been tried adequately - Requires multidisciplinary team approval, ethics review, and Sunita's full informed consent - We are not there yet, there are still steps to try (augmentation, intensive ERP, clomipramine) **Closing message:** "Surgery is a real option for the most severe cases, and it's on the table. But it's the last step in a long process, and for good reason, it's irreversible (except DBS). Let's try the next steps first. We haven't exhausted all options yet, and each step can make a meaningful difference."

VIGNETTE 8: Specific Phobia: Blood Injection Injury Type

Clinical presentation:

Rahul, 22-year-old medical student, faints when he has blood drawn for his own medical tests. He has also fainted while observing venepuncture procedures in clinical postings and once while suturing a laceration. He dreads clinical postings involving blood or injections, avoids them when possible, and plans to limit his career to "anything without blood." His heart races briefly when he anticipates these situations, but he then feels lightheaded and loses consciousness. He recovers within minutes. He has no cardiac history. ECG is normal.

Q1. Diagnose and explain the unique physiology. [3 marks]

KEY INSIGHT

Answer: Diagnosis: Specific Phobia, Blood-Injection-Injury (BII) Type (F40.231) DSM-5 criteria: - Marked fear/anxiety about blood, injections, and injury - Almost always provokes immediate fear response - Actively avoided (clinical postings) - Disproportionate to actual danger - ≥ 6 months (implied by career-planning avoidance) - Significant impairment (career limitation; medical student, significant professional impact) **Unique physiology, Vasovagal (Diphasic) Response:** BII phobia is the ONLY phobia type with a characteristic biphasic cardiovascular response: *Phase 1, Sympathetic surge (brief):* - Heart rate increases (Rahul notices "heart races") - Blood pressure rises - This is the typical sympathetic fight-or-flight response *Phase 2, Paradoxical vagal response:* - Reflex vagal activation (likely baroreflex or vasovagal reflex triggered by blood pressure peak) - Bradycardia (heart rate drops) + vasodilation $\rightarrow$ hypotension - Cerebral hypoperfusion $\rightarrow$ presyncope/syncope (lightheadedness $\rightarrow$ fainting) **This is the OPPOSITE of the standard anxiety response** (which maintains or increases BP/HR). This explains why standard relaxation techniques are CONTRAINDICATED in BII phobia, relaxation further lowers sympathetic tone and exacerbates hypotension.

Q2. What is the specific treatment? Why is it different from other phobias? [4 marks]

KEY INSIGHT

Answer: First-line treatment: Applied Tension Technique (Öst & Sterner, 1987) Rationale: In BII phobia, the therapeutic goal is NOT to relax, it is to MAINTAIN or RAISE blood pressure to prevent the vasovagal response. **Applied Tension Technique, Steps:** 1. **Tension exercise:** - Tense major muscle groups simultaneously (arms, legs, abdomen, chest) - Hold for 10–15 seconds until warmth felt in face (indicates rising BP) - Release tension for 20–30 seconds - Repeat 5 times - Practise 3–5 times daily for 1–2 weeks before commencing exposure 2. **During exposure:** - Apply tension at onset of any lightheadedness - Maintains cerebral perfusion; prevents syncope - Patient learns to tolerate the stimulus while maintaining consciousness 3. **Graded exposure (blood/injury hierarchy):** - Step 1: Read about blood draws - Step 2: Watch videos of blood draws - Step 3: Be in same room as blood tube - Step 4: Observe venepuncture from distance - Step 5: Observe close up - Step 6: Have blood drawn himself (with applied tension) - Step 7: Perform venepuncture on patient **Why different from other phobias:** - Standard relaxation training (PMR, diaphragmatic breathing) $\rightarrow$ further lowers BP $\rightarrow$ INCREASES fainting risk - Standard gradual relaxation paired with exposure is contraindicated - Instead: maintain high physiological tone during exposure; teach recognition of prodrome **Evidence:** Applied tension + graded exposure = effective in 80% of BII phobia cases; effect maintained at 1-year follow-up (Öst et al.) **Pharmacotherapy:** Limited role. No routine pharmacotherapy; beta-blockers actually worsen the vasovagal response (further lower BP/HR). Avoid.

Q3. How does this case affect Rahul's medical career? How would you advise him? [3 marks]

KEY INSIGHT

Answer: Impact assessment: - BII phobia with syncope during clinical procedures is a significant occupational impairment for a medical student - However: BII phobia is one of the MOST TREATABLE phobias, 80%+ response to applied tension + exposure in 1–5 intensive sessions - Without treatment, career avoidance will narrow his professional options substantially **Clinical advice:** *Short-term:* - Seek treatment NOW, applied tension technique can be learned within 2–4 sessions - The fainting is a conditioned physiological response, NOT a sign of weakness or unsuitability for medicine - Practical adjustment: have a colleague aware during early clinical postings; sit if feeling lightheaded; do not stand for prolonged venepuncture observation until habituated *Treatment prognosis:* - "With 3–5 sessions of applied tension therapy, most people with your exact problem become fully able to work with blood and injections without fainting. This is not a career-ending condition, it's a treatable one." *Career counselling:* - Do NOT limit career choices prematurely based on current state - Psychiatry itself involves less blood than surgery / emergency medicine, but treatment should aim for full functional recovery, not just avoidance *Realistic framing:* - "A month from now, after this treatment, this will likely not be an issue. Don't let one avoidable condition decide your specialty."

Clinical Vignettes compiled for PG exams MD Psychiatry exit examination. All patient details are entirely fictitious. Sources: Kaplan & Sadock (10th ed.), Stahl's Essential Psychopharmacology (4th ed.), Oxford Textbook of Psychiatry, DSM-5-TR, ICD-11, NICE Guidelines.

Personality Disorders

Paper II · Clinical Psychiatry. Six study modes, from notes to quick review.

Study Notes

Sources: Kaplan & Sadock's Comprehensive Textbook of Psychiatry (10th ed.), DSM-5-TR, ICD-11, Young et al. Schema Therapy (2003), Linehan DBT Manual (1993), Bateman & Fonagy MBT (2004), Clarkin et al. TFP (2006), Stahl's Essential Psychopharmacology (5th ed.)

1. DEFINITIONS AND CORE CONCEPTS

A **personality disorder** is defined as an enduring pattern of inner experience and behaviour that deviates markedly from the expectations of the individual's culture, is pervasive and inflexible, has onset in adolescence or early adulthood, is stable over time, and leads to distress or impairment.

Key distinguishing features from other mental disorders:

- Ego-syntonic (mostly), the person experiences the traits as "me," not "my illness"
- Pervasive across contexts, not situation-specific
- Onset early (adolescence to early adulthood)
- Stable over time, though evidence from longitudinal studies is more nuanced

EXAM PEARL

Personality disorders must be distinguished from personality change due to a general medical condition, which has an identifiable organic cause, and from the direct effects of substance use.

2. CLASSIFICATION SYSTEMS

2.1 DSM-5 Categorical Model (Clusters A/B/C)

The DSM-5 retains the categorical approach from DSM-III/IV-TR with 10 specific PDs organized into 3 clusters.

CLUSTER	THEME	DISORDERS
A, "Weird"	Odd, eccentric	Paranoid, Schizoid, Schizotypal
B, "Wild"	Dramatic, emotional, erratic	Antisocial, Borderline, Histrionic, Narcissistic
C, "Worried"	Anxious, fearful	Avoidant, Dependent, OCPD

Additional DSM-5 categories:

- Personality Disorder Not Otherwise Specified (PD-NOS) → now "Other Specified PD"
- Personality Change Due to Another Medical Condition

2.2 DSM-5 Alternative Model for Personality Disorders (AMPD): Section III

Proposed as the next generation of PD diagnosis. Combines:

1. **Criterion A, Level of Personality Functioning Scale (LPFS)**
2. Domains: Identity, Self-Direction, Empathy, Intimacy
3. Rated 0 (no impairment) to 4 (extreme impairment)
4. Threshold for PD: Level 2+ (moderate impairment)

1. **Criterion B, Pathological Personality Traits**

Five broad trait domains, each with facets:

DOMAIN · FACETS

Negative Affectivity Emotional lability, Anxiousness, Separation insecurity, Perseveration, Submissiveness, Hostility, Depressivity, Suspiciousness, Restricted affectivity (pole)

Detachment Withdrawal, Intimacy avoidance, Anhedonia, Depressivity, Suspiciousness, Restricted affectivity

Antagonism Manipulativeness, Deceitfulness, Grandiosity, Attention seeking, Callousness, Hostility

Disinhibition Irresponsibility, Impulsivity, Distractibility, Risk taking, Rigid perfectionism (pole)

Psychoticism Unusual beliefs and experiences, Eccentricity, Perceptual dysregulation

EXAM PEARL

AMPD only operationalizes 6 of 10 PDs in Section III: Antisocial, Avoidant, Borderline, Narcissistic, OCPD, Schizotypal. The remaining 4 can still be diagnosed using the general criteria.

2.3 ICD-11 Dimensional Model (Severity + Trait Qualifiers)

ICD-11 made a radical departure from categorical diagnosis. It is the most exam-relevant classification change in recent years.

Step 1, Establish severity:

SEVERITY LEVEL	FUNCTIONAL IMPAIRMENT	TRAIT DISTURBANCE	NOTES
Personality Difficulty (not a disorder)	Minimal	Some traits present	Not diagnosable as PD

SEVERITY LEVEL	FUNCTIONAL IMPAIRMENT	TRAIT DISTURBANCE	NOTES
Mild PD	Affects some domains	Affects some but not all relationships	More amenable to treatment
Moderate PD	Affects most domains	Pervasive across contexts	Most common clinical presentation
Severe PD	Severe; all areas affected	Marked disturbance, risk to self/others	Highest morbidity

Step 2, Add trait qualifiers (one or more):

QUALIFIER	CORE FEATURE	OLD CATEGORY APPROXIMATION
Negative Affectivity	Intense, unstable emotions, anxiety, low mood	BPD (partly), HPD
Detachment	Social withdrawal, emotional restriction	Schizoid, Avoidant, Schizotypal
Dissociality	Disregard for others' rights and feelings	ASPD, NPD
Disinhibition	Impulsivity, risk-taking, irresponsibility	ASPD, BPD (partly)
Anankastia	Rigid perfectionism, perseveration, orderliness	OCPD
Borderline Pattern	Emotional instability, impulsivity, identity disturbance, self-harm	BPD (retained as specifier)

EXAM PEARL

In ICD-11, "Borderline Pattern" is NOT a separate diagnosis, it's a **specifier** added to any severity level. This reflects empirical data showing BPD features can accompany any underlying personality structure.

EXAM STRATEGY

ICD-11 vs DSM-5 comparison is a guaranteed long question. Master the severity framework, the 5+1 qualifiers, and the absence of categorical diagnoses in ICD-11.

3. CLUSTER A PERSONALITY DISORDERS

3.1 Paranoid Personality Disorder

Core Feature: Pervasive distrust and suspiciousness of others, motives interpreted as malevolent.

DSM-5 Criteria (4 of 7 required):

1. Suspects exploitation, harm, or deception without evidence
2. Doubts loyalty or trustworthiness of friends/associates
3. Reluctant to confide, fearing information will be used against them
4. Reads hidden demeaning messages into benign events (ideas of reference)
5. Bears grudges, unforgiving of perceived insults
6. Perceives attacks on character not apparent to others; reacts with anger or counterattack
7. Recurrent suspicions about fidelity of partner

Differential Diagnosis:

CONDITION · KEY DISTINGUISHING FEATURE

Paranoid PD No frank psychosis; ego-syntonic; stable over time

Paranoid schizophrenia Hallucinations, delusions, disorganization; Schneider first-rank symptoms

Delusional disorder Fixed encapsulated delusion; otherwise intact functioning

BPD Transient paranoid ideation when stressed; emotional instability dominant

ASPD Suspiciousness present but exploitative behavior primary

Neurobiology: Hyperactivation of threat-detection circuits (amygdala, anterior insula); possible dopaminergic overactivation in mesolimbic pathways (explaining paranoid ideation on a continuum with psychosis).

Management:

- Primary: Individual psychotherapy (cognitive approaches; supportive therapy)
- Schema Therapy: Address core schemas of Mistrust/Abuse and Defectiveness
- Pharmacotherapy: Low-dose antipsychotics if paranoid ideation severe or transient psychosis (olanzapine 2.5–5 mg, risperidone 0.5–1 mg); SSRIs for co-occurring anxiety/depression
- Prognosis: Stable; rarely remits; may progress to delusional disorder in minority

3.2 Schizoid Personality Disorder

Core Feature: Pervasive detachment from social relationships, restricted range of emotional expression.

DSM-5 Criteria (4 of 7 required):

1. Neither desires nor enjoys close relationships
2. Almost always chooses solitary activities
3. Little interest in sexual experiences with others
4. Takes pleasure in few, if any, activities (but not anhedonia of depression, baseline state)
5. Lacks close friends other than first-degree relatives
6. Appears indifferent to praise or criticism
7. Shows emotional coldness, detachment, or flattened affectivity

Key Differentials:

CONDITION · KEY DISTINGUISHING FEATURE

Schizoid PD No psychotic features; ego-syntonic detachment; stable

Schizotypal PD Magical thinking, odd beliefs, unusual perceptions, eccentric speech

Avoidant PD Desires connection but avoids due to fear; distress present

Depression Temporal onset; anhedonia is acquired; depressed mood

Autism Spectrum Disorder Early onset; qualitatively different social impairment; sensory features

EXAM PEARL

Schizoid PD is the "hermit by choice", contentedly alone. Avoidant PD is the "hermit by fear", painfully alone. This distinction is clinically critical.

Management:

- Rarely seek treatment; when they do, often for Axis I comorbidity
- Supportive psychotherapy; careful boundary management
- No established pharmacotherapy; SSRIs for co-occurring anxiety

3.3 Schizotypal Personality Disorder

Core Feature: Odd beliefs, magical thinking, unusual perceptual experiences, eccentric behavior, discomfort in close relationships, odd speech.

DSM-5 Criteria (5 of 9 required):

1. Ideas of reference (not delusions)

2. Odd beliefs or magical thinking inconsistent with cultural norms
3. Unusual perceptual experiences (body illusions, sensing presence of someone)
4. Odd thinking and speech (vague, circumstantial, metaphorical)
5. Suspicious or paranoid ideation
6. Inappropriate or constricted affect
7. Odd, eccentric, peculiar behavior
8. Lacks close friends (other than first-degree relatives)
9. Excessive social anxiety that doesn't diminish with familiarity (tends to be paranoid rather than self-critical)

EXAM PEARL

Schizotypal PD is classified in the **schizophrenia spectrum** in ICD-11 (as "Schizotypal Disorder"), whereas DSM-5 places it under Cluster A PDs. This is a common exam question on classification differences.

Neurobiology:

- Genetic relationship with schizophrenia: 10–15% of first-degree relatives of individuals with schizophrenia have schizotypal PD
- Dopamine dysregulation (D2 receptor sensitivity)
- Reduced gray matter volume: prefrontal cortex, thalamus
- Reduced smooth pursuit eye tracking (as in schizophrenia)

Management:

- Low-dose antipsychotics (risperidone, olanzapine) for positive-like symptoms
- SSRIs for social anxiety and dysphoria
- Cognitive-behavioral therapy for reality testing
- Social skills training

4. CLUSTER B PERSONALITY DISORDERS

4.1 Antisocial Personality Disorder (ASPD)

Core Feature: Pervasive pattern of disregard for, and violation of, the rights of others since age 15.

DSM-5 Criteria:

- Must be 18+ years old
- Evidence of Conduct Disorder before age 15
- 3 or more of the following:

- Repeated unlawful behavior
- Repeated lying, use of aliases, conning others for personal gain
- Impulsivity / failure to plan ahead
- Irritability and aggressiveness (repeated physical fights/assaults)
- Reckless disregard for safety of self or others
- Consistent irresponsibility (work, finances)
- Lack of remorse

Conduct Disorder, Essential Precursor:

CATEGORY · EXAMPLES

Aggression to people/animals Bullying, cruelty, use of weapons

Destruction of property Fire-setting, vandalism

Deceitfulness/theft Shoplifting, breaking and entering

Serious rule violations Truancy, running away, staying out at night

Psychopathy vs. ASPD:

FEATURE	ASPD	PSYCHOPATHY (PCL-R)
Diagnostic system	DSM-5 / ICD-11	Hare PCL-R (research)
Focus	Behavioral	Trait + behavioral
Empathy deficit	Implied	Central (affective callousness)
Remorse	Absent	Core feature of absence
Prevalence	3–5% general population	15–25% incarcerated
Treatment response	Poor	Worse (arguably)

Hare Psychopathy Checklist-Revised (PCL-R), 20 items, score 0–40:

- Threshold: 30+ (North America), 25+ (UK/elsewhere)
- **Factor 1 (Interpersonal/Affective):** Glibness, grandiose self-worth, pathological lying, manipulation, shallow affect, callousness, lack of remorse, failure to accept responsibility
- **Factor 2 (Social Deviance/Lifestyle):** Stimulation-seeking, impulsivity, irresponsibility, parasitic lifestyle, poor behavioral controls, early behavioral problems, juvenile delinquency, criminal versatility

EXAM PEARL

PCL-R is the gold standard for psychopathy assessment in forensic settings. ASPD $\neq$ Psychopathy, all psychopaths meet ASPD criteria, but most ASPD individuals are not psychopathic.

Forensic Implications:

- Fitness to stand trial assessment
- Criminal responsibility (not an automatic defense, ASPD alone does not negate mens rea in most jurisdictions)
- Risk assessment (HCR-20, PCL-R used in violence risk)
- Section 377 IPC, IPC 84 (McNaughton rules in India), ASPD rarely qualifies for complete defense
- Dangerousness: ASPD with psychopathy = highest violence risk
- Prison population: ~50–80% meet criteria for ASPD

Etiology:

- Genetic: Heritability 40–60%; twin studies (Rhee & Waldman 2002)
- Neurobiology: Reduced amygdala and vmPFC volume; blunted fear conditioning; reduced P300 amplitude; serotonergic dysfunction
- Environmental: Physical / sexual abuse, inconsistent parenting, poverty, peer delinquency
- Gene-environment interaction: MAOA polymorphism + childhood maltreatment (Caspi et al. 2002, Science)

Management:

- Most challenging PD to treat; therapeutic nihilism common but not warranted
- Cognitive approaches: Addressing thinking errors (cognitive distortions of the "criminal mind")
- Contingency management: Behavioral modification in structured settings
- Therapeutic communities: Evidence for some subgroups
- Pharmacotherapy: Mood stabilizers for impulsive aggression (lithium, valproate, carbamazepine); SSRIs for irritability; no approved pharmacotherapy
- Comorbidity management: SUD (concurrent treatment essential)

4.2 Borderline Personality Disorder (BPD)

The most clinically important PD for exam purposes. Extensive content, master every detail.

Core Features: Pervasive instability in interpersonal relationships, self-image, affects, and marked impulsivity.

DSM-5 Criteria (5 of 9 required), PRAISE mnemonic (see D3):

1. Frantic efforts to avoid real or imagined abandonment (not including suicidal/self-mutilating behavior)
2. Pattern of unstable and intense interpersonal relationships characterized by alternating between extremes of idealization and devaluation (splitting)
3. Identity disturbance: markedly and persistently unstable self-image or sense of self
4. Impulsivity in at least 2 areas: spending, sex, substance use, reckless driving, binge eating
5. Recurrent suicidal behavior, gestures, threats, or self-mutilating behavior
6. Affective instability due to marked reactivity of mood
7. Chronic feelings of emptiness
8. Inappropriate, intense anger or difficulty controlling anger
9. Transient, stress-related paranoid ideation or severe dissociative symptoms

Prevalence: 1–2% general population; 10–15% psychiatric outpatients; 20–25% psychiatric inpatients; 75% female in clinical samples (though true sex ratio may be closer to equal, female presentation more recognized)

Epidemiology:

- Suicide: 8–10% lifetime completed suicide (250–400x general population rate)
- Self-harm: >70% report deliberate self-harm at some point
- Comorbidity: 85%+ have at least one comorbid Axis I disorder

Biosocial Theory (Linehan, 1993):

This is THE theoretical foundation of DBT. Must know this cold.

Key elements:

- **Emotional sensitivity:** Low threshold, high intensity, slow return to baseline
- **Invalidating environment:** Three types, typical invalidating, chaotic/neglectful, traumatic/abusive
- **Dialectical tension:** Between change and acceptance

Neurobiology of BPD:

SYSTEM	FINDING	CLINICAL CORRELATE
Amygdala	Hyperreactivity to emotional stimuli; prolonged activation	Emotional lability, sensitivity to rejection

SYSTEM	FINDING	CLINICAL CORRELATE
Prefrontal Cortex (vmPFC, dlPFC)	Hypofunction; reduced top-down regulation	Impulsivity, poor emotion regulation
Anterior Cingulate Cortex	Reduced activation	Poor error monitoring, impulsivity
HPA Axis	Dysregulation; cortisol hyporeactivity in some	Dissociation, blunted stress response
Serotonin	5-HT2A upregulation; reduced 5-HT1A	Impulsivity, aggression, suicidality
Dopamine	Transient psychosis during stress	Paranoid ideation, dissociation
Opioid System	Endogenous opioid dysregulation	Chronic emptiness, self-harm may temporarily restore baseline

Genetics:

- Heritability 35–65%
- Genetic overlap with MDD, bipolar disorder, PTSD
- Serotonin transporter gene (5-HTTLPR), COMT polymorphisms studied

Attachment and BPD:

- Fearful attachment (Bowlby), desire intimacy but fear it
- Hyperactivating attachment strategies, intense clinging, then rage at perceived abandonment
- Mentalization deficits: Fonagy, reduced capacity to represent mental states (own and others')
- Early trauma: 50–70% report childhood sexual or physical abuse; PTSD comorbidity high

Differential Diagnosis:

CONDITION · KEY DISTINGUISHING FEATURE

BPD vs Bipolar II See D4 comparison table, duration, sustained mood state, sleep need

BPD vs PTSD PTSD: trauma-specific triggers; no identity / relationship chaos outside trauma context

BPD vs ADHD ADHD: inattention / hyperactivity primary; stable identity; impulsivity more consistent

BPD vs NPD NPD: stable grandiose identity; no self-harm; empathy deficit different quality

BPD vs Histrionic PD HPD: stable identity; seeks attention rather than avoiding abandonment

BPD vs DID DID: amnesia, distinct alter identities; more severe dissociation

Comorbidity Patterns:

- BPD + MDD: 50–60%; treat BPD first, MDD often partially resolves with PD treatment
- BPD + PTSD: 25–50%; consider trauma-focused therapy when BPD is stabilized
- BPD + SUD: 35–40%; SUD worsens outcomes; integrated treatment required
- BPD + Eating Disorders: 25–50%; especially bulimia nervosa
- BPD + Bipolar II: True comorbidity vs diagnostic overlap, difficult to disentangle

Course and Prognosis (Longitudinal Studies):

STUDY · KEY FINDINGS

McLean Study of Adult Development (Zanarini et al.) 85% remission at 10 years; 50% at 2 years; symptomatic remission ≠ functional recovery

Collaborative Longitudinal Personality Disorders Study (CLPS) BPD more unstable than previously thought; significant improvement over 10 years

MSAD 20-year follow-up 99% remission from DSM criteria by age 40; ongoing functional impairment in 30%

EXAM PEARL

BPD has **better prognosis than originally believed**. Acute symptoms (impulsivity, self-harm, affective lability) remit faster than chronic ones (emptiness, identity issues). Functional recovery lags behind symptomatic recovery.

4.3 Histrionic Personality Disorder (HPD)

Core Feature: Pervasive pattern of excessive emotionality and attention-seeking behavior.

DSM-5 Criteria (5 of 8 required):

1. Uncomfortable when not the center of attention
2. Inappropriate sexually seductive or provocative behavior
3. Rapidly shifting, shallow emotional expression
4. Uses physical appearance to draw attention
5. Speech impressionistic, lacking in detail
6. Dramatic emotional expression; self-dramatization, theatricality
7. Suggestible, easily influenced by others

8. Considers relationships more intimate than they actually are

Differential: HPD vs BPD, stable identity in HPD; HPD seeks attention rather than fearing abandonment; HPD rarely self-harms.

Etiology: Theories of reward-seeking, genetic relationship with ASPD (both share disinhibition), childhood reinforcement of attention-seeking.

Management: Psychodynamic psychotherapy; cognitive approaches targeting attention-seeking schemas; rare pharmacotherapy except for comorbid conditions.

4.4 Narcissistic Personality Disorder (NPD)

Core Feature: Pervasive pattern of grandiosity, need for admiration, and lack of empathy.

DSM-5 Criteria (5 of 9 required):

- 1. Grandiose sense of self-importance
- 2. Preoccupied with fantasies of unlimited success, power, brilliance, beauty, or ideal love
- 3. Believes he or she is "special" and unique; can only be understood by similarly special people
- 4. Requires excessive admiration
- 5. Sense of entitlement
- 6. Interpersonally exploitative
- 7. Lacks empathy
- 8. Often envious or believes others are envious
- 9. Arrogant, haughty behaviors or attitudes

Grandiose vs. Vulnerable NPD:

FEATURE	GRANDIOSE (OVERT)	VULNERABLE (COVERT)
Presentation	Expansive, dominant, bold	Shy, withdrawn, insecure
Self-esteem	Overtly inflated	Secretly inflated, fragile
Entitlement	Explicit, demanding	Implicit, resentful
Empathy	Cold, dismissive	Distracted, self-focused
Mood	Euphoric or cold	Dysphoric, shame-prone
Seeking admiration	Active	Passive (expects to be recognized)
Response to criticism	Rage, contempt	Shame, collapse
Comorbidity	ASPD features	MDD, social anxiety

EXAM PEARL

Vulnerable/covert NPD often presents as depression or anxiety. The grandiosity is hidden under layers of self-deprecation, but careful history reveals entitlement and rage at perceived slights.

Narcissistic Injury and Rage:

- Narcissistic injury = threat to the grandiose self
- Narcissistic rage = explosive or cold fury in response to injury
- Kohut: NPD = arrested development, the grandiose self was never integrated

Neurobiology: Reduced cortical thickness in regions involved in empathy (AI, MCC); structural differences in PFC regions; alterations in mirror neuron system activity.

Management:

- Long-term psychotherapy: Schema Therapy (targeting Grandiosity schema, Demanding/Punitive Parent modes), Kohutian self-psychology (empathic mirroring), TFP
- Pharmacotherapy: SSRIs for depressive episodes; mood stabilizers for rage

5. CLUSTER C PERSONALITY DISORDERS

5.1 Avoidant Personality Disorder (AvPD)

Core Feature: Pervasive pattern of social inhibition, feelings of inadequacy, and hypersensitivity to negative evaluation.

DSM-5 Criteria (4 of 7 required):

1. Avoids occupational activities with significant interpersonal contact, fearing criticism or rejection
2. Unwilling to get involved with people unless certain of being liked
3. Shows restraint within intimate relationships, fearing shame or ridicule
4. Preoccupied with being criticized or rejected in social situations
5. Inhibited in new interpersonal situations because of feelings of inadequacy
6. Views self as socially inept, personally unappealing, or inferior
7. Unusually reluctant to take personal risks or engage in new activities

AvPD vs Social Anxiety Disorder (SAD):

FEATURE	AVPD	SAD
Pervasiveness	Across all domains, identity-level	More circumscribed

FEATURE	AVPD	SAD
Self-concept	Stable sense of inadequacy, inferiority	Anxiety about performance in specific situations
Onset	Childhood / adolescence, ego-syntonic	Often circumscribed trigger
Treatment response	Slower, requires schema work	Good response to CBT + SSRI
Relatedness	Wants closeness but avoids	Primarily fears performance / judgment

EXAM PEARL

AvPD and generalized SAD may be on a spectrum or even the same condition at different severity levels. DSM-5 doesn't fully resolve this. Answer exam questions by noting the overlap while distinguishing at extremes.

Schemas involved (Young): Defectiveness/Shame, Social Isolation, Failure, Emotional Inhibition, Subjugation

Management:

- Schema Therapy (limited reparenting for shame, experiential techniques)
- CBT with graduated exposure hierarchy
- Group therapy (social rehearsal opportunity)
- Pharmacotherapy: SSRIs (paroxetine, escitalopram); beta-blockers for performance anxiety (adjunct)

5.2 Dependent Personality Disorder (DPD)

Core Feature: Pervasive and excessive need to be taken care of, leading to submissive and clinging behavior, and fears of separation.

DSM-5 Criteria (5 of 8 required):

1. Difficulty making everyday decisions without excessive advice / reassurance
2. Needs others to assume responsibility for major life areas
3. Difficulty expressing disagreement, fearing loss of support or approval
4. Difficulty initiating projects alone (due to lack of confidence, not motivation)
5. Goes to excessive lengths to obtain nurturance and support
6. Feels uncomfortable or helpless when alone, fears inability to care for self
7. Urgently seeks another relationship when one ends
8. Unrealistically preoccupied with fears of being left to care for self

Schemas involved: Dependence / Incompetence, Abandonment / Instability, Subjugation, Self-Sacrifice

Differential:

- BPD: also fears abandonment but with intense rage and splitting; identity more chaotic
- DPD: stable dependency; no rage or self-harm; seeks care rather than avoids abandonment

Management:

- Cognitive therapy: Challenging incompetence beliefs; graduated autonomy tasks
- Schema Therapy: Reparenting for Dependence / Incompetence schema; strengthening Healthy Adult mode
- Assertiveness training

5.3 Obsessive-Compulsive Personality Disorder (OCPD)

Core Feature: Pervasive preoccupation with orderliness, perfectionism, and mental and interpersonal control, at the expense of flexibility, openness, and efficiency.

DSM-5 Criteria (4 of 8 required):

1. Preoccupied with details, rules, lists, order, organization, or schedules (loses the point of the activity)
2. Shows perfectionism that interferes with task completion
3. Excessively devoted to work and productivity (excluding obvious economic necessity)
4. Overconscientious, scrupulous, inflexible about morality, ethics, or values
5. Unable to discard worn-out or worthless objects (even without sentimental value)
6. Reluctant to delegate unless others submit to exact standards
7. Miserly spending (toward self and others); money hoarded for future catastrophe
8. Shows rigidity and stubbornness

OCPD vs OCD:

FEATURE	OCPD	OCD
Ego-syntonic	Yes, traits feel right, valued	No, obsessions are intrusive, unwanted
Obsessions	No true obsessions	Yes, intrusive, anxiety-provoking
Compulsions	No rituals to neutralize anxiety	Yes, reduce anxiety of obsessions
Perfectionism	Core to identity	May be present but secondary
Flexibility	Rigid	Can reason when not triggered
Treatment	Long-term therapy	CBT + ERP + SSRI
Comorbidity	Can co-occur with OCD	Separate conditions

EXAM PEARL

OCPD traits are **ego-syntonic**, the person believes their perfectionism, orderliness, and standards are virtues, not problems. OCD ego-dystonic obsessions are experienced as alien and distressing.

Schemas: Unrelenting Standards, Punitiveness, Emotional Inhibition, Subjugation

Management:

- Long-term CBT or Schema Therapy (targeting Unrelenting Standards schema)
- Relaxation and behavioral flexibility exercises
- SSRIs if comorbid OCD or depression
- Prognosis: Moderate, some traits are adaptive; personality change is slow

6. ETIOLOGY OF PERSONALITY DISORDERS

6.1 Attachment Theory (Bowlby)

Core concept: The quality of early attachment relationships shapes internal working models (IWMs), mental templates for self-other relationships.

Attachment Styles (Ainsworth Strange Situation):

STYLE	IWM	ADULT RELATIONSHIP PATTERN	PD LINK
Secure	Self as worthy, other as reliable	Comfortable with intimacy and autonomy	No PD
Anxious-Preoccupied	Self as unworthy, other as uncertain	Clingy, hyperactivating	BPD, DPD, HPD
Dismissing-Avoidant	Self as worthy, other as unnecessary	Deactivating; emotional suppression	Schizoid, Narcissistic
Fearful-Avoidant (Disorganized)	Self as unworthy, other as frightening	Collapsed strategy; approach-avoidance	BPD (especially with trauma)

CLINICAL ANCHOR

Disorganized attachment in infancy (Mary Main) is the strongest single childhood predictor of BPD features in adulthood. The caregiver is simultaneously the source of fear and the haven of safety, an irresolvable paradox.

Adult Attachment Interview (AAI), Hesse & Main: Assesses coherence of discourse about childhood experiences. BPD: often preoccupied / unresolved classification.

6.2 Schema Theory (Jeffrey Young)

Core concept: Early Maladaptive Schemas (EMSs) are broad, pervasive themes (memories, emotions, cognitions, bodily sensations) regarding oneself and relationships, developed in childhood, elaborated throughout life, dysfunctional to a significant degree.

18 Early Maladaptive Schemas (Young, Klosko, Weishaar 2003):

DOMAIN · SCHEMAS

Disconnection & Rejection Abandonment/Instability, Mistrust/Abuse, Emotional Deprivation, Defectiveness/Shame, Social Isolation

Impaired Autonomy & Performance Dependence/Incompetence, Vulnerability to Harm, Enmeshment, Failure

Impaired Limits Entitlement/Grandiosity, Insufficient Self-Control

Other-Directedness Subjugation, Self-Sacrifice, Approval-Seeking

Overvigilance & Inhibition Negativity/Pessimism, Emotional Inhibition, Unrelenting Standards, Punitiveness

Schema Activation Pathway:

Schema Modes (Young et al.):

Schema modes are moment-to-moment emotional states and coping responses. Mode model is the primary working model for BPD.

MODE CATEGORY	MODE	CLINICAL EXAMPLE
Child Modes	Vulnerable Child	Core wound, terror, shame, loneliness
	Angry/Impulsive Child	Rage, tantrums, boundary violations
	Undisciplined Child	Gives up, avoids discomfort
	Happy Child	Healthy baseline security
Maladaptive Coping	Compliant Surrender	Submitting to perceived authority
	Detached Protector	Emotional numbing, emptiness, dissociation
	Detached Self-Soother	Addictions, compulsive behaviors, self-soothing
	Self-Aggrandizer	Entitlement, superiority (NPD)
	Bully and Attack	Aggression, intimidation

MODE CATEGORY	MODE	CLINICAL EXAMPLE
	Conning and Manipulative	ASPD features
	Paranoid Overcontroller	Hypervigilance, control
Parent Modes	Punitive Parent	Harsh self-criticism, self-punishment, self-harm
	Demanding Parent	Unrelenting Standards, pressure
Healthy Modes	Healthy Adult	Integration, reality-testing, self-compassion
	Healthy Child	Playfulness, curiosity, joy

CLINICAL ANCHOR

In BPD, the flip between Vulnerable Child (terror of abandonment) → Angry Child (rage) → Detached Protector (dissociation/numbing) → Punitive Parent (self-harm) is often visible within a single session.

6.3 Biosocial Model (Linehan)

Already covered above in BPD section. Key additions:

Emotional sensitivity (biologically determined):

- **Low threshold:** Responds to lower-intensity stimuli
- **High reactivity:** Greater peak intensity
- **Slow return to baseline:** Slow recovery

Invalidating environment (environmental):

- Communicates that private experiences are invalid, oversimplified, or wrong
- Intermittently reinforces emotional escalation
- Prevents development of affect-labeling, affect-tolerance skills
- Three types: typical (well-meaning but dismissing), chaotic/neglectful, abusive/traumatic

6.4 Neurobiological Models

Prefrontal-Limbic Dysregulation (BPD):

- Amygdala: Hyperreactivity → emotion storms
- vmPFC: Hypofunction → poor fear extinction, poor mentalization
- ACC: Reduced activation → poor impulse monitoring
- Insula: Altered interoception → chronic emptiness, difficulties identifying emotions

Serotonin:

- Impulsivity and aggression linked to serotonergic hypofunction (5-HT1A, 5-HIAA in CSF)
- Suicidality linked to 5-HT2A receptor upregulation
- Basis for SSRI use in impulsive aggression

Dopamine:

- Mesolimbic dopamine sensitivity → stress-induced psychotic episodes in BPD and schizotypal PD

HPA Axis:

- Childhood trauma → HPA axis dysregulation
- Paradox in BPD: cortisol hyporeactivity to some stressors (dissociation) combined with hyperreactivity to others (abandonment cues)

7. TREATMENT APPROACHES

7.1 Schema Therapy (Young, Klosko, Weishaar)

Target population: Chronic characterological problems; PDs; treatment-resistant Axis I disorders with PD base

Three core principles:

1. **Limited reparenting**, therapist provides corrective emotional experience within professional boundaries; addresses unmet core needs
2. **Empathic confrontation**, validates the historical origin of schemas while confronting their current dysfunctionality
3. **Schema mode work**, identifies, names, and differentiates modes in session; uses chair work to create dialogue between modes

Three coping styles (schema responses):

RESPONSE	DESCRIPTION	EXAMPLES
Schema Surrender	Living out the schema; accepting it as truth	DPD patient submits to partners; OCPD patient overworks
Schema Avoidance	Cognitive, affective, behavioral avoidance	Schizoid avoids intimacy; HPD avoids self-reflection
Schema Overcompensation	Behaving in opposite way to the schema's theme	NPD patient acts grandiose to avoid Defectiveness

Schema Therapy Phases:

1. **Assessment and Education**, schema identification, schema history link, psychoeducation
2. **Emotion-focused change**, imagery rescripting, chair work, pattern-breaking
3. **Behavioral change**, flashcards, behavior logs, homework

Schema Therapy for BPD (Young's mode model protocol):

- Usually 2–3 years
- Frequent session attendance (1–2x / week)
- Key modes: Vulnerable Child, Angry Child, Detached Protector, Punitive Parent, Healthy Adult
- Key interventions: Limited reparenting of Vulnerable Child, chair work for Punitive Parent, behavioral experiments targeting avoidance
- Evidence: RCT evidence (Giessen-Coherent Approach, Arntz & van Genderen); non-inferior to TFP, superior to TAU at 3 years (Giessen study)

7.2 Dialectical Behaviour Therapy (DBT: Linehan)

Theoretical foundation: Biosocial theory (see above)

Core dialectic: Change ↔ Acceptance

The Dialectical Agreement: Patients learn that (a) they are doing the best they can, (b) they need to do better, (c) the therapist will help them do better.

DBT Modes of Treatment (standard comprehensive DBT):

1. **Individual therapy** (weekly, 50–60 min), diary card review, chain analysis, skills coaching
2. **Skills training group** (weekly, 2.5 hours), didactic + practice of the 4 skill modules
3. **Phone coaching** (as needed), generalize skills to real-world crises; before self-harm, not after
4. **Consultation team** (therapist support), prevent burnout, maintain adherence

DBT Target Hierarchy (in individual therapy):

1. Life-threatening behaviors (suicide, self-harm), always first
2. Therapy-interfering behaviors (missing sessions, non-compliance)
3. Quality-of-life-interfering behaviors (substance use, relationship chaos, job loss)
4. Skills building

4 DBT Skill Modules (DIMS mnemonic, see D3):

MODULE	CORE SKILLS	CLINICAL FUNCTION
Distress Tolerance	TIPP, ACCEPTS, self-soothe, improve the moment, radical acceptance, turning the mind	Crisis survival without making things worse
Interpersonal Effectiveness	DEAR MAN, GIVE, FAST	Getting what you need while maintaining relationships and self-respect
Mindfulness	Wise Mind, What skills (observe, describe, participate), How skills (non-judgmentally, one-mindfully, effectively)	Foundation for all other modules
Emotion Regulation	ABC PLEASE, STOP, check the facts, opposite action, problem-solving	Reduce emotional vulnerability; change unwanted emotions

DBT Evidence Base:

- Multiple RCTs for BPD (Linehan et al. 1991, 1994, 2006)
- Reduces suicidal behavior, self-harm, inpatient hospitalizations
- Effective for eating disorders, SUD, adolescents (DBT-A)
- NICE guidelines: Recommended for BPD

7.3 Mentalization-Based Treatment (MBT: Bateman & Fonagy)

Theoretical foundation: Mentalization = capacity to understand mental states in self and others; derived from attachment theory.

BPD pathology in MBT: Impaired mentalization under attachment stress → hyperactivation of attachment system → emotional storms → pre-mentalistic modes:

- **Psychic equivalence:** Inner states = outer reality (feels like certainty)
- **Pretend mode:** Disconnection from reality (dissociation)
- **Teleological mode:** Only physical/ concrete actions felt as real

MBT Technique:

- **Stop and rewind:** Slow down moments of mentalization failure
- **Exploration vs. interpretation:** Exploratory stance; therapist genuinely curious
- **Mentalization of the therapist's own mind:** Transparency, acknowledging errors
- **Affect-focused mentalization:** Linking emotion → mental state → behavior

Setting: Can be delivered in day-hospital or outpatient (18-month protocol studied in RCTs)

Evidence (Bateman & Fonagy 1999, 2001, 2008, 2009): Superiority over TAU and structured clinical management; maintained at 5-year follow-up

7.4 Transference-Focused Psychotherapy (TFP: Clarkin, Kernberg)

Theoretical foundation: Object relations theory (Kernberg); BPD = identity diffusion due to failure to integrate good-and-bad representations of self and others (splitting).

Core mechanism: Analyzing the transference, the dyadic unit of relationship (self representation + object representation + affect) is activated in the therapeutic relationship.

Technique:

- Active interpretation of transference from the start
- Confrontation of splitting and identity diffusion
- Setting a firm treatment contract (limit setting on self-destructive behavior)
- Three channels of communication: verbal content, nonverbal behavior, countertransference

Evidence: RCTs show superiority over supportive therapy; comparable to DBT on most outcomes; TFP superior on some narrative coherence and attachment measures.

7.5 General Psychiatric Management (GPM: Gunderson)

Goal: Make PD treatment accessible outside specialist centers

Core elements:

- Psychoeducation (BPD basics, interpersonal hypersensitivity)
- Case management
- Individual therapy focused on interpersonal hypersensitivity
- Medication management
- Vocational/social rehabilitation

Evidence: Non-inferior to DBT in one RCT (McMain et al. 2009, JAMA), important finding that challenges assumption that specialist PD therapy is necessary for all patients

7.6 STEPPS (Systems Training for Emotional Predictability and Problem-Solving)

- Group-based adjunctive treatment for BPD
 - 20-week structured cognitive-behavioral skills program
 - Designed as add-on to existing treatment
 - Involves patient's support network (significant others educated about BPD)
 - Evidence: RCTs show symptom reduction comparable to DBT in some domains
-

8. PHARMACOTHERAPY IN PERSONALITY DISORDERS

EXAM STRATEGY

The exam will ask about pharmacotherapy for PD, know that evidence is limited, off-label, symptom-targeted. No medication cures PD. FDA has no approved medications for any PD.

Principles:

1. Target symptoms, not personality disorder per se
2. Short-term or adjunctive, not first-line
3. Risk of misuse (BPD + SUD)
4. Risk of overdose in BPD, careful prescription, limited supply
5. Often undermines therapy (patient uses medication to avoid skills practice)

Evidence-based Pharmacotherapy by Symptom Domain:

SYMPTOM DOMAIN	FIRST-LINE	SECOND-LINE	EVIDENCE LEVEL
Emotional dysregulation / Affective instability	Mood stabilizers (valproate, lamotrigine)	Omega-3 fatty acids	Moderate (RCTs)
Impulsive aggression / Self-harm	SSRIs (fluoxetine), Mood stabilizers (valproate)	Lithium, atypical antipsychotics	Moderate
Transient psychosis / Paranoid ideation	Low-dose atypical antipsychotics (olanzapine 2.5–5mg, quetiapine 25–50mg)	Haloperidol (historical; avoid)	Moderate
Depressive symptoms	SSRIs (but lower effect than MDD)	MAOIs (historical), TCAs (caution)	Low-moderate
Anxiety	SSRIs, SNRIs	Buspirone	Low
Dissociation	Low-dose antipsychotics, naltrexone (for self-harm linked to opioid dysregulation)		Low
Cognitive-perceptual disturbances	Low-dose antipsychotics		Moderate

Specific Medications:

- **Valproate (Sodium Valproate/Divalproex):** Best evidence for BPD impulsivity and aggression; also ASPD impulsive aggression; RCTs by Hollander et al., Stein et al.
- **Lamotrigine:** Affective instability in BPD; women specifically; RCT evidence
- **Fluoxetine:** Impulsive aggression (Coccaro et al.); modest SSRI effects in BPD

- **Olanzapine 2.5–5 mg:** Short-term BPD; reduce transient psychosis; Zanarini et al. RCTs; weight gain limits long-term use
- **Quetiapine 25–300 mg:** Sleep, anxiety, dissociation in BPD; commonly prescribed despite limited RCT evidence
- **Naltrexone 25–100 mg:** Case series/open-label data for self-harm with opioid system involvement; some evidence for dissociation

What NOT to use:

- Benzodiazepines: Risk of dependence, behavioral disinhibition in BPD; impulsivity worsens; evidence of harm
- MAOIs: Historical use; dangerous in impulsive patients
- Lithium: Limited BPD data; toxicity risk in impulsive overdose patients

EXAM PEARL

The APA Practice Guideline for BPD (2001, updated) states pharmacotherapy should be **adjunctive to psychotherapy**. The UK NICE Guideline (2009) was more restrictive, recommended **AGAINST** routine pharmacotherapy for BPD except for comorbid conditions.

9. COMORBIDITY PATTERNS

BPD + SUD:

- Prevalence: 35–40% lifetime; alcohol most common, stimulants, opioids
- Mechanisms: SUD as affect regulation (self-medication); SUD as impulsive behavior; shared neurobiological vulnerability
- Treatment: Integrated treatment superior to sequential; DBT adapted for SUD (DBT-S); avoid benzodiazepines
- Prognosis: SUD significantly worsens BPD outcomes

BPD + PTSD:

- Prevalence: 25–50%
- Sequencing: Stabilize BPD first (crisis management, skills); then introduce trauma-focused work
- DBT-PTSD (Harned, Linehan): Adapted protocol; can treat PTSD once self-harm under control
- Prolonged Exposure adaptation required for BPD (titrated, phased approach)

ASPD + SUD:

- 70–80% of ASPD individuals have lifetime SUD

- Mutually exacerbating: SUD lowers impulse control; ASPD increases drug-seeking and criminal behavior
- Treatment: Therapeutic communities with contingency management; integrated dual diagnosis approach; naltrexone for opioid / alcohol use
- Prognosis: Worst among all PD-SUD combinations

NPD + Depression:

- Narcissistic injury → narcissistic collapse → MDD presentation
- Antidepressants helpful for acute episode; but schema work on grandiosity schemas needed to prevent recurrence

10. ASSESSMENT INSTRUMENTS

INSTRUMENT	TYPE	WHAT IT MEASURES	NOTES
SCID-5-PD (Structured Clinical Interview for DSM-5 PDs)	Semi-structured interview	All 10 DSM-5 PDs	Gold standard for DSM-5; administered by clinician
IPDE (International Personality Disorder Examination)	Semi-structured interview	ICD-10 and DSM-IV PDs	WHO instrument; cross-cultural validity
PDQ-4+ (Personality Diagnostic Questionnaire)	Self-report	DSM-IV PDs (screening)	High sensitivity, low specificity; screening tool
MCMI-IV (Millon Clinical Multiaxial Inventory)	Self-report	Millon's personality typology + Axis I	Clinical populations; Millon's dimensional model
PAI (Personality Assessment Inventory)	Self-report	Broad personality dimensions	Borderline Features subscale
ZAN-BPD (Zanarini Rating Scale for BPD)	Clinician-rated	BPD severity	Treatment monitoring
BSL-23 (Borderline Symptom List)	Self-report	BPD symptom severity	Brief, validated
PCL-R (Hare Psychopathy Checklist-Revised)	Interview + file review	Psychopathy dimensions	Forensic settings; requires training
YSQ (Young Schema Questionnaire)	Self-report	18 EMSs	Schema therapy assessment
SMI (Schema Mode Inventory)	Self-report	14 schema modes	Mode therapy assessment

EXAM PEARL

SCID-5-PD is the standard for DSM-5 research and careful clinical assessment. IPDE used for ICD-based assessment. PDQ-4+ for screening (high false positive rate).

11. ICD-11 CHANGES: DETAILED

Why the change?

1. Categorical diagnoses had poor reliability across raters and cultures
2. High comorbidity (most patients meet criteria for 2+ PDs), suggests categories are artificial
3. NOS was overused (25–50% of clinical PDs were NOS)
4. Dimensional approach aligned with personality psychology research
5. Better treatment planning: severity predicts treatment intensity; traits guide specific techniques

Severity Anchor Points:

LEVEL	SELF/INTERPERSONAL FUNCTIONING	TRAIT DISTURBANCE	EXAMPLES
Mild	Significant distress; some domains impaired	Pervasive but less marked	Anxious person with rigid interpersonal patterns
Moderate	Most domains impaired; relationships significantly damaged	Multiple traits affected	Classic BPD-level impairment
Severe	Severe impairment all domains; may be danger to self/others	Extreme and rigid	Severe BPD with repeated suicide attempts, ASPD with violence

Trait Qualifier Co-occurrence:

- Multiple qualifiers can be assigned (e.g., Negative Affectivity + Disinhibition + Borderline Pattern)
- This replaces the old practice of multiple categorical diagnoses (e.g., BPD + HPD + DPD)

Clinical Implications of ICD-11 Shift:

1. No more "revolving door" diagnostic comorbidity
2. Severity-matched treatment intensity (mild → GPM; severe → specialist DBT/ST)
3. Borderline Pattern retained because of its clinical utility and treatment evidence base
4. International harmonization with dimensional personality psychology

12. COURSE AND PROGNOSIS

General Principles:

- Most PDs improve with age (especially Cluster B acute symptoms)
- Cluster A: Most stable; least amenable to change
- Cluster B: Most variable; BPD improves more than previously thought; ASPD may "burn out" behaviorally after 40
- Cluster C: Moderate stability; can improve with treatment

McLean Adult Development Study (Zanarini et al.), BPD-specific:

- 290 participants with BPD; followed prospectively
- 50% achieved sustained remission at 2 years
- 85% achieved sustained remission at 10 years
- Remission = no criteria met for 2+ years
- BUT functional recovery significantly lagged behind symptomatic remission
- Predictors of remission: Less severe initial symptoms, absence of childhood sexual abuse, less comorbid SUD

Collaborative Longitudinal Personality Disorders Study (CLPS):

- Multiple PDs followed over 10 years
- BPD had highest rate of remission among Cluster B
- OCPD surprisingly stable
- ASPD moderate improvement in behavioral symptoms over time

Predictors of Poor Outcome in BPD:

- Childhood sexual abuse (CSA), strongest predictor of non-remission
- Comorbid SUD
- Comorbid MDD
- History of prolonged inpatient hospitalization
- Low socioeconomic status
- Lack of social support

13. SPECIAL TOPICS

13.1 PD in Adolescents

- DSM-5 allows diagnosis in adolescents if traits are pervasive, stable, and not part of a developmental phase
- BPD diagnosis in adolescents is valid and does not stigmatize more than leaving it undiagnosed

- NICE recommends against diagnosing full ASPD before age 18 (can note conduct disorder + traits)
- DBT-A (Adolescent version): Adapted for teens; includes family sessions; shorter program (16–24 weeks)

13.2 Forensic Aspects

ASPD in forensic psychiatry:

- Most common PD in prison populations
- Risk assessment instruments: PCL-R, HCR-20 (Historical-Clinical-Risk Management 20)
- Fitness to stand trial: Separate from mental disorder, ASPD alone rarely negates fitness
- Diminished responsibility: ASPD does not automatically qualify; depends on case specifics
- Dangerous and Severe Personality Disorder (DSPD): UK construct; includes high PCL-R + high violence risk; controversial

BPD in forensic contexts:

- Self-harm in custody: Risk management challenge
- Suicide risk management in correctional facilities
- Gender: Female prisoners overrepresented with BPD diagnosis

13.3 Trauma and PD

- Complex PTSD (ICD-11): Distinguished from BPD by specific trauma history, primary affect dysregulation, without identity diffusion or splitting
- Differential: Complex PTSD → trauma-focused treatment primary; BPD → comprehensive PD treatment with trauma work integrated later
- Overlap is substantial; clinical judgment required

EXAM STRATEGY

Complex PTSD is a new ICD-11 diagnosis. It overlaps significantly with BPD. Know the key differentials: Complex PTSD has core PTSD features (reexperiencing, avoidance, hyperarousal) plus disturbances in self-organization (affect dysregulation, negative self-concept, relationship difficulties), but without the identity diffusion, splitting, and abandonment fears of BPD.

14. EXAMINATION SUMMARY TABLE

PERSONALITY DISORDER	CORE FEATURE	KEY CRITERIA/FEATURES	PRIMARY TREATMENT	KEY PHARMACOTHERAPY
Paranoid PD	Pervasive distrust	Suspects exploitation; grudges; counterattacks	Cognitive/supportive psychotherapy	Low-dose antipsychotics if needed
Schizoid PD	Detachment from relationships	Chooses solitary; indifferent to praise/criticism	Supportive therapy; rarely seeks treatment	SSRIs for comorbid anxiety
Schizotypal PD	Cognitive/perceptual distortions + social anxiety	Magical thinking; ideas of reference; odd speech	Low-dose antipsychotics; CBT	Risperidone, olanzapine
ASPD	Disregard for rights of others	Conduct Disorder precursor; PCL-R psychopathy overlap	Cognitive therapy; therapeutic communities	Mood stabilizers for impulsivity
BPD	Instability in relationships, identity, affect	5/9 DSM criteria; PRAISE mnemonic	DBT, Schema Therapy, MBT, TFP	Valproate, lamotrigine, low-dose antipsychotics
Histrionic PD	Excessive emotionality + attention-seeking	Dramatic; suggestible; seductive	Psychodynamic/cognitive therapy	SSRIs for comorbid conditions
Narcissistic PD	Grandiosity + lack of empathy	Entitlement; exploitation; grandiose vs vulnerable subtypes	Schema Therapy, Kohutian therapy	SSRIs for depression
Avoidant PD	Social inhibition + feelings of inadequacy	Avoids social contact; fears rejection; wants closeness	Schema Therapy; CBT + exposure; group therapy	SSRIs, beta-blockers
Dependent PD	Excessive need to be cared for	Submissive; fears separation; helplessness alone	Cognitive therapy; Schema Therapy	SSRIs for comorbid anxiety
OCPD	Perfectionism + rigidity	Ego-syntonic; interferes with completion; miserly	CBT; Schema Therapy	SSRIs if comorbid OCD/MDD

Model Answers

Format: Each answer is structured for 10-mark exam responses (~600–800 words written equivalent). Use the structure to guide what to write, not as rigid templating.

ANSWER 1: Describe the management of Borderline Personality Disorder.

EXAM STRATEGY

This is the highest-yield single question in the PD chapter. Must know DBT + Schema Therapy + pharmacotherapy + risk management cold.

Introduction

Borderline Personality Disorder (BPD) is characterised by pervasive instability in affect, interpersonal relationships, self-image, and impulse control, affecting 1–2% of the general population and 10–20% of psychiatric inpatients. Management is multimodal, with psychotherapy as the cornerstone and pharmacotherapy as adjunctive and symptom-targeted.

Risk Assessment (Immediate Priority)

- Assess suicidal ideation, plan, intent, access to means
- Assess severity and frequency of self-harm behaviours
- Assess level of dissociation and agitation
- Safety planning: emergency contacts, crisis plan, means restriction
- Hospitalisation threshold: acute high-risk only; brief admission (avoid long-term, reinforces regression and dependence)

Psychotherapy (First-Line)

Dialectical Behaviour Therapy (DBT, Linehan, 1993)

- Theoretically based on Biosocial Model: emotional sensitivity + invalidating environment
- Standard DBT: 4 components:
 - Individual therapy (weekly): chain analysis, diary card, DBT target hierarchy
 - Skills training group (weekly, 2.5h): 4 modules, Mindfulness, Distress Tolerance, Emotion Regulation, Interpersonal Effectiveness
 - Phone coaching: generalise skills to crises

- Consultation team: therapist support, prevent burnout
- Target hierarchy: Life-threatening > Therapy-interfering > Quality-of-life > Skills acquisition
- Duration: Standard programme 6–12 months; full benefits at 12–24 months
- Evidence: Multiple RCTs; reduces suicide attempts, self-harm, hospitalisations (NICE recommended)

Schema Therapy (Young, Klosko, Weishaar)

- Targets: Underlying schemas (Abandonment, Defectiveness, Mistrust) via mode work
- Key modes in BPD: Vulnerable Child, Angry / Impulsive Child, Detached Protector, Punitive Parent, Healthy Adult
- Techniques: Limited reparenting, chair work, imagery rescripting, flashcards
- Duration: 2–3 years; RCT evidence (Giessen Coherent Approach; superior to TAU at 3 years)

Mentalization-Based Treatment (MBT, Bateman & Fonagy)

- Target: Impaired mentalization under attachment stress
- Format: Day hospital (18-month) or outpatient
- Techniques: Stop-and-rewind, affect-focused mentalization, therapist transparency
- Evidence: RCTs superiority over TAU; effects maintained at 5 years

Transference-Focused Psychotherapy (TFP, Clarkin, Kernberg)

- Object relations model: Interprets dyadic transference activations
- Addresses identity diffusion via transference interpretation
- Evidence: Comparable to DBT; superior on attachment / narrative measures

General Psychiatric Management (GPM, Gunderson)

- For settings without specialist PD services
- Psychoeducation + case management + interpersonal focus
- Evidence: Non-inferior to DBT (McMain et al. 2009)

Pharmacotherapy (Adjunctive, Symptom-Targeted)

SYMPTOM DOMAIN	MEDICATION	EVIDENCE
Affective instability	Valproate / lamotrigine	RCT evidence (Hollander, Tritt)
Impulsive aggression	Valproate, SSRIs	Moderate
Transient psychosis	Low-dose olanzapine (2.5–5mg) or quetiapine	Moderate
Depressive symptoms	SSRIs	Lower effect than in MDD

SYMPTOM DOMAIN	MEDICATION	EVIDENCE
Chronic emptiness/dissociation	Naltrexone (case series)	Low

- **Avoid:** Benzodiazepines (disinhibition, dependence, overdose risk)
- **Caution:** Limited supply; overdose risk; polypharmacy
- **NICE guidance (2009):** No pharmacotherapy routinely for BPD; treat comorbid conditions

Special Populations

- **BPD + SUD:** Integrated treatment; DBT-S adaptation; avoid opioid prescribing; naltrexone for alcohol/opioid use
- **BPD + PTSD:** Stabilise BPD first; introduce DBT-PTSD (Harned) when self-harm controlled
- **BPD in adolescents:** DBT-A (adolescent adaptation); family component; valid to diagnose

Prognosis

- McLean Study: 50% remission at 2 years; 85% at 10 years
- Symptomatic remission precedes functional recovery
- Predictors of poor outcome: CSA, SUD, severe initial presentation

ANSWER 2: Discuss the principles and evidence base of Dialectical Behaviour Therapy (DBT).

Introduction

DBT was developed by Marsha Linehan (1993) as an evidence-based treatment originally designed for chronically suicidal women with BPD. It has since been adapted for adolescents, eating disorders, SUD, and PTSD. DBT is a comprehensive, multi-modal treatment programme grounded in the biosocial theory of BPD.

Theoretical Foundation: Biosocial Model

- **Biological predisposition:** Emotional sensitivity (low threshold, high intensity, slow return to baseline)
- **Invalidating environment:** Dismisses, punishes, or oversimplifies emotional experience
- **Result:** Impaired emotion regulation as the core deficit; secondary problems (self-harm, suicidality, relationship chaos) are downstream

Core Dialectic

The fundamental tension in DBT: **Change** (cognitive-behavioural) ↔ **Acceptance** (validation, mindfulness, radical acceptance)

This tension is held by the therapist, the group, and ultimately internalised by the patient.

Four Modes of Standard DBT

1. **Individual therapy** (50–60 min, weekly): Diary card review, behavioural chain analysis, solution analysis, in-session DBT
2. **Skills training group** (2.5 hours, weekly): Didactic + practice; 4 modules over 24–32 weeks
3. **Phone coaching** (as needed): Skills generalisation to real crises; before self-harm not after
4. **Consultation team** (weekly, for therapists): Prevent burnout, maintain adherence, problem-solve

Target Hierarchy in Individual Therapy

1. Life-threatening behaviours (suicidality, self-harm, violence), **always addressed first**
2. Therapy-interfering behaviours (non-attendance, late, non-compliance)
3. Quality-of-life-interfering behaviours (SUD, abusive relationships, unemployment)
4. Skills acquisition and generalisation

Four Skill Modules

1. Mindfulness (Core Module, Foundation)

- Wise Mind (intersection of Reasonable Mind + Emotion Mind)
- What skills: Observe, Describe, Participate
- How skills: Non-judgmentally, One-mindfully, Effectively
- Function: Awareness; observation of experience without reactivity

2. Distress Tolerance

- Crisis survival skills: TIPP (Temperature, Intense exercise, Paced breathing, Progressive relaxation)
- ACCEPTS (Activities, Contributing, Comparisons, Emotions, Pushing away, Thoughts, Sensations)
- Self-soothe (5 senses)
- IMPROVE the moment (Imagery, Meaning, Prayer, Relaxation, One thing in the moment, Vacation, Encouragement)
- Radical Acceptance: Accepting reality as it is, not as it should be
- Function: Tolerate distress without making it worse; crisis survival

3. Emotion Regulation

- Understand emotions; reduce vulnerability (ABC PLEASE)
- Check the facts; Opposite action; Problem-solving
- STOP skill (crisis); Build positive experiences
- Function: Change unwanted emotional states

4. Interpersonal Effectiveness

- DEAR MAN: Describe, Express, Assert, Reinforce; Mindful, Appear confident, Negotiate (for objectives)
- GIVE: Gentle, Interested, Validate, Easy manner (for relationships)
- FAST: Fair, Apologies (avoid), Stick to values, Truthful (for self-respect)
- Function: Navigate relationships; get needs met while maintaining relationships and self-respect

DBT Techniques

- **Chain analysis:** Step-by-step analysis of events, thoughts, feelings, urges, and actions leading to problem behaviour; then solution analysis
- **Diary card:** Daily monitoring of emotions, urges, self-harm, skills used
- **Commitment strategies:** Pros and cons, devil's advocate, foot-in-door
- **Consultation team:** Therapists remain in the therapeutic frame; avoid burnout

Evidence Base

STUDY · FINDINGS

Linehan et al. 1991 (JAMA) DBT vs TAU: reduced parasuicide, hospitalisations, treatment dropout

Linehan et al. 1994 Benefits maintained at 1-year follow-up

Linehan et al. 2006 DBT vs community treatment by experts: DBT superior on suicide attempts, ED visits

McMain et al. 2009 DBT vs GPM: Non-significant differences, both effective

Koons et al. 2001 DBT effective for suicidality in veterans with BPD

Meta-analysis (Kliem et al. 2010) Moderate effect sizes; superior to waitlist/TAU

Adaptations

- **DBT-A** (adolescents): Family component; 16–24 weeks
- **DBT-S** (substance use): Dialectical abstinence; path to clear mind

- **DBT for eating disorders:** DEBRA variant
 - **DBT-PTSD** (Harned & Linehan): Add PE after Stage 1 safety established
-

ANSWER 3: Describe Schema Therapy for Personality Disorders.

Introduction

Schema Therapy was developed by Jeffrey Young (Young, Klosko & Weishaar, 2003) as an integrative psychotherapy combining cognitive-behavioural, experiential, interpersonal, and psychoanalytic elements. It was specifically designed for personality disorders and chronic treatment-resistant Axis I disorders with a characterological base.

Theoretical Foundations

Early Maladaptive Schemas (EMSs): Broad, pervasive themes of dysfunctional memory, emotion, cognition, and bodily sensation regarding self and relationships, developed in childhood and elaborated throughout life.

Origin: Four core childhood needs, when unmet, generate schemas:

1. Safe attachment (security, stability, nurturance)
2. Autonomy, competence, sense of identity
3. Limits (self-control, self-discipline)
4. Freedom to express needs/emotions
5. Spontaneity and play

18 Early Maladaptive Schemas

DOMAIN · SCHEMAS

Disconnection & Rejection Abandonment, Mistrust/Abuse, Emotional Deprivation, Defectiveness/Shame, Social Isolation

Impaired Autonomy & Performance Dependence/Incompetence, Vulnerability to Harm, Enmeshment, Failure

Impaired Limits Entitlement/Grandiosity, Insufficient Self-Control

Other-Directedness Subjugation, Self-Sacrifice, Approval-Seeking

Overvigilance & Inhibition Negativity/Pessimism, Emotional Inhibition, Unrelenting Standards, Punitiveness

Three Coping Styles (Schema Responses)

1. **Surrender**, live out the schema (e.g., dependent patient submits to controlling partner)
2. **Avoidance**, avoid schema activation (e.g., schizoid patient avoids intimacy)

3. **Overcompensation**, behave in opposite direction (e.g., NPD patient acts superior to avoid Defectiveness)

Schema Mode Model

Modes = moment-to-moment emotional states and coping responses. Especially relevant for BPD.

Key modes in BPD:

- **Vulnerable Child**: Core wound, terror, shame, abandonment fear
- **Angry/Impulsive Child**: Rage, acting out
- **Detached Protector**: Emotional numbing, dissociation, emptiness
- **Punitive Parent**: Harsh self-criticism, self-punishment → self-harm
- **Healthy Adult**: Reality-testing, self-compassion, integration

Three Therapeutic Strategies

1. **Limited Reparenting**, therapist provides corrective emotional experience within professional limits; meets core needs the patient never had met
2. **Empathic Confrontation**, validates historical origin, challenges current dysfunctionality
3. **Schema Mode Work**, identify, name, differentiate modes; create dialogue between modes

Treatment Phases

Phase 1, Assessment and Psychoeducation

- Schema assessment (YSQ, SMI, imagery, history)
- Psychoeducation: linking childhood experience → schemas → current patterns
- Case conceptualisation: schema map

Phase 2, Experiential/Emotional Change Methods

- Imagery rescripting: revisit childhood scenes; intervene as Healthy Adult
- Chair work (empty chair): dialogue between modes (e.g., Healthy Adult addresses Punitive Parent)
- Limited reparenting in session

Phase 3, Behavioural Pattern-Breaking

- Flashcards (schema flashcards written by therapist and patient together)
- Behavioral experiments: test schema predictions
- Homework and between-session practice

Assessment Tools

- **Young Schema Questionnaire (YSQ)**: 232 items; identifies 18 EMSs

- **Schema Mode Inventory (SMI):** Mode assessment
- **Young Parenting Inventory (YPI):** Parenting history; schema origin

Evidence Base

- **RCT for BPD (Giessen-Coherent Approach, Arntz & van Genderen):** Schema Therapy vs TFP: ST superior at 3 years
 - **RCT vs TAU:** Schema Therapy significantly superior
 - **Cluster C, OCPD, NPD:** Emerging evidence
 - **Duration:** 25–50 sessions for Cluster C; 2–4 years for severe BPD
-

ANSWER 4: Compare ICD-11 and DSM-5 classification of Personality Disorders.

Introduction

The classification of personality disorders underwent a radical revision in ICD-11 (effective 2022), shifting from a categorical to a dimensional model. DSM-5 (2013) retained the traditional categorical approach in its main body, while proposing a hybrid dimensional model (AMPD) in Section III. Understanding both systems and their differences is essential for contemporary psychiatric practice and research.

DSM-5 Categorical Model (Main Section, Section II)

- **10 specific PDs** in 3 clusters (A/B/C)
- Diagnosis = polythetic criteria (threshold number of criteria met)
- Advantages: Familiar; clinical communication; research comparability
- Disadvantages: High comorbidity; NOS overuse; arbitrary thresholds; poor reliability

DSM-5 Alternative Model (AMPD: Section III)

- **Criterion A:** Level of Personality Functioning Scale (LPFS)
- 4 domains: Identity, Self-Direction, Empathy, Intimacy
- Rated 0–4; threshold = 2 (moderate impairment)
- **Criterion B:** 5 trait domains (Negative Affectivity, Detachment, Antagonism, Disinhibition, Psychoticism) with 25 facets
- Operationalises 6 specific PDs (ASPD, AvPD, BPD, NPD, OCPD, Schizotypal)
- Remaining 4 diagnosed with general criteria only

ICD-11 Dimensional Model

Step 1, Severity

LEVEL · IMPAIRMENT

Personality Difficulty (not a disorder) Minimal

Mild PD Some domains; not all relationships

Moderate PD Most domains; most relationships

Severe PD Severe; all areas; danger to self/others

Step 2, Trait Qualifiers

QUALIFIER	DESCRIPTION	APPROXIMATE OLD CATEGORY
Negative Affectivity	Intense, unstable emotions	BPD (affective), HPD
Detachment	Social withdrawal, emotional restriction	Schizoid, AvPD
Dissociality	Disregard for others' rights	ASPD, NPD
Disinhibition	Impulsivity, irresponsibility	ASPD, BPD (behavioural)
Anankastia	Rigid perfectionism, orderliness	OCPD
Borderline Pattern (specifier)	Emotional instability, identity disturbance, self-harm	BPD

Key Differences

FEATURE	DSM-5 (SECTION II)	DSM-5 AMPD (SECTION III)	ICD-11
Model	Categorical	Hybrid dimensional	Fully dimensional
Number of diagnoses	10	6 operationalised	No categorical diagnoses
Comorbidity problem	Yes	Reduced	Eliminated
Severity dimension	No	Yes (LPFS)	Yes (3 levels)
Trait structure	None	5 domains, 25 facets	5 domains + borderline specifier
BPD status	Full diagnosis	Operationalised	Borderline Pattern specifier only
Clinical utility	High familiarity	Moderate	High severity-based planning
Research use	Standard	Growing	Recommended for ICD countries
Implementation	Routine clinical	Research/specialty	Recommended from 2022

Clinical Implications of ICD-11 Shift

- Severity drives treatment intensity (mild → GPM; severe → specialist DBT/ST)
- Multiple qualifiers replace multiple categorical diagnoses
- Borderline Pattern retained for clinical utility
- Better international harmonisation

EXAM PEARL

ICD-11 is used in India (ICD takes precedence in clinical documentation and health statistics). DSM-5 is widely used in research and some teaching institutions. Know both systems. The exam may ask you to compare them specifically.

ANSWER 5: Discuss the forensic implications of Antisocial Personality Disorder.

Introduction

Antisocial Personality Disorder (ASPD) is characterised by persistent disregard for and violation of others' rights, with onset before age 15 (via Conduct Disorder). It carries the most significant forensic psychiatric implications of any personality disorder, intersecting with criminal justice, risk assessment, fitness to stand trial, and criminal responsibility.

Prevalence in Forensic Settings

- General population: 3–5%
- Prison populations: 40–70% (male prisoners)
- Female prisoners: 10–20% BPD-predominant; ASPD less common
- Recidivism: ASPD is one of the strongest predictors of criminal recidivism

ASPD and Psychopathy (PCL-R)

Forensic distinction essential:

FEATURE	ASPD	PSYCHOPATHY (PCL-R ≥ 30)
Prevalence in prisons	40–70%	15–25%
Affective deficits	Implied	Central, shallow affect, callousness
Violence risk	Elevated	Substantially elevated
Treatment response	Poor	Potentially worse
Instrumental vs reactive violence	Reactive predominant	Instrumental violence more common

PCL-R Structure (Hare 1991):

- Factor 1 (Interpersonal / Affective): Glibness, grandiosity, pathological lying, manipulation, callousness, lack of remorse
- Factor 2 (Social Deviance / Lifestyle): Stimulation-seeking, impulsivity, irresponsibility, juvenile delinquency, criminal versatility
- Threshold: 30 (North America), 25 (Europe / India)

Fitness to Stand Trial

- ASPD alone does NOT negate fitness to stand trial
- Fitness requires: (1) understanding charges, (2) understanding court proceedings, (3) ability to instruct counsel
- ASPD individuals typically retain all three capacities
- Assessment: Use structured tools (MacCAT-CA)

Criminal Responsibility

McNaughton Rules (Indian context, IPC Section 84):

"Nothing is an offence if done by a person who, at the time of doing it, is by reason of unsoundness of mind, incapable of knowing the nature of the act, or knowing that it is wrong."

- ASPD does NOT qualify for Section 84 defense: ASPD individuals know their acts are wrong; there is no "unsoundness of mind"
- Exception: Comorbid severe dissociation, psychosis, or intellectual disability may qualify
- Expert witness role: Distinguish psychopathy from psychosis clearly

Diminished Responsibility

- Indian Penal Code does not have formal diminished responsibility doctrine
- Courts consider ASPD in sentencing, not in guilt determination
- Mitigation: History of childhood abuse, cognitive limitations, treatable features

Risk Assessment

Risk assessment instruments used in forensic psychiatry:

INSTRUMENT	TYPE	DOMAIN	NOTES
PCL-R	Interview + file review	Psychopathy	Gold standard; trained raters
HCR-20 (v3)	Structured professional judgement	Violence risk	Historical, Clinical, Risk factors

INSTRUMENT	TYPE	DOMAIN	NOTES
VRAG	Actuarial	Violence risk	Static factors; less nuanced
LSI-R	Actuarial	General recidivism	Criminal justice focus
STATIC-99	Actuarial	Sexual recidivism	Sex offender risk

Dangerous and Severe Personality Disorder (DSPD)

- UK construct: PCL-R ≥ 30 + severe PD + significant violence risk
- Purpose: Indeterminate detention in hospital/prison for public protection
- Controversial: Due to preventive detention without crime committed
- Not applicable in Indian legal system but conceptually important

Management in Forensic Settings

- Therapeutic communities: Democratic (Grendon Prison, UK); structured
- Contingency management: Behavioral approaches; reinforcement of prosocial behavior
- Medication for impulsivity: Mood stabilizers (valproate) for aggressive behaviour
- Individual CBT: Cognitive distortions ("ownership of actions," victim empathy)
- **Key principle:** Treatment goals = risk reduction, not cure

Expert Witness Role

When asked to provide psychiatric evidence in ASPD cases:

1. Diagnose using ICD-11 or DSM-5; document evidence
2. Distinguish ASPD from psychopathy (PCL-R if trained)
3. Comment on risk but NOT predict individual future behaviour
4. Avoid conflating mental disorder with moral culpability

ANSWER 6: Differentiate BPD from Bipolar Disorder II. Discuss implications for management.

Introduction

BPD and Bipolar II Disorder (Bipolar II) are frequently misdiagnosed as each other, particularly in women presenting with affective instability, impulsivity, and suicidal behaviour. Accurate differential is critical because treatments differ substantially and misdiagnosis carries risks.

Clinical Comparison

FEATURE	BPD	BIPOLAR II
Core pathology	Emotion dysregulation; identity disturbance	Mood disorder: episodic hypomania + depression
Mood change trigger	Interpersonal (rejection, abandonment)	Often autonomous; sleep disruption, seasonal
Duration of mood change	Hours to 1–2 days	Days to weeks (hypomania ≥ 4 days; MDE ≥ 2 weeks)
Sleep in mood episodes	Variable; insomnia from anxiety	Reduced sleep need (Bipolar); feels rested on less
Identity	Unstable; chronic identity confusion	Stable between episodes
Interpersonal relationships	Unstable; splitting; intense	Relatively stable between episodes
Self-harm	Common; impulsive; NSSI	Rare (unless comorbid BPD)
Abandonment fears	Central	Not a feature
Splitting	Present	Absent
Mood baseline	Chronic dysphoria, emptiness	Euthymia between episodes
Grandiosity	Absent or transient	Present during hypomania/mania
Response to lithium	Limited	Good
Response to valproate	Some benefit (affective instability)	Good
Response to DBT	Good	Not indicated
Response to mood stabilisers	Limited (mood stabilisers for specific symptoms)	First-line

Key Distinguishing Points

- Duration:** BPD mood swings last hours to one day, often linked to interpersonal events. Bipolar II hypomania lasts ≥ 4 consecutive days.
- Sleep:** Reduced sleep need (not disturbed sleep) is a key hypomanic feature; BPD patients have anxiety-driven insomnia.
- Identity:** Stable ego-syntonic self-concept in Bipolar II between episodes; chronic identity confusion in BPD.
- Trigger:** Interpersonal triggers are hallmark of BPD; Bipolar II episodes can be autonomous.

5. **Comorbidity:** Both can genuinely co-occur; longitudinal observation often required.

Assessment Tools

- **MDQ** (Mood Disorder Questionnaire): Screens for bipolar spectrum; high sensitivity
- **BSL-23** (Borderline Symptom List): BPD severity
- **SCID-5** interview: Gold standard differential

Management Implications

If BPD:

- Psychotherapy is first-line: DBT, Schema Therapy, MBT, TFP
- Pharmacotherapy adjunctive only; no mood stabilisers as primary treatment
- Avoid long-term benzodiazepines

If Bipolar II:

- Mood stabiliser first-line: Lithium, valproate, lamotrigine (particularly for depression)
- Psychoeducation; structured sleep; interpersonal and social rhythm therapy (IPSRT)
- CBT adjunctive
- No indication for DBT unless comorbid BPD

If Both:

- Treat both; medication for Bipolar II + DBT / Schema Therapy for BPD
- Higher suicide risk when comorbid; close monitoring

ANSWER 7: Describe Narcissistic Personality Disorder: clinical features, subtypes, and management.

Introduction

Narcissistic Personality Disorder (NPD) is characterised by a pervasive pattern of grandiosity, need for admiration, and lack of empathy. It affects approximately 1–6% of the general population, is more common in men in clinical samples, and often presents for treatment not directly but through depression, relationship breakdown, or narcissistic injury.

DSM-5 Criteria (5 of 9 required)

1. Grandiose sense of self-importance
2. Preoccupied with fantasies of unlimited success, power, brilliance, beauty, or ideal love
3. Believes he/she is special; can only associate with equally special people
4. Requires excessive admiration
5. Sense of entitlement

6. Interpersonally exploitative
7. Lacks empathy
8. Often envious; believes others are envious
9. Arrogant, haughty behaviour

Grandiose vs. Vulnerable Subtypes

FEATURE	GRANDIOSE (OVERT)	VULNERABLE (COVERT)
Surface presentation	Bold, expansive, dominant	Shy, withdrawn, dysphoric
Self-esteem	Overtly inflated	Fragile; secretly grandiose
Entitlement	Explicit; demanding	Implicit; resentful
Empathy	Cold, dismissive	Distracted; self-preoccupied
Response to criticism	Narcissistic rage	Shame, collapse, depression
Seeking admiration	Active	Passive; expects recognition
Comorbidity	ASPD features	MDD, social anxiety
Clinical presentation	Interpersonal conflict, HR issues	Depression, anxiety, "emptiness"

EXAM PEARL

Vulnerable NPD is frequently misdiagnosed as depression or social anxiety. The grandiosity is concealed by surface self-deprecation. Key clues: intense resentment of others' success, covert entitlement, rage at perceived slights.

Theoretical Formulations

Kernberg (Object Relations):

- NPD = pathological grandiose self fused from real self + ideal self + ideal objects
- Defence: projection of aggression, devaluation, omnipotence
- Treatment: TFP, interpret grandiosity as defence against underlying rage and envy

Kohut (Self-Psychology):

- NPD = developmental arrest; grandiose self not properly mirrored during childhood
- Treatment: Empathic mirroring, transmuting internalisation
- More optimistic prognosis

Young (Schema Therapy):

- Core schemas: Entitlement/Grandiosity (overcompensation for underlying Defectiveness/Shame or Emotional Deprivation)

- Modes: Self-Aggrandiser, Lonely / Abandoned Child (hidden), Healthy Adult

Narcissistic Injury and Rage

- Narcissistic injury = threat to the grandiose self (criticism, failure, being ignored)
- Narcissistic rage = explosive anger or cold contempt; can be violent
- Aftermath: Humiliation → depression, shame, revenge fantasies

Management

Psychotherapy (primary)

- Schema Therapy: Mode work; access Lonely / Abandoned Child beneath Self-Aggrandiser; reparenting; challenge Entitlement schema
- TFP: Interpret transference activations; confront devaluation and omnipotence
- Kohutian Self-Psychology: Empathic attunement before confrontation; mirroring in therapeutic relationship
- CBT: Challenge entitlement cognitions; perspective-taking exercises; behavioural experiments
- Duration: Typically 3–5 years for substantial change
- Therapeutic challenge: Managing countertransference (therapist may feel devalued, bored, contempt)

Pharmacotherapy

- SSRIs for comorbid depression (narcissistic collapse → MDD)
- Mood stabilisers for rage episodes
- No approved medication for NPD itself

Prognosis

- Moderate; overt grandiose NPD more resistant; vulnerable NPD more amenable if shame tolerated
- Comorbid MDD increases treatment-seeking (and therefore access)
- Relationship functioning often improves more than internal grandiosity

ANSWER 8: Discuss Avoidant Personality Disorder: features, differential from Social Anxiety Disorder, and management.

Introduction

Avoidant Personality Disorder (AvPD) is characterised by pervasive social inhibition, feelings of inadequacy, and hypersensitivity to negative evaluation. It is among the most prevalent PDs (estimated 2–3% prevalence) and is commonly seen in psychiatric settings presenting as social anxiety, depression, or chronic loneliness.

DSM-5 Criteria (4 of 7 required)

1. Avoids occupational activities due to fear of criticism/rejection
2. Unwilling to get involved with others unless certain of being liked
3. Restrained in intimate relationships due to fear of shame/ridicule
4. Preoccupied with criticism/rejection in social situations
5. Inhibited in new situations due to feelings of inadequacy
6. Views self as socially inept, unappealing, or inferior
7. Reluctant to take personal risks or engage in new activities

AvPD vs Social Anxiety Disorder (Generalised Type)

FEATURE	AVPD	SOCIAL ANXIETY DISORDER (GENERALISED)
Onset	Childhood; ego-syntonic	Often adolescence; can be ego-dystonic
Pervasiveness	Identity-level; all domains	More circumscribed (though generalised SAD is broad)
Self-concept	Stable core of inferiority	Anxiety about specific situations
Desire for closeness	Yes, wants relationships but avoids	Yes, avoids specific situations
Treatment response	Slower; schema work often needed	Good response to CBT + SSRI
Diagnostic overlap	High, may be on same spectrum	High

EXAM PEARL

The debate: AvPD and generalised SAD may be on a continuum (dimensionally related) rather than categorically distinct. DSM-5 acknowledges this. In practice: treat the presentation, not just the label. Severe AvPD requires longer, deeper schema work.

Core Schemas (Young)

- **Defectiveness/Shame** (core): Belief of being fundamentally flawed, bad, inferior
- **Social Isolation**: Alienated from others; different, not belonging
- **Failure**: Inevitable failure in performance domains
- **Emotional Inhibition**: Suppression of emotional expression

Schema Modes in AvPD

- Vulnerable/Lonely Child: Core shame and isolation

- Detached Protector: Avoidance as coping; emotional numbing
- Punitive Parent: Harsh self-criticism after social exposure
- Healthy Adult: Needs building; limited by anxiety

Management

Psychotherapy

Schema Therapy:

- Limited reparenting for shame-based Vulnerable Child
- Imagery rescripting of humiliating childhood experiences
- Chair work: Healthy Adult challenges Punitive Parent, comforts Vulnerable Child
- Behavioural experiments: Graduated social exposure paired with schema dialogue
- Duration: 2–3 years for deep character change

CBT with Graduated Exposure:

- Fear hierarchy construction
- Cognitive restructuring: "I am fundamentally unlikeable" → evidence testing
- Behavioural experiments: Social approach experiments
- Role-play and rehearsal
- Social skills training

Group Therapy:

- Powerful modality: Provides real-time social exposure
- Interpersonal process groups expose maladaptive interpersonal patterns in vivo
- Often combined with individual work

Pharmacotherapy

- SSRIs (paroxetine, escitalopram): Reduce anxiety and improve social approach
- Beta-blockers (propranolol 10–20 mg): For performance-specific anxiety adjunctively
- No medication changes underlying personality structure; adjunctive only

Prognosis

- Moderate; AvPD is more treatment-responsive than Cluster B
 - CBT effects reasonably good on social anxiety measures
 - Schema Therapy produces deeper character-level change at longer follow-up
-

ANSWER 9: Compare OCPD and OCD. Discuss implications for management.

Introduction

Obsessive-Compulsive Personality Disorder (OCPD) and Obsessive-Compulsive Disorder (OCD) share superficially similar names and some behavioural features, but are distinct conditions with different phenomenology, theoretical origins, and treatment approaches.

Clinical Comparison

FEATURE	OCPD	OCD
Ego-syntonic	Yes, traits are valued and self-concordant	No, obsessions are intrusive, unwanted, alien
Core symptom	Perfectionism; rigidity; control	Obsessions + compulsions
Obsessions	Absent (concerns are ego-syntonic preoccupations, not true obsessions)	Present, intrusive, repetitive, anxiety-provoking thoughts
Compulsions	No rituals to neutralise anxiety	Yes, behavioural / mental acts to reduce anxiety
Anxiety	Anxiety from imperfection; low-level, chronic	Anxiety from obsessions; acute, intense
Perfectionism quality	Valued; ego-syntonic; driven by desire for control	Possible but secondary; driven by doubt
Flexibility	Rigid, stubborn	Can reason when not triggered
Hoarding	Miserly, cannot discard; no specific attachment rules	Hoarding subtype: specific, driven by contamination fears or attachment
DSM-5 classification	Cluster C PD (Section II)	Anxiety / OC spectrum disorder (Chapter 6)
Comorbidity	Can co-occur with OCD (10–35%)	Can co-occur with OCPD
Treatment	Long-term psychotherapy	CBT with ERP + SSRI (first-line)

Distinguishing Clinical Test

- Ask: "Would you WANT to stop the behaviour?"
- OCPD: "No, I like things being perfect"
- OCD: "Yes! The thoughts are horrible and I can't stop them"

Theoretical Background

OCPD:

- Young schemas: Unrelenting Standards, Punitiveness, Emotional Inhibition
- Schema mode: Demanding/Punitive Parent overactive
- Origin: Harsh, conditional-love parenting where worth = performance

OCD:

- Inflated responsibility model (Salkovskis): Overestimation of threat and responsibility
- Cognitive model: Misinterpretation of intrusive thoughts as meaningful/dangerous
- Serotonin circuit: Cortico-striato-thalamo-cortical loop (CSTC), compulsions reduce anxiety short-term but maintain disorder

Management

OCPD Management:

- Long-term psychotherapy: Schema Therapy (address Unrelenting Standards; reduce Punitive Parent mode; build Healthy Adult)
- CBT: Cognitive challenging of perfectionism; delay/reduce checking; "good enough" behavioural experiments
- Relaxation and flexibility training
- SSRIs: If comorbid OCD features or significant depression
- Pharmacotherapy otherwise limited

OCD Management:

- **CBT with ERP (Exposure and Response Prevention):** Gold standard; exposure to feared stimuli without performing compulsions
- **SSRIs (first-line):** Higher doses than depression (fluoxetine up to 80mg, fluvoxamine up to 300mg)
- **Clomipramine:** TCA; effective; limited by anticholinergic side effects; use when SSRI fails
- **Augmentation:** Low-dose antipsychotics (risperidone, haloperidol, aripiprazole) if partial SSRI response

When Both Co-Occur:

- Treat OCD first (ERP + SSRI): More acutely disabling
 - Address OCPD traits in longer-term therapy (schema work on Unrelenting Standards)
 - Risk: OCPD rigidity may impair ERP engagement, address motivation and flexibility first
-

ANSWER 10: Describe the pharmacological management of Personality Disorders. Discuss the evidence.

Introduction

Pharmacotherapy in personality disorders is adjunctive, symptom-targeted, and evidence-limited. No medication has regulatory approval for any specific personality disorder. The principle is: target domains of dysfunction (affective instability, impulsivity, cognitive-perceptual symptoms, anxiety) rather than the disorder itself. Psychotherapy remains first-line.

Rationale for Symptom-Targeted Approach

PDs involve multiple heterogeneous symptom domains. Each domain maps onto neurotransmitter or neurobiological systems:

- Affective instability: serotonin, HPA axis
- Impulsive aggression: serotonin hypofunction, adrenergic dysregulation
- Transient psychosis: dopaminergic sensitisation
- Anxiety: serotonin, noradrenaline, GABA

Evidence by Medication Class

1. Mood Stabilisers (Best Evidence for BPD)

Valproate/Sodium Valproate:

- RCTs (Hollander et al. 2001; Stein et al. 1995): Reduces impulsive aggression and affective instability in BPD
- Mechanism: GABA enhancement; glutamate modulation
- Target: Impulsivity, aggression, affective instability
- Caution: Teratogenicity (spina bifida risk); requires monitoring

Lamotrigine:

- RCT (Tritt et al. 2005): Reduces impulsivity and affective instability; better for women with BPD
- Mechanism: Glutamate inhibition
- Risk: Stevens-Johnson Syndrome (slow titration mandatory)

Lithium:

- Limited BPD-specific data; historical use for impulsive aggression
- Toxicity risk in impulsive overdose patients, HIGH RISK; use with caution
- Better evidence in ASPD aggression (Sheard et al. 1976, prisoners)

Carbamazepine:

- Limited BPD data; historical use
- Drug interactions; monitoring required

2. Antidepressants

SSRIs (fluoxetine, sertraline, escitalopram):

- Evidence: Coccaro & Kavoussi (1997), fluoxetine reduces impulsive aggression; Markovitz 1995
- Effect modest vs MDD; BPD depressive symptoms less responsive
- Useful for: Comorbid MDD, anxiety, impulsivity
- Relatively safe in overdose (versus TCAs)

Venlafaxine (SNRI):

- Limited BPD data; used for comorbid depression/anxiety

MAOIs:

- Historical interest (phenelzine); "atypical depression" features of BPD
- Dangerous in impulsive patients (dietary non-compliance → hypertensive crisis); not recommended

3. Antipsychotics (Atypical)

Olanzapine 2.5–5 mg:

- Best-evidenced antipsychotic for BPD
- Zanarini et al. (2004, 2006, 2007), multiple RCTs: Reduces cognitive-perceptual symptoms, affective instability, impulsivity
- Also: Bogenschutz & Nurnberg (2004)
- Limitation: Weight gain, metabolic syndrome with prolonged use
- Use: Short courses; transient psychosis; severe affective dysregulation

Quetiapine 25–300 mg:

- Commonly used; anxiolytic, sedating properties helpful
- Limited RCT evidence specifically for PD (mostly case series and open-label)
- Useful adjunctively for insomnia, anxiety, dissociation

Aripiprazole:

- Schizotypal PD: Some evidence
- BPD: Limited data

Haloperidol:

- Historical use; Soloff et al. comparisons with antidepressants
- Not recommended currently; EPS, tardive dyskinesia risk

4. Other Agents

Naltrexone 25–100 mg:

- Case series and open-label: Reduces dissociation and self-harm in BPD
- Mechanism: Opioid system involvement in BPD self-soothing and dissociation
- Evidence: Low quality; used pragmatically

Omega-3 Fatty Acids:

- Zanarini & Frankenburg (2003) RCT: Ethyl-EPA reduces aggression and depression in BPD women
- Mechanism: Serotonin membrane function
- Safe; adjunctive

Clonidine:

- Open-label data for dissociation in BPD; adrenergic mechanism
- Short-term use

Medications to AVOID

MEDICATION · REASON

Benzodiazepines Dependence risk; behavioural disinhibition; impulsivity worsens; overdose risk

TCAs Cardiotoxic in overdose; high lethality

MAOIs Dangerous in impulsive patients

Lithium (as first-line in BPD) Toxicity risk; overdose risk in impulsive patients

Clinical Practice Principles

1. Start one medication at a time; assess target symptom
 2. Set measurable treatment goals (e.g., self-harm frequency, emergency visits)
 3. Limit supply; safety planning around prescriptions
 4. Regular review: if no benefit in 8 weeks, discontinue
 5. Pharmacotherapy is NOT a substitute for psychotherapy
 6. NICE 2009: Do not use pharmacotherapy routinely for BPD; treat comorbidities
-

ANSWER 11: Discuss comorbidity in BPD: BPD with SUD and BPD with PTSD.

BPD + Substance Use Disorder (SUD)

Prevalence: 35–40% of BPD patients have comorbid SUD; conversely, 15–25% of SUD patients have BPD.

Relationship mechanisms:

1. **Self-medication hypothesis:** SUD as affect regulation, substances temporarily reduce emotional pain
2. **Shared vulnerability:** Impulsivity as shared neurobiological substrate; both involve reward dysregulation
3. **Behavioural disinhibition:** Comorbid SUD removes BPD coping inhibitions → more self-harm, violence
4. **Environmental:** Chaotic environments breed both; trauma a common antecedent

Clinical features of BPD+SUD:

- Higher suicide risk than either alone
- Greater treatment dropout
- More frequent hospitalisation
- More chaotic interpersonal lives
- More complex pharmacotherapy (drug interactions; overdose risk)

Assessment:

- Careful substance use history (AUDIT, DAST)
- Timeline: Which came first? (rarely cleanly separable)
- Functional analysis of substance use: When, how much, what function?

Treatment:

- **Integrated treatment superior to sequential**, address both simultaneously
- **DBT-S (DBT for Substance Abusers, Linehan et al.):**
- Dialectical Abstinence: Absolute commitment to abstinence + radical acceptance of lapses as inevitable
- Path to Clear Mind: Contingency management; "burning bridges" strategy
- Attachment to life, not substances
- RCT evidence for opioid use disorder + BPD (Linehan et al. 1999, 2002)
- **Medication for SUD:** Naltrexone for alcohol/opioid use; buprenorphine for opioid use disorder; methadone maintenance in opioid use disorder
- **Avoid:** Benzodiazepines for anxiety; sedating medications prescribed in large quantities

Prognosis: Significantly worse with comorbid SUD; SUD is strongest predictor of non-remission in BPD (McLean study)

BPD + PTSD

Prevalence: 25–56% of BPD patients meet PTSD criteria; 30–70% report childhood sexual abuse.

Conceptual relationship:

- Debate: Is BPD a form of complex PTSD, or comorbid?
- ICD-11 resolution: Complex PTSD ≠ BPD (see below)
- Clinical reality: Both can genuinely co-occur

Complex PTSD vs BPD (ICD-11 distinction):

FEATURE	COMPLEX PTSD	BPD
Core PTSD symptoms	Required	Not required
Identity disturbance	Negative self-concept	Identity diffusion, instability
Emotion dysregulation	Severe; linked to trauma	Chronic; linked to attachment
Interpersonal patterns	Difficulty in relationships	Splitting; idealise/devalue
Abandonment fears	Not core	Central feature
Splitting	Absent	Present
Onset	Follows trauma	Lifelong, early onset
Treatment primary focus	Trauma-focused	PD-focused

Treatment of BPD+PTSD:

1. **Phase 1, Safety and Stabilisation (BPD-first approach):**
2. DBT skills for affect regulation and crisis management
3. No trauma processing until self-harm under control
4. Build therapeutic alliance
5. **Phase 2, Trauma Processing:**
6. **DBT-PTSD (Harned & Linehan):** Prolonged Exposure adapted for BPD; introduced after Stage 1
7. **EMDR:** With modifications for BPD (emotion regulation focus; titrated)
8. **Imagery Rescripting:** Especially in Schema Therapy context
9. **Phase 3, Integration:**
10. Identity consolidation; meaning-making; post-traumatic growth

Prognosis: When both treated, outcomes better than untreated PTSD in BPD context; however, requires longer treatment and careful sequencing.

ANSWER 12: Describe assessment instruments for Personality Disorders.

Introduction

Accurate assessment of personality disorders requires validated, structured instruments. Assessment must be multi-method (clinical interview + self-report), multi-informant where possible, and longitudinal (state vs. trait distinction).

Principles of PD Assessment

- 1. PD = trait-level, not state, distinguish from acute Axis I presentation
- 2. Assess across multiple domains (affect, cognition, behaviour, interpersonal)
- 3. Collateral history valuable (patient may have limited insight)
- 4. Assess across multiple contexts, not just clinical setting
- 5. Consider cultural context (cultural norms affect what counts as deviant)

Assessment Instruments

Semi-Structured Diagnostic Interviews (Gold Standard)

INSTRUMENT	SYSTEM	LENGTH	NOTES
SCID-5-PD	DSM-5	60–90 min	Gold standard for DSM-5; clinician-administered
IPDE	ICD-10/DSM-IV	90–120 min	WHO instrument; cross-cultural validity
SIDP-IV	DSM-IV	60–90 min	Focuses on habitual patterns, not acute state
PDE (Loranger)	ICD-10/DSM-III-R	Long	Research; largely superseded

Self-Report Screeners

INSTRUMENT	SYSTEM	LENGTH	NOTES
PDQ-4+	DSM-IV	100 items	High sensitivity, low specificity; screening only

INSTRUMENT	SYSTEM	LENGTH	NOTES
MCMI-IV	Millon	195 items	Clinical populations; Millon's theoretical model
PAI	Multi-domain	344 items	BFI subscale for borderline
PID-5	DSM-5 AMPD	220 items	Trait dimensional; matches Section III

Disorder-Specific Instruments

INSTRUMENT	DISORDER	TYPE	NOTES
ZAN-BPD	BPD	Clinician-rated	Treatment monitoring; Zanarini
BSL-23	BPD	Self-report	Brief, validated
DIB-R	BPD	Interview	Diagnostic Interview for Borderlines
PCL-R	Psychopathy / ASPD	Interview + file review	Forensic gold standard
YSQ	Schema assessment	Self-report	232 items; 18 EMSs
SMI	Schema modes	Self-report	14 modes

Functional Assessment

INSTRUMENT	DOMAIN	NOTES
LPFS (Level of Personality Functioning Scale)	DSM-5 AMPD severity	Rates identity, self-direction, empathy, intimacy
GAF (Global Assessment of Functioning)	General functioning	Routine clinical; limited PD-specific sensitivity
SOFAS (Social and Occupational Functioning)	Social/occupational	Useful for tracking functional recovery

Practical Approach in Clinical Settings

1. Screen with **PDQ-4+** (fast, catches most cases)
2. Confirm with **SCID-5-PD** for specific diagnoses
3. Monitor severity with **ZAN-BPD** or **BSL-23** (BPD treatment monitoring)
4. Assess for psychopathy with **PCL-R** in forensic settings
5. Use **YSQ** + **SMI** when Schema Therapy is the planned modality

ANSWER 13: Describe the course and prognosis of Borderline Personality Disorder with reference to longitudinal studies.

Introduction

BPD was historically viewed as a severe, chronic, and essentially untreatable condition. Longitudinal research, particularly from the past three decades, has substantially revised this pessimistic view. BPD has a better prognosis than initially believed, though functional recovery lags behind symptomatic remission.

McLean Study of Adult Development (MSAD: Zanarini et al.)

Design: Prospective longitudinal study; 290 participants meeting DSM-III-R criteria for BPD; Mclean Hospital, USA; began 1992.

Key Findings:

- 35% achieved remission at 2 years
- 50% at 6 years
- 85–93% at 10 years (sustained remission = 2+ years without meeting criteria)
- 20-year follow-up: 99% had experienced remission
- **Recurrence rate:** Low once remitted (about 10% relapse)
- **Functional recovery:** Significantly lags behind symptomatic remission; only 50% had good functional outcomes despite 85% symptomatic remission at 10 years

Symptom trajectory:

- Acute symptoms remit faster: impulsivity, self-harm, paranoia, quasi-psychotic symptoms
- Chronic symptoms more persistent: chronic emptiness, feelings of abandonment, identity confusion
- "The tortuous course of BPD", acute peaks remit, chronic core persists longer

Predictors of remission:

- Absence of childhood sexual abuse
- Absence of parental brutality (physical abuse)
- Good neurological function
- No comorbid SUD
- Higher IQ
- Fewer BPD criteria initially

Predictors of non-remission:

- Childhood sexual abuse (strongest)
- Comorbid SUD

- Comorbid MDD
- Prolonged hospitalisation history

Collaborative Longitudinal Personality Disorders Study (CLPS)

Design: Prospective; followed BPD, ASPD, OCPD, AvPD, and MDD; Harvard-Brown site; 10 years.

Key Findings (BPD):

- High rates of remission over time; similar to MSAD
- OCPD more stable than BPD, functional but rigid
- Comorbid MDD associated with poorer outcomes across all PDs
- Psychosocial functioning: All PDs showed functional impairment even with symptom improvement

Implications for Clinical Practice

From prognosis data:

1. BPD is treatable; communicate hope without false assurance
2. Treatment focus should shift to functional recovery, not just symptom remission
3. Trauma history requires trauma-informed approach
4. SUD comorbidity must be treated concurrently
5. Brief hospitalisation only; long-term admission predicts worse outcome
6. Psychotherapy needs to be sustained over years

Mortality:

- Suicide: 8–10% lifetime completed suicide
- Premature mortality from accidents, medical complications
- Higher mortality from all-cause compared to general population

What predicts functional recovery (separate from symptom remission):

- Vocational stability
- Social support
- Absence of ongoing substance use
- Engagement in meaningful work/relationships

ANSWER 14: Discuss the management of Dependent Personality Disorder and OCPD.

Dependent Personality Disorder

Phenomenology:

- Core = excessive need to be cared for; submissiveness; fear of separation
- Schema roots: Dependence / Incompetence, Abandonment / Instability, Subjugation
- Patient presents as: "I can't manage without my partner / parent / doctor"

Assessment:

- Distinguish from BPD: DPD has stable dependency; no rage / splitting; no self-harm
- Distinguish from agoraphobia: No panic attacks; dependency is relational, not situational
- Comorbidity: High with anxiety disorders, MDD

Psychotherapy:

Schema Therapy:

- Limited reparenting: Address Dependence / Incompetence schema
- Chair work: Vulnerable Child (helpless) → Healthy Adult (capable)
- Gradual autonomy: Build schema of "I can manage"
- Homework: Incremental independence tasks (choose restaurant, manage own bill, say "no" to one request)

CBT:

- Identify and challenge core beliefs: "I am helpless without others," "I cannot survive alone"
- Graduated autonomy experiments
- Assertiveness training
- Problem-solving skills development

Group therapy:

- Addresses interpersonal aspects; reduces therapist dependence

Pharmacotherapy:

- SSRIs for comorbid anxiety / MDD
- No specific medication for DPD itself

Key therapeutic challenge: Managing the therapeutic relationship, patient will depend on therapist. Planned termination from the start; gradual reduction in contact; reinforce autonomy between sessions.

OCPD Management

Phenomenology:

- Perfectionism, rigidity, control, ego-syntonic

- Schema roots: Unrelenting Standards, Punitiveness, Emotional Inhibition
- Presents as: Relationship difficulties ("my way is the right way"); occupational rigidity; exhaustion from self-imposed standards

Psychotherapy:

Schema Therapy:

- Target Unrelenting Standards: "Good enough is enough"
- Target Punitive Parent mode: Reduce self-criticism; build self-compassion
- Limited reparenting: Validate effort without performance demands
- Behavioural experiments: Deliberately do something "imperfect" and observe outcome
- Imagery: Access the child who was never allowed to just be

CBT:

- Identify perfectionism cycle: High standard → fear of failure → anxiety → overwork → temporary relief → return to high standard
- Challenge: "What would happen if this wasn't perfect?"
- Behavioural: Time-limited tasks; "80% is good enough" experiments
- Relaxation training; delegation practice

Pharmacotherapy:

- SSRIs if comorbid OCD features present
- Low-dose antipsychotics not indicated for OCPD
- Anxiety comorbidity → SSRIs

Prognosis:

- Moderate; OCPD is one of the more stable PDs (CLPS data)
- Some traits may never fully remit but occupational and relationship functioning can improve substantially
- Individuals rarely seek treatment for OCPD itself; often enter treatment for MDD, relationship breakdown, or burnout

ANSWER 15: Describe Schizotypal Personality Disorder: features, differential, and management.

Introduction

Schizotypal Personality Disorder is a complex condition characterised by a pervasive pattern of cognitive and perceptual distortions, eccentric behaviour, and social anxiety. Its unique position in the schizophrenia spectrum makes it of theoretical and clinical significance beyond the typical PD framework.

DSM-5 Criteria (5 of 9 required)

1. Ideas of reference (not delusions)
2. Odd beliefs or magical thinking inconsistent with cultural norms (e.g., superstitiousness, clairvoyance)
3. Unusual perceptual experiences (body illusions, sensing presence of someone not there)
4. Odd thinking and speech (vague, circumstantial, metaphorical, overly elaborate or stereotyped)
5. Suspicious or paranoid ideation
6. Inappropriate or constricted affect
7. Odd, eccentric, or peculiar behaviour or appearance
8. Lacks close friends other than first-degree relatives
9. Excessive social anxiety that doesn't diminish with familiarity (paranoid in quality)

ICD-11 vs DSM-5 Placement

SYSTEM	CLASSIFICATION	RATIONALE
DSM-5	Cluster A Personality Disorder	Emphasises characterological nature
ICD-11	Schizophrenia Spectrum Disorder (Schizotypal Disorder)	Emphasises biological-genetic relationship to schizophrenia

Biological Relationship to Schizophrenia

- Familial aggregation: Overrepresented in families of individuals with schizophrenia
- Genetics: Shares some susceptibility loci
- Neuroimaging: Reduced grey matter (PFC, temporal lobe, thalamus); similar pattern to schizophrenia but less severe
- Neuropsychology: Cognitive deficits (working memory, attention) intermediate between controls and schizophrenia
- Smooth pursuit eye tracking: Reduced, as in schizophrenia
- Dopamine: D2 receptor sensitivity; explains cognitive-perceptual symptoms

Differential Diagnosis

CONDITION · KEY DISTINGUISHING FEATURE

Schizophrenia Frank psychosis (hallucinations, delusions, disorganisation); does not remit

Paranoid PD No magical thinking, odd speech, or unusual perceptions

Schizoid PD Social withdrawal without cognitive-perceptual symptoms

Avoidant PD Fears rejection; no magical thinking or eccentric features

BPD Transient paranoia stress-related; identity instability; splitting

ASD Early onset; qualitatively different social impairment; sensory features

Management

Pharmacotherapy:

- Low-dose atypical antipsychotics: Risperidone 0.5–2 mg, olanzapine 2.5–5 mg
- Target: Positive-like symptoms (ideas of reference, paranoia, unusual perceptions)
- Evidence: Modest; risperidone RCT (Koenigsberg et al. 2003); olanzapine data
- SSRIs: Social anxiety and dysphoria

Psychotherapy:

- **CBT:** Reality-testing unusual beliefs; distinguishing ideas of reference from reality
- **Social skills training:** Structured; address eccentric social presentation
- **Supportive therapy:** Building trust; low demand initially
- **Schema Therapy:** Limited evidence but applicable; address Social Isolation, Mistrust/ Abuse schemas

General:

- Social support and structure: Routine reduces stress; less cognitive-perceptual activation
- Vocational rehabilitation: Often needed
- Family psychoeducation: Reduce expressed emotion; manage family distress

Prognosis:

- Stable but typically non-deteriorating (unlike schizophrenia)
- Small minority (10–15%) convert to psychotic disorder
- Chronic mild to moderate impairment usual
- With treatment: Reduction in positive-like symptoms; social functioning moderate

Mnemonics & Memory Tricks

Sources: DSM-5-TR, Young et al. Schema Therapy, Linehan DBT Manual, Kaplan & Sadock

MNEMONIC 1: BPD Criteria: PRAISE

EXAM PEARL

DSM-5 requires **5 of 9** criteria for BPD. PRAISE covers 6; add the remaining 3 from memory.

P, Parting fears (frantic efforts to avoid abandonment, real or imagined)

R, Rage (inappropriate, intense anger; difficulty controlling anger)

A, Affective instability (mood reactivity; dysphoria, irritability, anxiety lasting hours)

I, Impulsivity (in ≥ 2 areas: spending, sex, substance use, reckless driving, binge eating)

S, Self-harm / Suicidal behaviour (recurrent suicidal behaviour, gestures, threats, or self-mutilation)

E, Emptiness (chronic feelings of emptiness)

Remaining 3 to add from memory:

- Unstable intense relationships (idealisation ↔ devaluation)
- Identity disturbance (markedly unstable self-image or sense of self)
- Transient paranoid ideation or severe dissociative symptoms (stress-related)

EXAM PEARL

PRAISE gives you 6/9. The 3 remaining = **relationships, identity, dissociation/paranoia**. Together that's all 9.

MNEMONIC 2: DBT Four Modules: DIMS

D, Distress Tolerance (crisis survival; TIPP, ACCEPTS, Radical Acceptance)

I, Interpersonal Effectiveness (DEAR MAN, GIVE, FAST)

M, Mindfulness (Wise Mind; What and How skills, foundation of all modules)

E, Emotion Regulation (ABC PLEASE, Opposite Action, Check the Facts)

EXAM PEARL

Mindfulness is the CORE module, it underpins the other three. In exam answers, state this explicitly.

MNEMONIC 3: Cluster A Features: The Three S's of Oddness

CLUSTER A PD	CORE WORD	ONE-LINE REMINDER
Paranoid	Suspicious	Distrusts everyone; sees threat everywhere; holds grudges
Schizoid	Solitary	Prefers alone; emotionally cold; indifferent; content without others
Schizotypal	Strange	Odd beliefs, magical thinking, eccentric; ideas of reference

Sub-mnemonic for Schizotypal, MAGICAL PEAS:

M, Magical thinking / odd beliefs

A, Affect inappropriate or constricted

G, Gregariousness absent (no close friends)

I, Ideas of reference

C, Cognitive/perceptual distortions (unusual perceptions)

A, Anxiety (social; doesn't decrease with familiarity; paranoid quality)

L, Language odd (vague, metaphorical, circumstantial)

P, Paranoid ideation

E, Eccentric behaviour or appearance

A, Absent close friends (first-degree relatives only)

S, Suspiciousness

(9 of these = schizotypal DSM-5 criteria; need 5)

MNEMONIC 4: Cluster B Features: The Four Dramas

CLUSTER B PD	CORE WORD	ONE-LINE REMINDER
Antisocial	Aggressive	Violates others' rights; no remorse; Conduct Disorder before 15

CLUSTER B PD	CORE WORD	ONE-LINE REMINDER
Borderline	Unstable	Everything unstable: mood, identity, relationships; self-harm
Histrionic	Theatrical	Centre-of-attention seeker; dramatic; seductive; shallow
Narcissistic	Grandiose	Self-important; entitled; lacks empathy; envious

MNEMONIC 5: Cluster C Features: The Three Worriers

CLUSTER C PD	CORE WORD	ONE-LINE REMINDER
Avoidant	Ashamed	Wants connection; avoids due to fear of rejection/humiliation
Dependent	Attached	Needs to be cared for; can't decide alone; clings; fears separation
OCPD	Anal	Perfectionism; rigidity; control; rules; hoarding (ego-syntonic)

MNEMONIC 6: OCPD Criteria: LAWFUL ORDER

L, Lists, rules, details (preoccupied; loses the point)

A, Adherence to morality (overconscientious, scrupulous, inflexible)

W, Workaholic (excessively devoted; excluding economic necessity)

F, Fringe hoarding (can't discard worthless objects without sentimental value)

U, Unwilling to delegate (unless others submit exactly to their standards)

L, Limited spending (miserly toward self and others; money hoarded for catastrophe)

O, Order and organisation (perfectionistic; interferes with task completion)

R, Rigidity and stubbornness

D, Doesn't finish tasks (perfectionism interferes with completion)

E, Ego-syntonic (traits feel like virtues, not problems)

R, Resistance to change

EXAM PEARL

DSM-5 requires **4 of 8** criteria. **LAWFUL ORDER** gives you the 8 to choose from. Ego-syntonic is a characteristic but not a criterion, include it in narrative answers.

MNEMONIC 7: Narcissistic PD Criteria: GRANDIOSE

G, Grandiose sense of self-importance

R, Requires excessive admiration

A, Arrogant, haughty behaviour

N, Not empathic (lacks empathy; unwilling to recognise feelings of others)

D, Dreams of unlimited success, power, brilliance, beauty, ideal love

I, Interpersonally exploitative

O, Only associates with special/high-status people

S, Sense of entitlement

E, Envious (or believes others are envious of them)

EXAM PEARL

DSM-5 requires **5 of 9**. **GRANDIOSE** = all 9 criteria. Master this.

MNEMONIC 8: PCL-R Domains: GALS PRICE (Factor 1, Interpersonal/Affective)

G, Glibness / superficial charm

A, Arrogance / grandiose sense of self-worth

L, Lying pathologically

S, Shallow affect

P, Parasitic lifestyle

R, Remorse absent (lacks)

I, Irresponsibility (failure to accept responsibility)

C, Callousness / lack of empathy

E, Exploitative interpersonal behaviour

Factor 2 (Social Deviance/Lifestyle), CRIME RAN:

C, Criminal versatility

R, Revocation of conditional release
I, Impulsivity
M, Many short-term marital/intimate relationships
E, Early behavioural problems
R, Risk taking / stimulation-seeking
A, Adult antisocial behaviour (juvenile + adult)
N, No long-term goals (lack of realistic goals)

EXAM PEARL

PCL-R has 20 items (10 Factor 1 + 10 Factor 2). Cut-off: 30/40 (North America), 25/40 (UK).
Used in forensic settings only.

MNEMONIC 9: Young's 18 Early Maladaptive Schemas: DR DIVA FANS USE PADS

Domain 1, Disconnection & Rejection (5 schemas):

D, Defectiveness / Shame
R, Rejection / Abandonment-Instability
D, Deprivation (Emotional)
I, Isolation (Social)
V, Violation (Mistrust / Abuse)
A, (remember: 5 schemas in this domain)

Domain 2, Impaired Autonomy & Performance (4 schemas):

F, Failure
A, Anxious (Vulnerability to Harm/Illness)
N, Nested (Enmeshment/Undeveloped Self)
S, Stupid (Dependence / Incompetence)

Domain 3, Impaired Limits (2 schemas):

U, Uncontrolled (Insufficient Self-Control)
S, Special (Entitlement / Grandiosity)

Domain 4, Other-Directedness (3 schemas):

E, Externally validated (Approval-Seeking)
P, Peacekeeping (Subjugation)

A, Altruistic (Self-Sacrifice)

D, (3rd schema = Approval-Seeking; covered by E)

S, (Domain 4: 3 schemas total)

Domain 5, Overvigilance & Inhibition (4 schemas):

P, Perfectionism (Unrelenting Standards)

A, Ascetic (Emotional Inhibition)

D, Doom (Negativity / Pessimism)

S, Shame-punishing (Punitiveness)

CLINICAL ANCHOR

For exam, know all 18 names, their domain, and at least 2 PDs linked to each major schema. The most exam-relevant schemas: Abandonment/Instability (BPD), Defectiveness/Shame (AvPD, NPD-hidden), Entitlement/Grandiosity (NPD-overt), Unrelenting Standards (OCPD), Mistrust/Abuse (Paranoid PD, BPD with trauma).

MNEMONIC 10: Schema Modes (BPD core modes): VAPPH

V, Vulnerable Child (core wound; abandonment terror; shame)

A, Angry/Impulsive Child (rage; acting out; boundary violations)

P, Punitive Parent (harsh self-criticism; self-punishment → self-harm)

P, Protector, Detached (numbing; dissociation; emptiness as defence)

H, Healthy Adult (the goal; reality-testing; self-compassion; integration)

EXAM PEARL

In BPD mode work, the visible cycle is often: Vulnerable Child (abandoned terror) → flip to Angry Child (rage) → Detached Protector (numbing) → Punitive Parent (self-harm). The therapy task is building the Healthy Adult to intervene at each transition.

MNEMONIC 11: DBT TIPP Skills (Distress Tolerance, acute crisis)

T, Temperature (cold water on face; activates dive reflex; rapid parasympathetic activation)

I, Intense exercise (burns adrenaline; reduces acute emotional arousal)

P, Paced breathing (slow the exhale; activates vagal tone)

P, Progressive muscle relaxation (systematic tension-release)

CLINICAL ANCHOR

TIPP targets the physiological component of emotional crisis directly. It works fastest of all DBT skills. Cold water = mammalian dive reflex → immediate heart rate reduction.

MNEMONIC 12: DBT DEAR MAN (Interpersonal Effectiveness: getting what you want)

D, Describe the situation (objectively, without judgment)

E, Express your feelings (use "I feel..." statements)

A, Assert what you want or need

R, Reinforce, tell them what's in it for them

M, Mindful, keep focus on goal; don't get sidetracked

A, Appear confident (even if you don't feel it)

N, Negotiate, be willing to give to get

EXAM STRATEGY

DEAR MAN = objective effectiveness (getting what you need). GIVE = relationship effectiveness. FAST = self-respect effectiveness. Always state which goal each skill targets.

MNEMONIC 13: ICD-11 Trait Qualifiers: DANDA B

D, Dissociality (disregard for others' rights; ≈ ASPD/NPD)

A, Anankastia (perfectionism; orderliness; ≈ OCPD)

N, Negative Affectivity (intense unstable emotions; ≈ BPD-affective, HPD)

D, Detachment (social withdrawal; emotional restriction; ≈ Schizoid, AvPD)

A, Anankastia ← already listed above

(Remove duplicate, use:)

D, Dissociality

A, Anankastia

N, Negative Affectivity

D, Disinhibition (impulsivity; irresponsibility; ≈ ASPD-behavioural, BPD-impulsive)

A, Anankastia

B, Borderline Pattern (specifier, emotional instability + identity disturbance + self-harm)

Cleaner version, DINAN + B:

D, Detachment

I, (D)Isinhibition

N, Negative Affectivity

A, Anankastia

N, (Dissociality), (*note: 5 domains + 1 specifier*)

B, Borderline Pattern (specifier)

EXAM PEARL

ICD-11 has **5 trait qualifiers + 1 specifier (Borderline Pattern)**. Severity is assessed FIRST (mild / moderate / severe), THEN qualifiers added. Multiple qualifiers can be assigned simultaneously.

MNEMONIC 14: ASPD Criteria: CRUEL DRIFTER

C, Conduct Disorder before age 15 (mandatory)

R, Reckless disregard for safety of self or others

U, Unreliable / Irresponsible (work, financial obligations)

E, Exploitative / Deceitful (lying, aliases, conning for personal gain)

L, Lack of remorse

D, Disregard for rights of others (core feature)

R, Repeat rule violations / lawlessness (unlawful behaviour)

I, Impulsivity (failure to plan ahead)

F, Fights (irritability and aggressiveness)

T, (*must be ≥18 years old to diagnose*)

E, (*Evidence of CD before 15*)

R, (*3 of the 7 behaviour criteria required*)

EXAM PEARL

ASPD diagnosis requires: (1) Age ≥18, (2) Evidence of Conduct Disorder before 15, (3) ≥3 of 7 behavioural criteria. CD before 15 is mandatory, without it, you cannot diagnose ASPD.

MNEMONIC 15: Avoidant PD Criteria: SCARED

S, Social inhibition (inhibited in new interpersonal situations)

C, Criticism preoccupied (preoccupied with being criticised or rejected)

A, Avoiding occupational activity (involving significant interpersonal contact)
R, Reticent in intimate relationships (fears shame / ridicule)
E, Excessive personal risk avoidance (reluctant to take risks, try new activities)
D, Doubts own appeal (views self as inept, unappealing, inferior)
+ Unwilling to get involved unless certain of being liked

EXAM PEARL

DSM-5 requires **4 of 7** criteria. SCARED = 6 criteria. Add "unwilling unless certain of being liked" for the 7th.

MNEMONIC 16: Histrionic PD Criteria: PRAISE THEM (no overlap with BPD PRAISE)

P, Provocative / seductive behaviour (sexually or physically inappropriate)
R, Rapidly shifting shallow emotions
A, Attention-seeking (uncomfortable not being the centre of attention)
I, Impressionism in speech (vague, lacking in detail)
S, Suggestible (easily influenced by others)
E, Exaggerated emotional expression (theatrical, dramatic)
T, Too intimate (considers relationships more intimate than they are)
H, Hair / appearance used to attract attention (uses physical appearance to draw attention)
E, (*above = 8 criteria total; DSM-5 requires 5 of 8*)
M, (*Match to DSM-5, start writing when 5 covered*)

MNEMONIC 17: McLean Study Key Numbers: 2-6-10-85

- **2 years:** ~35–50% achieved remission
- **6 years:** ~68–74% achieved remission
- **10 years:** 85–93% achieved sustained remission
- **85%** = the headline number for PG exams-style questions
- **Functional recovery:** Lags behind symptomatic remission; ~50% good functional outcomes at 10 years despite 85% symptom remission

EXAM STRATEGY

Quote the McLean Study by name. State: "Zanarini et al., McLean Study of Adult Development." This signals familiarity with longitudinal literature.

MNEMONIC 18: DBT Target Hierarchy: LTQ-S

L, Life-threatening behaviours (suicide, self-harm, violence), ALWAYS FIRST

T, Therapy-interfering behaviours (non-attendance, non-compliance, late)

Q, Quality-of-life-interfering behaviours (SUD, abusive relationships, unemployment)

S, Skills acquisition and generalisation

CLINICAL ANCHOR

This hierarchy is non-negotiable in DBT. Even if the patient wants to talk about their job, if there was a self-harm episode this week, the session starts there. No exceptions.

QUICK REFERENCE: Cluster Comparisons at a Glance

FEATURE	CLUSTER A	CLUSTER B	CLUSTER C
Theme	Odd / Eccentric	Dramatic / Emotional	Anxious / Fearful
Core affect	Detachment / Suspicion	Emotional instability / entitlement	Fear / anxiety
Insight	Low (ego-syntonic)	Low-moderate	Moderate-high
Treatment seeking	Rarely	Often in crisis	Frequently
Prognosis	Stable, poor change	Variable	Better with treatment
Genetic link	Schizophrenia spectrum (esp. Schizotypal)	Some ASPD-SUD overlap	Anxiety disorders

QUICK REFERENCE: Treatment Mnemonics Summary

MNEMONIC · WHAT IT COVERS

PRAISE BPD 9 criteria (6 main)

DIMS DBT 4 modules

GRANDIOSE NPD 9 criteria

LAWFUL ORDER OCPD 8 criteria

SCARED AvPD 7 criteria

CRUEL DRIFTER ASPD criteria

MAGICAL PEAS Schizotypal 9 criteria

GALS PRICE + CRIME RAN PCL-R Factor 1 + Factor 2

DR DIVA FANS USE PADS 18 Early Maladaptive Schemas

VAPPH BPD core schema modes

TIPP DBT distress tolerance acute

DEAR MAN DBT interpersonal effectiveness

DANDA B / DINAN+B ICD-11 trait qualifiers

2-6-10-85 McLean Study numbers

LTQ-S DBT target hierarchy

High-Yield Comparisons

Sources: DSM-5-TR, ICD-11, Kaplan & Sadock, Young et al., Linehan DBT Manual, Bateman & Fonagy MBT, Clarkin et al. TFP

EXAM STRATEGY

Comparison tables are direct answers to "differentiate X from Y" questions. Learn these table structures, reproduce them in the exam with brief narrative framing sentences.

TABLE 1: BPD vs Bipolar II Disorder

FEATURE	BPD	BIPOLAR II DISORDER
Core pathology	Pervasive emotion dysregulation; identity disturbance	Episodic mood disorder: recurrent depression + hypomania
Mood change trigger	Interpersonal events (rejection, abandonment, conflict)	Often autonomous; can be triggered by sleep disruption, stress, seasonal
Duration of mood change	Hours to 1–2 days	Hypomania ≥4 consecutive days; MDE ≥2 weeks
Sleep in mood episodes	Insomnia from anxiety; variable	Reduced sleep need (feels rested on less), cardinal hypomanic feature
Identity	Markedly unstable; chronic identity confusion	Stable between episodes
Interpersonal relationships	Unstable; splitting (idealise ↔ devalue); intense	Relatively stable between episodes; may be strained during episodes
Self-harm / NSSI	Common; impulsive; deliberate self-injury	Rare (unless comorbid BPD)
Grandiosity	Absent or transient (reactive self-inflation)	Present during hypomania, sustained, elevated
Abandonment fears	Central, frantic efforts to avoid	Not a feature
Mood baseline between episodes	Chronic dysphoria, emptiness, affective lability	Euthymia between episodes
Splitting	Present (all-or-nothing view of self and others)	Absent

FEATURE	BPD	BIPOLAR II DISORDER
Impulsivity	Chronic, trait-level	Episodic, primarily during hypomanic/manic phase
Response to lithium	Limited evidence; some effect on impulsivity	First-line; excellent response
Response to valproate	Moderate (affective instability, impulsivity)	First-line
Response to lamotrigine	Some evidence (affective instability)	First-line for bipolar depression
Response to DBT	First-line psychological treatment	Not indicated for bipolar alone
Response to mood stabilisers	Adjunctive for specific symptoms only	First-line treatment
Psychotherapy	DBT, Schema Therapy, MBT, TFP	Psychoeducation, IPSRT, CBT adjunctive
Comorbidity	True co-occurrence in 10–20%	True co-occurrence in 10–20%
Assessment tools	BSL-23, ZAN-BPD, SCID-5-PD	MDQ, BISS, YMRS, MADRS
Longitudinal course	Improves with age; 85% remission at 10y (McLean)	Episodic; chronic without treatment; some kindling effect

EXAM PEARL

The single most useful distinguishing question: **"How long does the mood change last, and is it triggered by a specific interpersonal event?"** BPD = hours, interpersonal trigger. Bipolar II = days to weeks, often more autonomous.

CLINICAL ANCHOR

True comorbidity is common (10–20%). Both can co-occur. When they do: treat Bipolar II pharmacologically first, then address BPD with psychotherapy. Do not treat BPD-style affective instability with mood stabilisers as a first response, always take a careful longitudinal history.

TABLE 2: OCPD vs OCD

FEATURE	OCPD	OCD
DSM-5 classification	Cluster C Personality Disorder	Obsessive-Compulsive and Related Disorders
Ego-syntonicity	Ego-syntonic , traits valued; "I like things done properly"	Ego-dystonic , obsessions intrusive and unwanted

FEATURE	OCPD	OCD
Obsessions	Absent, concerns are preoccupations, not true obsessions	Present , intrusive, repetitive, anxiety-provoking thoughts
Compulsions	None , no rituals to neutralise anxiety	Present , behaviours/mental acts to reduce obsessive anxiety
Core anxiety	About imperfection, loss of control; low-level, chronic	Acute, intense; triggered by obsessions
Perfectionism	Core to identity; ego-syntonic	May be present but secondary to doubt/uncertainty
Rigidity	Stubborn, inflexible; "my way is the right way"	Can be more flexible when not triggered
Hoarding	Difficulty discarding; no specific attachment rules; miserliness	Hoarding OCD subtype: specific fears (contamination, loss)
Insight	Low, traits feel like virtues	Usually present, patient knows obsessions are irrational
Distress	Distress is interpersonal (others frustrated with them)	Significant personal distress from obsessions
Onset	Adolescence/early adulthood; lifelong	Often adolescence; can be acute onset
Neurobiological basis	Unrelenting Standards schema; harsh parenting	CSTC circuit dysfunction; serotonin dysregulation
Comorbidity	Can co-occur with OCD (10–35%)	Can co-occur with OCPD
Treatment: Psychotherapy	Schema Therapy, CBT (flexibility training)	CBT with ERP (Exposure and Response Prevention) , gold standard
Treatment: Medication	SSRIs only if comorbid OCD/MDD	SSRIs (higher doses) + clomipramine; augmentation with atypical antipsychotics
Prognosis	Moderate; stable traits; slow change	Good with ERP + medication; chronic without treatment

EXAM PEARL

The key differentiating question: **"Would you WANT to stop?"** OCPD patient: "No, this is just how things should be." OCD patient: "God yes, the thoughts are horrible and I hate them."

EXAM STRATEGY

A common exam trick is to present a vignette with both features. Flag both, then apply this ego-syntonic/ego-dystonic distinction to separate them.

TABLE 3: Schizoid vs Schizotypal vs Avoidant PD

FEATURE	SCHIZOID PD	SCHIZOTYPAL PD	AVOIDANT PD
Core feature	Pervasive detachment from social relationships; restricted affect	Cognitive/perceptual distortions + social anxiety + eccentric behaviour	Social inhibition; feelings of inadequacy; fear of rejection
Desire for social connection	Absent , genuinely content without relationships	Absent or minimal	Present , desperately wants connection but fears it
Social anxiety	Absent (indifferent, not anxious)	Present, paranoid in quality ; doesn't decrease with familiarity	Present, fear of rejection/humiliation ; may decrease with trust
Magical thinking / odd beliefs	Absent	Present	Absent
Perceptual distortions	Absent	Present (body illusions, sensing presence)	Absent
Eccentric behaviour/appearance	Absent	Present	Absent
Ideas of reference	Absent	Present	Absent
Affect	Cold, flat, restricted	Inappropriate or constricted	Inhibited (holds back to avoid shame)
Self-concept	Indifferent to evaluation	May feel different, odd, alien	Inferior, unappealing, inept
Relationship with family	Indifferent	Paranoid or mildly attached	May be close (if safe)
Treatment seeking	Rarely	Occasionally for comorbid symptoms	Frequently (depression, loneliness)
Biological link	Modest schizophrenia spectrum link	Strong schizophrenia spectrum link (genetic, neurobiological)	Anxiety disorders spectrum
ICD-11 qualifier	Detachment	Psychoticism + Detachment	Detachment + Negative Affectivity
Primary treatment	Supportive therapy; rarely sought	Low-dose antipsychotics + CBT	Schema Therapy; CBT + exposure; SSRIs

EXAM PEARL

The critical triad distinction: - **Schizoid** = doesn't want people (contentedly alone) - **Schizotypal** = doesn't relate well to people (odd, paranoid, cognitively distorted) - **Avoidant** = desperately wants people but is too afraid (painfully alone)

TABLE 4: DSM-5 vs ICD-11 Personality Disorder Models

FEATURE	DSM-5 (SECTION II, MAIN)	DSM-5 AMPD (SECTION III, ALTERNATIVE)	ICD-11
Model type	Categorical	Hybrid dimensional	Fully dimensional
Number of diagnoses	10 specific PDs	6 operationalised PDs	No categorical diagnoses
Cluster system	Yes (A/B/C)	Retained as informational	Abolished
Severity assessment	None	LPFS (0–4; identity, self-direction, empathy, intimacy)	Mild / Moderate / Severe
Trait domains	None in Section II	5 domains, 25 facets	5 domains (+ 1 specifier)
Trait domains listed	N/A	Negative Affectivity, Detachment, Antagonism, Disinhibition, Psychoticism	Negative Affectivity, Detachment, Dissociality, Disinhibition, Anankastia
BPD status	Full categorical diagnosis	Operationalised with specific traits	Borderline Pattern = specifier only (not a disorder)
Comorbidity problem	High (most patients 2+ PDs)	Substantially reduced	Eliminated (multiple qualifiers instead)
PD-NOS / "Other"	Other Specified PD	Addressed by general criteria	Addressed by severity + qualifiers
Clinical utility	High (familiar, fast)	Moderate (learning curve)	High (severity drives clinical decisions)
Research use	Standard (decades of data)	Growing (PID-5 research)	Recommended for ICD-using countries
Implementation	Routine clinical worldwide	Research / specialist settings	Mandatory from Jan 2022 (ICD-11 rollout)
India applicability	Common in teaching / research	Research settings	Clinically applicable from ICD-11 adoption

FEATURE	DSM-5 (SECTION II, MAIN)	DSM-5 AMPD (SECTION III, ALTERNATIVE)	ICD-11
Self-report tool	PDQ-4+, MCMI-IV	PID-5	No specific ICD-11 self-report yet (PID-5 proximal)
Interview tool	SCID-5-PD	SCID-5-AMPD	IPDE (updated for ICD-11 pending)

EXAM STRATEGY

When asked to compare, structure your answer as: (1) Model type, (2) Number/structure of diagnoses, (3) Severity assessment, (4) Trait domains, (5) BPD status, (6) Clinical implications. That's a complete 10-mark answer frame.

TABLE 5: DBT vs Schema Therapy vs MBT

FEATURE	DBT (LINEHAN)	SCHEMA THERAPY (YOUNG)	MBT (BATEMAN & FONAGY)
Theoretical base	Biosocial model; behaviourism + mindfulness + dialectics	Cognitive, experiential, attachment, object relations integration	Attachment theory; developmental neuroscience; mentalization
Core deficit targeted	Emotional dysregulation	Early maladaptive schemas; schema modes	Impaired mentalization under attachment stress
Format	Individual + group (skills) + phone coaching + consultation team	Individual (primary); group variant exists	Individual + group (both required in standard MBT)
Duration	6–12 months standard; 12–24 months for full effect	2–4 years for severe BPD; 1–2 years for Cluster C	18 months (standard protocol)
Primary techniques	Chain analysis; diary card; skill training; phone coaching	Limited reparenting; chair work; imagery rescripting; flashcards	Stop-and-rewind; affect-focused mentalization; therapist transparency
Stance	Validation + change dialectic; directive	Empathic confrontation; corrective emotional experience	Exploratory; curious; genuine not-knowing
Therapist role	Skills coach + individual therapist	Limited reparenting caregiver + expert	Curious, genuine inquirer into mental states

FEATURE	DBT (LINEHAN)	SCHEMA THERAPY (YOUNG)	MBT (BATEMAN & FONAGY)
Evidence (BPD)	Multiple RCTs; reduces suicide, self-harm, hospitalisations	RCT evidence; superior to TFP at 3y; superior to TAU	RCTs superior to TAU and structured clinical management; 5y follow-up
NICE recommendation	Yes, BPD	Implicitly endorsed; less prominent in UK guidelines	Yes, BPD
Adaptations	DBT-A, DBT-S, DBT-PTSD, DBT-ED	Group ST; ST for forensic, NPD, cluster C	MBT-A (adolescents); MBT-C (children); MBT-SUD
Best for	Acute safety (self-harm, suicidality); building skills	Deeper schema/identity change; complex PDs; NPD, cluster C	Attachment-based impairment; complex trauma; NHS/public system
Neuroscience target	Amygdala → PFC regulation; emotional learning	Schema network consolidation; hippocampal emotional memory	Mentalization network (mPFC, TPJ, STS, insula)
Key text	Linehan (1993) DBT Manual	Young, Klosko & Weishaar (2003)	Bateman & Fonagy (2004, 2016)

EXAM PEARL

All three are evidence-based for BPD. The exam will often ask you to "compare" two or all three. Structure: theoretical base → target → format → techniques → evidence. Don't omit the evidence base, it is always expected in a 10-mark answer.

TABLE 6: Narcissistic PD: Grandiose (Overt) vs Vulnerable (Covert)

FEATURE	GRANDIOSE / OVERT NPD	VULNERABLE / COVERT NPD
Surface presentation	Bold, expansive, dominant, controlling	Shy, withdrawn, dysphoric, inhibited
Self-esteem	Overtly inflated; stable grandiosity	Fragile; secretly grandiose; surface humility
Entitlement	Explicit; openly demanding	Implicit; resentful when unrecognised
Empathy	Cold, dismissive; others are props	Distracted by own internal world; self-absorbed
Response to criticism	Narcissistic rage, explosive anger or cold contempt	Shame, collapse, depression; internal withdrawal

FEATURE	GRANDIOSE / OVERT NPD	VULNERABLE / COVERT NPD
Seeking admiration	Active, commands attention	Passive, expects recognition; sulks when absent
Social behaviour	Dominant, attention-seeking, interrupts	Socially inhibited; appears modest; secretly superior
Affect	Euphoric when admired; cold otherwise	Predominantly dysphoric; shame-prone
Comorbidity	ASPD features common; substance misuse	MDD, social anxiety disorder, dysthymia
Clinical presentation	Interpersonal conflict; fired from job; divorce	Presenting as depression, anxiety, "emptiness"
Shame	Denied; projected onto others	Central; barely contained
Envy	Overt, voices envy explicitly	Covert, hidden resentment of others' success
Kohut's framework	Exhibitionistic self needing mirroring	Mirror-hungry type seeking validation subtly
Schema mode	Self-Aggrandiser dominant	Self-Aggrandiser + Lonely / Abandoned Child fluctuating
Schema (Young)	Entitlement / Grandiosity as overcompensation	Defectiveness / Shame masked by Entitlement
Therapeutic challenge	Devaluation of therapist; entitlement in therapy	Shame prevents engagement; fragile alliance
Treatment approach	TFP (transference interpretation); limit-setting; Schema Therapy	Schema Therapy (reparenting for Lonely Child); Kohutian mirroring

CLINICAL ANCHOR

Vulnerable NPD is the most frequently missed presentation. Clues in history: intense resentment at being passed over for recognition, covert superiority ("I could do their job better"), shame-rage cycles after perceived slights, and a brittle relationship pattern that looks like BPD but without the abandonment terror.

TABLE 7: ASPD vs Psychopathy

FEATURE	ASPD	PSYCHOPATHY (HARE PCL-R)
Diagnostic system	DSM-5 / ICD-11 (clinical)	PCL-R (research/forensic instrument)

FEATURE	ASPD	PSYCHOPATHY (HARE PCL-R)
Basis	Behavioural criteria	Trait + behavioural (interpersonal, affective, lifestyle, antisocial)
Core affective deficit	Implied (lack of remorse)	Central , shallow affect, callousness, absence of empathy
Empathy	Impaired but variable	Severely impaired , affective empathy absent; predatory
Remorse	Absent	Core criterion, grandiose dismissal of harm caused
Deceit	Present	Pathological , glibness, chronic lying, manipulation as art
General population prevalence	3–5%	~1% (PCL-R ≥30)
Prison population prevalence	40–70%	15–25%
Violence type	Reactive aggression predominant	Instrumental (predatory) violence more characteristic
Intelligence	Variable	Often average to above average
Risk to others	Elevated	Substantially higher
Treatment response	Poor	Potentially worse; some evidence for risk reduction not character change
Relationship	All psychopaths meet ASPD criteria	Most ASPD do NOT meet psychopathy threshold
Conduct Disorder link	Mandatory precursor	Often present; PCL-R doesn't require it formally
Assessment tool	SCID-5-PD, clinical interview	PCL-R (trained rater + file review + interview)
Forensic risk	High	Highest of all personality presentations
Key theorist	American Psychiatric Association	Robert Hare (1980, 1991)

EXAM PEARL

"All psychopaths meet ASPD criteria, but most individuals with ASPD are not psychopaths."
This sentence alone will secure marks in any forensic psychiatry question.

TABLE 8: Cluster A vs Cluster B vs Cluster C: Overview

FEATURE	CLUSTER A ("WEIRD")	CLUSTER B ("WILD")	CLUSTER C ("WORRIED")
Theme	Odd, eccentric, suspicious	Dramatic, emotional, erratic	Anxious, fearful, inhibited
Core affect	Detachment, suspicion, paranoia	Emotional intensity, instability, entitlement	Fear, anxiety, shame
Interpersonal style	Withdrawn, guarded	Stormy, intense, dramatic	Dependent, avoidant, rigid
Ego-syntonicity	Typically ego-syntonic	Mixed; BPD often ego-dystonic in crises	Variable (OCPD ego-syntonic; AvPD ego-dystonic)
Self-harm	Rare	Common (BPD)	Rare
Psychosis risk	Highest (Schizotypal → schizophrenia)	BPD: Transient stress-related psychosis	Minimal
Forensic relevance	Low	High (ASPD, BPD)	Low
Treatment seeking	Rarely	Often in crisis	Frequently
Treatment response	Moderate-poor	Variable: BPD good, ASPD poor	Generally best of the three clusters
Heritability	Moderate (schizophrenia spectrum)	Moderate (temperamental link)	Moderate (anxiety spectrum)
Biological link	Schizophrenia spectrum	Impulsivity / aggression neurobiology	Anxiety, autonomic dysregulation
ICD-11 qualifiers	Detachment; Psychoticism (Schizotypal)	Disinhibition; Dissociality; Negative Affectivity; Borderline Pattern	Negative Affectivity; Anankastia; Detachment (AvPD)
Key disorders	Paranoid, Schizoid, Schizotypal	ASPD, BPD, Histrionic, Narcissistic	Avoidant, Dependent, OCPD

TABLE 9: Attachment Styles and PD Links

ATTACHMENT STYLE (AINSWORTH)	INTERNAL WORKING MODEL	ADULT PRESENTATION	PD LINK
Secure	Self = worthy; other = reliable	Comfortable with intimacy and autonomy	No PD
Anxious-Preoccupied	Self = unworthy; other = uncertain	Clingy, hyperactivating attachment; emotional escalation	BPD, DPD, HPD

ATTACHMENT STYLE (AINSWORTH)	INTERNAL WORKING MODEL	ADULT PRESENTATION	PD LINK
Dismissing-Avoidant	Self = worthy; other = unnecessary	Deactivating strategies; emotional suppression; self-reliance	Schizoid PD, NPD (grandiose)
Fearful-Avoidant (Disorganised)	Self = unworthy; other = frightening	Approach-avoidance collapse; cannot regulate via attachment figure	BPD (especially with trauma); AvPD

CLINICAL ANCHOR

Disorganised attachment (Main & Hesse) is the strongest single childhood predictor of BPD features in adolescence and adulthood. The caregiver is simultaneously the source of fear and the haven of safety, an irresolvable paradox that creates the BPD pattern of intense approach + rage + collapse.

TABLE 10: Pharmacotherapy Evidence Summary: Personality Disorders

MEDICATION	SYMPTOM DOMAIN	EVIDENCE LEVEL	KEY STUDY/AUTHOR	NOTES
Valproate	Impulsive aggression, affective instability	Moderate (RCTs)	Hollander et al. 2001; Stein et al.	Best evidence for BPD pharmacotherapy
Lamotrigine	Affective instability, impulsivity	Moderate (RCT)	Tritt et al. 2005	Women with BPD; SJS risk, slow titration
Olanzapine 2.5–5mg	Cognitive-perceptual symptoms, affective dysregulation	Moderate (RCTs)	Zanarini et al. 2004, 2006, 2007	Weight gain limits long-term use
Quetiapine 25–300mg	Anxiety, insomnia, dissociation	Low-moderate (open-label)	Clinical series	Commonly used; limited RCT data for PD specifically
Fluoxetine	Impulsive aggression	Moderate	Coccaro & Kavoussi 1997	Modest; less effective than in MDD
Naltrexone 25–100mg	Dissociation, self-harm	Low (case series, open-label)	Roth et al., Bohus et al.	Opioid system hypothesis; pragmatic use

MEDICATION	SYMPTOM DOMAIN	EVIDENCE LEVEL	KEY STUDY/AUTHOR	NOTES
Omega-3 (EPA)	Aggression, depression	Low-moderate (RCT)	Zanarini & Frankenburg 2003	Safe, adjunctive; 1g EPA/day
Lithium	Impulsive aggression (ASPD)	Low-moderate	Sheard et al. 1976	Prison study; toxicity risk in BPD
Risperidone 0.5–2mg	Schizotypal PD symptoms	Moderate (RCT)	Koenigsberg et al. 2003	Schizotypal positive-like symptoms
Benzodiazepines	(Attempted: anxiety)	Contraindicated	Multiple studies of harm	Disinhibition, dependence, overdose risk
TCAs	(Attempted: depression)	Caution/avoid	Cardiotoxic in overdose	High lethality in impulsive patients

EXAM STRATEGY

When asked about pharmacotherapy in PD, open with: "Pharmacotherapy in PD is adjunctive and symptom-targeted. No medication is approved for any PD. Psychotherapy remains first-line." Then provide the evidence table by symptom domain.

PYQ Frequency Analysis

Series: MD Psychiatry Postgraduate Examinations (PG exams and equivalent Indian PG Psychiatry boards)

Data coverage: Representative question patterns across 17+ years of examination data

Purpose: Guide revision priority, identify high-yield zones, flag question format patterns

SECTION 1: Topic Frequency Heat Map

TOPIC	FREQUENCY	MARKS FORMAT	PRIORITY
BPD, Management (DBT, psychotherapy)	★★★★★ Very High	10 marks	P1, Master first
DBT, Principles and modules	★★★★★ Very High	10 marks	P1
BPD, Clinical features / DSM criteria	★★★★☆ High	5–10 marks	P1
ICD-11 vs DSM-5 PD classification	★★★★☆ High	10 marks	P1 (recent + rising)
Schema Therapy for PD	★★★★☆ High	10 marks	P1
ASPD, Forensic implications	★★★★☆ High	10 marks	P1
BPD vs Bipolar II differential	★★★☆☆ Moderate-High	5–10 marks	P2
Pharmacotherapy in PD	★★★☆☆ Moderate-High	5–10 marks	P2
Narcissistic PD	★★★☆☆ Moderate	5–10 marks	P2
OCPD vs OCD	★★★☆☆ Moderate	5–10 marks	P2
Avoidant PD vs Social Anxiety	★★★☆☆ Moderate	5 marks	P2
Attachment theory and PD	★★★☆☆ Moderate	5 marks	P2
MBT (Mentalization-Based Treatment)	★★☆☆☆ Moderate	5 marks	P3

TOPIC	FREQUENCY	MARKS FORMAT	PRIORITY
Cluster A PDs (overview)	★★☆☆☆ Moderate	5 marks	P3
Schizotypal PD	★★☆☆☆ Moderate	5 marks	P3
Longitudinal course / McLean Study	★★☆☆☆ Moderate	5 marks	P3
Biosocial Theory (Linehan)	★★★☆☆ Moderate	5 marks	P2
PCL-R and Psychopathy	★★☆☆☆ Moderate	5 marks	P3
Assessment instruments (SCID-5-PD, IPDE)	★★☆☆☆ Moderate	5 marks	P3
Dependent PD	★★☆☆☆ Low-Moderate	5 marks	P3
TFP (Transference-Focused Psychotherapy)	★★☆☆☆ Low-Moderate	5 marks	P3
GPM (General Psychiatric Management)	★★☆☆☆ Low-Moderate	5 marks	P3
Histrionic PD	★★☆☆☆ Low	5 marks	P3
18 Early Maladaptive Schemas (Young)	★★★☆☆ Moderate	5–10 marks	P2
Schema modes (mode model)	★★★☆☆ Moderate	5 marks	P2
BPD comorbidity (SUD, PTSD)	★★☆☆☆ Low-Moderate	5 marks	P3

Priority Key: P1 = Must master; P2 = Should know; P3 = Good to know; P4 = Awareness level

SECTION 2: Reconstructed Question Bank by Topic

EXAM PEARL

Note: These represent question patterns drawn from reported exam formats. Exact wording reconstructed for study purposes.

BPD: Management (P1)

1. Describe the management of Borderline Personality Disorder. (10 marks)
2. Discuss the role of Dialectical Behaviour Therapy in Borderline Personality Disorder. (10 marks)
3. Outline the psychological management of BPD. (10 marks)
4. A 24-year-old woman presents with recurrent self-cutting, intense relationships, and chronic emptiness. Describe management. (10 marks)
5. Write short notes on: DBT for BPD. (5 marks)
6. Enumerate the pharmacological approaches in management of BPD with their evidence. (5 marks)
7. What are the components of standard DBT? Describe the target hierarchy. (5 marks)

EXAM STRATEGY

Q1 above appears in some form almost every other year. There is no excuse for being underprepared for this question. The structure: Risk assessment → Psychotherapy (DBT, ST, MBT, TFP) → Pharmacotherapy (symptom-targeted, adjunctive) → Special populations → Prognosis.

Classification of PD (ICD-11 vs DSM-5) (P1: Rising Priority)

1. Compare ICD-11 and DSM-5 classification of Personality Disorders. (10 marks)
2. Write short notes on: ICD-11 dimensional model of Personality Disorders. (5 marks)
3. What are the advantages of the dimensional approach to Personality Disorders? (5 marks)
4. Describe the severity levels in ICD-11 Personality Disorder classification. (5 marks)
5. Discuss the Alternative Model for Personality Disorders (AMPD) in DSM-5. (5 marks)
6. Compare categorical and dimensional approaches to Personality Disorders. (10 marks)

EXAM PEARL

ICD-11 was adopted in 2022. Questions on the new classification system have increased in frequency since 2022–23. Master the severity levels (mild/moderate/severe), the 5 trait qualifiers + Borderline Pattern specifier, and the contrast with DSM-5 clusters.

Schema Therapy (P1)

1. Describe Schema Therapy for Personality Disorders. (10 marks)
2. What are Early Maladaptive Schemas? Classify them with examples. (10 marks)
3. Write short notes on: Schema modes in BPD. (5 marks)
4. Discuss the role of limited reparenting in Schema Therapy. (5 marks)

5. Enumerate Young's Early Maladaptive Schemas. (5 marks)
 6. Describe the mode model in Schema Therapy. (5 marks)
-

ASPD and Forensic Psychiatry (P1)

1. Discuss the forensic implications of Antisocial Personality Disorder. (10 marks)
 2. Describe the features of ASPD and its management. (10 marks)
 3. What is the Hare Psychopathy Checklist-Revised (PCL-R)? Describe its structure and forensic relevance. (10 marks)
 4. Compare ASPD and Psychopathy. (5 marks)
 5. Write short notes on: Conduct Disorder as a precursor to ASPD. (5 marks)
 6. Discuss the risk assessment tools used in forensic psychiatry. (5 marks)
 7. What are the McNaughton Rules? How does ASPD relate to criminal responsibility? (5 marks)
-

BPD vs Bipolar II (P2)

1. Differentiate BPD from Bipolar Disorder II. What are the implications for management? (10 marks)
 2. A 27-year-old woman presents with emotional lability, impulsivity, and relationship difficulties. How do you differentiate BPD from Bipolar II? (10 marks)
 3. Write short notes on: Differential diagnosis of BPD. (5 marks)
-

Pharmacotherapy in PD (P2)

1. Discuss the pharmacological management of Personality Disorders with evidence. (10 marks)
 2. What is the role of mood stabilisers in Personality Disorders? (5 marks)
 3. Write short notes on: Pharmacotherapy in BPD. (5 marks)
 4. What medications should be avoided in BPD and why? (5 marks)
-

Narcissistic PD (P2)

1. Describe Narcissistic Personality Disorder. Distinguish grandiose from vulnerable subtypes. (10 marks)
 2. Write short notes on: Narcissistic injury and narcissistic rage. (5 marks)
 3. Describe the management of NPD. (5 marks)
-

OCPD vs OCD (P2)

1. Differentiate OCPD from OCD. Discuss implications for management. (10 marks)
 2. Write short notes on: OCPD, clinical features. (5 marks)
 3. A 35-year-old professional presents with extreme perfectionism affecting his work and marriage. Distinguish OCPD from OCD. (5 marks)
-

Attachment Theory and PD (P2)

1. Describe Bowlby's attachment theory and its relevance to Personality Disorders. (10 marks)
 2. Write short notes on: Disorganised attachment and BPD. (5 marks)
 3. How does attachment theory inform the treatment of BPD? (5 marks)
-

Avoidant PD vs Social Anxiety (P2)

1. Compare Avoidant Personality Disorder with Social Anxiety Disorder. (5 marks)
 2. Describe the management of Avoidant PD. (5 marks)
-

Biosocial Theory (P2)

1. Describe Linehan's Biosocial Theory of BPD. (5 marks)
 2. What is the invalidating environment? How does it contribute to BPD? (5 marks)
-

Mentalization-Based Treatment (P3)

1. Describe Mentalization-Based Treatment (MBT) for BPD. (5–10 marks)
 2. What is mentalization? How is it impaired in BPD? (5 marks)
-

Cluster A PDs (P3)

1. Describe the Cluster A Personality Disorders with their distinguishing features. (10 marks)
 2. Write short notes on: Schizotypal Personality Disorder. (5 marks)
 3. Compare Schizoid and Avoidant Personality Disorders. (5 marks)
 4. Discuss the relationship between Schizotypal PD and schizophrenia. (5 marks)
-

Longitudinal Course (P3)

1. Describe the course and prognosis of BPD with reference to longitudinal studies. (10 marks)
2. Write short notes on: McLean Study of Adult Development. (5 marks)

3. What factors predict poor prognosis in BPD? (5 marks)

SECTION 3: Format Analysis

Question Type Distribution

QUESTION TYPE	APPROXIMATE FREQUENCY	MARKS
"Describe the management of..."	30%	10
"Differentiate/Compare X from Y"	25%	5–10
"Discuss/Describe X" (single topic)	25%	5–10
"Write short notes on..."	15%	5
Clinical vignette-based	5%	5–10

EXAM STRATEGY

"Management" questions dominate. Every "management" answer follows the same structure: (1) brief intro/definition, (2) risk assessment if relevant, (3) psychological/psychosocial treatment, (4) pharmacotherapy, (5) special considerations, (6) prognosis. Drill this structure until it's automatic.

Marks Allocation Pattern

MARKS	EXPECTED CONTENT	WORD COUNT EQUIVALENT
5 marks	Definition + 3–4 key points + brief conclusion	~300–400 words
10 marks	Full structured answer: intro + all major sections + conclusion	~600–900 words
15 marks (rare)	Comprehensive essay with evidence citations	~900–1200 words

SECTION 4: Emerging and High-Priority Topics (2023–2026)

These topics have either appeared recently for the first time or are expected based on curricular updates:

TOPIC · WHY HIGH PRIORITY NOW

ICD-11 dimensional model ICD-11 adopted 2022; examiners testing contemporary knowledge

Complex PTSD vs BPD New ICD-11 diagnosis; differential increasingly tested

DBT adaptations (DBT-A, DBT-S) Expanding evidence base; clinical applications growing

Schema modes in BPD Schema Therapy increasingly recognised; mode model is board-level knowledge

Vulnerable NPD Clinical relevance; misdiagnosis as depression increasingly highlighted

GPM (General Psychiatric Management) McMain et al. study; important that GPM = non-inferior to DBT

MBT-A (adolescent MBT) BPD diagnosis in adolescents now more accepted; MBT-A emerging

BPD + PTSD management Sequencing treatment; DBT-PTSD protocol; practical clinical importance

SECTION 5: Answer Skeleton Templates

Template A: Management Question (10 marks)

Template B: Comparison / Differentiation Question (10 marks)

Template C: "Describe X" Short Note (5 marks)

SECTION 6: Examiner Intelligence: What Scores High Marks

Based on patterns across question papers and standard marking expectations:

ELEMENT · MARKS IMPACT

Citing named studies (McLean, Zanarini, Linehan 1991, Bateman & Fonagy) +2 marks

Using correct diagnostic criteria (DSM-5 number required: e.g., 5/9) +1 mark

Structured headings (not a prose wall) +1 mark

Including pharmacotherapy with evidence, not just listing drugs +1 mark

Mentioning NICE guidance or APA guideline recommendation +1 mark

Distinguishing psychotherapy as first-line from pharmacotherapy as adjunctive +1 mark

Clinical vignette: linking features to diagnosis AND management +2 marks

ICD-11 and DSM-5 both mentioned where relevant +1 mark

Prognosis with specific data (e.g., "85% remission at 10 years, McLean Study") +1 mark

EXAM STRATEGY

In a 10-mark answer, you are competing for approximately 10 content points. The elements above tell you exactly what examiners are counting. Structure your answer to hit as many as possible.

SECTION 7: Common Exam Mistakes to Avoid

MISTAKE · CORRECTION

Writing "BPD has poor prognosis" BPD has **better prognosis than previously believed**, cite McLean Study

Saying pharmacotherapy is first-line for BPD Pharmacotherapy is **adjunctive**; psychotherapy is first-line

Confusing OCPD and OCD as the same condition OCPD = ego-syntonic; OCD = ego-dystonic; different treatment

Listing DBT skills without explaining the module structure Always name the 4 modules (DIMS) and explain the target hierarchy

Describing ICD-11 as having 10 categorical PDs ICD-11 abolished categorical PDs; uses severity + qualifiers

Calling all Cluster A patients "schizophrenia spectrum" Only **Schizotypal** is in schizophrenia spectrum (ICD-11); not Paranoid or Schizoid

Missing the Conduct Disorder requirement for ASPD CD before age 15 is **mandatory** for ASPD diagnosis, always state this

Conflating psychopathy with ASPD All psychopaths have ASPD, most ASPD ≠ psychopathy

Recommending benzodiazepines for BPD anxiety Benzodiazepines are **contraindicated** in BPD (disinhibition, dependence, overdose)

Not naming the biosocial theory when discussing DBT DBT is explicitly grounded in the Biosocial Theory (Linehan), always state the theory

SECTION 8: Paper II Context: Where PD Sits

Paper II: Clinical Psychiatry covers:

- Neurotic, stress-related, and somatoform disorders
- Mood disorders
- **Personality disorders** ← this notebook
- Psychosexual disorders
- Child and adolescent psychiatry
- Old age psychiatry
- Consultation-liaison psychiatry
- Substance use disorders

PD questions typically appear as:

- 1–2 long questions (10 marks each) per paper
- 1–2 short notes (5 marks each) per paper
- Embedded in vignette-based questions across topics

Strategy: BPD management + DBT + Schema Therapy = guaranteed marks if you know them cold. ICD-11 classification = rising priority. ASPD + forensics = reliable 10-marker every 2–3 years.

Quick Review

All names, identifying details, ages, and circumstances are entirely fictitious. Any resemblance to real persons is coincidental.

Purpose: Build diagnostic reasoning under exam conditions. Each vignette is followed by structured analysis: diagnosis, differential, key diagnostic reasoning, and management outline.

EXAM STRATEGY

Vignette questions test your ability to (1) identify the correct diagnosis from clinical features, (2) apply diagnostic criteria, (3) discuss the differential, and (4) outline management. Practice stating your diagnostic reasoning explicitly, examiners reward visible clinical thinking.

VIGNETTE 1: The Young Woman Who Cuts

Presentation:

Priya, 22 years old, is brought to the psychiatric emergency by her boyfriend after she cut her forearm with a razor blade. The precipitant was a text message from her boyfriend saying he would be 30 minutes late. She describes this as feeling "like he was abandoning me." She has a history of three similar episodes in the past year, each triggered by perceived rejection. She describes her relationships as intense, "either he's perfect or he's the worst person alive." Her mood changes rapidly, sometimes within hours, from tearful to enraged. She endorses chronic feelings of emptiness and reports that cutting temporarily relieves the emptiness. She sometimes feels "unreal" when very stressed. She has changed jobs three times in 18 months and has few stable friendships.

Mental State Examination:

- Affect: Labile, tearful → irritable within the interview
- Mood: "I don't know what I am"
- Cognition: Intact
- No formal thought disorder; no hallucinations; no sustained delusional beliefs

Diagnostic Analysis

Primary Diagnosis: Borderline Personality Disorder (DSM-5)

DSM-5 criteria met (5/9 required):

1. Frantic efforts to avoid abandonment (text message → cutting) ✓

2. Unstable intense relationships (idealisation ↔ devaluation of boyfriend) ✓
3. Identity disturbance ("I don't know what I am") ✓
4. Impulsivity, self-harm and job changes ✓
5. Recurrent self-mutilating behaviour ✓
6. Affective instability (rapid mood shifts within hours) ✓
7. Chronic feelings of emptiness ✓
8. Dissociative symptoms under stress ("unreal") ✓

8/9 criteria met, diagnosis well-established.

Differential Diagnosis:

DIAGNOSIS	REASON TO CONSIDER	WHY NOT PRIMARY
Bipolar II	Mood instability; impulsivity	Mood changes last hours not days; no sustained hypomanic episode; interpersonal trigger dominant
MDD with NSSI	Cutting; low mood	No sustained depressive episode; emptiness is baseline not acquired; identity instability not explained
PTSD/Complex PTSD	Dissociation; hyperreactivity	No specific trauma reexperiencing; dissociation stress-related in context of abandonment; identity diffusion present
ADHD	Impulsivity; job instability	No inattention/hyperactivity history; emotional instability more severe and relationship-centred

ICD-11 Formulation:

- Severity: **Moderate PD** (most domains impaired; all relationships affected)
- Qualifier: **Borderline Pattern + Negative Affectivity + Disinhibition**

Management Outline

Immediate:

- Safety assessment: wound care; suicide risk stratification (chronic elevated risk, not acute suicidal intent here)
- Safety plan: crisis contacts, distress tolerance steps, means restriction (remove razors)
- Brief supportive engagement; psychoeducation about BPD (normalise, de-shame)
- Avoid admission unless acute high risk, brief admission only if needed

Psychological (First-Line):

- **DBT:** Standard comprehensive programme; individual + skills group; phone coaching
- Priority: distress tolerance for abandonment cues (TIPP, ACCEPTS, Radical Acceptance)
- Chain analysis of cutting episode
- **Schema Therapy:** When stabilised; address Abandonment/Instability schema; Vulnerable Child mode; Punitive Parent mode
- Duration: 12–24 months minimum

Pharmacotherapy (Adjunctive):

- If affective instability severe: low-dose lamotrigine (titrate slowly) or valproate
- If transient paranoid ideation: low-dose quetiapine 25–50 mg PRN
- Avoid benzodiazepines

Prognosis: Guarded but not poor, 85% achieve sustained remission at 10 years (McLean Study). Communicate hope alongside realism.

VIGNETTE 2: The Man Who Feels Above the Rules

Presentation:

Rohan, 34 years old, is referred by his employer's occupational health service following multiple complaints from junior colleagues about "bullying." He presents as composed, articulate, and dismissive of the referral: "I'm not the problem, they can't handle feedback." He describes a career trajectory of early success followed by increasing conflict at work. He has been married and divorced twice. His ex-wives both cited emotional cruelty and his inability to acknowledge their needs. He says, "Women just aren't capable of keeping up with me." He describes his management style as "high standards" and says he has never met anyone who truly understands him. He denies any psychological distress. He does not believe he has a problem.

MSE: Euthymic; no psychosis; no depressive features; no anxiety. Affect: Restricted, cold. Speech: Verbose, self-referential. Insight: Absent.

Diagnostic Analysis

Primary Diagnosis: Narcissistic Personality Disorder, Grandiose Subtype (DSM-5)

DSM-5 criteria met:

1. Grandiose sense of self-importance ("they can't handle feedback") ✓
2. Preoccupied with unlimited success ("early career success") ✓
3. Believes only equally special people understand him ✓
4. Interpersonally exploitative (workplace bullying; marriages) ✓

5. Lacks empathy (ex-wives' needs dismissed; "women can't keep up") ✓
6. Sense of entitlement (referral is "not his problem") ✓
7. Arrogant, haughty behaviour (dismissive of occupational referral) ✓

7/9 criteria met, threshold exceeded.

Differential:

DIAGNOSIS	REASON TO CONSIDER	WHY NOT PRIMARY
ASPD	Workplace misconduct; callousness	No Conduct Disorder history; no criminal behaviour; identity stable and grandiose not dissocial
Grandiose Bipolar I	Expansiveness, overconfidence	No episodic course; no discrete hypomanic / manic episodes; stable personality presentation
Paranoid PD	Distrust of colleagues	Dismissiveness is about superiority not threat; no ideas of persecution
OCPD	High standards, rigid	OCPD ego-syntonic perfectionism lacks grandiosity; no entitlement; would be distressed about imperfection not others' incompetence

Management Outline

Engagement: The most significant challenge. He does not believe he has a problem. Avoid confrontation early. Use motivational stance.

Psychological (Primary):

- **Schema Therapy:**
 - Initial phase: Validate his experience before challenging it
 - Build alliance with the Healthy Adult while respecting the Self-Aggrandiser
 - Gradually access the Lonely / Abandoned Child beneath the grandiosity
 - Chair work: dialogue between Self-Aggrandiser and Vulnerable Child
 - Empathic confrontation: "Your standards make sense given your history, AND they are costing you relationships"
 - Duration: 3–5 years; NPD is among the more treatment-resistant PDs
- **TFP (Transference-Focused Psychotherapy):** Interpret idealisations and devaluations in the transference; work with object relations pathology
- **Motivational Interviewing:** To build engagement before formal therapy

Pharmacotherapy:

- No indication currently
- SSRIs if depressive episode develops (narcissistic collapse with NPD is common trigger for MDD)

Forensic/Occupational: If workplace behaviour escalates to harassment, occupational psychiatry report may be needed; boundaries of confidentiality discussion

Prognosis: Guarded. Overt grandiose NPD is among the harder PD subtypes to engage. If engagement achieved, meaningful change is possible over years.

VIGNETTE 3: The Man Who Cannot Stop Lying

Presentation:

Kiran, 28 years old, is referred from the prison mental health team. He is serving a 4-year sentence for fraud. He has three previous convictions (theft age 16, assault age 20, drug dealing age 24). He is charming and articulate at interview. He minimises his offences: "The judge didn't understand the full picture." He describes childhood conduct problems, truancy, fighting, fire-setting, from age 12. He shows no apparent distress about his victims. He is currently working in the prison library and has received multiple warnings for manipulating other prisoners. He sleeps well, has normal appetite, and reports no psychiatric symptoms.

MSE: Normal cognition; no mood disorder; no psychosis; no anxiety. **Affect:** Shallow. **Insight:** Minimal regarding impact on others.

Diagnostic Analysis

Primary Diagnosis: Antisocial Personality Disorder (DSM-5)

Mandatory requirements:

- Age ≥ 18 ✓ (28)
- Evidence of Conduct Disorder before 15 ✓ (truancy, fighting, fire-setting from age 12)

DSM-5 behavioural criteria (≥ 3 required):

1. Repeated unlawful behaviour ✓ (4 convictions)
2. Repeated lying, conning for personal gain ✓ (fraud; manipulating prisoners)
3. Impulsivity (implied in pattern) ✓
4. Reckless disregard for safety (assault) ✓
5. Consistent irresponsibility ✓
6. Lack of remorse ✓ ("judge didn't understand")

6/7 behavioural criteria met.

Psychopathy assessment: Shallow affect, charm, manipulation, pathological lying, lack of remorse, callousness, PCL-R assessment warranted in forensic context.

Differential:

DIAGNOSIS	REASON TO CONSIDER	WHY NOT PRIMARY
Schizophrenia	Prison population; consider all diagnoses	No psychotic symptoms; clear history
SUD with antisocial behaviour	Criminal history often drug-linked	SUD may be comorbid but antisocial traits predate substance use
Bipolar I (hypomanic disinhibition)	Manipulation; charm	No episodic history; stable antisocial pattern from childhood

EXAM PEARL

Always distinguish ASPD from malingering in prison settings. ASPD patients may malingering for secondary gain (medication, transfer, privileges), MSE and PCL-R help distinguish.

Management Outline

Risk Assessment:

- PCL-R (trained rater): Factor 1 (interpersonal/ affective) and Factor 2 (social deviance)
- HCR-20 (v3): Historical, Clinical, Risk Management factors for violence risk
- Violence risk: HIGH given PCL-R features; instrumental manipulation

Psychological:

- **Therapeutic community** (democratic model): Group-based; peer accountability; contingency management
- **CBT adapted for offenders:** Cognitive distortions ("ownership," victim empathy, consequential thinking)
- **Reasoning and Rehabilitation (R&R) Programme:** Structured cognitive skills programme for offenders
- Not suitable for standard individual psychotherapy until motivation established

Pharmacotherapy:

- **Valproate/lithium** for impulsive aggression if relevant
- Treat comorbid SUD if present (naltrexone, buprenorphine)
- No medication changes underlying antisocial personality

Forensic/Legal:

- Expert witness report if requested: Document diagnosis; comment on risk; do NOT predict specific future behaviour
- McNaughton Rules: ASPD alone does NOT fulfil Section 84 IPC criteria

Prognosis: Guarded. Behavioural antisocial symptoms may decrease after 40 ("burning out"). Character change is rare. Goal = risk reduction, not cure.

VIGNETTE 4: The Woman Who Cannot Say No

Presentation:

Ananya, 38 years old, presents with depression following the breakdown of her 12-year marriage. She describes her marriage as one in which she "did whatever he wanted." She could not refuse her husband's demands, even when they were unreasonable, for fear of losing him. Since his departure, she has been paralysed, unable to make basic decisions (what to eat, whether to call a plumber), phoning her sister 15–20 times daily for reassurance. She describes herself as "helpless without someone to tell me what to do." She has returned to live with her parents at age 38. She describes a similar pattern in her relationship before her marriage. She cannot imagine living alone.

Diagnostic Analysis

Primary Diagnosis: Dependent Personality Disorder (DSM-5)

DSM-5 criteria (≥5 of 8 required):

1. Difficulty making everyday decisions without reassurance (calling sister 15–20x) ✓
2. Needs others to assume responsibility for major life areas ✓
3. Difficulty expressing disagreement (could not refuse husband) ✓
4. Difficulty initiating alone (paralysed without partner) ✓
5. Goes to excessive lengths to obtain nurturance (compliance with unreasonable demands) ✓
6. Feels helpless when alone ✓
7. Urgently seeks new relationship after one ends (implicit in fear of being alone) ✓

7/8 criteria met.

Comorbidity: Current MDD (secondary to relationship loss + schema activation)

Differential:

DIAGNOSIS	REASON TO CONSIDER	WHY NOT PRIMARY
BPD	Fears abandonment; clingy	BPD: rage, splitting, self-harm, identity instability, none prominent here; DPD has stable submissive dependency
Adjustment disorder	Depressive response to loss	Pattern predates current loss; lifelong across relationships
Agoraphobia	Cannot manage independently	No panic attacks; no avoidance of specific situations; dependency is relational
MDD alone	Current depression	MDD is secondary; personality pattern is lifelong, predates current episode

Management Outline

Immediate:

- Treat acute MDD: SSRI (escitalopram 10–20 mg)
- Psychoeducation: Explain DPD; normalise; de-shame
- Safety assessment: Passive suicidal ideation common in MDD + DPD

Psychological (Primary):

- **Schema Therapy:** Target Dependence/Incompetence schema; Abandonment/Instability schema
- Limited reparenting: Build sense of internal reliability
- Graduated autonomy exercises: Make one decision alone each week
- Chair work: Vulnerable Child (helpless) ↔ Healthy Adult (capable)
- **CBT:** Challenge core beliefs: "I cannot manage without others" → graduated experiments in independence
- **Assertiveness training:** Structured practice of expressing disagreement
- **Group therapy:** Reduces therapist dependency; provides peer feedback

Therapeutic Pitfall: Dependency will transfer to the therapist. Manage from the start: planned tapering, explicit focus on autonomy-building, no unscheduled calls between sessions (or structure them).

Pharmacotherapy:

- SSRI for acute MDD
- No specific medication for DPD traits

Prognosis: Moderate. DPD is more treatment-responsive than Cluster B. MDD resolves with treatment; personality change slower but achievable with sustained schema work.

VIGNETTE 5: The Engineer Who Cannot Finish Anything

Presentation:

Suresh, 42 years old, is referred by his wife following marital conflict. He is a software engineer who frequently works 16-hour days but repeatedly misses deadlines because "it's never good enough." His wife describes him as controlling at home, he re-does tasks she has done because she doesn't do them "correctly." He becomes agitated if objects are not in precise positions. He has not taken a holiday in 8 years ("I can't leave the project in someone else's hands"). He has not discarded any work documents since 1998. He says he cannot understand why his wife is unhappy, he works hard, he is reliable, he does not understand emotions well. He does not experience his traits as problems. "I just have high standards."

Diagnostic Analysis

Primary Diagnosis: Obsessive-Compulsive Personality Disorder (DSM-5)

DSM-5 criteria (≥4 of 8 required):

1. Preoccupied with details/ rules/ schedules (to point of losing the point) ✓
2. Perfectionism interfering with task completion (misses deadlines) ✓
3. Excessively devoted to work, excluding relationships ✓
4. Overconscientious, rigid about values ("correct" way to do things) ✓
5. Unable to discard worthless objects (documents since 1998) ✓
6. Reluctant to delegate (cannot leave project to others) ✓
7. Rigid and stubborn ✓

7/8 criteria met; ego-syntonic ("I just have high standards").

No OCD: No intrusive ego-dystonic obsessions; no anxiety-driven compulsions; traits are valued not distressing to the patient himself.

Differential:

DIAGNOSIS	REASON TO CONSIDER	WHY NOT PRIMARY
OCD	Checking; re-doing tasks	No intrusive obsessions; re-doing is to maintain standards, not neutralise anxiety; ego-syntonic
ASPD	Controlling behaviour	No disregard for others' rights; no deception; conscientious

DIAGNOSIS	REASON TO CONSIDER	WHY NOT PRIMARY
ASD	Rigidity; social difficulties	ASD: qualitatively different social communication; sensory features; lifelong developmental history
Avoidant PD	Work focus; social constriction	AvPD: wants relationships but fears rejection; emotional inhibition here is about control not fear

Management Outline

Engagement: Patient sees traits as virtues. Motivation for change usually driven by relationship consequences. Use MI.

Psychological:

- **Schema Therapy:** Target Unrelenting Standards schema; Demanding/Punitive Parent mode
- "Good enough" experiments: Deliberately do something at 80% and observe consequences
- Limited reparenting for the child who only received approval for achievement
- Chair work: Demanding Parent ↔ Vulnerable Child ↔ Healthy Adult
- **CBT:** Perfectionism cycle psychoeducation; time-limited task experiments; delegation hierarchy
- **Couples/Marital therapy:** Address impact of rigidity on relationship

Pharmacotherapy:

- SSRIs if comorbid OCD features emerge or significant anxiety
- No specific pharmacotherapy for OCPD traits

Prognosis: CLPS data: OCPD is one of the more stable PDs. Some functional improvement with sustained therapy. Traits may persist; occupational and relationship impact can reduce.

VIGNETTE 6: The Young Man With Unusual Beliefs

Presentation:

Aakash, 26 years old, is referred by his GP after his family expressed concern. He lives alone and works in data entry. He has no friends, "I prefer my own company." He describes believing he can sense when people are "thinking about him." He carries rituals, he does not walk on certain streets because he believes they are "energetically wrong." He speaks in elaborate, vague sentences that are hard to follow. His affect is incongruent, he smiles when

discussing serious topics. He denies hearing voices. He denies believing he has special powers "in the sense others mean it." His family describes him as always being "odd" and socially avoidant since childhood. No acute deterioration. No Axis I psychiatric history.

Diagnostic Analysis

Primary Diagnosis: Schizotypal Personality Disorder (DSM-5)

DSM-5 criteria (≥ 5 of 9 required):

1. Ideas of reference (sensing when people think about him) ✓
2. Odd beliefs / magical thinking (energetically wrong streets; rituals) ✓
3. Unusual perceptual experiences (sensing presences), implied ✓
4. Odd thinking and speech (vague, hard to follow, elaborate) ✓
5. Social anxiety that doesn't decrease with familiarity, implied ✓
6. Inappropriate or constricted affect (incongruent smiling) ✓
7. Odd, eccentric behaviour or appearance (rituals, isolation) ✓
8. Lacks close friends ✓

8/9 criteria met.

ICD-11 Note: Classified under Schizophrenia Spectrum Disorders, not PD category.

Relevant distinction for exam.

Differential:

DIAGNOSIS	REASON TO CONSIDER	WHY NOT PRIMARY
Schizophrenia	Odd beliefs; unusual perceptions	No frank delusions; no hallucinations; no disorganisation; stable for years without deterioration
Schizoid PD	Social isolation; restricted affect	Schizoid: no magical thinking, no odd speech, no ideas of reference; content with isolation
OCD with magical thinking	Rituals; street avoidance	OCD: ego-dystonic; specific obsessions; anxiety-driven; would want to stop
ASD	Social difficulties; routines	ASD: qualitatively different social impairment; sensory sensitivity; explicit social communication deficits
Paranoid PD	Suspiciousness; ideas of reference	Paranoid PD: no magical thinking; no odd speech/appearance; no unusual perceptions

Management Outline

Pharmacotherapy:

- Low-dose risperidone 0.5–1 mg: Targets positive-like symptoms (ideas of reference, paranoid ideation, unusual perceptions)
- SSRIs for social anxiety and dysphoria if present
- Regular monitoring: Metabolic, EPS

Psychological:

- **CBT:** Reality-testing unusual beliefs; distinguishing ideas of reference from reality; cognitive restructuring
- **Social skills training:** Address eccentric presentation; build social confidence
- **Supportive therapy:** Build therapeutic alliance; low demand initially; validate his experience of the world

Social/Vocational:

- Occupational support: Structured, low-demand environment suits schizotypal features
- Psychoeducation for family: Reduce expressed emotion; avoid pressure for "normal" social functioning

Monitoring:

- Annual review for conversion to psychotic disorder (10–15% lifetime risk)
- No routine antipsychotic without active positive-like symptoms

Prognosis: Stable but chronic mild impairment. Non-deteriorating unlike schizophrenia. Small minority convert to psychotic disorder.

VIGNETTE 7: The Reluctant Doctor

Presentation:

Meera, 29 years old, a junior doctor, seeks help for "social anxiety." She avoids ward rounds where she might be asked questions publicly. She has declined three promotion opportunities because "I'll just embarrass myself." She has no close friendships at work despite being described by colleagues as "warm and thoughtful." She reveals she secretly wants deeper friendships but is terrified of being "found out as inadequate." She does not initiate conversations for fear of saying something stupid. She describes herself as "fundamentally unlikeable." She has been to two parties in the past year and left early both times, feeling overwhelmed. She is not depressed. She is not psychotic.

Diagnostic Analysis

Primary Diagnosis: Avoidant Personality Disorder (DSM-5)

DSM-5 criteria (≥ 4 of 7 required):

1. Avoids occupational activities (declined promotions; avoids ward rounds) ✓
2. Shows restraint in intimate relationships (fears shame / ridicule) ✓
3. Preoccupied with criticism / rejection in social situations ✓
4. Inhibited in new interpersonal situations ✓
5. Views self as inept / inferior ("fundamentally unlikeable") ✓
6. Reluctant to take personal risks (avoids conversations) ✓
7. Unwilling to get involved unless certain of being liked (implied) ✓

7/7 criteria met; significant functional impairment (career; social).

Key feature: She WANTS connections, this is the crucial distinction from Schizoid PD.

Differential:

DIAGNOSIS	REASON TO CONSIDER	WHY NOT PRIMARY
Social Anxiety Disorder (generalised)	Social avoidance; fear of embarrassment	High overlap; AvPD may be severe end of same spectrum; AvPD: identity-level inferiority, more pervasive, ego-syntonic
Schizoid PD	Social avoidance; few friendships	Schizoid: does not want connections; no fear; indifferent; no distress
Dependent PD	Fears criticism; needs reassurance	DPD: submissive clinging; needs to be cared for; AvPD avoids rather than clings
MDD	Low self-esteem; social withdrawal	No depressive episode; avoidance is lifelong not acquired

Management Outline

Psychological (First-Line):

- **Schema Therapy:** Core schemas: Defectiveness/Shame, Social Isolation, Failure
- Imagery rescripting: Early humiliation scenes in school or family
- Chair work: Healthy Adult comforts shamed Vulnerable Child
- Limited reparenting: Empathic validation without performance demands
- Behavioural experiments: Graduated social approach with schema diary
- **CBT with graduated exposure:**
- Fear hierarchy: Simple social interactions → ward round participation → party attendance
- Cognitive restructuring: "I am fundamentally unlikeable" → evidence testing

- Role-play: Social scenario practice
- **Group therapy:** In-vivo exposure; interpersonal process

Pharmacotherapy:

- Escitalopram 10–20 mg or paroxetine 20 mg: Reduces social anxiety; improves approach behaviour
- Beta-blocker (propranolol 10–20 mg) for performance-specific anxiety as adjunct

Prognosis: Better than Cluster B. Good response to CBT for social anxiety component. Schema work produces deeper identity-level change over 1–3 years.

VIGNETTE 8: The Man Who Has Always Been Different

Presentation:

Vikram, 32 years old, presents after his wife's persistent encouragement. He describes preferring solitary activities, he collects vintage stamps and spends most evenings in his study. He has been married for 3 years but describes marriage as "a practical arrangement." He has no interest in sexual activity with his wife ("it never interested me"). He does not discuss feelings and cannot understand why his wife needs emotional conversations. He has no friends; he says he "doesn't see the point." When his father died last year, he felt nothing. He is not distressed by any of this. He does not believe he has a problem.

Diagnostic Analysis

Primary Diagnosis: Schizoid Personality Disorder (DSM-5)

DSM-5 criteria (≥4 of 7 required):

1. Neither desires nor enjoys close relationships ✓
2. Almost always chooses solitary activities ✓
3. Little interest in sexual experiences ✓
4. Lacks close friends ✓
5. Indifferent to praise or criticism ✓
6. Emotional coldness, detachment, flattened affectivity ✓
7. *(Takes pleasure in few activities, partial; he enjoys stamps)*

6/7 criteria met. Ego-syntonic, he does not believe he has a problem.

Key distinguishing feature: He is **content** with his solitary life, this is the crucial Schizoid–Avoidant distinction.

Differential:

DIAGNOSIS	REASON TO CONSIDER	WHY NOT PRIMARY
Avoidant PD	Social isolation; few friendships	AvPD desperately wants connection; this patient is genuinely indifferent; no fear
Schizotypal PD	Social isolation; restricted affect	No magical thinking; no odd speech; no ideas of reference; no unusual perceptions
ASD	Restricted affect; social difficulties	ASD: qualitative communication differences; sensory features; childhood developmental history pattern
Depressive disorder	Anhedonia; withdrawal	Anhedonia in Schizoid is longstanding baseline not acquired; no depressed mood; no neurovegetative features

Management Outline

Reality check: He is not presenting voluntarily; his wife is distressed. Goal is not to transform his personality but to reduce the relational impact.

Psychological:

- **Supportive therapy:** Build minimal therapeutic alliance; low demand; do not push emotional expression
- **Couples therapy:** Help wife understand his experience; negotiate needs; reframe his style as difference not deficit
- **Psychoeducation:** Schizoid PD; why emotional conversation is difficult; what he can offer

Pharmacotherapy:

- SSRIs if comorbid anxiety or dysphoria emerges
- No specific pharmacotherapy for Schizoid traits

Prognosis: Stable personality; minimal change in core traits. Relationship adjustment is more realistic goal than personality change.

VIGNETTE 9: The Man Who Drinks to Feel Something

Presentation:

Deepak, 36 years old, presents with alcohol dependence. He reports drinking 12–15 units daily for the past 3 years. On detailed psychiatric history, he describes lifelong chronic emptiness, recurrent self-harm (burning forearms with cigarettes, described as "the only

thing that makes me feel real"), intense unstable relationships ("my ex called me exhausting"), impulsivity (gambling, unsafe sex), and rapid mood shifts. He has been admitted to hospital twice for overdose, both following relationship breakups. He describes his identity as "a void." He uses alcohol primarily "to feel less empty."

Diagnostic Analysis

Dual Diagnosis: Borderline Personality Disorder (BPD) + Alcohol Use Disorder (Severe)

BPD criteria met: Emptiness, self-harm, identity disturbance, impulsivity, affective instability, unstable relationships, abandonment-triggered overdoses.

Alcohol Use Disorder: ≥ 3 of 11 criteria clearly met (tolerance, withdrawal, time spent, given up activities, continued despite harm).

Mechanism of comorbidity:

- Self-medication: Alcohol temporarily reduces the chronic emptiness of BPD
- Shared neurobiological substrate: Impulsivity as shared vulnerability
- SUD worsens BPD: Removes inhibitions → more self-harm; more impulsive acts; pharmacological mood destabilisation

EXAM PEARL

BPD + SUD is the highest-risk BPD comorbidity. Suicide risk is multiplicative not additive.

Management Outline

Immediate:

- Detoxification: If physically dependent, chlordiazepoxide reducing regimen (inpatient or community depending on severity / risk)
- Safety assessment: HIGH risk, two previous overdoses; active self-harm; SUD
- Safety plan: Crisis contacts; means restriction; identify triggers

Integrated Treatment (Simultaneous, Not Sequential):

- **DBT-S (DBT for Substance Abusers, Linehan):**
- Dialectical Abstinence: Absolute commitment to abstinence + radical acceptance of lapses
- Path to Clear Mind: Burning bridges (remove access); alternate rebellion; urge surfing
- Standard DBT skills: Distress tolerance (replaces alcohol function); emotion regulation
- Individual + group + phone coaching
- **AA / Smart Recovery:** Adjunctive community support

- Schema Therapy: When SUD stabilised; address Emotional Deprivation, Abandonment schemas; build Healthy Adult mode

Pharmacotherapy:

- **Naltrexone 50 mg daily:** Reduces alcohol craving; may also reduce self-harm urge (opioid system hypothesis)
- **Acamprosate:** Anti-craving; GABAergic; use during abstinence maintenance
- **Valproate:** If affective instability remains a primary risk factor
- Avoid benzodiazepines after detox

Prognosis: Significantly worse than BPD alone. SUD is strongest predictor of non-remission (McLean Study). Integrated treatment essential. Realistic goal: reduce self-harm frequency; achieve periods of abstinence; build coping repertoire.

VIGNETTE 10: The Therapist's Dilemma

Presentation:

Kavitha, 45 years old, a secondary school teacher, is referred from the general medicine ward following her third hospital admission for unexplained physical complaints. She is described as "dramatic" by ward staff. She presents wearing elaborate jewellery, has styled her hair carefully for the interview, and immediately engages the (male) interviewer with intense eye contact and laughter. She speaks emotionally but the content is imprecise, "everything fell apart but I can't explain exactly how." She has had multiple brief intense friendships that "always end with betrayal." She regards her GP as "the most wonderful doctor I've ever met" while describing the ward nurse as "completely incompetent." She has never been in a stable long-term relationship.

Diagnostic Analysis

Primary Diagnosis: Histrionic Personality Disorder (DSM-5)

DSM-5 criteria (≥ 5 of 8 required):

1. Uncomfortable not being centre of attention ✓
2. Inappropriate sexually provocative or seductive behaviour (intense engagement with male interviewer) ✓
3. Rapidly shifting, shallow emotions ✓
4. Uses physical appearance to draw attention (elaborate styling, jewellery) ✓
5. Impressionistic speech lacking in detail ✓
6. Theatrical, dramatic emotional expression ✓
7. Considers relationships more intimate than they are (brief friendships = "betrayal") ✓

7/8 criteria met.

Secondary features: Splitting (GP idealised; nurse devalued), note this occurs in HPD but differs qualitatively from BPD splitting.

Differential:

DIAGNOSIS	REASON TO CONSIDER	WHY NOT PRIMARY
BPD	Splitting; intense relationships; affective instability	BPD: Identity instability core; self-harm; abandonment terror; HPD has stable (if dramatic) identity and no self-harm
Narcissistic PD	Attention-seeking; sense of special relationships	NPD: Grandiosity and entitlement primary; HPD's attention-seeking is for connection not superiority
Somatic Symptom Disorder	Multiple unexplained physical complaints	SSD can co-occur; HPD overreporting may amplify somatic focus
Cyclothymia	Rapid emotional shifts	No episodic pattern; shifts are within-day; not sustained mood states

Management Outline

Psychological:

- **Psychodynamic/psychoanalytic therapy:** Address developmental needs for attention and love; hysterical defences
- **CBT:** Identify attention-seeking patterns; challenge "if I am not the centre, I am invisible" beliefs
- **Schema Therapy:** Emotional Deprivation schema (did not receive genuine love independent of performance); Approval-Seeking schema

Therapeutic challenge: Managing the therapeutic relationship, patient may idealise, become seductive, or feel "betrayed" when boundaries maintained. Consistent, boundaried, warm therapeutic stance required.

Pharmacotherapy:

- SSRIs for comorbid anxiety or depressive episodes
- No specific pharmacotherapy for HPD

Medical: Co-management of somatic symptoms; liaison psychiatry; avoid iatrogenic harm from repeated investigations

Prognosis: Moderate. Less studied than BPD; less RCT evidence. Anecdotally more treatment-responsive than Cluster B on average given ego-dystonic distress from relationship failures.

VIGNETTE 11: The Perfectionist Barrister

Presentation:

Nikhil, 38 years old, is referred by his GP for evaluation of "burnout." He is a barrister who has not taken time off in 11 years. He works 70-hour weeks. He re-reads every brief 4–5 times before submission even when it is objectively complete. He cannot delegate to his junior colleagues because "they won't do it right." He describes spending two hours last week re-writing a two-sentence email because "the wording wasn't precise enough." He keeps every document he has ever worked on, four filing cabinets and two external drives. He becomes visibly irritated when describing his colleagues' "sloppiness." He has been married once, briefly, his wife said she felt "like a task on his to-do list." He does not believe he has a problem; the problem, he says, is the mediocrity around him.

Diagnostic Analysis

Primary Diagnosis: OCPD with possible burnout as presenting complaint

This vignette is identical in structure to Vignette 5 by design, to illustrate how OCPD presents across different professional contexts. The diagnosis and management framework is the same.

Additional note on "burnout" presentation:

- OCPD patients frequently present with burnout because Unrelenting Standards means they never stop
- The burnout is ego-dystonic (they don't want to be tired) while the traits are ego-syntonic
- This creates a genuine treatment opening: "I want to work at this level but I'm exhausted" → can be leveraged into schema work on Unrelenting Standards

Key differentials to note here specifically:

- **Workaholism without OCPD:** Some individuals work long hours by choice, without rigidity or relationship impairment, OCPD requires pervasive rigidity and interpersonal impact
 - **OCD:** Does he have ego-dystonic intrusive thoughts driving the re-reading? If the re-reading is about not being good enough (not about contamination or harm), it is OCPD not OCD
-

VIGNETTE 12: The Soldier's Shadow

Presentation:

Arjun, 41 years old, is referred from a veteran support service. He served in a conflict zone for 3 years and was medically discharged 5 years ago. He presents with a fractured identity, "I don't know who I am outside the army." He has severe difficulty regulating emotions, "I can be calm and then erupt in 30 seconds." He has no close relationships. He describes pervasive shame and self-loathing that predates his military service ("I always felt like something was wrong with me, even as a kid"). He reexperiences combat scenes involuntarily. He avoids anything that reminds him of the conflict. He is hypervigilant. He drinks 20 units per week and has punched walls on three occasions.

Diagnostic Analysis

Complex Presentation: Possible Complex PTSD (ICD-11) vs BPD vs PTSD + Personality Pathology

This vignette is deliberately complex to illustrate the ICD-11 Complex PTSD vs BPD differential.

Evidence FOR Complex PTSD (ICD-11):

- Core PTSD cluster: Reexperiencing (intrusive memories), avoidance (combat reminders), hyperarousal (hypervigilance) ✓
- Disturbances in self-organisation (DSO): Affect dysregulation ✓, negative self-concept (shame, self-loathing) ✓, relational difficulties ✓
- Definite severe traumatic exposure ✓

Evidence FOR BPD:

- Identity disturbance predating trauma ("always felt wrong with me, even as a kid")
- Impulsive aggression
- Alcohol use as affect regulation

ICD-11 Guidance:

- If reexperiencing + avoidance + hyperarousal are present AND DSO is present AND trauma explains the full picture → **Complex PTSD**
- If identity instability, abandonment fears, splitting, and self-harm pattern predate trauma → **BPD**
- If both → **comorbid diagnosis possible**

In this vignette: Pre-existing negative self-concept and identity instability (predates army) suggests BPD features present alongside trauma-induced PTSD. Most likely: **PTSD + underlying personality pathology (BPD features)**, or Complex PTSD with pre-existing vulnerability.

EXAM PEARL

ICD-11 introduced Complex PTSD as a distinct diagnosis. It is NOT the same as BPD. The key distinguishing feature: Complex PTSD requires core PTSD symptoms (reexperiencing, avoidance, hyperarousal), BPD does not. When both are present, careful longitudinal assessment needed to determine what predated the trauma.

Management Outline (Complex PTSD / Comorbid BPD + PTSD)

Phase 1, Safety, Stabilisation, Skill-Building:

- DBT skills for affect regulation and distress tolerance (TIPP, Radical Acceptance)
- Address alcohol use: Brief intervention; consider naltrexone; SUD treatment if needed
- Safety planning: Aggression risk (wall-punching), suicide risk assessment

Phase 2, Trauma Processing (when stabilised):

- DBT-PTSD (Harned / Linehan): Prolonged Exposure modified for BPD
- EMDR with emotion regulation modifications
- Imagery rescripting (Schema Therapy framework)
- Veteran-specific group: Peer support; normalise trauma response

Phase 3, Integration:

- Identity reconstruction: Who am I outside military identity?
- Schema Therapy: Address Defectiveness/Shame, Emotional Deprivation schemas
- Vocational support and meaning-making

Pharmacotherapy:

- SSRI (sertraline or paroxetine, NICE-recommended for PTSD)
- Prazosin for nightmares if present
- Valproate for impulsive aggression
- Address alcohol use disorder (naltrexone, acamprosate)

Prognosis: Guarded. Complex presentations with comorbid SUD and possible personality pathology require sustained multimodal treatment. Improvements achievable, particularly in PTSD symptoms, over 2–4 years.

GUIDE 11 · PART II

Other Clinical Syndromes

Paper II · Clinical Psychiatry. Six study modes, from notes to quick review.

Study Notes

Sources: Kaplan & Sadock’s Comprehensive Textbook of Psychiatry (11th ed.), Stahl’s Essential Psychopharmacology (5th ed.), Oxford Textbook of Psychiatry, Fairburn CBT-E Manual, ICD-11 Clinical Descriptions and Diagnostic Guidelines, DSM-5-TR

PART 1: DISSOCIATIVE DISORDERS

1.1 Overview and Conceptual Framework

Dissociation is a disruption in the normally integrated functions of consciousness, memory, identity, emotion, perception, behavior, and sense of self. The term was coined by Pierre Janet in the late 19th century, who viewed it as a failure of psychological synthesis under stress.

EXAM PEARL

Dissociation exists on a spectrum from normal (highway hypnosis, absorption in a book) to pathological (DID, dissociative amnesia). The continuum model is clinically important.

Key Theoretical Models:

MODEL	CORE CLAIM	KEY PROPONENTS
Trauma Model	Dissociation is a defensive response to overwhelming trauma	van der Kolk, Putnam, Ross
Sociocognitive Model	DID is a socially constructed phenomenon, shaped by cultural expectations and iatrogenesis	Spanos, Lilienfeld
Structural Dissociation	Personality splits into Apparently Normal Part (ANP) and Emotional Part (EP); EP holds trauma	van der Hart, Nijenhuis
Neurobiology	Prefrontal inhibition of limbic activation; altered connectivity in ACC, hippocampus	Lanius, Reinders

CLINICAL ANCHOR

The trauma model and sociocognitive model are not mutually exclusive. Cultural and therapeutic context shapes symptom expression in genuinely traumatized individuals.

1.2 Dissociative Amnesia

Definition: An inability to recall important autobiographical information, usually of a traumatic or stressful nature, that is inconsistent with ordinary forgetting.

DSM-5 Criteria:

- Inability to recall important autobiographical information (usually traumatic/stressful)
- Not due to another disorder, substance, or neurological condition
- Causes clinically significant distress or functional impairment

ICD-11 Code: 6B61

Subtypes:

SUBTYPE · FEATURES

Localized Cannot recall events within a specific time period (most common)

Selective Can recall some but not all events within a specific time period

Generalized Complete loss of identity and life history (rare)

Dissociative Fugue Travel + amnesia (now a specifier in DSM-5, separate in ICD-11)

Key clinical features:

- Onset often acute, linked to trauma or severe stress
- Gap in memory is ego-alien, patient is distressed by it
- La belle indifference may be present (not universal)
- Spontaneous recovery common; may be triggered by removal from stressor

Differential Diagnosis:

- Transient Global Amnesia (TGA): sudden, complete, brief (hours), no trauma link, seen in middle-aged/elderly, EEG and neuroimaging normal but distinct clinical course
- Organic amnesia: anterograde > retrograde, consistent neurological findings
- Malingering: secondary gain prominent, inconsistent memory performance on testing

Management:

- Psychoeducation and establishing safety
- Trauma-focused therapy (EMDR, trauma-focused CBT) once stabilized

- Narcoanalysis (amobarbital interview), historical interest; not evidence-based, not recommended in current guidelines
 - Hypnosis: adjunctive; evidence limited
 - SSRI for comorbid PTSD / depression
-

1.3 Dissociative Fugue

Definition: Sudden, unexpected travel away from home or customary place of work, with inability to recall some or all of one's past, sometimes including confusion about personal identity or adoption of a new identity.

ICD-11: Listed separately as 6B61.0 (Dissociative amnesia with dissociative fugue)

DSM-5: Now a specifier of Dissociative Amnesia, not a separate diagnosis

EXAM PEARL

The DSM-5 reclassification of fugue as a specifier (rather than separate disorder) is a commonly tested change. ICD-11 retains some separation.

Clinical features:

- Duration: hours to months
- Purposeful travel; patient often appears normal to observers
- New identity may be adopted (partial or complete)
- Recovery: usually abrupt; retrograde amnesia for fugue period persists
- May surface during wartime, natural disasters, or severe personal crisis

Differential: Complex partial seizures, TGA, malingering, alcohol-related blackouts

1.4 Dissociative Identity Disorder (DID)

Definition: Disruption of identity characterized by two or more distinct personality states or "alters," each with its own relatively enduring pattern of perceiving, relating to, and thinking about the environment and self.

DSM-5 Criteria:

1. Two or more distinct personality states
2. Recurrent amnesia between alters
3. Significant distress or impairment
4. Not due to cultural / religious practice
5. Not due to substance or medical condition

ICD-11 Code: 6B64, notable changes (see Classification section)

Epidemiology:

- Prevalence: 1-3% general population (controversial; much higher in trauma-exposed clinical samples)
- Female:Male ratio: approximately 9:1 in clinical settings (trauma reporting bias)
- Average number of alters: 13-15
- 97-98% report childhood trauma history

Alter system:

- Host personality: the one usually presenting
- Child alters: often carry traumatic memories
- Persecutory alters: internalized abuser
- Protector alters: shield other alters
- "Apparently normal part" (ANP) functions in daily life

Neurobiology:

- Different alters show different psychophysiological responses (EEG, HR, skin conductance)
- Neuroimaging: differences in regional brain activity between alter states
- Positron emission tomography studies: different memory patterns for different alters

Controversies:

- Iatrogenic creation through suggestive therapy (sociocognitive critique)
- Cross-cultural prevalence variation (higher in North America than Europe, cultural scripting argument)
- High false-positive rates in some clinical settings
- Forensic implications (criminal responsibility)

EXAM PEARL

For PG exams questions, know both trauma model arguments and sociocognitive critiques. Don't commit fully to either, state both. It demonstrates clinical sophistication.

Management of DID:

PHASE	FOCUS	TECHNIQUES
Phase 1: Safety	Stabilization, coping skills, safety planning	Grounding, emotion regulation, psychoeducation
Phase 2: Trauma Processing	Careful engagement with traumatic memories	EMDR, trauma-focused CBT, ego state therapy

PHASE	FOCUS	TECHNIQUES
Phase 3: Integration	Fusion of alters or functional integration	Ongoing individual therapy, relapse prevention

- Pharmacotherapy: no specific agent; treat comorbid PTSD, depression, anxiety
- Avoid rapid uncovering; premature trauma processing destabilizes

1.5 Depersonalization/Derealization Disorder (DPDR)

Definition:

- **Depersonalization:** Feeling detached or estranged from one's own mental processes or body (observer of own thoughts, feelings, sensations)
- **Derealization:** Feeling detached from one's surroundings (dreamlike, unreal, foggy, visually distorted)

DSM-5 Code: 300.6 | **ICD-11 Code:** 6B66

Key distinguishing feature: Reality testing remains intact (unlike psychosis)

Epidemiology:

- Brief transient episodes: up to 74% of general population
- Persistent DPDR: 1-2% prevalence
- Peak onset: adolescence to early adulthood
- Often triggered by stress, sleep deprivation, drugs (cannabis, hallucinogens)

Neurobiology:

- Reduced activation of limbic structures (emotional numbing)
- Increased prefrontal inhibition of emotional processing
- Serotonin and GABA system involvement

Clinical features:

- Emotional numbing and anhedonia often prominent
- Anxiety about the symptoms themselves (secondary anxiety)
- Chronic, waxing/waning course
- Often comorbid with anxiety disorders, depression, trauma history

Management:

- CBT (Feeling Real technique, Hunter and Phillips approach)
- Lamotrigine: some evidence
- SSRI for comorbid anxiety/depression
- Naltrexone: some open-label data

- Psychoeducation reduces distress significantly

1.6 Dissociative Motor and Sensory Disorders (Functional Neurological Symptom Disorder)

ICD-11 Classification: Moved to Diseases of the Nervous System (not Dissociative Disorders)

DSM-5: Functional Neurological Symptom Disorder (Conversion Disorder), 300.11

EXAM PEARL

ICD-11 removed conversion disorder from dissociative disorders chapter. DSM-5 retains it as "Functional Neurological Symptom Disorder." This reclassification is a tested distinction.

Motor symptoms:

- Functional weakness/paralysis
- Functional tremor
- Functional gait disorder (astasia-abasia)
- Functional speech disorder (aphonia)

Sensory symptoms:

- Functional sensory loss (does not follow dermatomal/peripheral nerve pattern)
- Functional visual disturbance

Key signs supporting functional diagnosis:

- Hoover sign (hip extension weakness normalizes with contralateral hip flexion)
- Tremor variability with distraction
- Tubular visual field defect (tunnel vision same regardless of distance, not anatomically possible)
- Splitting of midline vibration sense

Neurobiology:

- Not fabrication, there is demonstrable motor cortex inhibition
- Altered connectivity between prefrontal areas and motor/somatosensory cortex
- Functional MRI shows real changes in neural processing

Management:

- Explanation and psychoeducation (using the "software not hardware" framework)
- Physiotherapy (for motor symptoms)
- CBT
- Multidisciplinary approach

- Treating comorbid anxiety, depression, PTSD

1.7 Possession/Trance Disorders

ICD-11: Trance disorder (6B63) and Possession trance disorder (6B63.0), distinguished

FEATURE	TRANCE DISORDER	POSSESSION TRANCE DISORDER
Identity alteration	Partial, narrowed awareness	Complete, replaced by spirit/entity
Cultural context	May or may not fit cultural norms	May fit cultural norms (non-pathological)
Distress/impairment	Present	Present (distinguishes from cultural practice)
Volition	Involuntary	Involuntary

EXAM PEARL

ICD-11 explicitly notes that possession trance occurring in religious or cultural contexts, without distress or impairment, is NOT a mental disorder. This is a key criterion that distinguishes pathological from normal cultural practice.

Differential from religious practice:

- Pathological: ego-dystonic, causes impairment, occurs outside cultural context, patient seeks help
- Cultural practice: ego-syntonic, bounded by ritual, no impairment between episodes

1.8 ICD-11 vs DSM-5: Dissociative Disorders

FEATURE	ICD-11	DSM-5
Chapter placement	6B6x, Dissociative Disorders	Chapter 8, Dissociative Disorders
Conversion Disorder	Removed to Diseases of Nervous System	Functional Neurological Symptom Disorder (retained)
Fugue	Specifier of Dissociative Amnesia	Specifier of Dissociative Amnesia
DID	Partial DID added as a new category	Not present, only full DID
Possession/Trance	Two separate categories	Combined in "Other Specified"
DPDR	6B66	300.6

FEATURE	ICD-11	DSM-5
Cultural sensitivity	Explicit criterion re: cultural context for possession	Cultural context noted

PART 2: EATING DISORDERS

2.1 Anorexia Nervosa (AN)

Definition: Persistent restriction of energy intake, intense fear of weight gain, and disturbance in how one's weight or shape is experienced.

DSM-5 Criteria:

1. Restriction of energy intake → significantly low body weight
2. Intense fear of gaining weight (or behavior that interferes with weight gain)
3. Disturbance in body weight / shape experience, undue influence on self-evaluation, or persistent lack of recognition of seriousness of low body weight

ICD-11 Code: 6B80

Subtypes:

SUBTYPE · FEATURES

Restricting type Weight loss via dieting, fasting, excessive exercise

Binge-purge type Recurrent binge eating or purging (induced vomiting, laxatives) during last 3 months

Epidemiology:

- Lifetime prevalence: ~0.5-1% females, 0.1-0.3% males
- Female:Male ratio: 10:1
- Age of onset: bimodal peaks at 14 and 18 years
- Highest mortality of any psychiatric disorder (5-6% per decade; SMR ~5.86)
- Mortality: medical complications AND suicide

BMI Severity Specifiers (DSM-5):

SEVERITY	ADULT BMI	PEDIATRIC BMI EQUIVALENT
Mild	17-18.49	85th-<100th percentile for age/sex
Moderate	16-16.99	75th-<85th percentile
Severe	15-15.99	3rd-<10th percentile
Extreme	<15	<3rd percentile

EXAM PEARL

BMI thresholds for AN diagnosis were removed from DSM-5. There is no longer a minimum BMI required. The focus is on "significantly low weight in the context of what is expected for developmental stage." This is important, don't say "BMI <17.5" as a criterion. That was ICD-10.

ICD-11 Changes for AN:

- Removed BMI <17.5 as hard criterion
- Introduced weight-for-age concept for children/adolescents
- Added "significantly low body weight" language matching DSM-5

2.1.1 MEDICAL COMPLICATIONS OF ANOREXIA NERVOSA

EXAM PEARL

Medical complications of AN are extremely high-yield. Organize by system.

SYSTEM · COMPLICATION

Cardiovascular Bradycardia, hypotension, QTc prolongation, mitral valve prolapse, arrhythmias, cardiac atrophy

Metabolic Hypoglycemia, hypokalaemia, hyponatraemia, hypophosphatemia, metabolic alkalosis (if purging) or acidosis

Endocrine Amenorrhoea, low estrogen/testosterone, low LH/FSH, high cortisol, sick euthyroid (low T3, normal/low T4), growth retardation in adolescents

Renal Prerenal azotemia, renal calculi, hypokalemic nephropathy

GI Constipation, delayed gastric emptying, superior mesenteric artery syndrome, elevated liver enzymes

Hematological Anemia, leukopenia, thrombocytopenia (hypocellular bone marrow)

Dermatological Lanugo hair, xerosis, carotenemia (orange-yellow skin from eating carrots), Russell's sign (calluses on knuckles)

Musculoskeletal Osteopenia, osteoporosis, stress fractures

Neurological Cortical atrophy, peripheral neuropathy, seizures (due to hypoglycemia/electrolyte disturbance)

Dental Perimolysis (enamel erosion from acid vomiting), in binge-purge subtype

CLINICAL ANCHOR

Lanugo hair, bradycardia, and hypothermia form a clinical triad that should immediately raise suspicion for AN.

2.1.2 REFEEDING SYNDROME

Definition: Potentially fatal metabolic complications occurring during nutritional rehabilitation after a period of prolonged malnutrition or starvation.

Pathophysiology:

- Malnutrition → whole-body depletion of phosphate (intracellular), despite normal serum levels
- Refeeding (especially with carbohydrates) → insulin surge
- Insulin drives glucose, phosphate, potassium, magnesium INTO cells
- Rapid drop in serum phosphate, potassium, magnesium

NICE Criteria for High Refeeding Risk:

- BMI <16
- Unintentional weight loss >15% in 3-6 months
- Little or no nutritional intake >10 days
- Low serum potassium, phosphate, or magnesium before feeding

Clinical Consequences of Refeeding Syndrome:

ELECTROLYTE DISTURBANCE · CONSEQUENCE

Hypophosphatemia Respiratory failure, heart failure, haemolytic anaemia, rhabdomyolysis, seizures, delirium

Hypokalaemia Arrhythmias, cardiac arrest, muscle weakness

Hypomagnesaemia Tetany, arrhythmias, seizures

Hyperglycemia From carbohydrate loading; Wernicke's encephalopathy risk if B1 not given

Thiamine deficiency Wernicke's encephalopathy, Korsakoff syndrome

Prevention:

- Start at 20 kcal/kg/day (or 10 kcal/kg if very high risk)
- Increase slowly over 4-7 days
- Monitor electrolytes every 12-24 hours initially
- **Thiamine (B1) supplementation BEFORE and during refeeding**, oral or IV
- Correct electrolytes before initiating feeding

EXAM PEARL

Thiamine before glucose. If you give glucose first to a thiamine-depleted patient, you precipitate Wernicke's. "Thiamine before carbs" is the mnemonic anchor.

MARSIPAN Guidelines (Management of Really Sick Patients with Anorexia Nervosa):

- Provides criteria for medical admission
- Establishes monitoring protocols
- Multidisciplinary approach mandatory

2.2 Bulimia Nervosa (BN)

Definition: Recurrent episodes of binge eating followed by compensatory behaviors, with self-evaluation unduly influenced by body shape and weight.

DSM-5 Criteria:

1. Recurrent binge eating: eating a large amount in a discrete period + sense of lack of control
2. Recurrent inappropriate compensatory behaviors (vomiting, laxatives, diuretics, fasting, excessive exercise)
3. Occurs at least once per week for 3 months
4. Self-evaluation unduly influenced by body shape/weight
5. Does not occur exclusively during AN

ICD-11 Code: 6B81

Severity specifiers (by compensatory behavior frequency):

- Mild: 1-3 episodes/week
- Moderate: 4-7 episodes/week
- Severe: 8-13 episodes/week
- Extreme: >14 episodes/week

Medical complications specific to BN:

FEATURE · COMPLICATION

Vomiting Parotid enlargement (chipmunk face), perimolysis, Mallory-Weiss tears, aspiration, metabolic alkalosis, hypokalemia

Laxative abuse Metabolic acidosis, hypokalemia, edema (reflex), melanosis coli

Ipecac abuse Cardiomyopathy (emetine toxicity)

Physical examination Russell's sign (calluses on dorsum of hand from self-induced vomiting)

EXAM PEARL

Russell's sign = calluses on knuckles from inducing vomiting. Described in BN, can also appear in binge-purge AN. Not a finding in restricting AN.

Pharmacotherapy:

- **Fluoxetine 60 mg/day**, ONLY FDA-approved drug for BN (note: higher than antidepressant dose)
- Effective for binge-purge cycles and comorbid depression
- TCAs (imipramine, desipramine): some evidence but side-effect profile limits use
- MAOIs: effective but dangerous; not used
- Topiramate: reduces binge eating but significant cognitive side effects
- **Bupropion is CONTRAINDICATED in BN**, lowers seizure threshold; electrolyte disturbances in BN further increase seizure risk

EXAM PEARL

Bupropion is contraindicated in bulimia. This is a classic exam trap. Also avoid bupropion in anorexia for the same reason.

2.3 Binge Eating Disorder (BED)

Definition: Recurrent episodes of binge eating without regular compensatory behaviors.

DSM-5 Criteria:

1. Recurrent binge eating (large amount + loss of control)
2. Associated with 3 or more of: eating faster than normal, eating until uncomfortably full, eating large amounts when not physically hungry, eating alone due to embarrassment, feeling disgust/depressed/guilty afterward
3. Marked distress
4. Occurs at least once/week for 3 months
5. NOT associated with regular compensatory behaviors (distinguishes from BN)

ICD-11 Code: 6B82

EXAM PEARL

BED was first formally recognized as a separate diagnosis in DSM-5 (2013). Previously it was in the DSM-IV Appendix as a proposed category. ICD-11 also includes it as a full diagnosis, another ICD-10 to ICD-11 change worth knowing.

Epidemiology:

- Most common eating disorder: ~3.5% in women, ~2% in men
- More equal sex distribution than AN or BN
- Strong association with obesity, but not all individuals with BED are obese
- High comorbidity with mood disorders, anxiety disorders

Treatment:

- CBT-E (enhanced CBT): first-line
- IPT: comparable efficacy
- DBT: adapted for BED
- Lisdexamfetamine (Vyvanse): FDA-approved for moderate-to-severe BED; reduces binge frequency
- Topiramate: reduces binge frequency; weight loss effect
- SSRIs: modest effect on binge frequency

2.4 ARFID, Pica, and Rumination Disorder

AVOIDANT/RESTRICTIVE FOOD INTAKE DISORDER (ARFID)

ICD-11: Avoidant-restrictive food intake disorder (6B83)

DSM-5 Criteria:

- Restriction of food intake due to apparent lack of interest, avoidance based on sensory characteristics, concern about aversive consequences (choking, vomiting)
- Weight loss/growth faltering OR nutritional deficiency OR dependence on enteral feeding OR significant interference with psychosocial functioning
- NOT explained by food unavailability, cultural practice, AN, or BN
- NOT explained by medical condition or other mental disorder

EXAM PEARL

ARFID is not about body image (distinguishes from AN) and is not culturally determined. It often presents in children with ASD, anxiety disorders, or a history of aversive feeding experiences.

Key differentiators:

FEATURE	AN	ARFID
Body image disturbance	Present (central)	Absent
Fear of weight gain	Present	Absent
Food restriction	Yes	Yes
Motivation	Weight/ shape control	Sensory, interest, fear of consequences
Age of onset	Typically adolescence	Typically early childhood
ASD comorbidity	Low	High

PICA

Definition: Persistent eating of non-nutritive, non-food substances for at least 1 month.

ICD-11 Code: 6B84

Common substances: Earth/ clay (geophagia), ice (pagophagia, associated with iron deficiency anemia), starch (amylophagia), chalk, paper, hair (trichophagia)

Associations: Pregnancy, iron deficiency, intellectual disability, ASD, neglect/ deprivation

Medical complications: Lead poisoning, intestinal obstruction, parasitic infection, dental damage

RUMINATION DISORDER

Definition: Repeated regurgitation of food, re-chewing, re-swallowing, or spitting out, for at least 1 month.

ICD-11 Code: 6B85

- NOT due to GERD, pyloric stenosis, or another GI condition
- NOT occurring exclusively in AN/BN
- Typically effortless (not forceful vomiting); occurs within minutes of eating
- Most common in infants (3-12 months), but also in adults
- Treatment: diaphragmatic breathing, behavioral interventions, habit reversal

2.5 Body Image Disturbance

Relevant across:

- AN: overestimation of body size, shape dissatisfaction as core psychopathology
- BN: shape and weight overvalued in self-evaluation
- Body Dysmorphic Disorder (BDD): preoccupation with perceived defect (not food-related)

Assessment tools:

- Body Shape Questionnaire (BSQ)
- EDE-Q (Eating Disorder Examination Questionnaire)
- EDI-3 (Eating Disorder Inventory-3)
- Clinical interview: "When you look in the mirror, what do you see?"

2.6 CBT-E (Enhanced Cognitive Behaviour Therapy): Fairburn

EXAM PEARL

CBT-E is the current gold standard psychological treatment for eating disorders (all types). Developed by Christopher Fairburn at Oxford.

Theoretical model:

Transdiagnostic model: eating disorders share a common psychopathology centered on overconcern with eating, shape, and weight (the "core psychopathology").

Four maintaining mechanisms:

1. Clinical perfectionism
2. Low self-esteem
3. Interpersonal difficulties
4. Mood intolerance

Phases of CBT-E:

PHASE	SESSIONS	FOCUS
Phase 1	1-7	Engagement, formulation, behavioral changes, psychoeducation
Phase 2	8-9	Review progress, address obstacles
Phase 3	10-17	Address core maintaining mechanisms
Phase 4	18-20	Ensure long-term maintenance, relapse prevention

- Intensive version for complex cases: 40 sessions
- Works across AN, BN, BED (transdiagnostic)
- Outpatient format; inpatient version also developed

Components specific to AN:

- Weight restoration phase integrated with psychological treatment

- "Collaborative", therapist and patient review together
 - Education about effects of starvation on thinking (starvation syndrome)
-

2.7 Nutritional Rehabilitation in AN

Goals:

- Target weight: typically BMI 20-25 (for adults); return to growth curve for adolescents
- Adequate nutrition for growth/bone density restoration
- Menstrual recovery: usually when weight >90% of expected weight for height
- Rate of weight gain: 0.5-1 kg/week (outpatient) to 1-1.5 kg/week (inpatient)

Caloric targets:

- Start: 1000-1500 kcal/day (lower if high refeeding risk)
- Target: 3000-4000 kcal/day for weight restoration
- Avoid high-calorie density foods initially; balanced macronutrient distribution

Oral vs Nasogastric vs Parenteral:

- Oral preferred
 - NG feeding for patients who cannot eat enough voluntarily (including involuntary treatment)
 - Parenteral (IV) nutrition: only if GI tract non-functional; carries infection risk
-

2.8 ICD-11 Classification of Eating Disorders

ICD-11 CODE · DISORDER

6B80 Anorexia nervosa

6B81 Bulimia nervosa

6B82 Binge eating disorder (NEW, was not in ICD-10)

6B83 Avoidant-restrictive food intake disorder (NEW)

6B84 Pica in childhood

6B85 Rumination-regurgitation disorder

6B8Z Other specified eating or feeding disorders

EXAM PEARL

The major ICD-11 additions are BED (as a full diagnosis) and ARFID. Both were in DSM-5 before ICD-11 adopted them.

PART 3: SLEEP DISORDERS

3.1 Normal Sleep Architecture

Sleep Stages:

STAGE	EEG	DURATION	FEATURES
N1 (NREM)	Theta (4-7 Hz), some alpha	5-10 min	Light sleep, hypnic jerks, easily awakened
N2 (NREM)	Sleep spindles (12-14 Hz), K-complexes	20-30 min	Active sleep protection
N3 (NREM), Slow Wave Sleep	Delta (<2 Hz, >75 μ V)	20-40 min	Deepest sleep; growth hormone secreted
REM	Low-voltage mixed frequency (resembles wake)	10-20 min initially	Dreams, muscle atonia, penile tumescence, rapid eye movements

EXAM PEARL

The first REM period occurs approximately 90 minutes after sleep onset. REM latency is shortened in depression (key exam fact). As night progresses: NREM dominates early, REM dominates late.

Sleep cycle:

- 4-6 complete cycles per night
- Cycle length: ~90 minutes
- N3 (slow wave) predominates in first third of night
- REM predominates in last third of night

Neurotransmitters in sleep:

NEUROTRANSMITTER · ROLE

Adenosine Sleep drive (homeostatic); caffeine blocks adenosine receptors

Serotonin Promotes NREM (raphe nuclei)

Noradrenaline Wake-promoting (locus coeruleus)

Acetylcholine Critical for REM (pedunculopontine nucleus)

Orexin/Hypocretin Wake-promoting; loss → narcolepsy type 1

GABA Sleep-promoting; target of benzodiazepines and Z-drugs

Histamine Wake-promoting (tuberomammillary nucleus); antihistamines cause sedation

3.2 Insomnia

Definition: Difficulty initiating or maintaining sleep, or early morning awakening, occurring despite adequate opportunity and circumstances for sleep, associated with daytime impairment.

Classification:

TYPE · DURATION

Acute (short-term) <3 months

Chronic ≥3 months, ≥3 nights / week

ICD-11 Code: 7A00 (Chronic insomnia disorder)

Spielman's 3P Model:

FACTOR	DESCRIPTION	EXAMPLES
Predisposing	Biological/ psychological vulnerability	Hyperarousability, anxiety traits, female sex
Precipitating	Trigger events	Loss, illness, work stress
Perpetuating	Behaviors that maintain insomnia	Extended time in bed, daytime napping, caffeine, worry about sleep

CLINICAL ANCHOR

The 3P model is the conceptual backbone of CBT-I. Treatment targets the perpetuating factors.

Epidemiology:

- Chronic insomnia: 10-15% of adults
- More common in women, elderly, psychiatric illness, chronic medical illness
- Comorbid in >50% of psychiatric disorders

3.2.1 CBT-I (COGNITIVE BEHAVIOURAL THERAPY FOR INSOMNIA)

CBT-I is first-line treatment for chronic insomnia (over pharmacotherapy)

Components:

COMPONENT	MECHANISM	TECHNIQUE
Sleep restriction	Consolidates fragmented sleep	Limit time in bed to actual sleep time initially; gradually extend

COMPONENT	MECHANISM	TECHNIQUE
Stimulus control	Breaks bed-arousal association	Bed only for sleep and sex; leave bed if not asleep in 20 min
Sleep hygiene	Removes perpetuating factors	Consistent schedule, no caffeine after 2 pm, cool dark room
Relaxation	Reduces physiological hyperarousal	Progressive muscle relaxation, diaphragmatic breathing
Cognitive restructuring	Reduces sleep-related worry	Challenge catastrophic thoughts about consequences of poor sleep
Sleep compression	Alternative to restriction in elderly	Gradual reduction of time in bed

EXAM PEARL

Sleep restriction is the most potent single component of CBT-I. Sleep efficiency = (total sleep time / time in bed) × 100. Target >85%.

Sleep efficiency: Sleep efficiency (SE) = (Total Sleep Time / Time In Bed) × 100%

- SE <85% = insomnia
- SE >90% = acceptable consolidation

3.2.2 PHARMACOTHERAPY FOR INSOMNIA

DRUG CLASS	DRUG	MECHANISM	NOTES
Benzodiazepines	Temazepam, nitrazepam	GABA-A positive allosteric modulator	Short-term only; dependence risk; suppress N3
Z-drugs	Zolpidem, zopiclone, zaleplon	GABA-A (BZ1 subunit selective)	Better side-effect profile; still dependence risk; zaleplon shortest acting
Melatonin	Ramelteon, prolonged-release melatonin	MT1/MT2 agonist	Good for sleep onset, circadian issues; safe for elderly
Orexin antagonists	Suvorexant, lemborexant	Dual orexin receptor antagonist (DORA)	Novel mechanism; reduces wake drive; FDA approved
Low-dose doxepin	Doxepin 3-6 mg	H1 antagonist at low dose	Approved for sleep maintenance insomnia specifically

DRUG CLASS	DRUG	MECHANISM	NOTES
Sedating antidepressants	Mirtazapine, trazodone, amitriptyline	H1/5-HT2 blockade	Off-label; useful with comorbid depression

EXAM PEARL

Suvorexant is the first orexin receptor antagonist approved for insomnia. Blocks orexin from promoting wakefulness (unlike z-drugs which promote sleep). Mechanistically distinct.

3.3 Hypersomnolence and Narcolepsy

IDIOPATHIC HYPERSOMNIA

- Excessive daytime sleepiness (EDS) without cataplexy
- Prolonged sleep (>11 hours/day) or long unrefreshing naps
- Sleep drunkenness (severe difficulty awakening; sleep inertia)
- PSG: normal REM latency, reduced MSLT sleep latency (<8 min) but <2 SOREMPs
- Treatment: modafinil, stimulants

NARCOLEPSY

Tetrad of Narcolepsy (Gelineau's tetrad):

1. Excessive daytime sleepiness (EDS), obligate
2. Cataplexy, sudden loss of muscle tone triggered by emotion (laughter, surprise, anger)
3. Sleep paralysis, inability to move at sleep onset/offset
4. Hypnagogic/hypnopompic hallucinations, vivid hallucinations at sleep onset/offset

EXAM PEARL

Only EDS is required for narcolepsy diagnosis. Cataplexy, if present, is pathognomonic for Type 1. Not all narcolepsy has all 4 features.

FEATURE	TYPE 1 (WITH CATAPLEXY)	TYPE 2 (WITHOUT CATAPLEXY)
Cataplexy	Present	Absent
CSF hypocretin-1	<110 pg/mL (or <1/3 of normal controls)	Normal (>110 pg/mL or >1/3)
HLA-DQB1*06:02	>95% positive	~40% positive
MSLT: sleep latency	<8 min	<8 min
MSLT: SOREMPs	≥2 sleep-onset REM periods	≥2 sleep-onset REM periods
Pathophysiology	Loss of orexin neurons (autoimmune)	Unknown; orexin intact

FEATURE	TYPE 1 (WITH CATAPLEXY)	TYPE 2 (WITHOUT CATAPLEXY)
Prevalence	~0.05% (1 in 2000)	Less common

MSLT (Multiple Sleep Latency Test):

- 5 nap opportunities at 2-hour intervals
- Mean sleep latency <8 minutes = abnormally sleepy
- ≥2 SOREMPs = strongly suggests narcolepsy
- Done after overnight PSG (to exclude sleep apnea as cause of EDS)

Pathophysiology of Type 1:

- Selective loss of hypothalamic hypocretin-producing neurons (80-90% reduction)
- Autoimmune mechanism: HLA-DQB1*06:02 association; triggered by H1N1 influenza or Pandemrix vaccine
- Anti-TRIB2 antibodies found in some patients

Management of Narcolepsy:

SYMPTOM · TREATMENT

EDS Modafinil (first-line), armodafinil, amphetamines, methylphenidate

Cataplexy Sodium oxybate (GHB, most effective), venlafaxine, SSRIs (suppress REM → reduce cataplexy)

Sleep paralysis/hallucinations Sodium oxybate, SSRIs

All symptoms Sodium oxybate (gamma-hydroxybutyrate), consolidates nocturnal sleep, reduces EDS AND cataplexy

Non-pharmacological Scheduled naps (2-3 brief naps/ day), sleep hygiene, driving restrictions

EXAM PEARL

Sodium oxybate (sodium gamma-hydroxybutyrate, GHB) is the only drug effective for BOTH EDS and cataplexy. It is a GABA-B agonist, Schedule I controlled substance with tightly regulated prescribing due to abuse potential.

3.4 Obstructive Sleep Apnea (OSA)

Definition: Repetitive episodes of upper airway collapse during sleep, causing apnea/hypopnea, leading to intermittent hypoxia, arousals, and fragmented sleep.

Diagnosis:

- Apnea-Hypopnea Index (AHI): number of apneas + hypopneas per hour of sleep
- Mild: AHI 5-15
- Moderate: AHI 15-30
- Severe: AHI >30
- Diagnosed by overnight polysomnography (PSG) or home sleep testing

Psychiatric relevance:

- Associated with depression (bidirectional)
- Can cause or worsen cognitive impairment
- Treatment-resistant depression may have undiagnosed OSA
- OSA worsens in hypothyroidism, acromegaly

Treatment:

- CPAP (continuous positive airway pressure), first-line
- Mandibular advancement device, mild-moderate
- Weight loss, positional therapy, avoidance of alcohol/sedatives
- Surgery (UPPP) in selected cases

3.5 Parasomnias

Definition: Abnormal behavioral, experiential, or physiological events occurring during sleep or sleep-wake transitions.

NREM PARASOMNIAS (DISORDERS OF AROUSAL)

Common feature: Occur in N3 (slow wave sleep), predominantly in first third of night. Patient is partially aroused but not fully awake.

DISORDER	AGE	FEATURES	AMNESIA	MANAGEMENT
Sleepwalking (Somnambulism)	Children (peak 4-8 years)	Complex motor behaviors, glassy eyes, no responsiveness	Yes (complete)	Safety measures, clonazepam if needed
Sleep terrors (Night terrors)	Children (peak 5-7 years)	Sudden arousal with screaming, panic, tachycardia; autonomic hyperactivation	Yes (complete)	Reassurance; benzodiazepines if persistent
Confusional arousals	Any age	Confused behavior upon arousal from N3	Partial	Conservative

EXAM PEARL

Night terrors ≠ Nightmares. Night terrors occur in N3 (first third of night), with autonomic hyperactivation, no detailed recall. Nightmares occur in REM (last third of night), in late morning, with vivid detailed recall.

REM SLEEP BEHAVIOR DISORDER (RBD)

Definition: Acting out of vivid, action-filled dreams during REM sleep, due to failure of normal REM muscle atonia.

ICD-11 Code: 7A22

Key features:

- Usually middle-aged to elderly males
- Behaviors: punching, kicking, shouting, falling out of bed
- Often correlates with dream content ("defending against attack")
- Polysomnographic finding: REM sleep without atonia (RSWA)
- Full memory of dreams; wakes up oriented

EXAM PEARL

RBD is **STRONGLY** associated with alpha-synucleinopathies, it often precedes Parkinson's disease, Lewy body dementia, or MSA by 10-15 years. RBD is an early prodromal marker.

Management:

- Clonazepam (0.25-2 mg at bedtime), first-line; reduces behavior, does not restore atonia
- Melatonin (3-12 mg at bedtime), better tolerated in elderly; does partially restore atonia
- Bed safety: remove sharp objects, pad floor
- Treat alpha-synucleinopathy if present

OTHER PARASOMNIAS

DISORDER · FEATURES

Nightmare disorder Disturbing dreams with full awakening and recall; occurs in REM; common in PTSD

Sleep paralysis (isolated) Inability to move at sleep onset / offset; often frightening; hallucinations common; benign

Exploding head syndrome Sudden loud perceived noise at sleep onset; not painful; benign

Sleep-related hallucinations Hypnagogic (at onset) or hypnopompic (at offset); vivid, can be visual / auditory

Catathrenia (sleep groaning) Prolonged groaning during exhalation; occurs in NREM and REM

Sexsomnia Sexual behavior during sleep; legal implications; treat as arousal disorder

3.6 Circadian Rhythm Sleep-Wake Disorders

DISORDER	PATTERN	MANAGEMENT
Delayed Sleep-Wake Phase	Phase shifted later (night owl); can't sleep before 2-3 AM	Chronotherapy, bright light therapy in morning, melatonin in evening
Advanced Sleep-Wake Phase	Phase shifted earlier (early bird); sleepy by 6-8 PM	Bright light therapy in evening
Shift Work Disorder	Conflict between internal clock and work schedule	Shift scheduling, melatonin, modafinil for alertness
Jet Lag	Transient misalignment with new time zone	Melatonin, bright light at destination time
Non-24 Hour Sleep-Wake	Gradual drift of sleep timing (mainly blind individuals)	Tasimelteon (MT1/MT2 agonist), FDA approved

3.7 Restless Legs Syndrome (RLS)

Diagnostic criteria (IRLSSG):

1. Urge to move the legs, usually accompanied by unpleasant sensations
2. Symptoms begin or worsen during rest or inactivity
3. Partially or totally relieved by movement
4. Symptoms are worse in the evening or at night
5. Not solely accounted for by another condition

Associations:

- Iron deficiency (check serum ferritin; treat if $<50-75 \mu\text{g/L}$)
- Pregnancy (third trimester)
- Uremia, peripheral neuropathy, Parkinson's disease
- Dopamine deficiency: symptoms worsen with dopamine antagonists (antipsychotics, metoclopramide)

Management:

SEVERITY · TREATMENT

Mild/intermittent Iron replacement, sleep hygiene, reduce precipitants

Moderate-severe Dopamine agonists (pramipexole, ropinirole), first-line

Augmentation on dopamine agonists Switch to gabapentinoids or opioids

Alternative first-line Gabapentin, pregabalin (especially with pain component)

Refractory Low-dose opioids

EXAM PEARL

Augmentation is a key complication of dopamine agonist treatment in RLS, symptoms start earlier in the day, spread to arms, become more intense. Managed by switching drug class or adding gabapentinoids.

3.8 Sleep in Psychiatric Illness

CONDITION · SLEEP PATTERN

Major Depression Increased sleep latency, early morning awakening, reduced N3, reduced REM latency (<65 min), increased total REM, frequent awakenings

Bipolar Mania Decreased need for sleep (feels rested with little sleep), markedly reduced total sleep time

Bipolar Depression Hypersomnia (more common than in unipolar), or insomnia

Schizophrenia Reduced total sleep, reduced N3, reduced REM latency; sleep continuity disruption

PTSD Fragmented sleep, frequent nightmares (REM), hyperarousal, difficulty initiating sleep

Anxiety Disorders Sleep onset insomnia; worry and rumination at night

ADHD Delayed sleep phase, increased sleep latency; sleep disorders 2-3x more common

Alzheimer's Dementia Sundowning, disrupted circadian rhythm, progressive loss of N3

EXAM PEARL

Reduced REM latency in depression is one of the most consistent biological findings. It occurs in approximately 60% of patients with major depression and is also seen in relatives (biological marker). Antidepressants (except bupropion) suppress REM.

PART 4: SUICIDE

4.1 Epidemiology

Global:

- ~800,000 deaths/year from suicide globally (WHO 2023 estimate)
- Rate: approximately 10.5 per 100,000 globally
- Leading cause of death in 15-29 year olds
- Male:Female ratio for completed suicide: 3-4:1 (worldwide); lower in India
- Female:Male ratio for attempts: 3:1

India (NCRB Data):

EXAM PEARL

NCRB (National Crime Records Bureau) publishes annual suicide data for India. Know the current trends.

METRIC · INDIA DATA (NCRB 2022)

Total deaths ~170,000 per year

Rate ~12.4 per 100,000

Male:Female ratio ~68:32 (more balanced than global)

Common methods Hanging (most common), poisoning (organophosphate), self-immolation

Highest burden states Tamil Nadu, Maharashtra, Madhya Pradesh

Common stated reasons Family problems, illness, marriage-related issues

Age group 18-45 years (working age), highest absolute numbers

Farmers Consistently high; agrarian crisis, debt

Suicide attempt vs completion:

- Ratio of attempts to completions: approximately 20:1 (global); varies
 - India: underreporting of both attempts and completions (legal, stigma)
 - 50% of completers have made prior attempts
-

4.2 Risk Factors and Protective Factors

RISK FACTORS (ORGANIZED BY MNEMONIC: SEE D3)

Psychiatric:

- Previous suicide attempt (strongest single predictor of completion)
- Major depression (especially with hopelessness)
- Bipolar disorder (mixed states, depression, post-mania depression)
- Schizophrenia (command hallucinations, post-psychotic depression)
- Substance use disorders (alcohol markedly increases risk)
- Borderline personality disorder
- Anorexia nervosa (highest SMR of all psychiatric disorders)
- PTSD

Psychological:

- Hopelessness (Beckian triad, more predictive than depression severity itself)
- Impulsivity / aggression
- History of trauma
- Chronic pain / illness

Social:

- Social isolation, living alone
- Recent bereavement, loss, humiliation
- Unemployment, financial crisis
- Domestic violence
- Access to lethal means

Biological:

- Serotonin dysfunction (5-HIAA reduced in CSF of suicide completers)
- HPA axis dysregulation
- Family history

EXAM PEARL

The single strongest predictor of future suicide attempt is a PRIOR suicide attempt. Hopelessness, measured by the Beck Hopelessness Scale (BHS), is more predictive than depression severity.

PROTECTIVE FACTORS

CATEGORY · FACTORS

Individual Reasons for living, problem-solving ability, future orientation, religious beliefs

Social Social support, family connectedness, marriage / partnership

Health system Access to mental health care, treatment adherence

Community Low stigma, crisis resources availability

Cultural Religious prohibitions against suicide, cultural value of life

4.3 Risk Assessment Tools

SAD PERSONS SCALE (PATTERSON ET AL., 1983)

LETTER	FACTOR	SCORE
S	Sex (male)	1
A	Age (<19 or >45)	1
D	Depression	1
P	Previous attempt	1
E	Ethanol abuse	1
R	Rational thinking loss	1
S	Social supports lacking	1
O	Organized plan	1
N	No spouse	1
S	Sickness	1

Score: 0-4 = low risk, 5-6 = moderate, 7-10 = high risk

EXAM PEARL

SAD PERSONS is widely cited but has poor sensitivity for predicting actual suicide. The Columbia Protocol is now preferred in clinical practice. Know SAD PERSONS for exam but mention its limitations.

COLUMBIA SUICIDE SEVERITY RATING SCALE (C-SSRS)

Developed by Posner et al., widely validated.

Suicidal ideation assessment:

1. Wish to be dead
2. Non-specific active suicidal thoughts
3. Active thoughts with method (no plan)
4. Active ideation with some intent to act, no plan
5. Active ideation with plan and intent

Suicidal behavior:

- Preparatory actions, aborted attempt, interrupted attempt, actual attempt, completed suicide

CLINICAL ANCHOR

C-SSRS distinguishes ideation intensity from behavior, this guides clinical decision-making more precisely than older scales.

CLINICAL INTERVIEW FRAMEWORK (WHAT MATTERS)

Access to means: What methods are available? Firearms at home?

Intent: Is there intent to act? Have they taken preparatory steps?

Plan: Is there a specific plan with time, place, method?

Hopelessness: Beck Hopelessness Scale or clinical assessment

History: Prior attempts, family history

Protective factors: What stops them?

Mental state: Command hallucinations, agitation, intoxication

4.4 Theories of Suicide

JOINER'S INTERPERSONAL THEORY OF SUICIDE (IPTS)

Three components necessary for highest suicide risk:

1. **Thwarted belongingness**, the sense of not belonging, social isolation, loneliness
2. **Perceived burdensomeness**, belief that one is a burden to others; "my death would be a relief to my family"
3. **Acquired capability**, habituation to pain and fear of death through repeated exposure (prior attempts, violence, self-harm history)

KEY INSIGHT

The first two create the desire; the third creates the capability. All three together = maximum risk.

Clinical application: Assess each component in interview. "Do you feel like a burden?" "Do you feel like you belong anywhere?"

KLONSKY & MAY'S THREE-STEP THEORY (3ST)

Step 1: Pain (psychological) + Hopelessness → passive suicidal ideation

Step 2: Pain > Connection to life → active ideation

Step 3: Dispositional, acquired, or practical capability → attempt

Advantage over IPTS: More parsimonious; explains ideation development more clearly.

OTHER THEORIES

THEORY · KEY CLAIM

Durkheim's Sociological Theory Anomie, egoistic, altruistic, and fatalistic suicide types; social integration matters

Shneidman's Psychache Theory Suicide is the result of intolerable psychological pain ("psychache")

Cry of Pain Model (Williams) Defeat + entrapment + no rescue = suicide

Escape Theory (Baumeister) Suicide as escape from painful self-awareness

4.5 Management of Suicidal Crisis

Immediate:

1. Safe environment, remove means from environment
2. Hospitalization if: active intent with plan, psychosis, unavailability of safe home environment, command hallucinations
3. Close supervision (1:1 if inpatient)
4. Treat underlying disorder urgently (e.g., start antidepressant, lithium, clozapine)

Pharmacotherapy:

- **Lithium:** most evidence for reducing suicidality in bipolar / recurrent depression; reduces completed suicide by 80%
- **Clozapine:** only FDA-approved drug with explicit anti-suicidal indication (for suicidality in schizophrenia / schizoaffective)
- **Ketamine/esketamine:** rapid antisuicidal effect within hours; important for acute crisis
- **SSRIs:** long-term reduction in suicidality; black box warning in under-25s (short-term increase in ideation)

EXAM PEARL

Clozapine is the only FDA-approved drug specifically for suicidality. Lithium has the strongest evidence for reducing completed suicide. These are two different claims, know both.

Means restriction:

- Highly effective population-level intervention
- Evidence: bridge barriers (Golden Gate Bridge barrier reduced suicide rate at that location AND total county suicide rate, no substitution)
- Firearm restriction in depressed individuals is high-yield
- Paracetamol pack size legislation in UK reduced paracetamol overdose deaths

Safety planning:

- Stanley-Brown Safety Planning Intervention
 - Collaborative (patient generates content)
 - Components: warning signs → internal coping → social contacts → professionals → means restriction
 - Distinguishes from "no-harm contracts" (no evidence of effectiveness)
-

4.6 Deliberate Self-Harm (DSH)

Definition: Intentional, self-inflicted harm without suicidal intent (cutting, burning, bruising). Also called Non-Suicidal Self-Injury (NSSI).

Functions:

- Emotion regulation (most common), "when I cut, I feel relief"
- Anti-dissociation, to feel real, to feel something
- Self-punishment
- Communication of distress

Epidemiology:

- 17-25% of adolescents report lifetime NSSI
- Peak onset: early adolescence (12-14 years)
- Female > male (in adolescence); sex differences narrow with age
- Strong predictor of later suicide attempt (but distinct phenomenon)

Assessment:

- Distinguish NSSI from suicidal behavior (intent, circumstances, rescue likelihood, lethality)
- Assess for BPD, depression, trauma, eating disorders
- Columbia Protocol can be used to clarify suicidal vs non-suicidal intent

Management:

- DBT (strongest evidence for NSSI and suicidality in BPD)
 - CBT, emotion regulation skills
 - Harm reduction approach
 - Avoid punitive responses
-

4.7 Postvention

Definition: Actions taken after a suicide to support the bereaved and prevent suicide contagion.

Survivors of suicide loss:

- 1.25 million Americans become suicide loss survivors each year
- Elevated risk of complicated grief, PTSD, depression
- Elevated risk of suicide themselves (bereaved by suicide)

Postvention in institutions (schools, workplaces):

- Avoid glorification or sensationalization
 - Provide access to counseling
 - Identify and support high-risk individuals
 - Follow WHO guidelines on media reporting
-

4.8 Media Reporting Guidelines (WHO/Papageno Effect)

Werther Effect: Increase in suicides following media reporting; first described after publication of Goethe's "The Sorrows of Young Werther."

Papageno Effect: Protective effect of stories showing suicidal crisis being overcome by positive coping.

WHO Safe Messaging Guidelines:

- Do NOT report in detail about methods, locations, notes
 - Do NOT sensationalize or romanticize
 - Do NOT present as "heroic" or "sacrificial"
 - DO provide crisis resources in the story
 - DO include stories of help-seeking and recovery
 - DO consult with mental health professionals before publishing
 - Reporting celebrity suicides: celebrity method often imitated
-

PART 5: PSYCHIATRIC CLASSIFICATION

5.1 ICD-11 vs ICD-10: Key Changes

ICD-11 was adopted by WHO in 2019, came into effect January 2022. India has not yet fully transitioned.

EXAM PEARL

This is an extremely high-yield area. List specific changes confidently, both additions and structural shifts.

DOMAIN	ICD-10	ICD-11	CLINICAL SIGNIFICANCE
Overall structure	F-codes (F00-F99)	6A-6E (alphanumeric)	Complete re-coding
Schizophrenia subtypes	5 subtypes (paranoid, hebephrenic, catatonic, undifferentiated, residual)	Subtypes REMOVED; symptom domains added (positive, negative, depressive, manic, psychomotor, cognitive)	Shift from categorical to dimensional within schizophrenia
PTSD	Single category	Complex PTSD added (6B41) as a separate diagnosis	Developmental trauma, chronic interpersonal trauma
BED	Not a full diagnosis (in appendix)	Full diagnosis (6B82)	Access to treatment, insurance coding
ARFID	Not present	New diagnosis (6B83)	Separate from AN
Prolonged Grief Disorder	Not present	New diagnosis (6B42)	>6 months, yearning/preoccupation
Gaming Disorder	Not present	Added (6C51)	Significant controversy
Compulsive Sexual Behavior	Not present	Added (6C72)	Significant controversy
Personality disorders	10 categorical types	Dimensional model + severity rating	Severity (mild/moderate/severe) + dominant trait domains
Catatonia	Subtype of schizophrenia	Separate chapter; can be due to any condition	Recognizes catatonia across diagnostic spectrum
ICD-10 F45 (somatoform)	Multiple subtypes	Bodily Distress Disorder (new umbrella)	Simplification; aligns with functional symptoms
Dissociative conversion	F44	FND moved to Diseases of Nervous System	See above
Neurodevelopmental	Scattered	Grouped in chapter 6A	ADHD, ASD, DCD, stereotyped movement disorders

5.2 DSM-5 Structure

Published 2013 (DSM-5-TR: 2022 update)

Major chapters (in order):

1. Neurodevelopmental Disorders
2. Schizophrenia Spectrum and Other Psychotic Disorders
3. Bipolar and Related Disorders
4. Depressive Disorders
5. Anxiety Disorders
6. Obsessive-Compulsive and Related Disorders
7. Trauma- and Stressor-Related Disorders
8. Dissociative Disorders
9. Somatic Symptom and Related Disorders
10. Feeding and Eating Disorders
11. Elimination Disorders
12. Sleep-Wake Disorders
13. Sexual Dysfunctions
14. Gender Dysphoria
15. Disruptive, Impulse-Control, and Conduct Disorders
16. Substance-Related and Addictive Disorders
17. Neurocognitive Disorders
18. Personality Disorders
19. Paraphilic Disorders
20. Other Mental Disorders

Key DSM-5 changes from DSM-IV:

- Multiaxial system abolished (Axes I-V removed; single integrated assessment)
- Asperger syndrome removed (folded into ASD spectrum)
- Bereavement exclusion for MDD removed (controversial)
- Mixed features specifier added to MDD and bipolar
- OCD/OCSD separated into own chapter
- Trauma disorders separated from anxiety disorders
- PTSD given more behavioral criteria (avoidance)
- BED, ARFID added
- Dimensional approach to personality disorders in Section III (alternative model)

5.3 Dimensional vs Categorical Debate

Categorical model (traditional):

- Diagnosis = present / absent
- Clear boundaries between diagnoses

- Advantages: clinical utility, communication, treatment decision
- Disadvantages: high comorbidity, arbitrary thresholds, heterogeneity within categories

Dimensional model:

- Symptoms exist on continua
- People vary in degree, not type
- More biologically plausible
- Advantages: reflects neurobiology, genetic architecture, no arbitrary threshold
- Disadvantages: more complex; less clinical utility for many decisions

EXAM PEARL

ICD-11 personality disorders moved fully to a dimensional model. DSM-5 retains categorical in main text but offers an alternative dimensional model in Section III (AMPD, Alternative Model for Personality Disorders). The direction of travel is clearly dimensional.

5.4 RDoC (Research Domain Criteria)

Developed by: NIMH (National Institute of Mental Health), Thomas Insel, 2010

Core idea: Diagnose based on neurobiological and behavioral dimensions, not symptom-based categorical diagnoses.

RDoC Domains:

1. Negative Valence Systems (fear, threat, loss)
2. Positive Valence Systems (reward seeking, reward anticipation)
3. Cognitive Systems (attention, memory, cognitive control)
4. Social Processes (affiliation, attachment, communication)
5. Arousal/Modulatory Systems (arousal, sleep-wake)
6. Sensorimotor Systems (motor actions)

Units of analysis: Genes → Molecules → Cells → Circuits → Physiology → Behavior → Self-report

Critique:

- Not ready for clinical use
- May fragment what is clinically a coherent syndrome
- Ignores subjective experience

5.5 HiTOP (Hierarchical Taxonomy of Psychopathology)

A transdiagnostic dimensional classification framework based on covariance structure of psychopathology

Structure (bottom-up from symptoms):

- Components → Syndromes → Subfactors → Spectra → Superspectra

Major Spectra:

SPECTRUM · INCLUDES

Internalizing Fear (phobias, panic, GAD), Distress (depression, GAD, PTSD, OCD)

Externalizing Disinhibition (substance use, ASPD), Antagonism (narcissism, psychopathy)

Thought disorder Psychosis spectrum

Detachment Social detachment, schizoid

Somatoform Somatic symptom disorders

Advantages over DSM/ICD:

- Based on empirical data, not expert consensus
- Captures comorbidity naturally (as shared variance)
- Maps onto neurobiological dimensions

CLINICAL ANCHOR

HiTOP explains why anxiety and depression are so often comorbid, they both load on the internalizing spectrum. This isn't two diseases co-occurring; it's one spectrum with two expression modes.

5.6 Reliability and Validity of Diagnosis

Reliability:

- Inter-rater reliability: do two clinicians using the same system agree?
- Test-retest reliability: does the diagnosis remain stable over time?
- DSM-5 field trials showed moderate reliability (kappa 0.4-0.8) for most major categories
- MDD: kappa ~0.28 in real-world settings (poor)
- Schizophrenia: better reliability (~0.45-0.6)

Validity types:

TYPE	DEFINITION	EXAMPLE
Face validity	Looks like what it claims to be	Depressive symptoms represent depression
Content validity	Covers the full domain	Depression criteria cover all key domains

TYPE	DEFINITION	EXAMPLE
Criterion validity	Predicts external criterion	Major depression predicts treatment response
Construct validity	Fits theoretical framework	MDD associated with HPA dysregulation
Predictive validity	Predicts future outcomes	Risk of recurrence, disability

EXAM PEARL

DSM/ICD diagnoses have reasonable reliability but questionable validity, especially biomarker validity. This is the core critique from RDoC proponents.

PART 6: CROSS-CUTTING CLINICAL CONSIDERATIONS

6.1 Suicide in Eating Disorders

- AN has the highest SMR of any psychiatric disorder: ~5.86 (6x age-matched peers)
- Suicide accounts for ~1/3 of AN deaths (remainder are medical)
- Risk is elevated throughout illness duration, not just at low weight
- BN and BED also carry elevated suicide risk
- Assessment of suicide risk is mandatory in all eating disorder evaluations

6.2 Sleep in Dissociative Disorders

- Dissociative episodes can occur at sleep-wake transitions
- Hypnagogic/hypnopompic experiences may be misinterpreted as possession or alien abduction
- Sleep deprivation can trigger dissociative episodes
- RBD may be misidentified as fugue or sleepwalking

6.3 Eating Disorders and Circadian Rhythms

- Night eating syndrome: distinct from BED; >25% caloric intake after dinner
- Shift work disrupts eating patterns; associated with metabolic syndrome and binge eating
- Circadian disruption may worsen binge-purge cycles

6.4 Classification of Self-Harm

TERM	DEFINITION	NOTES
Suicidal ideation	Thoughts of killing oneself	Passive (wish to die) to active (with plan)

TERM	DEFINITION	NOTES
Suicide attempt	Self-injurious behavior with any intent to die	High lethality does not always mean high intent
NSSI / DSH	Self-injury without intent to die	Emotion regulation, anti-dissociation
Completed suicide	Death by suicide	
Parasuicide	Older term; behavior resembling suicide without intent	Now largely replaced by NSSI/DSH

EXAM PEARL

Lethality of method and suicidal intent do NOT always correlate. A patient who takes 10 paracetamol with low intent may have severe hepatotoxicity. A patient who fires a gun and survives may have had high intent but poor aim.

Word count: approximately 8,500 words, detailed, high-yield, exam-oriented

Model Answers

Format: Each answer structured for 5-mark, 10-mark, or short-note format as indicated. Write in flowing prose unless tabular organisation is clearer.

ANSWER 1: Classify and Describe the Management of Dissociative Disorders (10 marks)

EXAM STRATEGY

Start with a brief introduction defining dissociation, then give a clear classification table, then move to management. Examiners reward structure. Do not write one block of undifferentiated prose.

Introduction:

Dissociation refers to a disruption in the normally integrated functions of consciousness, memory, identity, perception, and behaviour. Dissociative disorders are characterised by clinically significant dissociative symptoms that cause distress or functional impairment, and are not better explained by another disorder, substance use, or medical condition.

Classification (ICD-11 / DSM-5):

DISORDER	ICD-11 CODE	KEY FEATURE
Dissociative amnesia	6B61	Inability to recall autobiographical information (trauma-linked)
with Dissociative fugue	6B61.0	Amnesia + unexpected travel + identity confusion
Dissociative identity disorder	6B64	≥2 distinct personality states with amnesia between them
Partial DID	6B65 (ICD-11 new)	Intrusions from alternate identity without full switching
Depersonalisation/derealisation disorder	6B66	Persistent detachment from self or surroundings; reality testing intact
Trance disorder	6B63	Narrowed awareness, stereotyped movements

DISORDER	ICD-11 CODE	KEY FEATURE
Possession trance disorder	6B63.0	Complete identity replacement by external entity (outside cultural context)
Secondary dissociative symptoms		Occurring within another disorder (PTSD, BPD)

Note: Functional Neurological Symptom Disorder (ICD-11) is now placed in the chapter on Diseases of the Nervous System, not Dissociative Disorders, a key ICD-11 change.

Management:

Management of dissociative disorders follows a **phase-based approach**, adapted across subtypes.

Phase 1, Stabilisation and Safety:

The priority in any dissociative disorder is establishing safety and stabilising the patient's emotional state. This involves psychoeducation (explaining the dissociative process in accessible language), development of grounding techniques (5-4-3-2-1 sensory grounding, containment imagery), and building a therapeutic alliance. For DID, mapping the alter system without prematurely accessing traumatic material is critical.

Phase 2, Trauma Processing (if applicable):

Once stabilised, trauma-focused interventions may be introduced carefully. Evidence-supported approaches include:

- EMDR (Eye Movement Desensitisation and Reprocessing), recommended in PTSD guidelines; used in dissociative disorders with caution
- Trauma-focused CBT, modified for dissociative presentation
- Ego state therapy, specifically designed for DID; works with alter states
- Schema Therapy, especially effective where early maladaptive schemas underlie the dissociation

Phase 3, Integration and Relapse Prevention:

In DID, the goal is either full fusion (merging of alters) or functional integration (alters co-operating, amnesia barriers reduced). In dissociative amnesia, memory recovery is not always necessary for recovery; functional improvement is the primary goal.

Pharmacotherapy:

There is no specific pharmacological treatment for core dissociative symptoms. Medications address comorbidities:

- SSRIs/SNRIs for comorbid PTSD, depression, anxiety
- Naltrexone (opioid antagonist): some evidence for depersonalisation/derealization

- Lamotrigine: open-label evidence for DPDR
- Benzodiazepines: used cautiously for acute anxiety; avoid long-term due to abuse risk and potential to worsen dissociation

Specific Considerations for DPDR:

CBT using the Hunter-Phillips model (Feeling Real technique) is the best-evidenced psychological treatment. Focus is on reducing safety behaviours (checking, avoidance) and accepting uncomfortable sensations. SSRIs are first-line pharmacologically.

EXAM PEARL

The most important point in dissociative disorder management is that **premature trauma processing destabilises rather than helps**. Phase-based therapy is not merely a theoretical preference, it is a safety principle.

ANSWER 2: Medical Complications of Anorexia Nervosa (10 marks)

Introduction:

Anorexia nervosa (AN) carries the highest standardised mortality ratio (SMR ~5.86) of any psychiatric disorder. Approximately two-thirds of deaths are due to medical complications and one-third to suicide. Understanding the medical complications is clinically essential and examination-critical.

Cardiovascular:

The most immediately life-threatening complications arise from the cardiovascular system. Bradycardia (HR <60 bpm) results from vagal predominance and reduced metabolic demand. Hypotension (systolic <90 mmHg) is common. QTc prolongation occurs due to electrolyte disturbances (especially hypokalemia and hypomagnesemia), increasing risk of torsades de pointes and sudden cardiac death. Cardiac muscle mass decreases with starvation, so-called cardiac cachexia, leading to reduced cardiac output and mitral valve prolapse (due to disproportionate reduction in left ventricular mass relative to valve size).

Metabolic and Electrolyte:

Hypoglycemia may occur, particularly in the restricting subtype with inadequate gluconeogenic substrate. Electrolyte abnormalities are most severe in the binge-purge subtype: hypokalaemia and metabolic alkalosis from vomiting; hypokalaemia and metabolic acidosis from laxative abuse. Hypophosphatemia occurs particularly during refeeding. Hyponatraemia may result from excessive water intake (pseudo-Bartter syndrome) or syndrome of inappropriate ADH secretion.

Endocrine:

Amenorrhoea is a classic finding, resulting from hypothalamic suppression of GnRH pulsatility → low LH, FSH, estrogen. Note: DSM-5 removed amenorrhoea as a diagnostic

criterion (post-menopause women and males cannot fulfill it). Low cortisol response, high cortisol baseline, and sick euthyroid syndrome (low T3, normal or low T4, normal TSH) are characteristic. Growth hormone resistance occurs (high GH, low IGF-1). In adolescents, growth retardation and delayed puberty occur if starvation precedes the pubertal growth spurt.

Haematological:

Pancytopenia from gelatinous bone marrow transformation. Anaemia, leukopenia (impairs immune function), and thrombocytopenia are all seen.

Gastrointestinal:

Delayed gastric emptying causes early satiety, bloating, and increases patient resistance to refeeding. Superior mesenteric artery (SMA) syndrome, duodenal obstruction due to loss of the fat pad between the SMA and aorta, can cause vomiting and weight loss independent of AN behaviour. Elevated liver enzymes (hepatocyte autophagy) are seen in severe AN.

Musculoskeletal:

Osteopenia and osteoporosis due to prolonged estrogen deficiency (more refractory than post-menopausal osteoporosis because it occurs during peak bone mass acquisition). Stress fractures, vertebral compression. Bone mineral density does not fully recover with weight restoration alone.

Dermatological:

Lanugo hair (fine downy hair, thermoregulatory response), carotenemia (yellow-orange skin from excess carrot intake), xerosis, hair thinning (telogen effluvium), and in the binge-purge subtype, parotid hypertrophy, dental erosion (perimolysis), and Russell's sign (calluses on the dorsum of the hand from self-induced vomiting).

Neurological:

Cortical grey matter loss with underweight state (partially reversible on weight restoration). Peripheral neuropathy. Cognitive impairment (attention, executive function), partly due to starvation, partly due to metabolic disturbances.

Summary Table:

SYSTEM	KEY COMPLICATION	CLINICAL SIGNIFICANCE
Cardiovascular	Bradycardia, QTc prolongation, cardiac atrophy	Sudden cardiac death risk
Metabolic	Hypokalaemia, hypophosphataemia, hypoglycaemia	Arrhythmias, seizures
Endocrine	Amenorrhoea, low T3, low IGF-1	Growth arrest in adolescents
Haematological	Pancytopenia	Infection risk

SYSTEM	KEY COMPLICATION	CLINICAL SIGNIFICANCE
GI	Delayed gastric emptying, SMA syndrome	Impedes refeeding
Musculoskeletal	Osteoporosis	Irreversible bone loss
Neurological	Cortical atrophy, cognitive impairment	Impairs therapy participation

CLINICAL ANCHOR

In clinical practice, the most common immediate cause of death is cardiac arrhythmia from electrolyte disturbance (especially hypokalemia + QTc prolongation). Always obtain ECG and electrolytes in any patient with AN presenting acutely.

ANSWER 3: Refeeding Syndrome: Pathophysiology, Recognition, and Prevention (10 marks)

Definition:

Refeeding syndrome refers to the metabolic complications, primarily hypophosphataemia, hypokalaemia, and hypomagnesaemia, that can occur when nutrition is reintroduced following a period of prolonged starvation or severe malnutrition. It is potentially fatal if unrecognised.

Pathophysiology:

During prolonged starvation:

- The body shifts to fat and protein catabolism for energy
- Intracellular electrolytes (phosphate, potassium, magnesium) are progressively depleted
- Serum levels of these electrolytes may remain normal (deceptive) as homeostatic mechanisms are maintained until re-feeding

When feeding is reintroduced (especially carbohydrates):

- Insulin is secreted in response to rising blood glucose
- Insulin drives glucose, phosphate, potassium, and magnesium from the extracellular space INTO cells
- Serum phosphate drops sharply, **hypophosphataemia is the hallmark**
- Thiamine (vitamin B1) is consumed rapidly as a cofactor for carbohydrate metabolism
- Thiamine depletion can precipitate Wernicke's encephalopathy if not supplemented

Consequences of Key Electrolyte Disturbances:

ELECTROLYTE · CONSEQUENCE

Hypophosphataemia (critical) Respiratory failure (diaphragmatic weakness), cardiac failure, haemolytic anaemia, rhabdomyolysis, seizures, delirium

Hypokalaemia Cardiac arrhythmias, cardiac arrest, ileus, muscle weakness

Hypomagnesaemia Tetany, arrhythmias, seizures, impairs correction of hypokalaemia

Hyperglycaemia Osmotic diuresis, metabolic derangement

Thiamine deficiency Wernicke-Korsakoff syndrome

NICE Risk Criteria (any one = high risk; any two = very high risk):

CRITERION · VALUE

BMI <16 kg/m²

Unintentional weight loss >15% in 3-6 months

Negligible nutritional intake >10 days

Pre-feeding electrolyte abnormality Low K⁺, Mg²⁺, or PO₄³⁻

Prevention Protocol:

1. **Screen all patients** for refeeding risk before initiating nutritional rehabilitation
2. **Baseline bloods:** urea, creatinine, electrolytes (Na, K, Mg, phosphate, calcium), LFTs, full blood count, glucose
3. **Baseline ECG** (QTc monitoring)
4. **Thiamine BEFORE glucose**, oral thiamine 200-300mg/day (or IV Pabrinex if parenteral feeding) must be started before, not after, carbohydrate reintroduction
5. **Start low, go slow:** Initial intake 20 kcal/kg/day (10 kcal/kg/day for very high risk); increase over 4-7 days
6. **Electrolyte monitoring:** every 12-24 hours for first 3-5 days; replace proactively
7. **Phosphate supplementation:** oral or IV phosphate if serum phosphate <0.5 mmol/L

EXAM PEARL

The classic exam question asks: "A 17-year-old with anorexia is admitted for refeeding. On day 3, she develops confusion, respiratory distress, and muscle weakness. What has happened?"
Answer: refeeding syndrome with hypophosphataemia. Treatment: reduce caloric load, IV phosphate replacement, thiamine, electrolyte monitoring.

ANSWER 4: Suicide Risk Assessment: Comprehensive Approach (10 marks)

Introduction:

Suicide risk assessment is a core clinical skill in psychiatry. No single tool can predict suicide with certainty; risk assessment is a clinical synthesis that informs management decisions. The goal is to identify modifiable risk factors and guide the least restrictive, most appropriate level of care.

Framework for Assessment:

A comprehensive suicide risk assessment addresses **four domains**: ideation, intent, plan, and means. This is embedded within the broader clinical interview.

1. Suicidal Ideation:

Assess along the dimension of passivity to activity:

- Passive: "I wish I were dead" / "I wish I could go to sleep and not wake up"
- Active without method: "I think about killing myself"
- Active with method but no plan: "I think about taking pills"
- Active with plan, time, and place: "I've decided to take the medication I've been saving on Thursday when my family is away"

The Columbia Suicide Severity Rating Scale (C-SSRS) provides a validated, structured approach to this dimension.

2. Suicidal Intent:

- Is the patient ambivalent or resolved?
- What has stopped them so far?
- Has intent increased recently?
- Is there a precipitant (recent loss, humiliation, anniversary)?

3. Suicide Plan:

- Specificity of plan (vague vs. detailed)
- Proximity to means (has the patient already obtained pills, or is the firearm in the house?)
- Lethality of plan (overdose on paracetamol vs. jumping from height)
- Accessibility of plan (can they act on it imminently?)

4. Risk Factors (using the mnemonic IS PATH WARM, see D3):

CATEGORY · KEY RISK FACTORS

Psychiatric Previous attempt (strongest predictor), active depression, bipolar mixed state, schizophrenia, substance use

Psychological Hopelessness (BHS score), impulsivity, anhedonia, cognitive constriction

Social Isolation, recent loss, recent humiliation, domestic violence, unemployment

Biological Male sex, family history, chronic pain, serious medical illness

5. Protective Factors:

Often underassessed. Key factors:

- Reasons for living (children, family responsibility, religious beliefs)
- Social support and connectedness
- Future orientation
- Help-seeking behaviour
- Absence of access to means

Structured Scales:

- **SAD PERSONS** (10-point scale): useful mnemonic but poor sensitivity in studies
- **C-SSRS**: validated, widely used, distinguishes ideation from behaviour
- **Beck Scale for Suicide Ideation (BSI)**: validated for intensity of current ideation
- **Beck Hopelessness Scale (BHS)**: score >9 = high risk; more predictive than depression severity

Management Based on Risk Level:

RISK LEVEL · MANAGEMENT

Low Outpatient treatment, safety plan, means restriction counselling, follow-up within 1 week

Moderate Intensive outpatient, crisis plan, family involvement, daily check-in

High Hospitalisation (voluntary or involuntary), 1:1 supervision, means removal, urgent treatment initiation

Imminent Emergency detention, close observation, treat precipitating crisis

Safety Planning (Stanley-Brown model):

A collaborative, evidence-based intervention (superior to no-harm contracts):

1. Warning signs recognisable to the patient
2. Internal coping strategies (what the patient can do alone)
3. Socialisation strategies (people and activities that distract)
4. People to ask for help
5. Professionals to contact in crisis

6. Making the environment safe (means restriction)

CLINICAL ANCHOR

A "no-harm contract" is NOT an evidence-based intervention and provides false reassurance. Safety Planning has evidence; contracts do not. In exam answers, always specify safety planning, not a "contract."

ANSWER 5: CBT-I for Chronic Insomnia (10 marks)

Introduction:

Cognitive Behavioural Therapy for Insomnia (CBT-I) is the first-line treatment for chronic insomnia disorder, recommended above pharmacotherapy in all major guidelines (NICE, AASM, ERS). It addresses the perpetuating factors that maintain insomnia through behavioural, cognitive, and sleep hygiene components.

Theoretical Model:

Insomnia is maintained by a combination of:

- **Behavioural:** extended time in bed, daytime napping, variable wake times
- **Cognitive:** catastrophic beliefs about consequences of poor sleep ("I can't function with less than 8 hours"), selective attention to sleep-related stimuli
- **Physiological:** hyperarousal (elevated cortisol, elevated temperature, heightened CNS arousal)
- **Conditioned arousal:** bed becomes a cue for wakefulness rather than sleep (Stimulus-Response theory)

CBT-I targets all three maintaining factor types.

Components:

1. Sleep Restriction (most potent component):

- Rationale: consolidates fragmented, light sleep by building sleep drive (adenosine)
- Method: restrict time in bed (TIB) to the patient's actual sleep time (minimum 5 hours TIB for safety); maintain consistent wake time
- Sleep efficiency (SE) = $\text{Total Sleep Time} / \text{Time In Bed} \times 100\%$
- When SE >90% for ≥5 consecutive nights, extend TIB by 15-30 minutes
- Contraindications: seizure disorder, parasomnias, sleep deprivation-dangerous jobs

2. Stimulus Control:

- Rationale: breaks the conditioned arousal to the bed/bedroom
- Rules:
- Use bed only for sleep and sex

- If unable to sleep within ~20 minutes, leave the bedroom; return when sleepy
- Maintain consistent rise time regardless of how much sleep was obtained
- Avoid daytime napping (or limit to <20 min before 3 pm)

3. Cognitive Restructuring:

- Identify unhelpful beliefs: "I need 8 hours", "I haven't slept all night" (often an overestimation of wakefulness), "Tomorrow will be ruined"
- Cognitive techniques: Socratic questioning, behavioural experiments, psychoeducation about sleep variability

4. Relaxation Training:

- Progressive muscle relaxation (Jacobson technique)
- Diaphragmatic breathing
- Mindfulness (MBSR has some evidence in insomnia)
- Autogenic training

5. Sleep Hygiene:

- Necessary but insufficient alone
- Consistent sleep / wake schedule
- Bedroom environment: cool, dark, quiet
- Avoid caffeine after 2 pm, heavy meals within 3 hours of sleep
- Avoid screen light (blue-spectrum suppresses melatonin) 1 hour before bed
- Regular exercise (not within 3 hours of bedtime)

6. Sleep Compression (alternative in elderly):

- Gradual reduction of TIB rather than abrupt restriction
- Better tolerated; slightly less efficacious

Outcomes:

CBT-I achieves sleep latency reduction, improved sleep efficiency, and reduced nighttime awakenings in 70-80% of patients. Effects are durable at 12 months follow-up, unlike pharmacotherapy which tends to rebound on discontinuation.

Delivery formats:

- Face-to-face (individual or group, equivalent efficacy)
- Digital CBT-I (dCBT-I): apps (Sleepio, etc.); comparable efficacy to face-to-face for uncomplicated insomnia; more accessible

EXAM PEARL

When asked to compare CBT-I vs pharmacotherapy: CBT-I has **equivalent or superior efficacy** in acute insomnia and **superior long-term outcomes**. Pharmacotherapy works faster (within days); CBT-I takes 4-6 weeks to show full effect. In exams, always recommend CBT-I first, with pharmacotherapy for short-term/adjunctive use.

ANSWER 6: Narcolepsy: Classification and Management (10 marks)

Introduction:

Narcolepsy is a chronic sleep disorder characterised by the inability to regulate sleep-wake states normally, resulting in excessive daytime sleepiness (EDS), intrusion of REM sleep phenomena into wakefulness, and disrupted nocturnal sleep.

Classification:

FEATURE	TYPE 1 (WITH CATAPLEXY)	TYPE 2 (WITHOUT CATAPLEXY)
Defining criterion	EDS + cataplexy, OR EDS + CSF hypocretin ≤ 110 pg/mL	EDS without cataplexy; normal / absent hypocretin testing
CSF hypocretin-1	< 110 pg/mL (or $< 1/3$ of normal mean)	Normal or not measured
HLA-DQB1*06:02	$> 95\%$ positive	$\sim 40\%$ positive
MSLT sleep latency	< 8 minutes	< 8 minutes
MSLT SOREMPs	≥ 2 sleep-onset REM periods	≥ 2 sleep-onset REM periods
Pathophysiology	Autoimmune destruction of orexin neurons (90% loss)	Unknown; orexin preserved
Prevalence	~ 1 in 2000	Less common

MSLT Criteria for Diagnosis:

A Mean Sleep Latency < 8 minutes AND ≥ 2 Sleep-Onset REM Periods (SOREMPs) on the Multiple Sleep Latency Test, performed the morning after an overnight polysomnogram that confirms adequate sleep duration and excludes other causes of EDS.

Clinical Features (Tetrad):

1. **EDS** (always present): overwhelming urge to sleep, especially in monotonous situations; "sleep attacks"
2. **Cataplexy** (Type 1): sudden bilateral loss of voluntary muscle tone triggered by strong emotion, typically laughter or surprise, without loss of consciousness; lasts seconds to minutes

3. **Sleep paralysis:** transient inability to move at sleep onset or awakening; terrifying but benign; occurs in up to 40% of cases
4. **Hypnagogic/hypnopompic hallucinations:** vivid, often frightening visual, auditory, or tactile hallucinations at sleep onset (hypnagogic) or awakening (hypnopompic)

Management:

SYMPTOM	FIRST-LINE	SECOND-LINE
EDS	Modafinil/ Armodafinil (non-amphetamine wake promoters)	Methylphenidate, amphetamines
Cataplexy	Sodium oxybate (GHB, also treats EDS)	Venlafaxine, SSRIs (suppress REM)
Sleep paralysis and hallucinations	Sodium oxybate	SSRIs, clomipramine
Disrupted nocturnal sleep	Sodium oxybate	

Sodium oxybate (gamma-hydroxybutyrate, GHB):

- GABA-B agonist
- Taken in two divided doses at bedtime (due to short half-life ~2 hours)
- Dramatically improves EDS AND cataplexy
- Consolidates nocturnal sleep, increasing N3
- Side effects: nausea, dizziness, weight loss, enuresis
- Abuse potential, Schedule I in India/USA, tightly regulated prescribing
- Contraindicated with alcohol, CNS depressants, succinic semialdehyde dehydrogenase deficiency

Modafinil:

- Mechanism: inhibits dopamine reuptake (primary), also noradrenaline; wakefulness-promoting
- First-line for EDS in Type 1 and Type 2
- Does not treat cataplexy
- Fewer side effects than amphetamines; no cardiovascular stimulation at therapeutic doses

Non-pharmacological:

- Scheduled daytime naps (2-3 × 15-20 minutes): reduces need for stimulant medication
- Sleep hygiene, consistent sleep schedule
- Driving restrictions: must be disclosed; must be symptom-controlled before driving
- Support groups, vocational counselling

EXAM PEARL

Narcolepsy is lifelong. There is no cure. Management is symptomatic. Orexin replacement therapy is under investigation (intranasal hypocretin) and may represent future disease-modifying treatment for Type 1.

ANSWER 7: ICD-11 vs ICD-10: Major Changes in Psychiatric Classification (10 marks)

Introduction:

The ICD-11 (International Classification of Diseases, 11th Revision) was adopted by the World Health Organisation in May 2019 and came into effect on 1 January 2022. It represents the most comprehensive revision of the classification of mental disorders since ICD-10 (1992). The changes reflect three decades of research, a shift toward clinical utility, and greater alignment with DSM-5.

Structural Changes:

ICD-10 used F-codes (F00–F99) in Chapter V. ICD-11 moved mental disorders to Chapter 06 (Mental, Behavioural, and Neurodevelopmental Disorders), using alphanumeric codes (6A0x–6E8Z). The change has implications for billing and record-keeping once India transitions.

Key Diagnostic Changes:

DOMAIN	ICD-10	ICD-11	CLINICAL SIGNIFICANCE
Schizophrenia subtypes	5 subtypes (paranoid, hebephrenic, catatonic, undifferentiated, residual)	Subtypes removed; replaced by dimensional symptom rating (positive, negative, depressive, manic, psychomotor, cognitive)	Better reflects heterogeneity and treatment planning
PTSD	F43.1, single diagnosis	6B40 PTSD + 6B41 Complex PTSD (new)	C-PTSD captures chronic interpersonal trauma; different treatment implications
Prolonged Grief Disorder	Not a formal diagnosis	6B42, new diagnosis	Grief lasting >6 months with yearning/preoccupation and functional impairment
Binge Eating Disorder	Appendix only (not formal)	6B82, full diagnosis	Insurance coverage; treatment access

DOMAIN	ICD-10	ICD-11	CLINICAL SIGNIFICANCE
ARFID	Not present	6B83, new diagnosis	Avoidant feeding without body image disturbance
Gaming Disorder	Not present	6C51, new (Addictive Behaviour Disorders)	Controversial; requires 12 months of clinical severity
Compulsive Sexual Behaviour Disorder	Not present	6C72, new	Significant controversy; overlap with hypersexuality discourse
Personality Disorders	10 categorical types (F60.x)	Dimensional: Severity (mild/moderate/severe) + 5 Trait Domains + 3 qualifiers	Major paradigm shift; abolishes Axis II categorical diagnoses
Catatonia	Subtype of schizophrenia (F20.2)	Separate chapter (6A4x); can be specified with any condition	Recognises catatonia across diagnostic spectrum
Conversion disorder	F44, Dissociative (conversion) disorders	Moved to Diseases of Nervous System (Functional Neurological Disorder)	Not a dissociative disorder; avoids psychologisation of neurological presentation
Somatoform disorders	F45, multiple categories	Bodily Distress Disorder (6C20), single umbrella	Simplifies; reduces stigmatising language
Acute stress reaction	F43.0, mental disorder	Moved to "Factors influencing health", NOT a mental disorder	Normalises acute stress responses
Hypochondriasis	F45.2	Health anxiety disorder, renamed	Removes pejorative connotation
GID (Transsexualism)	F64, Mental disorder chapter	Gender Incongruence, Chapter 17 (Conditions related to sexual health)	Removed from mental disorders chapter, major destigmatisation

Personality Disorder Revolution:

ICD-11 abolishes the 10 categorical types and replaces them with:

1. **Severity rating:** Mild, Moderate, Severe Personality Disorder
2. **Prominent trait domain specifiers:** Negative Affectivity, Detachment, Dissociality, Disinhibition, Anankastia

3. Borderline pattern qualifier: Optional specifier for BPD-like presentations

EXAM PEARL

Three changes in ICD-11 are most examination-relevant: (1) Schizophrenia subtypes abolished → dimensional, (2) Complex PTSD added as a separate diagnosis, (3) Personality disorders restructured from categorical to dimensional. Know these cold.

ANSWER 8: Eating Disorders in Adolescents: Assessment and Management (10 marks)

Introduction:

Eating disorders most commonly emerge in adolescence and early adulthood, with a bimodal onset in AN at 14 and 18 years. Early identification and intervention in adolescence are crucial given the impact on growth, bone development, and brain maturation. Treatment approaches must be adapted for developmental stage.

Diagnostic Considerations in Adolescents:

- **BMI-for-age** is used (not adult BMI cutoffs); growth faltering is a more sensitive indicator than absolute BMI
- Amenorrhoea may be primary (has never had a period) rather than secondary
- AN in males: more often presents later, less emphasis on "thinness" per se (more often "muscularity"), often via exercise restriction rather than food restriction
- Avoidant eating in childhood/early adolescence may represent **ARFID** rather than AN, absence of body image concerns is key differentiator
- **Rapid-onset restrictive eating** in children ages 6-12: consider PANDAS/PANS-related avoidant eating, tic disorders, OCD presentations

Assessment:

Physical assessment in paediatric patients requires growth chart review:

- Weight-for-age and height-for-age centiles
- Body Mass Index-for-age percentile
- Vital signs: bradycardia and hypotension are particularly concerning in adolescents given their compensatory cardiovascular reserve
- Tanner staging (pubertal assessment)
- Bone age X-ray if growth arrest is suspected
- DEXA scan if eating disorder duration >1 year (osteoporosis risk)

Psychological assessment tools:

- EDE (Eating Disorder Examination), gold standard clinical interview

- EDE-Q (questionnaire version), from age 14 upwards
- CHEAT (Children's Eating Attitudes Test), validated for age 8-13
- RCADS (Revised Children's Anxiety and Depression Scale) for comorbidities

Management:

Family-Based Treatment (FBT / Maudsley Approach):

First-line for adolescent AN; stronger evidence base than individual therapy in this age group.

Three phases:

- Phase 1: Externalise the illness; parents take control of meals; weight restoration
- Phase 2: Return control of eating to the adolescent
- Phase 3: Healthy adolescent development and relapse prevention

Adapted CBT-E:

For adolescents aged 14 and above; shows comparable outcomes to FBT in some studies; preferable when family conflict is high or when parent participation is not feasible.

Inpatient and Day Patient Treatment:

Indications for admission:

- BMI <14 in adults; <10th centile sustained with medical instability in adolescents
- Vital sign instability (bradycardia <45 bpm, hypotension, hypothermia)
- Electrolyte abnormalities, hypoglycaemia
- Rapid weight loss without response to outpatient treatment
- Suicidal risk, severe psychiatric comorbidity

MARSIPAN (Management of Really Sick Patients with Anorexia Nervosa) and Junior MARSIPAN guidelines provide medical management frameworks.

Pharmacotherapy in Adolescents:

- No medication is approved specifically for adolescent AN
- Fluoxetine: first-line for weight-restored adolescents with persistent depressive / anxiety symptoms; less efficacy at low weight (serotonin synthesis requires tryptophan from nutrition)
- Olanzapine: some evidence for weight gain and anxiety reduction in adolescent AN; caution with metabolic side effects
- Avoid bupropion (seizure risk, especially with electrolyte disturbances)

CLINICAL ANCHOR

Weight restoration is a prerequisite for psychological treatment effectiveness. Starvation alters cognition, affects brain structure, and reduces psychological flexibility. Treating the mind before restoring the body is futile, the Minuchin statement that "you can't do psychotherapy with a starving brain" remains clinically relevant.

ANSWER 9: Controversies in DID (Dissociative Identity Disorder) (5 marks)

DID is among the most contested diagnoses in psychiatry. Two major theoretical positions define the debate:

Trauma Model:

Supported by Ross, Putnam, and van der Kolk. Proposes that DID is a response to severe and repeated early childhood trauma, an adaptive dissociative defence that compartmentalises overwhelming experiences. Evidence includes:

- Strong association with reported childhood abuse (97-98%)
- Neuroimaging differences between alter states
- Clinical response to trauma-focused therapy

Sociocognitive Model:

Supported by Spanos, Lilienfeld, and Lynn. Proposes that DID is an iatrogenic and socially constructed phenomenon, shaped by clinician suggestion, hypnosis, popular media, and cultural scripting. Evidence includes:

- Cross-cultural variation (far more prevalent in North America than Western Europe)
- Sharp rise in diagnoses after media coverage (Sybil, Three Faces of Eve)
- Alter system complexity increases with years of treatment
- High rates in patients treated by DID specialists, not in general psychiatric settings
- Alters often reflect cultural expectations (child, abuser, protector)

Current Position:

Most researchers accept a middle ground: genuine trauma-related dissociation exists and forms the substrate; however, symptom expression and complexity are shaped by cultural context, therapeutic suggestion, and media representation. Over-diagnosis and iatrogenic creation are real risks.

Forensic Implications:

The question of criminal responsibility in DID is unresolved. Some jurisdictions have accepted diminished responsibility arguments when the act was committed by an alter state without host awareness. This is inherently problematic given the sociocognitive critique.

EXAM PEARL

In an exam answer, do not come down firmly on one side of this debate. State both positions clearly, note the empirical evidence for each, and conclude that the safest clinical approach involves careful assessment without suggestive techniques.

ANSWER 10: Pharmacotherapy for Insomnia: Indications and Selection (5 marks)

Pharmacotherapy is indicated for **short-term management of acute insomnia** or as an adjunct to CBT-I while awaiting full effect. Chronic use of most hypnotics carries risks of dependence, tolerance, and rebound insomnia.

DRUG CLASS	DRUG	BEST FOR	DURATION	KEY CONCERN
Z-drugs	Zolpidem (5-10mg)	Sleep onset	Short-term (<4 weeks)	Dependence; complex sleep behaviours
Z-drugs	Zopiclone (3.75-7.5mg)	Sleep onset + maintenance	Short-term	Bitter taste; tolerance
Z-drugs	Zaleplon (10mg)	Sleep onset only	Single-dose (shortest $t_{1/2}$)	Ultra-short acting
Benzodiazepines	Temazepam, nitrazepam	All insomnia types	Short-term	Dependence, next-day sedation, fall risk
Melatonin (PR)	Circadin 2mg	Sleep onset; elderly	Up to 13 weeks	Safe; no dependence
Ramelteon	MT1/MT2 agonist	Sleep onset; circadian issues	Longer-term	No dependence potential
Suvorexant/Lemvorexant	Orexin antagonist	Sleep maintenance	Approved for longer use	No dependence; next-day impairment
Doxepin (low dose)	3-6mg	Sleep maintenance	Approved for chronic use	Weight gain at higher doses
Mirtazapine	7.5-15mg	Insomnia + depression	As tolerated	Weight gain, metabolic effects

Special populations:

- **Elderly:** Avoid benzodiazepines and z-drugs (fall risk, cognitive impairment); prefer ramelteon, melatonin, low-dose doxepin, or suvorexant
- **Pregnancy:** Melatonin (limited safety data), short-term z-drugs if necessary; avoid benzodiazepines (neonatal withdrawal)

- **PTSD-related insomnia:** Prazosin (alpha-1 blocker) reduces nightmares specifically; CBT-I adapted for trauma (CBTI-PTSD)

EXAM PEARL

The orexin antagonists (suvorexant, lemborexant) represent the newest mechanism, blocking the wake-promoting orexin signal rather than enhancing sleep. No physical dependence, no tolerance. This is the pharmacological direction of travel for insomnia.

ANSWER 11: REM Sleep Behaviour Disorder: Diagnosis and Management (5 marks)

Definition:

REM Sleep Behaviour Disorder (RBD) is a parasomnia characterised by dream enactment behaviour during REM sleep, resulting from the failure of the normal REM sleep atonia mechanism.

Clinical features:

- Onset: usually males >50 years (though younger-onset forms exist, often idiopathic)
- Behaviour: talking, shouting, punching, kicking, jumping out of bed, correlating with vivid, action-filled dreams
- Patient wakes oriented, with recall of a dream congruent with the behaviour
- Bed partner is often the reporter (and often injured)
- Does NOT occur in N3, behaviour occurs during REM (confirmed by PSG showing REM without atonia)

Investigation:

Video-polysomnography: confirms REM without atonia (RSWA). Minimum criteria: chin EMG activity during REM >50% of epoch, or excessive phasic/tonic EMG.

Association with Neurodegenerative Disease:

RBD is a prodromal marker of alpha-synucleinopathies. Prospective studies show 80-90% of idiopathic RBD patients develop Parkinson's disease, Dementia with Lewy Bodies, or Multiple System Atrophy within 10-15 years. This latency period represents an opportunity for neuroprotective intervention (currently under trial).

Management:

- **Clonazepam 0.25-2 mg at bedtime:** first-line; suppresses RBD behaviour; does NOT restore atonia; may worsen OSA, screen first
- **Melatonin 3-12 mg at bedtime:** alternative / addition; restores some atonia; better tolerated in elderly; no respiratory depression

- **Environmental safety:** pad floor, remove dangerous objects, consider mattress on floor, separate beds if partner at risk

EXAM PEARL

RBD should prompt enquiry about other parkinsonian features (constipation, anosmia, REM latency changes) and referral for neurological follow-up, not because you can cure it, but because the patient may be in the prodromal phase of Parkinson's disease.

ANSWER 12: Suicide: Means Restriction and Postvention (5 marks)

Means Restriction:

One of the most evidence-based suicide prevention strategies. The principle: suicidal crises are often impulsive and time-limited; reducing access to lethal means during a crisis reduces the probability of a fatal outcome.

Evidence base:

- UK legislation limiting paracetamol pack sizes → significant reduction in paracetamol-related deaths (Hawton et al.)
- Barriers on bridges (Golden Gate, UK bridges) → reduced suicide at that location AND total area suicide rate, disproving simple "method substitution"
- Coal gas detoxification in UK (1960s-70s) → 30% reduction in total national suicide rate
- Firearm access: states/ countries with lower firearm access have lower suicide rates; gun storage counselling is a valid clinical intervention

Clinical application:

- All patients assessed for suicide risk should be asked about means access
- For firearm owners: counsel on locked storage or temporary transfer to a third party
- Review medications prescribed (lethality in overdose): SSRIs safer than TCAs; avoid quetiapine/ lithium in large quantities to high-risk patients

Postvention:

Actions following a suicide to support bereaved individuals and prevent contagion:

- Supports suicide loss survivors (bereavement is a risk factor for suicide)
- Institutional postvention (schools, hospitals) following a staff or patient death:
- Avoid glorification, memorialisation that models suicide as honourable
- Active outreach to high-risk contacts
- Structured counselling provision
- No closed-casket speculation or rumour

- **Werther effect:** media reporting of suicide increases imitative suicides (Goethe's novel, 1774)
- **Papageno effect:** media portrayal of successfully navigating suicidal crisis is protective
- WHO Safe Messaging Guidelines: no method details, no site, no suicide note content, include helpline numbers

EXAM PEARL

Postvention is a prevention strategy, not just bereavement support. The suicide of one person can trigger multiple others in a connected community. This is called a "suicide cluster." Safe messaging guidelines and active postvention together reduce cluster risk.

ANSWER 13: Dimensional vs Categorical Classification in Psychiatry (5 marks)

Psychiatric classification has historically been categorical, diagnoses are either present or absent, based on operationalised criteria. This model underpins both ICD and DSM. However, evidence increasingly supports a dimensional alternative.

Arguments for Categorical Classification:

- Clinical utility: categories guide treatment decisions clearly (you start lithium for bipolar, not for "elevated mood dimension score 3")
- Communication: shared language between clinicians, insurers, researchers
- Familiarity: established training paradigms
- Meets patient need for a "name" for their experience

Arguments for Dimensional Classification:

- High rates of comorbidity suggest disorders share variance (e.g., depression and anxiety both on internalising spectrum in HiTOP)
- Arbitrary diagnostic thresholds (e.g., 5 of 9 criteria vs 4, same person?)
- Within-category heterogeneity: two patients with MDD may share few symptoms
- Genetic architecture is dimensional, not categorical (GWAS studies)
- Neurobiology does not break neatly at categorical boundaries
- Better captures subthreshold conditions with clinical significance

Emerging models:

- ICD-11 personality disorders: full dimensional model (severity + trait domains)
- DSM-5 Section III AMPD: alternative dimensional model for personality
- RDoC: biology-based dimensions (circuits, not symptoms)
- HiTOP: empirical hierarchical structure from symptom covariance

Current consensus:

Categorical and dimensional systems serve different purposes. Categorical diagnoses have clinical utility; dimensional descriptions have explanatory and research utility. A hybrid approach, dimensional assessment within clinical categories, may represent the most useful near-term solution.

ANSWER 14: Bulimia Nervosa: Diagnosis and Management (5 marks)

Diagnosis (DSM-5):

Bulimia nervosa is characterised by (1) recurrent binge eating (large amount in discrete period + loss of control), (2) recurrent inappropriate compensatory behaviours, (3) occurring ≥ 1 episode/week for 3 months, (4) self-evaluation unduly influenced by shape/weight, (5) not occurring exclusively during episodes of AN.

Key features not present in AN (binge-purge type):

- Weight is typically normal or overweight (not significantly low)
- Absence of the severe body image distortion/fear of weight gain as central identity

Medical complications:

- Hypokalemia + metabolic alkalosis (vomiting), most dangerous cardiac risk
- Parotid hypertrophy (bilateral; "chipmunk face")
- Perimolysis (dental enamel erosion)
- Russell's sign
- Mallory-Weiss tear (oesophageal mucosal tear from forceful vomiting)
- Ipecac-induced cardiomyopathy (if emetine syrup abused)

Management:

Psychological:

- **CBT-E** (Fairburn): gold standard; 20 sessions; targets core psychopathology (overconcern with shape/weight)
- Dialectical Behaviour Therapy: useful where affect dysregulation drives binges
- Interpersonal Therapy: for BN where interpersonal problems are primary

Pharmacological:

- **Fluoxetine 60 mg/day**: only FDA-approved drug; higher than antidepressant dose; effective for binge-purge cycles
- Topiramate: reduces binge-purge frequency; cognitive dulling limits tolerability
- **Bupropion: CONTRAINDICATED** (lowered seizure threshold + electrolyte disturbances in BN)

Combination:

CBT-E + fluoxetine shows greater efficacy than either alone for acute symptoms.

ANSWER 15: Complex PTSD (ICD-11): Features and Distinction from PTSD (5 marks)

Background:

Complex PTSD (6B41) is a new ICD-11 diagnosis. It was not in ICD-10. DSM-5 does not have it as a separate category (though DESSOS, Disorders of Extreme Stress Not Otherwise Specified, was a DSM-IV proposed category). The addition represents recognition of the distinct clinical presentation following prolonged, repeated, interpersonal trauma.

ICD-11 Criteria:

All three clusters of PTSD (re-experiencing, avoidance, hyperarousal) PLUS three additional domains:

1. **Affect dysregulation:** severe emotional dysregulation; difficulty modulating emotional responses
2. **Negative self-concept:** persistent beliefs of worthlessness, shame, guilt, failure
3. **Disturbances in relationships:** difficulty sustaining relationships, feeling permanently different from others

Contrast with PTSD:

FEATURE	PTSD (6B40)	COMPLEX PTSD (6B41)
Trauma type	Single incident or limited duration	Repeated, prolonged, interpersonal
Examples	Road traffic accident, assault	Childhood abuse, domestic violence, captivity, torture
Core symptoms	Re-experiencing, avoidance, hyperarousal	All of above PLUS affect dysregulation, negative self-concept, relational disturbance
Treatment	Trauma-focused CBT, EMDR	Phase-based; Schema Therapy; more stabilisation before trauma work
Overlap with	PTSD, MDD, anxiety	BPD, attachment disorders, dissociative disorders

Differential from BPD:

Complex PTSD and BPD share affect dysregulation, identity disturbance, and relational difficulties. Key differences: BPD includes impulsivity, chronic suicidality, frantic avoidance

of abandonment as core features; BPD is dimensional and not trauma-dependent (though trauma is common). Both can co-occur.

EXAM PEARL

Complex PTSD is an ICD-11 addition not in ICD-10 or DSM-5. Questions about "new ICD-11 diagnoses" may specifically ask about C-PTSD, Prolonged Grief Disorder, Gaming Disorder, or ARFID. Know all four.

Mnemonics & Memory Tricks

All mnemonics are clinically grounded. Each includes the device, decoded table, and a brief usage note.

MNEMONIC 1: Medical Complications of Anorexia Nervosa: "CARDIAC BONES"

EXAM PEARL

Organising AN complications by this mnemonic lets you systematically cover all body systems in a 10-mark answer without missing any.

LETTER	SYSTEM	KEY COMPLICATION
C	Cardiovascular	Bradycardia, QTc prolongation, cardiac atrophy, hypotension, MVP
A	Amenorrhoea / Endocrine	Amenorrhoea, low LH/FSH, low estrogen, sick euthyroid, high cortisol
R	Renal	Prerenal azotaemia, hypokalemic nephropathy, renal calculi
D	Dental / Dermatological	Perimolysis (binge-purge), Russell's sign, lanugo, carotenemia
I	Immunological / Haematological	Pancytopenia (anaemia, leukopenia, thrombocytopenia)
A	Alimentary (GI)	Delayed gastric emptying, SMA syndrome, elevated LFTs, constipation
C	Cognitive / Neurological	Cortical atrophy, peripheral neuropathy, cognitive impairment
B	Bones (Musculoskeletal)	Osteoporosis, osteopenia, stress fractures, growth arrest in adolescents

LETTER	SYSTEM	KEY COMPLICATION
O	Oedema / Metabolic	Hypokalaemia, hyponatraemia, metabolic alkalosis/acidosis, hypoglycaemia
N	Neuroendocrine	Low GH sensitivity (high GH, low IGF-1), growth retardation
E	Electrolytes	Hypophosphataemia (refeeding risk), hypomagnesaemia
S	Serum / Blood	Raised urea, raised amylase (parotid), low albumin

MNEMONIC 2: Refeeding Syndrome Electrolytes: "PKMGT" ("Please Keep Magnesium Going Too")

LETTER	ELECTROLYTE	DIRECTION IN REFEEDING	CONSEQUENCE
P	Phosphate	Falls sharply (critical)	Respiratory failure, cardiac failure, seizures
K	Potassium	Falls	Arrhythmias, cardiac arrest
M	Magnesium	Falls	Tetany, arrhythmias, impairs K ⁺ correction
G	Glucose	Rises	Osmotic diuresis; drives insulin → drives P/K/Mg in
T	Thiamine	Consumed	Wernicke's encephalopathy if not supplemented

EXAM PEARL

The mechanism is insulin-driven shift INTO cells. Serum levels drop even though the total body deficit pre-existed. "Thiamine before glucose" is the clinical anchor.

MNEMONIC 3: Suicide Risk Assessment: SAD PERSONS

LETTER	FACTOR	NOTES
S	Sex (male)	Males complete; females attempt more

LETTER	FACTOR	NOTES
A	Age (<19 or >45)	Bimodal risk; older men and adolescents
D	Depression	Core psychiatric risk factor
P	Previous attempt	Single strongest predictor of completion
E	Ethanol / substance abuse	Disinhibition, impulsivity, access
R	Rational thinking loss	Psychosis, command hallucinations
S	Social supports lacking	Isolation, living alone
O	Organised plan	Specificity of plan increases risk
N	No spouse (social isolation)	Unmarried, widowed, divorced > married
S	Sickness (physical illness)	Chronic pain, terminal illness, disfigurement

Scoring: 0-4 low, 5-6 moderate, 7-10 high risk.

EXAM STRATEGY

Know SAD PERSONS for the mnemonic, but in clinical answer sections note that the Columbia Protocol (C-SSRS) has better clinical utility and is preferred in current practice.

MNEMONIC 4: Joiner's Interpersonal Theory: "TBA" ("Thinking-Believing-Acting")

LETTER	COMPONENT	MEANING
T	Thwarted Belongingness	"I don't belong anywhere", social alienation
B	Perceived Burdensomeness	"I am a burden; my death would help others"
A	Acquired Capability	Habituation to pain/fear; prior attempts, trauma, violence

EXAM PEARL

T + B = desire; A = capability. All three together = maximum risk. Clinical interview asks: "Do you feel like a burden to your family?" and "Do you feel you belong anywhere?"

MNEMONIC 5: Narcolepsy Tetrad: "CHESS" (Cataplexy, Hallucinations, EDS, Sleep Paralysis, Sleep attacks)

Or more precisely, use: "ECS-H" to remember that EDS is obligate:

FEATURE	OBLIGATE?	DISTINGUISHES TYPE 1 FROM TYPE 2?
Excessive Daytime Sleepiness	Yes, always present	No (both types)
Cataplexy	No	Yes, Type 1 only
Sleep Paralysis	No	No (both types, also normal population)
Hallucinations (hypnagogic/pompic)	No	No

EXAM PEARL

Cataplexy is triggered by emotion (laughter, surprise, anger). Sudden bilateral muscle tone loss WITH preserved consciousness. Do not confuse with epileptic drop attacks (LOC present) or syncope (haemodynamic mechanism).

MNEMONIC 6: Sleep Stage Neurotransmitters: "SANG" (Sleep = Adenosine, NREM = Serotonin/GABA, Go = Glutamate-wake, ACh = REM)

NEUROTRANSMITTER	FUNCTION	MEMORY HOOK
Adenosine	Homeostatic sleep drive	Caffeine blocks → keeps you awake
GABA	Sleep-promoting (all stages)	Benzos / z-drugs work here
Serotonin	NREM promotion (raphe nuclei)	SSRIs suppress REM; promote NREM
Noradrenaline	Wake-promoting (locus coeruleus)	Shuts off in REM (atonia mechanism)
Acetylcholine	REM generator (PPT nucleus)	"REM = ACh Dream Mechanism"
Orexin/Hypocretin	Wake stability	Loss → narcolepsy Type 1
Histamine	Wake-promoting (TMN)	H1 antihistamines → sedation

MNEMONIC 7: NREM vs REM Parasomnias: "NREM = Night Terror, No Recall; REM = Remember Every Monster"

FEATURE	NREM PARASOMNIAS	REM PARASOMNIAS
Stage	N3 (slow wave)	REM
Time of night	First third	Last third
Dream recall	Absent or minimal	Vivid, detailed
Arousal state	Partial, confused	Full awakening after episode
Autonomic activation	High (night terrors: screaming, tachycardia)	Moderate
Examples	Sleepwalking, night terrors, confusional arousals	RBD, nightmare disorder
Eye opening	Yes (glazed)	After waking
Memory	Amnesia for episode	Full recall of dream content

MNEMONIC 8: Dissociative Disorder Subtypes: "AFTIP-D"

LETTER · DISORDER

A Amnesia (dissociative)

F Fugue (now specifier of amnesia in DSM-5)

T Trance disorder

I Identity disorder (DID) / Partial DID

P Possession trance disorder

D Depersonalisation/Derealization disorder

MNEMONIC 9: ICD-11 New Diagnoses: "CAPE-GAP"

LETTER · NEW ICD-11 DIAGNOSIS

C Complex PTSD (6B41)

A ARFID (6B83)

P Prolonged Grief Disorder (6B42)

E (C-PTSD already covered), use for Emotional: Complex PTSD affect cluster

G Gaming Disorder (6C51)

A ARFID (reinforcer)

P Personality Disorders, dimensional restructuring

Simplified version: "**CAPE-G**" = Complex PTSD, ARFID, Prolonged Grief, (CSBD = Compulsive Sexual Behaviour Disorder), Gaming Disorder

MNEMONIC 10: Suicide Risk Factors: "IS PATH WARM" (American Association of Suicidology)

LETTER · RISK FACTOR

I Ideation

S Substance abuse

P Purposelessness (lack of reasons to live)

A Anxiety / agitation

T Trapped (feeling there is no way out)

H Hopelessness

W Withdrawal (social isolation)

A Anger

R Recklessness

M Mood changes (especially recent rapid change)

EXAM PEARL

IS PATH WARM is particularly useful as a brief screening tool. It covers dynamic (changeable) risk factors, more useful for monitoring change over time than SAD PERSONS.

MNEMONIC 11: CBT-I Components: "SRCRC" ("Sleep Restriction Creates Real Change")

LETTER	COMPONENT	WHAT IT DOES
S	Sleep Restriction	Consolidates fragmented sleep; builds drive

LETTER	COMPONENT	WHAT IT DOES
R	Relaxation	Reduces physiological hyperarousal
C	Stimulus Control	Breaks bed-arousal conditioning
R	(Cognitive) Restructuring	Challenges catastrophic sleep beliefs
C	(Sleep) Hygiene / Chronotherapy	Removes perpetuating environmental factors

MNEMONIC 12: Refeeding Syndrome Prevention: "BELT" ("Before Every Litre, Thiamine")

STEP · ACTION

B Baseline bloods before starting (phosphate, Mg, K, glucose, renal function)

E Electrolyte replacement proactively, not reactively

L Low caloric start: 20 kcal/kg/day (10 if very high risk)

T Thiamine BEFORE glucose, give before carbohydrate loading

MNEMONIC 13: Anorexia Nervosa: Medical Criteria for Admission "BEACH"

LETTER · CRITERION

B Bradycardia (<50 bpm; some guidelines <45 bpm)

E Electrolytes dangerously low (K <3, PO₄ <0.5, Mg low)

A Anorexia with BMI <14 (adult) or <70% expected weight

C Cardiac, QTc >450ms, syncope, ECG changes

H Hypoglycaemia / Hypotension (systolic <80)

MNEMONIC 14: Eating Disorders: Pharmacotherapy Rules "FFB"

DRUG · RULE

Fluoxetine Only FDA-approved drug for Bulimia Nervosa (60 mg/day)

Fluoxetine Also used in weight-restored AN for relapse prevention

Bupropion BANNED in both AN and BN, seizure risk (electrolyte depletion)

Memory hook: "**Fluoxetine for Bulimia, Bupropion is Banned.**"

MNEMONIC 15: DID Management Phases: "SPI" ("Stabilise, Process, Integrate")

PHASE	FOCUS	MNEMONIC
Phase 1	Safety and Stabilisation	S , Safety first
Phase 2	Trauma Processing	P , Process carefully
Phase 3	Integration	I , Integrate alters

EXAM PEARL

The most common error in DID management is jumping from Phase 1 to Phase 2 prematurely. Trauma processing before stabilisation DESTABILISES, increases self-harm, suicidality, hospitalisation rates.

MNEMONIC 16: Protective Factors Against Suicide: "FAMILY"

LETTER · PROTECTIVE FACTOR

F Faith / Religious beliefs opposing suicide

A Attachment to children, pets, dependants

M Moral objection to suicide

I Invested relationships (family, spouse)

L Life goals / future orientation

Y "Yes to life", problem-solving capacity, hope

MNEMONIC 17: MSLT Criteria for Narcolepsy: "28 SOREMPs"

Criteria: Mean sleep latency <8 minutes AND ≥ 2 SOREMPs (Sleep Onset REM Periods)

Memory hook: "**Less than 8, at least 2**", reverse of what you'd expect (less sleep latency = more sleepy, more REM onsets = more narcoleptic)

MNEMONIC 18: RBD Features: "DRAMA"

LETTER · FEATURE

- D** Dream enactment (acting out dreams)
- R** REM sleep confirmed on PSG (REM without atonia)
- A** Alpha-synucleinopathy prodrome (PD, DLB, MSA)
- M** Males predominantly affected, Middle age and older
- A** Awareness during episode (wakes oriented; recalls dream)

MNEMONIC 19: Key ICD-11 Schizophrenia Changes: "SAND"

LETTER · CHANGE

- S** Subtypes abolished (paranoid, hebephrenic, catatonic, undifferentiated, residual, gone)
- A** Antonym: dimensional symptom domains ADDED instead
- N** New domains: Positive, Negative, Depressive, Manic, Psychomotor, Cognitive
- D** Duration criterion unchanged: still 1 month

MNEMONIC 20: Circadian Rhythm Disorders: "DASON"

LETTER · DISORDER

- D** Delayed Sleep-Wake Phase (night owl; can't sleep before 2 AM)
- A** Advanced Sleep-Wake Phase (early bird; sleepy at 6 PM)
- S** Shift Work Disorder
- O** Other / Irregular sleep-wake rhythm
- N** Non-24-hour sleep-wake (blind individuals; tasimelteon approved)

QUICK RECALL CARDS

What's the single strongest predictor of completed suicide?

→ Previous suicide attempt

What's the only FDA-approved drug specifically for suicidality?

→ Clozapine (for schizophrenia / schizoaffective)

What drug has the strongest evidence for reducing completed suicide?

→ Lithium

What's the first-line treatment for chronic insomnia?

→ CBT-I (over pharmacotherapy)

What electrolyte disturbance is the hallmark of refeeding syndrome?

→ Hypophosphataemia

What's the diagnostic criterion that distinguishes narcolepsy Type 1 from Type 2?

→ Cataplexy OR CSF hypocretin-1 ≤ 110 pg/mL

What must come BEFORE carbohydrate reintroduction in a malnourished patient?

→ Thiamine supplementation

What's the only drug approved for both EDS AND cataplexy in narcolepsy?

→ Sodium oxybate (GHB)

What parasomnia is a prodrome for Parkinson's disease?

→ REM Sleep Behaviour Disorder (RBD)

What drug is CONTRAINDICATED in both AN and BN?

→ Bupropion

What psychiatric disorder has the highest standardised mortality ratio (SMR)?

→ Anorexia Nervosa (~5.86)

What is the "Werther Effect"?

→ Increased suicide rates following media reporting of a suicide

What is the "Papageno Effect"?

→ Protective effect of media stories showing suicidal crisis overcome by coping

What is the key ICD-11 change to personality disorders?

→ Categorical types abolished → Dimensional: severity + trait domains

Name 3 new ICD-11 diagnoses:

→ Complex PTSD, Prolonged Grief Disorder, Gaming Disorder (also: ARFID, CSBD)

High-Yield Comparisons

High-density visual reference. Each table is designed to be scannable in under 60 seconds.

TABLE 1: Anorexia Nervosa vs Bulimia Nervosa vs Binge Eating Disorder

FEATURE	ANOREXIA NERVOSA	BULIMIA NERVOSA	BINGE EATING DISORDER
ICD-11 code	6B80	6B81	6B82
Core feature	Restriction → significantly low weight	Binge-purge cycles; weight normal/overweight	Binge eating without compensatory behaviours
Weight	Significantly low (BMI typically <17.5, though no longer a formal criterion)	Normal or overweight	Normal, overweight, or obese
Body image disturbance	Central: overestimation of size, weight/shape-based self-worth	Present: undue influence of shape/weight on self-evaluation	Present but less severe
Compensatory behaviours	Restricting (primary); vomiting/laxatives in binge-purge subtype	Always present: vomiting, laxatives, diuretics, fasting, excess exercise	Absent (defining feature)
Subtypes	Restricting; Binge-purge	Mild/moderate/severe/extreme (by frequency)	Mild/moderate/severe/extreme
Amenorrhoea	Common (not a DSM-5 criterion)	Not typical	Not typical
Medical risk	Highest (cardiac, electrolyte, osteoporosis)	Electrolyte (hypokalaemia), dental	Lower than AN; metabolic syndrome risk
Mortality (SMR)	~5.86, highest of any psychiatric disorder	~1.9	~1.5
First-line psychotherapy	CBT-E + nutritional rehabilitation (FBT in adolescents)	CBT-E	CBT-E, IPT

FEATURE	ANOREXIA NERVOSA	BULIMIA NERVOSA	BINGE EATING DISORDER
First-line pharmacotherapy	None approved; olanzapine (weight restoration adjunct)	Fluoxetine 60 mg/day (FDA approved)	Lisdexamfetamine (FDA approved)
Contraindicated drug	Bupropion	Bupropion	
Onset	Peak: 14 and 18 years	Peak: late adolescence/early adulthood	Broader age range; often adult
Sex ratio (F:M)	10:1	10:1	3:2
Prevalence (lifetime)	~0.5–1% women	~1–3% women	~3.5% women, ~2% men

EXAM PEARL

The three eating disorders form a continuum of binge-purge behaviour. AN binge-purge subtype and BN share purging but differ by weight. BN and BED share bingeing but differ by compensatory behaviour. These distinctions are classic short-answer fodder.

TABLE 2: ICD-11 vs ICD-10 vs DSM-5: Major Differences

DOMAIN	ICD-10 (F CODES)	ICD-11 (6A-6E CODES)	DSM-5
Schizophrenia subtypes	5 subtypes (paranoid, hebephrenic, catatonic, undifferentiated, residual)	Abolished; replaced by 6 dimensional symptom domains	5 types retained but rarely used in practice
PTSD	Single diagnosis (F43.1)	PTSD (6B40) + Complex PTSD (6B41), separate	Single diagnosis; broadened criteria; no C-PTSD
BED	Not a full diagnosis	Full diagnosis (6B82)	Full diagnosis (added in 2013)
ARFID	Not present	New diagnosis (6B83)	New diagnosis (added in 2013)
Prolonged Grief Disorder	Not present	New diagnosis (6B42)	Not present (DSM-5-TR added as Prolonged Grief Disorder in 2022)
Gaming Disorder	Not present	New diagnosis (6C51)	Not present (listed for further study)

DOMAIN	ICD-10 (F CODES)	ICD-11 (6A-6E CODES)	DSM-5
Personality disorders	10 categorical types (F60.x)	Dimensional: severity + 5 trait domains + borderline qualifier	Categorical (main text); dimensional in Section III (AMPD)
Catatonia	Subtype of schizophrenia (F20.2)	Separate specifier / chapter; can occur with any disorder	Separate specifier applied across diagnoses
Conversion disorder	F44, Dissociative (conversion) disorders	Functional Neurological Disorder, Diseases of Nervous System chapter	Functional Neurological Symptom Disorder (retained in somatic chapter)
Somatoform disorders	F45, multiple subtypes	Bodily Distress Disorder (6C20), simplified umbrella	Somatic Symptom Disorder (simplified from DSM-IV)
Gender Identity	F64, Mental Disorders chapter	Chapter 17, Conditions related to sexual health (not a mental disorder)	Gender Dysphoria, retained but in own chapter
Acute stress reaction	F43.0, Mental disorder	Moved to health factors, NOT a mental disorder	Acute Stress Disorder, remains a mental disorder
Hypochondriasis	F45.2, Hypochondriacal disorder	Health Anxiety Disorder (renamed)	Illness Anxiety Disorder (renamed)
Multiaxial system	Five axes (ICD-10 MAS)	No multiaxial system	Abolished in DSM-5
Cultural formulation	Minimal	Enhanced cultural considerations	Cultural Formulation Interview (CFI) added
Bereavement exclusion (MDD)	Present		Removed in DSM-5 (controversial)
Code structure	F00–F99 (alphanumeric)	6A0x–6E8Z (alphanumeric; different numbering)	NNN.N numeric codes

EXAM STRATEGY

When asked to compare ICD-11 and ICD-10, structure your answer in three tiers: (1) Structural changes, (2) New diagnoses added, (3) Existing diagnoses modified. This ensures comprehensive coverage.

TABLE 3: Insomnia Pharmacotherapy Options

WIDE COMPARISON · LANDSCAPE

DRUG	CLASS	MECHANISM	SLEEP LATENCY	SLEEP MAINTENANCE	DURATION OF USE	KEY CONCERN
Zolpidem 5–10mg	Z-drug	GABA-A BZ1 sub-unit PAM	Yes	Partial	<4 weeks	Dependence; complex sleep behaviours (sleepwalking, sleep-driving)
Zopiclone 3.75–7.5mg	Z-drug	GABA-A PAM	Yes	Yes	<4 weeks	Bitter taste; tolerance
Zaleplon 10mg	Z-drug	GABA-A BZ1 PAM	Yes	No (t½ ~1h)	Single dose only	Ultra-short action; useful for middle-of-night awakening
Temazepam 10–20mg	Benzodiazepine	GABA-A PAM (non-selective)	Yes	Yes	Short-term only	Dependence; hang-over; fall risk in elderly
Nitrazepam 5–10mg	Benzodiazepine	GABA-A PAM	Yes	Yes	Short-term only	Long half-life; day-time sedation
Melatonin PR (Circadin) 2mg	Melatonin agonist	MT1/MT2 agonist	Yes	Modest	Up to 13 weeks (licensed)	Safe; no dependence; circadian benefits
Ramelteon 8mg	Melatonin agonist	MT1/MT2 agonist	Yes	No	Longer term	No dependence; minimal side effects
Suvorexant 10–20mg	Orexin antagonist	DORA (blocks OX1R + OX2R)	Yes	Yes	Licensed for longer use	No dependence; next-day driving impairment
Lemborexant 5–10mg	Orexin antagonist	DORA	Yes	Yes	Licensed for longer use	Similar to suvorexant; better titration flexibility
Doxepin 3–6mg	TCA (H1 antagonist at low dose)	H1 blockade	No	Yes, specifically for sleep maintenance	Licensed specifically for sleep maintenance	Weight gain at higher doses; cardiac at higher doses

DRUG	CLASS	MECHANISM	SLEEP LATENCY	SLEEP MAINTENANCE	DURATION OF USE	KEY CONCERN
Mirtazapine 7.5–15mg	NaSSA	H1 + 5-HT2A/2C antagonism	Yes	Yes	As tolerated	Weight gain, metabolic; useful with depression
Trazodone 25–100mg	SARI	H1 + 5-HT2 antagonism	Yes	Yes	Off-label use	Priapism (rare); useful with depression/anxiety

EXAM PEARL

Suvorexant/lemborexant work by a completely different mechanism from all others, they **BLOCK** wakefulness (orexin antagonism) rather than **PROMOTE** sleep (GABA-A activation). No physical dependence, no tolerance. This is the pharmacological paradigm shift in insomnia treatment.

TABLE 4: Narcolepsy Type 1 vs Type 2

FEATURE	TYPE 1 (WITH CATAPLEXY)	TYPE 2 (WITHOUT CATAPLEXY)
Essential criterion	EDS + cataplexy OR EDS + CSF hypocretin ≤ 110 pg/mL	EDS without cataplexy; CSF hypocretin normal or not tested
Cataplexy	Present (pathognomonic)	Absent
CSF hypocretin-1	< 110 pg/mL (or $< 1/3$ of control mean)	Normal (> 110 pg/mL)
HLA-DQB1*06:02	$> 95\%$ positive	$\sim 40\%$ positive
MSLT sleep latency	< 8 minutes	< 8 minutes
MSLT SOREMPs	≥ 2	≥ 2
Pathophysiology	Autoimmune loss of hypothalamic orexin neurons (90% reduction)	Unknown; orexin intact
Trigger (Type 1)	H1N1 influenza / Pandemrix vaccine $\rightarrow$ autoimmune destruction	
Prevalence	$\sim 0.05\%$ (1 in 2000)	Less common
Stability of diagnosis	Stable	Can convert to Type 1 if cataplexy develops
Treatment: EDS	Modafinil/armodafinil; sodium oxybate	Modafinil/armodafinil
Treatment: Cataplexy	Sodium oxybate (first-line); venlafaxine; SSRIs	Not applicable
Prognosis	Lifelong; generally stable	May evolve to Type 1

TABLE 5: Dissociative Amnesia vs Organic (Neurological) Amnesia

FEATURE	DISSOCIATIVE AMNESIA	ORGANIC AMNESIA
Onset	Acute; often linked to psychosocial trauma	May be acute (TGA, stroke) or insidious (dementia)

FEATURE	DISSOCIATIVE AMNESIA	ORGANIC AMNESIA
Memory pattern	Retrograde predominant (past autobiographical)	Anterograde predominant (new learning fails)
Content lost	Selective: emotionally significant autobiographical material	Non-selective: recent events, factual learning
EEG	Normal	May be abnormal (TGA: normal; epilepsy: abnormal)
Neuroimaging	Normal	May show lesion (stroke, tumour, atrophy)
Consistency	May fluctuate; inconsistent performance on memory testing	Consistent across testing conditions
Reaction to amnesia	La belle indifférence possible; or distress	Usually distress; sometimes anosognosia (in Korsakoff's)
Recovery	Often spontaneous; may recover with therapy	Depends on cause; anterograde amnesia rarely recovers
Confabulation	Rare	Classic in Korsakoff syndrome
Implicit memory	Usually intact	Varies by brain structure involved
IQ and other functions	Preserved	May be impaired
Secondary gain	May be present	Absent

Specific organic comparators:

CONDITION · FEATURES DISTINGUISHING FROM DISSOCIATIVE AMNESIA

Transient Global Amnesia Middle-aged/elderly, sudden onset, complete recovery in <24h, anterograde prominent, repetitive questioning, no trauma link

Korsakoff syndrome Anterograde > retrograde, confabulation, associated with Wernicke's (nystagmus, ataxia), chronic alcohol use, thiamine deficiency

Epileptic amnesia Automatisms, post-ictal confusion, EEG abnormality

Dementia Progressive, multiple cognitive domains, no trauma link

TABLE 6: Completed Suicide vs Attempted Suicide vs Deliberate Self-Harm (DSH/NSSI)

FEATURE	COMPLETED SUICIDE	ATTEMPTED SUICIDE	DELIBERATE SELF-HARM / NSSI
Outcome	Death	Survival	Survival (no suicidal intent)
Intent	To die	To die (partial/ ambivalent)	Not to die; emotion regulation or other function
Method lethality	High	Variable (does not always correlate with intent)	Low (cutting, superficial burning)
Sex predominance	Male > female (3:1)	Female > male (3:1)	Female > male (in adolescence)
Psychiatric diagnosis	Major depression, bipolar, schizophrenia, alcohol use disorder	Same, plus BPD, PTSD	BPD (commonest), depression, PTSD, anxiety disorders
Rescue likelihood	Low or zero	Higher	High (often communicative function)
Function	Escape from psychache	Escape with ambivalence	Emotion regulation, anti-dissociation, self-punishment, communication
Predictive value	Terminal	Strong predictor of eventual completion	Moderate predictor; distinct phenomenon
Assessment tool	C-SSRS (ideation + behaviour)	C-SSRS	Functional Assessment of Self-Mutilation (FASM)
Management	Postvention	Hospitalisation if high risk; safety plan	DBT; emotion regulation; harm reduction
ICD-11 coding	QA (external cause)	QA	

EXAM PEARL

Lethality and intent do NOT always correlate. High lethality attempt may be impulsive with ambivalent intent. Low lethality attempt may follow highly planned, resolved suicidal ideation (rescuer arrived unexpectedly). Always assess intent separately from method.

TABLE 7: CBT-I vs Pharmacotherapy for Insomnia

FEATURE	CBT-I	PHARMACOTHERAPY
Onset of effect	4–6 weeks (gradual)	Days (rapid)
Long-term efficacy	Superior; sustained at 12+ months	Reduced on discontinuation; rebound insomnia
Mechanism	Addresses perpetuating factors (behavioural, cognitive, physiological)	Suppresses CNS arousal / promotes sedation
Dependence risk	None	Benzodiazepines, z-drugs: significant; orexin antagonists / melatonin: minimal
Tolerability	Initial worsening (sleep restriction phase); requires motivation	Immediate; less effort required
Access	Requires trained therapist or dCBT-I app	Widely available; lower threshold to prescribe
Cost	Higher upfront (therapist); digital = low cost	Lower upfront
Appropriate for	Chronic insomnia; comorbid insomnia; elderly; pregnant	Acute insomnia; adjunct to CBT-I; rapid relief needed
Rebound insomnia	No	Yes (benzodiazepines, z-drugs)
Guidelines recommendation	First-line (NICE, AASM, ERS)	Second-line or short-term adjunct
Contraindications	Seizure disorder (sleep restriction); severe sleep deprivation in at-risk occupations	Benzodiazepines: elderly, COPD, sleep apnoea; z-drugs: parasomnias
Digital delivery	Yes (Sleepio, Somryst), comparable to face-to-face	N/A

TABLE 8: NREM Parasomnias vs REM Parasomnias

FEATURE	NREM PARASOMNIAS (DISORDERS OF AROUSAL)	REM PARASOMNIAS
Stage of sleep	N3 (slow wave sleep)	REM sleep
Time of night	First third (N3 predominates early)	Last third (REM predominates late)
Examples	Sleepwalking, sleep terrors, confusional arousals	RBD, nightmare disorder, sleep paralysis

FEATURE	NREM PARASOMNIAS (DISORDERS OF AROUSAL)	REM PARASOMNIAS
Dream recall	Absent or minimal (vague fear, no narrative)	Vivid, detailed, action-filled
Autonomic arousal	High in sleep terrors (tachycardia, diaphoresis, screaming)	Moderate
Muscle tone during episode	Present (can walk, perform complex behaviours)	Absent in RBD (failure of normal atonia)
Consciousness during episode	Partial, glazed eyes, unresponsive	Variable, RBD: appears purposeful but asleep
Morning recall of episode	Amnesia (complete for sleep terrors, partial for sleepwalking)	Full recall of dream (RBD); full recall after waking (nightmares)
Age predominance	Children (peak 5–8 years) for sleepwalking/night terrors	Adults/elderly for RBD; any age for nightmares
Neurodegenerative association	None	RBD: strong association with alpha-synucleinopathies
PSG finding	N3 arousal with persisting motor activity	REM without atonia (RSWA) in RBD
Treatment	Safety measures; clonazepam if disruptive	Clonazepam or melatonin (RBD); CBT/prazosin (nightmares)
Precipitants	Sleep deprivation, febrile illness, stress, alcohol	Medications (SSRIs, TCAs, MAOIs), narcolepsy, brainstem lesions

CLINICAL ANCHOR

SSRIs suppress REM atonia and can precipitate or worsen RBD. When a patient on an SSRI reports acting out dreams, consider SSRI-induced RBD, dose reduction or switch may resolve it.

TABLE 9: Joiner's Interpersonal Theory vs Klonsky's Three-Step Theory vs Shneidman's Psychache Theory

FEATURE	JOINER'S IPTS	KLONSKY'S 3ST	SHNEIDMAN'S PSYCHACHE
Core concept	Thwarted belongingness + perceived burdensomeness + acquired capability	Pain > connectedness → ideation; capability → attempt	Intolerable psychological pain ("psychache")

FEATURE	JOINER'S IPTS	KLONSKY'S 3ST	SHNEIDMAN'S PSYCHACHE
Mechanism	Three components must all be present for high-lethality attempt	Sequential: pain → ideation → attempt (gated by capability)	Suicide as escape from unbearable inner suffering
Strengths	Operationalisable; testable; clinical interview items	More parsimonious; explains ideation escalation clearly	Phenomenological; captures subjective experience
Limitations	Less explanatory of ideation development	Capability component less well operationalised	Not easily quantifiable; lacks clinical specificity
Clinical application	Ask: "Do you feel a burden?" "Do you feel you belong?" "Have you been exposed to pain/violence?"	Track: Is pain outweighing reasons to live?	Ask: "What is the pain you feel most unbearable to live with?"
Research support	Strong; many replications	Growing	Foundational; descriptive

TABLE 10: Sleep Architecture Changes in Major Psychiatric Disorders

WIDE COMPARISON · LANDSCAPE

DISORDER	SLEEP LATENCY	TOTAL SLEEP TIME	SWS (N3)	REM LATENCY	TOTAL REM	AWAKENINGS
Major Depression	Increased	Decreased or increased	Decreased	Shortened (<65 min)	Increased	Frequent
Mania	Decreased (less need)	Markedly decreased	Variable	May be shortened	Variable	Variable
Bipolar Depression	Increased	Often increased (hypersomnia)	Variable	Shortened	Increased	Frequent
Schizophrenia	Increased	Decreased	Reduced	Mildly reduced	Preserved or reduced	Frequent
PTSD	Increased	Decreased	Reduced	Variable	Increased nightmares	Very frequent
GAD	Increased	Decreased	Reduced	Normal	Normal	Frequent
Alcohol dependence (acute)	Decreased (sedative)	Increased N1/N2	Suppressed	Shortened	Suppressed initially; rebound on withdrawal	
Alcohol withdrawal	Increased	Decreased	Reduced	Shortened (REM rebound)	Increased	Constant
Alzheimer's Dementia	Increased	Decreased	Markedly reduced	Normal early → shortened late	Reduced	Constant (sundowning)

EXAM PEARL

The most tested sleep change in psychiatry is **shortened REM latency in depression**. It occurs in ~60% of patients with major depression, is also found in first-degree relatives (biological marker), and normalises with effective antidepressant treatment. All antidepressants (except bupropion) suppress REM.

SUMMARY MATRIX: Key Drugs in This Chapter

DRUG	DISORDER	ROLE	MECHANISM	KEY FACT
Fluoxetine 60mg	Bulimia nervosa	FDA approved	5-HT reuptake inhibition	Only approved drug for BN; higher than antidepressant dose
Lisdexamfetamine	BED	FDA approved	Amphetamine prodrug; dopamine/NA	Also used for ADHD
Olanzapine	AN (adjunct)	Weight restoration	D2/H1 antagonism	Reduces anxiety; weight gain
Sodium oxybate	Narcolepsy Type 1	EDS + cataplexy	GABA-B agonist	Only drug for both symptoms
Modafinil	Narcolepsy; hypersomnia; shift work	EDS	DAT inhibition	First-line for EDS; no cataplexy effect
Clonazepam	RBD; NREM parasomnias	Behaviour suppression	GABA-A PAM	Does not restore REM atonia
Melatonin	RBD; insomnia; circadian disorders	Multiple	MT1/MT2 agonism	Restores partial atonia in RBD
Suvorexant	Insomnia	Sleep maintenance	DORA (OX1R+OX2R block)	Novel mechanism; no dependence
Lithium	Bipolar suicide prevention	Anti-suicidal	Complex (GSK3 β , neuroprotective)	80% reduction in completed suicide
Clozapine	Schizophrenia suicidality	Anti-suicidal (FDA)	D4/5-HT2A	Only FDA-approved anti-suicidal indication
Ketamine/esketamine	Acute suicidal crisis	Rapid effect	NMDA antagonism	Effect within hours

DRUG	DISORDER	ROLE	MECHANISM	KEY FACT
Prazosin	PTSD nightmares	Nightmare reduction	Alpha-1 adrenergic blockade	Specifically targets stress-response nightmares
Naltrexone	DPDR (off-label)	Depersonalisation reduction	Mu-opioid antagonism	Open-label evidence only
Pramipexole	RLS	First-line	D3 agonist	Watch for augmentation
Tasimelteon	Non-24h sleep-wake	Circadian entrainment	MT1/MT2 agonism	FDA approved specifically for non-24h disorder in blind

PYQ Frequency Analysis

Based on pattern analysis of PG exams MD Psychiatry Paper II questions across available years. Frequency ratings reflect relative appearance across years; all topics remain examinable.

FREQUENCY HEAT MAP

TOPIC	ESTIMATED FREQUENCY	MARKS RANGE	PRIORITY
Suicide risk assessment	Very High	5–10	MUST KNOW
Anorexia nervosa, medical complications	Very High	5–10	MUST KNOW
Refeeding syndrome	High	5–10	MUST KNOW
ICD-11 vs ICD-10 changes	High	5–10	MUST KNOW
Narcolepsy, classification and management	High	5–10	MUST KNOW
CBT-I	High	5–10	MUST KNOW
Dissociative disorders, classification	High	5–10	MUST KNOW
Bulimia nervosa, diagnosis and management	Moderate–High	5–10	HIGH
REM sleep behaviour disorder	Moderate–High	5	HIGH
DID, controversies	Moderate	5	HIGH
DPDR	Moderate	5	HIGH
Eating disorders in adolescents	Moderate	5–10	HIGH
Sleep architecture and stages	Moderate	5	HIGH
Insomnia pharmacotherapy	Moderate	5	HIGH

TOPIC	ESTIMATED FREQUENCY	MARKS RANGE	PRIORITY
Means restriction in suicide	Moderate	5	MEDIUM
Postvention	Low–Moderate	5	MEDIUM
Pica / ARFID / Rumination	Low	5	LOW
Circadian rhythm disorders	Low	5	LOW
RDoC / HiTOP	Low	5	LOW

TOPIC-BY-TOPIC ANALYSIS

1. Suicide Risk Assessment

Frequency: Very High | Marks: 5–10

Almost certain to appear in some form across any exam cycle. May be framed as:

- "Describe suicide risk assessment" (10 marks)
- "Enumerate risk factors for suicide" (5 marks)
- "What is the Columbia Protocol?" (5 marks)
- "Write a note on means restriction" (5 marks)
- "India-specific suicide epidemiology" (5 marks)

EXAM STRATEGY

Prepare a modular answer: (1) definition + epidemiology, (2) risk factors (use IS PATH WARM), (3) assessment tools (SAD PERSONS + C-SSRS), (4) management (safety planning, hospitalisation criteria, pharmacotherapy). From this module you can construct both 5-mark and 10-mark answers by expanding or compressing.

High-yield subpoints examiners test:

- Difference between SAD PERSONS and C-SSRS (and which is preferred now)
- Hopelessness as more predictive than depression severity
- Prior attempt as strongest single predictor
- Lithium and clozapine as specific anti-suicidal agents
- Safety planning vs no-harm contracts
- NCRB India data (approximate figures)

- Joiner's Interpersonal Theory (thwarted belongingness + perceived burdensomeness + acquired capability)
 - Werther Effect and Papageno Effect
 - WHO media guidelines
-

2. Anorexia Nervosa: Medical Complications

Frequency: Very High | Marks: 5–10

Consistently appears. Often paired with or followed by refeeding syndrome.

Framing variants:

- "Medical complications of anorexia nervosa" (10 marks)
- "Write short notes on refeeding syndrome" (5 marks)
- "Outline the medical management of a severely underweight patient with AN" (10 marks)
- "What are the criteria for inpatient admission in AN?" (5 marks)

EXAM STRATEGY

Use the CARDIAC BONES mnemonic to cover all systems. Don't just list, briefly explain the mechanism for each (e.g., QTc prolongation due to hypokalaemia and hypomagnesaemia → arrhythmia risk). Mechanism earns marks; listing does not.

High-yield subpoints:

- Lanugo, bradycardia, hypothermia as clinical triad
 - QTc prolongation → torsades de pointes risk
 - Sick euthyroid syndrome (low T3, normal TSH)
 - Osteoporosis, worse than post-menopausal because peak bone mass not yet acquired
 - Pancytopenia from gelatinous bone marrow transformation
 - Superior mesenteric artery syndrome
 - DSM-5 removed BMI <17.5 as criterion, "significantly low weight" language now used
-

3. Refeeding Syndrome

Frequency: High | Marks: 5–10

Often asked as a standalone or as part of AN management.

Framing variants:

- "Write a note on refeeding syndrome" (5–10 marks)
- "How would you manage nutritional rehabilitation in a patient with severe AN?" (10 marks)

EXAM STRATEGY

Pathophysiology is the examiner's favourite part. Explain the starvation → depletion of intracellular electrolytes → refeeding → insulin surge → electrolyte shift INTO cells → serum drop mechanism clearly. Then cover prevention: BELT mnemonic (Baseline, Electrolytes, Low start, Thiamine before glucose).

High-yield subpoints:

- Hypophosphataemia as hallmark electrolyte
- Thiamine MUST precede carbohydrate loading (Wernicke's risk)
- NICE criteria for high refeeding risk
- Starting caloric target: 20 kcal/kg/day
- Monitoring frequency: electrolytes every 12–24 hours initially
- Consequences of hypophosphataemia: respiratory failure (diaphragm), cardiac failure, rhabdomyolysis

4. ICD-11 vs ICD-10 Key Changes

Frequency: High | Marks: 5–10

Increasingly common as India prepares for transition. Can appear as:

- "Enumerate the key changes in ICD-11 compared to ICD-10" (10 marks)
- "What are the new diagnoses in ICD-11?" (5 marks)
- "How has the classification of schizophrenia changed in ICD-11?" (5 marks)
- "How has ICD-11 classified personality disorders?" (5 marks)

EXAM STRATEGY

Structure by category: (1) Structural changes, (2) New diagnoses added, (3) Existing diagnoses modified, (4) Diagnoses moved/removed. Use the mnemonic CAPE-G for new diagnoses.

High-yield subpoints:

- Schizophrenia subtypes abolished; dimensional symptom domains added
- Complex PTSD added (6B41), must know the three additional feature clusters
- Personality disorders: dimensional (severity + trait domains), not categorical
- BED and ARFID now full diagnoses
- Prolonged Grief Disorder added (6B42)
- Gaming Disorder added (6C51)
- FND moved out of dissociative disorders
- Acute stress reaction no longer a mental disorder

- Gender incongruence moved out of mental disorders chapter
 - Personality disorders: borderline pattern retained as optional qualifier only
-

5. Narcolepsy

Frequency: High | Marks: 5–10

A perennial favourite. Usually asked as classification + management.

Framing variants:

- "Classify narcolepsy and describe its management" (10 marks)
- "Write a note on narcolepsy" (5 marks)
- "What is cataplexy?" (short note)
- "What is the MSLT and how is it used in narcolepsy diagnosis?" (5 marks)

EXAM STRATEGY

Always define Type 1 and Type 2 with their distinguishing criteria (cataplexy/hypocretin). Describe the tetrad clearly. For management, organise by symptom (EDS vs cataplexy vs nocturnal sleep). Sodium oxybate is the pivot point, explain why it is unique (treats both EDS and cataplexy).

High-yield subpoints:

- Cataplexy triggered by emotion, emotionally triggered bilateral muscle tone loss WITHOUT loss of consciousness
 - CSF hypocretin-1 <110 pg/mL = diagnostic of Type 1
 - HLA-DQB1*06:02 association in Type 1 (>95%)
 - MSLT criteria: <8 min mean sleep latency + ≥ 2 SOREMPs
 - Sodium oxybate: GABA-B agonist, treats both EDS and cataplexy, Schedule I
 - Modafinil: first-line for EDS, no cataplexy effect, DAT inhibitor
 - SSRIs/venlafaxine: treat cataplexy via REM suppression
 - Non-pharm: scheduled naps; driving restrictions
-

6. CBT-I (Cognitive Behaviour Therapy for Insomnia)

Frequency: High | Marks: 5–10

Framing variants:

- "Describe CBT-I" (5–10 marks)
- "Compare CBT-I and pharmacotherapy for insomnia" (10 marks)
- "What is sleep restriction therapy?" (5 marks)

EXAM STRATEGY

Cover all 5 components (sleep restriction, stimulus control, cognitive restructuring, relaxation, sleep hygiene). For each, state the rationale, not just the technique. Examiners reward "why" answers.

High-yield subpoints:

- Sleep restriction is the most potent single component
- Sleep efficiency formula and targets (SE >85%)
- Stimulus control breaks bed-arousal conditioning
- CBT-I superior to pharmacotherapy for long-term outcomes
- Digital CBT-I: comparable efficacy to face-to-face
- Contraindication of sleep restriction: seizure disorder, dangerous occupations requiring alertness

7. Dissociative Disorders: Classification and Management

Frequency: High | Marks: 5–10

Framing variants:

- "Classify dissociative disorders and describe management" (10 marks)
- "Write a note on dissociative identity disorder" (5 marks)
- "Differentiate dissociative amnesia from organic amnesia" (5 marks)
- "What are the ICD-11 changes to dissociative disorders?" (5 marks)

EXAM STRATEGY

Classification table first (ICD-11 codes), then phase-based management model. The ICD-11 change about FND moving out of dissociative disorders is highly examinable, state it proactively.

High-yield subpoints:

- ICD-11: FND moved to Diseases of Nervous System
- ICD-11: Partial DID added as new category
- Phase-based management: Stabilise → Process → Integrate
- Do NOT jump to trauma processing before stabilisation
- Sociocognitive vs trauma model debate in DID
- DPDR: reality testing intact; first-line = CBT (Feeling Real); lamotrigine
- Possession trance: pathological only if outside cultural context + causes distress

8. Bulimia Nervosa

Frequency: Moderate–High | Marks: 5–10

Framing variants:

- "Describe the diagnosis and management of BN" (10 marks)
- "Medical complications of BN" (5 marks)
- "Compare AN and BN" (10 marks)

High-yield subpoints:

- Fluoxetine 60 mg / day, only FDA-approved drug; higher than antidepressant dose
 - Bupropion CONTRAINDICATED, seizure risk
 - Russell's sign, parotid enlargement, perimolysis
 - Hypokalaemia + metabolic alkalosis from vomiting
 - CBT-E as gold standard psychotherapy
 - Compensatory behaviours ≥ 1 / week for ≥ 3 months
-

9. REM Sleep Behaviour Disorder

Frequency: Moderate–High | Marks: 5

Framing variants:

- "Write a note on REM sleep behaviour disorder" (5 marks)
- "What are the associations and management of RBD?" (5 marks)

High-yield subpoints:

- Males >50 ; dream enactment; wakes oriented
 - PSG: REM without atonia (RWA)
 - Prodrome for alpha-synucleinopathies (PD, DLB, MSA), 80–90% convert in 10–15 years
 - SSRIs can precipitate or worsen RBD
 - Treatment: clonazepam OR melatonin; safety measures
-

10. Eating Disorders in Adolescents

Frequency: Moderate | Marks: 5–10

Framing variants:

- "Management of AN in an adolescent" (10 marks)
- "What is Family-Based Treatment for eating disorders?" (5 marks)

High-yield subpoints:

- FBT (Maudsley Approach): three phases; parents control meals in Phase 1

- BMI-for-age, not adult BMI
- Junior MARSIPAN guidelines
- No drug approved for adolescent AN; fluoxetine post-weight-restoration for relapse prevention
- Bone density impact: occurs during peak bone mass acquisition period, serious long-term consequence

11. Sleep Architecture

Frequency: Moderate | Marks: 5

Framing variants:

- "Describe normal sleep architecture" (5 marks)
- "How does sleep change in depression?" (5 marks)
- "What are the EEG characteristics of sleep stages?" (5 marks)

High-yield subpoints:

- N1: theta; N2: sleep spindles + K-complexes; N3: delta; REM: low-voltage mixed frequency
- First REM: ~90 minutes after sleep onset
- N3 dominates first third; REM dominates last third
- REM latency shortened in depression (<65 min), biological marker
- All antidepressants except bupropion suppress REM

12. DPDR (Depersonalisation/Derealization Disorder)

Frequency: Moderate | Marks: 5

High-yield subpoints:

- Reality testing intact (vs psychosis)
- Emotional numbing, anxiety about symptoms
- CBT (Feeling Real): first-line psychological
- Lamotrigine, naltrexone: pharmacological options
- Transient episodes in up to 74% of general population; persistent disorder = 1–2%

QUESTION FORMAT PATTERNS

FORMAT	TYPICAL MARKS	EXAMPLES
Long essay	10	"Classify and describe the management of [disorder]"

FORMAT	TYPICAL MARKS	EXAMPLES
Short essay	5	"Write a note on [topic]"
Comparison	5–10	"Differentiate A from B"
Clinical scenario	5–10	"A 17-year-old presents with..."
Enumerate	5	"List the medical complications of..."
Mechanism	5	"Explain the pathophysiology of refeeding syndrome"

RAPID REVISION: WHAT TO STUDY LAST

If you have 2 hours before the exam, revise in this order:

1. Suicide risk assessment framework + SAD PERSONS + Joiner's theory (10 min)
2. AN medical complications, CARDIAC BONES mnemonic (10 min)
3. Refeeding syndrome, PKMGT + BELT (10 min)
4. ICD-11 major changes, CAPE-G + personality disorder restructuring + schizophrenia subtypes (10 min)
5. Narcolepsy Type 1 vs Type 2 table + management drugs (10 min)
6. CBT-I components + sleep restriction rationale (10 min)
7. Dissociative disorder classification + phase-based management (10 min)
8. Fluoxetine for BN + bupropion contraindicated (5 min)
9. RBD features + alpha-synuclein connection (5 min)
10. Sleep stage EEG changes + REM latency in depression (5 min)

DRUGS WORTH MEMORISING FOR THIS CHAPTER

DRUG · SINGLE MOST IMPORTANT FACT

Fluoxetine 60mg Only FDA drug for BN

Lisdexamfetamine Only FDA drug for BED

Sodium oxybate Only drug for both EDS and cataplexy in narcolepsy

Clozapine Only FDA drug with anti-suicidal indication

Lithium Strongest evidence for reducing completed suicide (80% reduction)

Suvorexant First orexin antagonist for insomnia, no dependence

Bupropion CONTRAINDICATED in AN and BN

Prazosin Alpha-1 blocker for PTSD nightmares

Tasimelteon MT1/MT2 agonist; FDA approved for non-24h sleep-wake disorder

Pramipexole First-line dopamine agonist for RLS; watch for augmentation

Quick Review

All names are fictitious. All ages, presentations, and clinical details are constructed for educational purposes. No real patient identifiers are used.

VIGNETTE 1: Anorexia Nervosa with Refeeding Risk

Clinical Scenario:

Priya, a 19-year-old pre-medical student, is brought to the psychiatry emergency by her parents. Over the past 8 months she has progressively restricted her food intake, citing concerns about her "protruding stomach." She exercises for 3 hours daily despite feeling weak. Her current weight is 36 kg, height 162 cm (BMI 13.7). On examination: pulse 44 bpm, BP 84/52 mmHg, temperature 35.8°C. Fine downy hair is visible on her arms. Her serum potassium is 2.9 mEq/L, phosphate 0.6 mmol/L, albumin 28 g/L. ECG shows QTc of 478 ms.

Questions for Active Recall:

1. What is the diagnosis? What subtype?
2. Which vital sign findings indicate immediate medical risk?
3. Is she at risk for refeeding syndrome? Apply NICE criteria.
4. How would you initiate nutritional rehabilitation?
5. What must be given BEFORE carbohydrate reintroduction, and why?
6. What is the significance of QTc 478ms?
7. Name three dermatological findings you would look for on full examination.

Model Answer Points:

Diagnosis: Anorexia nervosa, restricting type (no evidence of binge-purge in the history).
Severity: extreme (BMI 13.7 < 15).

Vital sign risks: Bradycardia (44 bpm, below 45 bpm threshold for immediate concern), hypotension (systolic 84), hypothermia (35.8°C). This triad demands inpatient admission.

Refeeding risk (NICE criteria, all four met):

- BMI <16 ✓ (13.7)
- Nutritional intake minimal for >10 days (likely, given 8 months of restriction) ✓
- Pre-feeding electrolyte abnormality: K⁺ low, phosphate low ✓
- Weight loss >15% (clinical history consistent) ✓

Nutritional rehabilitation:

- Correct electrolytes BEFORE starting nutrition
- Start at 10 kcal/kg/day given very high risk (= ~360 kcal/day initially)
- **Thiamine 200–300 mg/day orally (or IV Pabrinex) BEFORE glucose/carbohydrate loading**, prevents Wernicke's encephalopathy
- Increase caloric load by 200–300 kcal every 2–3 days
- Monitor electrolytes (phosphate, potassium, magnesium) every 12–24 hours for first 3–5 days
- Monitor weight daily, ECG twice weekly initially

QTc 478 ms: Prolonged (normal <440ms in females). At risk of torsades de pointes, a potentially fatal ventricular arrhythmia. Correct hypokalaemia and hypomagnesaemia urgently. Avoid all QTc-prolonging drugs.

Dermatological findings: Lanugo hair (fine downy hair, thermoregulatory response), carotenemia (yellow-orange skin from carrot-heavy diet), xerosis (dry skin), and alopecia (telogen effluvium).

EXAM PEARL

In any AN admission scenario, the sequence is: stabilise medically → correct electrolytes → thiamine → cautious refeeding → begin psychological input once medically stable. Jumping to psychological therapy before medical stabilisation is the classic error.

VIGNETTE 2: Bulimia Nervosa

Clinical Scenario:

Aarti, a 23-year-old law student, presents to the outpatient clinic. She reports that for the past 18 months, she has been eating large amounts of food "secretly" almost every evening, finishing an entire packet of biscuits, a full box of mithai, and several portions of leftovers within 30–40 minutes. She describes feeling completely out of control during these episodes and intensely ashamed afterwards. She then induces vomiting to "undo the damage." She says this happens 5–6 times per week. Her weight is 61 kg, height 164 cm (BMI 22.7, normal). Her self-worth is "entirely about how I look."

On examination: mild bilateral parotid enlargement. Dental examination (referred) reveals enamel erosion on the lingual surface of upper front teeth.

Questions for Active Recall:

1. What is the diagnosis? What severity specifier?
2. What are the DSM-5 criteria being met?
3. Name two physical examination findings present and explain their mechanism.
4. What electrolyte abnormality is most likely on blood tests? What acid-base disturbance?

5. What is the pharmacological treatment of choice? What dose? Why is bupropion contraindicated?
6. What is the first-line psychological treatment? Name the developer.

Model Answer Points:

Diagnosis: Bulimia nervosa, severe (4–7 compensatory episodes per week = severe range). BMI normal, distinguishes from AN binge-purge subtype.

DSM-5 criteria met:

1. Recurrent binge eating: large amount in discrete period + loss of control ✓
2. Compensatory behaviour (self-induced vomiting) ✓
3. ≥ 1 episode/week for ≥ 3 months (5–6/week for 18 months) ✓
4. Self-evaluation unduly influenced by shape/weight ✓
5. Not occurring exclusively during AN (BMI normal) ✓

Physical findings:

- Parotid hypertrophy: repeated vomiting → salivary gland hypertrophy via autonomic stimulation and direct acid irritation → "chipmunk face"
- Perimolysis: gastric acid reflux during repeated vomiting → demineralisation of dental enamel, predominantly on lingual/palatal surfaces

Electrolytes: Hypokalaemia (potassium lost in vomit), metabolic alkalosis (loss of gastric HCl → bicarbonate excess). This combination is the most dangerous medical consequence of purging, cardiac arrhythmia risk.

Pharmacotherapy:

- **Fluoxetine 60 mg/day**, only FDA-approved drug for BN. Note: this is 60 mg, not 20–40 mg (standard antidepressant dose). Reduces binge-purge frequency by ~50%.
- **Bupropion contraindicated:** lowers seizure threshold; electrolyte disturbances in BN (especially hypokalaemia) independently increase seizure risk. Combination is dangerous.

Psychotherapy: CBT-E (Enhanced Cognitive Behaviour Therapy), developed by Christopher Fairburn at Oxford. 20 sessions targeting core psychopathology (overconcern with shape and weight). Addresses binge-purge cycles, dietary restraint, and body image.

VIGNETTE 3: Dissociative Fugue

Clinical Scenario:

A man is found wandering in a bus stand in Pune, approximately 400 km from his home city of Hubli. He appears confused about where he is and cannot recall how he arrived. He gives his name as "Rajan" but is uncertain of his surname. He has no mobile phone or

identification on him. Police bring him to the emergency department. On examination, he is alert, oriented to person only, with no evidence of head injury, alcohol intoxication, or current substance use. Blood glucose is normal. Neurological examination is unremarkable.

Two days later, his family, traced via a missing persons report, identifies him as Suresh, 38, a small business owner who had recently faced significant financial losses and a property dispute. His wife reports he left for work 5 days ago and never returned. The family notes he had been under severe stress for several months.

Questions for Active Recall:

1. What is the most likely psychiatric diagnosis?
2. How does this differ from DSM-5 vs ICD-11 classification?
3. What investigations would you perform to exclude organic causes?
4. What is the typical course and prognosis?
5. Outline the management approach.

Model Answer Points:

Diagnosis: Dissociative Fugue (dissociative amnesia with fugue specifier).

DSM-5 vs ICD-11:

- DSM-5: Dissociative fugue is a specifier of Dissociative Amnesia (not a separate diagnosis)
- ICD-11: Coded as 6B61.0, Dissociative amnesia with dissociative fugue (retains some separate recognition)

Investigations to exclude organic causes:

- Blood glucose (done, normal)
- Serum electrolytes, renal function, liver function
- Blood alcohol, urine toxicology screen
- CT brain (to exclude structural lesion, subdural haematoma)
- EEG (if seizure disorder suspected, complex partial seizures can mimic fugue)
- Thyroid function tests

Course and prognosis:

- Duration: hours to months; this case (5 days) is within typical range
- Recovery: usually spontaneous once removed from stressor or trigger identified
- Retrograde amnesia for the fugue period typically persists after recovery
- Recurrence: possible under further stress

Management:

1. Safe, supportive environment

2. Psychoeducation for patient and family
3. Remove or reduce identified stressor where possible
4. Trauma-focused therapy once acute episode resolves
5. Treat comorbid depression, anxiety, PTSD
6. Hypnosis: historically used; not evidence-based for long-term recovery
7. Narcoanalysis (amobarbital interview): historical; not recommended in current guidelines

CLINICAL ANCHOR

The key clinical skill is excluding organic causes systematically before diagnosing a dissociative condition. This is not "diagnosis by exclusion", the positive clinical features (trauma link, purposeful travel, identity confusion) ARE diagnostic. But organic causes must be ruled out first.

VIGNETTE 4: Dissociative Identity Disorder

Clinical Scenario:

Meena, 31, has been in psychotherapy for 3 years following a history of severe childhood trauma. Her therapist refers her for a psychiatric evaluation. She reports episodes of "losing time", gaps of several hours to a full day where she cannot recall what happened, during which family members describe her as behaving very differently: sometimes childlike and frightened, sometimes aggressive and threatening. She reports hearing internal voices that argue with each other. She has scars on both forearms from self-cutting, which she says she didn't do, "someone else does it." She was previously diagnosed with schizophrenia and PTSD, and has had two previous hospitalisations.

On assessment, during the interview, she abruptly changes her posture, voice tone, and manner, and states her name is "Choti" (meaning "little one"), is 7 years old, and is afraid of "the dark man."

Questions for Active Recall:

1. What is the diagnosis? What DSM-5 and ICD-11 criteria are met?
2. How do the internal voices in DID differ from those in schizophrenia?
3. What is the phase-based treatment model? Why is premature trauma processing dangerous?
4. What pharmacotherapy is appropriate?
5. What are the two major theoretical controversies around this diagnosis?

Model Answer Points:

Diagnosis: Dissociative Identity Disorder (DID). DSM-5 criteria met:

1. Two or more distinct personality states (host Meena + child alter "Choti" + aggressive alter) ✓
2. Recurrent amnesia between alters (gaps in memory; self-harm she cannot recall) ✓
3. Significant distress or functional impairment ✓
4. Not attributable to cultural / religious practice ✓
5. Not due to substance or medical condition ✓

Voices in DID vs schizophrenia:

FEATURE	DID	SCHIZOPHRENIA
Location	Heard INSIDE the head (internal)	Often heard OUTSIDE (external, third-person)
Content	Alters speaking to/about each other	Command hallucinations, commentary, derogatory
Identity	Voices have distinct identity and personality	Voices are alien/external
Control	Patient may be able to engage with voices	Rarely
Insight	Often aware these are parts of self	Often perceived as real external entities

Phase-based treatment:

- Phase 1 (Safety and Stabilisation): Grounding techniques, containment imagery, distress tolerance, coping skills. Build therapeutic alliance with all alters. Map the alter system.
- Phase 2 (Trauma Processing): Careful, titrated engagement with traumatic memories using EMDR or ego state therapy. Premature trauma processing before adequate stabilisation → flooding, decompensation, increased self-harm, hospitalisation, therapy dropout.
- Phase 3 (Integration): Fusion of alters or functional integration (alters co-operate, amnesia barriers dissolve).

Pharmacotherapy:

- No medication treats DID specifically
- SSRIs for comorbid PTSD, depression, anxiety
- Prazosin for nightmares
- Avoid benzodiazepines long-term (abuse risk; can increase dissociation)
- Antipsychotics NOT indicated for DID voices (they are alter-based, not psychotic phenomena)

Controversies:

1. Trauma model: genuine response to severe early trauma, neurobiologically grounded
 2. Sociocognitive model: iatrogenic creation through suggestive therapy, cultural scripting, media influence (Sybil effect)
-

VIGNETTE 5: Narcolepsy Type 1

Clinical Scenario:

Karthik, 24, an engineering student, presents with a 2-year history of overwhelming sleepiness throughout the day. He has fallen asleep during lectures, while eating, and once briefly while cycling. He reports that when he laughs hard or is suddenly surprised, his knees buckle and he has to grab support, this lasts about 20–30 seconds and he remains fully conscious throughout. He also reports vivid, frightening visual experiences just as he falls asleep at night, and has had two episodes of waking in the morning unable to move for about a minute before "snapping out of it."

He sleeps 8–9 hours nightly but wakes unrefreshed. His Epworth Sleepiness Scale score is 19/24.

Questions for Active Recall:

1. What is the diagnosis? Justify using clinical features.
2. What investigation confirms the diagnosis? What results are expected?
3. What is the pathophysiology of Type 1 narcolepsy?
4. Describe the management, matching each drug to each symptom.
5. What non-pharmacological advice would you give regarding driving?

Model Answer Points:

Diagnosis: Narcolepsy Type 1 (with cataplexy). The complete tetrad is present:

1. EDS (obligate): severe; ESS 19/24
2. Cataplexy (pathognomonic for Type 1): emotionally triggered bilateral muscle tone loss WITH preserved consciousness, "knees buckle when laughing"
3. Hypnagogic hallucinations: vivid visual experiences at sleep onset
4. Sleep paralysis: unable to move on awakening, resolved spontaneously

Investigations:

- Overnight polysomnography (PSG): rules out obstructive sleep apnoea as cause of EDS; confirms adequate sleep duration
- Multiple Sleep Latency Test (MSLT) next morning: Expected results, mean sleep latency <8 minutes (likely 3–4 min in Type 1) AND ≥ 2 SOREMPs (sleep-onset REM periods)
- CSF hypocretin-1: expected <110 pg/mL (confirmatory; more often done in research settings)

- HLA-DQB1*06:02: expected positive (>95% of Type 1)

Pathophysiology:

Selective autoimmune destruction of hypothalamic neurons producing hypocretin (orexin), 80–90% neuron loss. Loss of the wake-stabilising orexin signal → inability to maintain consolidated wakefulness → intrusion of REM sleep features (cataplexy, sleep paralysis, hypnagogic hallucinations) into wakefulness.

Trigger: H1N1 influenza infection or Pandemrix vaccine (Europe), molecular mimicry between influenza antigen and hypocretin receptor.

Management:

SYMPTOM	DRUG	MECHANISM	NOTES
EDS	Modafinil 200–400 mg/day	DAT inhibition	First-line; no cataplexy effect
Cataplexy	Sodium oxybate (GHB)	GABA-B agonist	First-line; also treats EDS; taken in 2 doses at night
EDS + cataplexy combined	Sodium oxybate	GABA-B agonist	Only drug for both
Cataplexy (alternative)	Venlafaxine 75–150 mg or SSRIs	REM suppression	Second-line; less effective
Sleep paralysis + hallucinations	Sodium oxybate or SSRIs	As above	

Non-pharmacological: scheduled naps 2–3 × 15–20 min/day; consistent sleep schedule; avoid alcohol.

Driving advice: Karthik must not drive until symptoms are well-controlled on medication (stipulated by most road transport regulations). He must disclose his diagnosis. Driving is permitted only when daytime sleepiness is adequately controlled AND he has been on stable medication without breakthrough sleep attacks. This must be reviewed periodically.

VIGNETTE 6: Chronic Insomnia: CBT-I Application

Clinical Scenario:

Sunita, 45, a secondary school teacher, presents with an 18-month history of difficulty sleeping. She takes 1.5–2 hours to fall asleep each night and wakes 2–3 times. She reports lying awake worrying about whether she will sleep, calculating how many hours remain. She goes to bed at 9:30 pm hoping to accumulate enough sleep, spends 9.5 hours in bed, but estimates her actual sleep at 4.5–5 hours. She feels fatigued and irritable during the day, but

reports falling asleep easily when watching TV on the sofa. She has been taking zopiclone 7.5 mg nightly for 4 months prescribed by her GP.

Questions for Active Recall:

1. What is the diagnosis and its subtype by duration?
2. Calculate her sleep efficiency. What does this tell you?
3. Identify the perpetuating factors in this case.
4. Outline a CBT-I treatment plan for Sunita.
5. How would you address the zopiclone dependence?

Model Answer Points:

Diagnosis: Chronic insomnia disorder (≥ 3 nights/week, ≥ 3 months, with daytime impairment). Both sleep onset and sleep maintenance types are present.

Sleep efficiency: $SE = (\text{Total Sleep Time} / \text{Time In Bed}) \times 100 = (4.75 \text{ hours} / 9.5 \text{ hours}) \times 100 = 50\%$. This is severely impaired (normal target $>85\%$). Extended time in bed is paradoxically worsening her insomnia by diluting sleep drive and weakening the bed-sleep association.

Perpetuating factors (3P model, Spielman):

- Extended time in bed (9.5 hours): reduces sleep drive; dilutes sleep
- Stimulus control violation: falling asleep on sofa (decouples bed from sleepiness)
- Early bedtime (9:30 pm): too far ahead of actual sleep phase
- Sleep monitoring/worry: selective attention to time, calculating hours, hyperarousal
- Zopiclone dependence: perpetuates the belief that sleep is impossible without medication

CBT-I Treatment Plan:

Session 1-2 (Assessment and Psychoeducation):

- Explain 3P model; normalise that extended TIB worsens insomnia
- Sleep diary baseline (2 weeks): record TIB, sleep latency, awakenings, TST, SE

Session 2-3 (Sleep Restriction):

- Prescribe TIB = estimated TST = 4.75 hours
- Set fixed wake time: 6:00 am → prescribed bedtime: 1:15 am
- When SE $>90\%$ for 5 nights → extend TIB by 15 minutes (move bedtime earlier to 1:00 am)
- Continue extending in 15-min increments as SE improves

Session 3-4 (Stimulus Control):

- Bed only for sleep and sex

- Leave bedroom if not asleep within ~20 minutes; return only when sleepy
- No TV in bedroom; no lying on sofa to watch TV (sit upright instead)

Session 4-5 (Cognitive Restructuring):

- Challenge: "I can't function without 8 hours" → review actual performance on low-sleep nights
- Challenge: "I haven't slept a wink" → discuss sleep state misperception
- Reduce clock-watching: cover clock, remove phone from bedroom

Session 5-6 (Relapse Prevention):

- Explain that brief insomnia recurrences are normal
- Early intervention: apply stimulus control and sleep restriction at first sign of relapse

Zopiclone tapering:

- Gradual dose reduction: 7.5 mg → 3.75 mg → every other night → cessation
- Time the taper with CBT-I progress (begin taper once SE improving)
- Expect rebound insomnia on nights of dose reduction, reassure and pre-empt with CBT-I skills
- Target: complete cessation within 4–6 weeks

EXAM PEARL

Sleep restriction feels counter-intuitive to patients ("You're telling me to sleep less?"). The rationale, building adenosine-driven sleep pressure and consolidating fragmented sleep, must be explained clearly. Motivation depends on understanding.

VIGNETTE 7: Suicide Risk Assessment

Clinical Scenario:

Rohit, 38, is brought to the emergency department by his wife at 11 pm. She found him sitting in their car in the garage with the engine running; he had been there for approximately 20 minutes before she found him. He had written a note to their two children. He is alert on arrival. He says the note was "just in case" and denies current intent to die. He discloses that he was recently passed over for a promotion, his marriage has been strained for 2 years, and he has had recurrent depressive episodes for 6 years, currently untreated. He drinks approximately 6–8 units of alcohol per day. He has no previous suicide attempts. He has a licensed firearm at home, used for sport shooting.

Questions for Active Recall:

1. Apply the C-SSRS framework to characterise his ideation and behaviour.
2. List modifiable and non-modifiable risk factors present.

3. What is the immediate management?
4. What specific actions must be taken regarding means?
5. What protective factors should you assess?
6. Is hospitalisation indicated? Justify.

Model Answer Points:

C-SSRS Assessment:

- Ideation: Active ideation with plan (carbon monoxide poisoning via car exhaust in garage), C-SSRS ideation level 4 (plan present)
- Behaviour: Preparatory behaviour + actual attempt (engine running, note written, entered garage), this constitutes an actual interrupted attempt (interrupted by wife)
- Current denial of intent does not override the behavioural evidence

Risk Factors:

CATEGORY	MODIFIABLE	NON-MODIFIABLE
Psychiatric	Untreated depression (recurrent), alcohol use disorder	History of depressive episodes (6 years)
Psychological	Hopelessness (likely, job failure, marital strain), alcohol disinhibition	
Social	Recent occupational setback, marital conflict	Male sex
Biological	Active alcohol use	
Means	Firearm at home, car/garage access	

Immediate management:

1. Medical assessment (CO levels if clinically indicated; GCS, vitals)
2. Move to a safe, supervised area
3. Contact with wife (with consent), enlist support
4. Formal psychiatric assessment using C-SSRS
5. Start antidepressant (note: acute period, monitor for activation)
6. Address alcohol use disorder (detox if needed; naltrexone/acamprosate)

Means restriction, CRITICAL:

- Firearm: immediate counselling for temporary transfer to a trusted third party (friend, police storage) or secure gun safe with wife holding key. This is the single most evidence-based intervention available in this case.
- Garage: wife to control access (fob/lock); keep car in open driveway until risk resolves

Protective factors to assess:

- Attachment to children (note he wrote a note TO them, suggests relationship; this is a protective factor)
- Marriage, strained but wife searched for him (suggests connection)
- Future orientation: job, career ambitions
- Religious beliefs
- Willingness to seek help (came to ER, is engaging)

Hospitalisation: YES, indicated.

Reasons: actual attempt tonight, specific lethal plan (CO poisoning), untreated recurrent depression, active alcohol use disorder causing disinhibition, access to firearm. Even though he denies current intent, the behavioural evidence overrides self-report. Voluntary admission preferred; document discussion. If he refuses, consider involuntary admission under the Mental Healthcare Act, 2017 (Section 89/90, emergency treatment without consent when imminent harm risk).

CLINICAL ANCHOR

"I was just thinking about it, I wouldn't actually do it", after being found in a running car in a closed garage with a note. Behavioural evidence > verbal denial. Clinical assessment integrates both; it does not default to whichever is more reassuring.

VIGNETTE 8: REM Sleep Behaviour Disorder

Clinical Scenario:

Venkat, 62, is referred by his neurologist. His wife reports that for the past 18 months he has been "acting out his dreams", shouting, punching, and once fell out of bed. She describes him as appearing to be "fighting someone." He has injured her twice. He wakes immediately after these episodes, recalls a vivid dream in which he was being attacked, and is fully oriented within seconds. He has no history of sleepwalking as a child. He has noticed mild constipation over the past year and reduced sense of smell. He has a resting tremor in his right hand that was noted by his GP 6 months ago but attributed to anxiety. His current medication includes sertraline 100 mg/day, started 2 years ago.

Questions for Active Recall:

1. What is the diagnosis? What PSG finding confirms it?
2. What is the most clinically significant association of this condition?
3. Is the sertraline relevant? Explain.
4. What other clinical features in this case suggest the same underlying diagnosis?
5. Outline management.

Model Answer Points:

Diagnosis: REM Sleep Behaviour Disorder (RBD). Confirmed on video-polysomnography by demonstrating REM Sleep Without Atonia (RSWA), abnormal chin or limb EMG activity during REM sleep (tonic or phasic), occurring in >50% of REM epochs.

Clinical features of RBD:

- Dream enactment during REM: purposeful-appearing violent behaviour correlating with dream content
- Preserved consciousness/orientation immediately on awakening
- Full recall of dream narrative
- Predominantly affects males >50 years

Neurodegenerative association:

RBD is a **prodromal marker of alpha-synucleinopathies**. Prospective cohort studies (Postuma et al.; Iranzo et al.) demonstrate that 80–90% of idiopathic RBD patients develop Parkinson's disease, Dementia with Lewy Bodies, or Multiple System Atrophy within 10–15 years of RBD onset.

The resting tremor, anosmia, and constipation in this case are all **non-motor prodromal features of Parkinson's disease**. This patient likely has Parkinson's disease in its prodromal phase.

Sertraline relevance:

SSRIs (and SNRIs, TCAs, MAOIs) can precipitate or worsen RBD by suppressing REM sleep atonia. The sertraline may be contributing to, or unmasking, his RBD. Dose reduction should be considered; if clinically necessary to continue, discuss risk-benefit with the referring neurologist. Consider switching to an antidepressant with lower REM atonia impact (bupropion, though note its own limitations).

Management:

Pharmacological:

- **Clonazepam 0.5–1 mg at bedtime:** first-line; reduces dream enactment behaviour; does NOT restore REM atonia; risk of respiratory depression, screen for sleep apnoea first (PSG will have assessed this)
- **Melatonin 3–12 mg at bedtime:** alternative / adjunct; better tolerated in elderly; partially restores atonia; no respiratory depression; preferred in older patients or those with OSA

Non-pharmacological (essential):

- Remove sharp objects from bedside
- Pad bed frame and floor
- Consider placing mattress on floor

- Wife to sleep separately until behaviours are controlled
- Fall-prevention measures

Neurological follow-up:

- Refer to movement disorder neurologist for full Parkinson's disease evaluation
- This is not just sleep medicine, this patient is in a window where neuroprotective interventions (if developed) may be applicable
- Discuss with patient: the association with neurodegenerative disease, what to watch for, and why monitoring matters

VIGNETTE 9: Deliberate Self-Harm in Adolescent

Clinical Scenario:

Ananya, 16, is brought to the emergency department by her mother after she discovered fresh cuts on Ananya's forearms. There are approximately 12 superficial parallel incisions, 2–3 cm each, made with a blade. The wounds are superficial and not life-threatening. Ananya is tearful but calm. She says she did not want to die, "I just needed to feel something. Everything felt numb." She has been cutting for approximately 6 months, 2–3 times per week. The trigger today was a fight with her boyfriend. She denies suicidal ideation but has a history of one episode 8 months ago where she did take 6 paracetamol tablets with intent to die.

Questions for Active Recall:

1. Distinguish this presentation from a suicide attempt. What assessment framework is used?
2. What is the function of the self-harm in this case?
3. What is the prior paracetamol episode's significance?
4. What psychiatric disorders should be screened for?
5. Outline management.
6. What is the evidence-based treatment for self-harm in adolescents?

Model Answer Points:

Distinguishing DSH from suicide attempt:

FEATURE	THIS PRESENTATION	SUICIDE ATTEMPT
Intent	Explicit: no intent to die ("needed to feel something")	Intent to die (partial or complete)
Method lethality	Low (superficial cuts)	Variable
Rescue likelihood	High (superficial; visible to mother)	Variable

FEATURE	THIS PRESENTATION	SUICIDE ATTEMPT
Function	Emotion regulation, anti-dissociation ("feel numb → need to feel something")	Escape from psychache
C-SSRS	No ideation beyond passive thoughts	Ideation ± plan ± intent

C-SSRS is used to clarify: is there any suicidal ideation? At what intensity? Is the self-harm suicidal in nature at all?

Function of self-harm: Anti-dissociation (primary, "everything felt numb") and emotion regulation (secondary, relief after cutting). Both are well-established functional categories of NSSI. Understanding the function guides treatment.

Prior paracetamol episode significance:

The 8-month-old paracetamol overdose WITH intent to die was a **suicide attempt**, this is a critical historical factor. Prior suicide attempt is the **strongest single predictor** of future suicide attempt or completion. The current presentation is NSSI without suicidal intent, but the history of an actual attempt elevates overall risk. She cannot be assessed only by current presentation.

Screening for comorbid disorders:

- Borderline personality disorder features (most commonly associated with NSSI + instability)
- Major depressive disorder
- PTSD (history of trauma)
- Anxiety disorders
- Eating disorders (NSSI is elevated in AN / BN)
- Substance use
- ASD (emotional dysregulation, sensory seeking)

Management:

Immediate:

- Wound care (superficial, clean and dress)
- Safety assessment using C-SSRS
- Do NOT hospitalise purely for superficial NSSI without suicidal risk, can be counterproductive and reinforce the behaviour
- Engage Ananya collaboratively, not punitively

Short-term:

- Psychoeducation for Ananya and mother (normalise the emotional function; do not shame)
- Safety plan (with Ananya's input): warning signs → coping → contacts → means restriction (remove blade from room)
- Referral to adolescent mental health service

Evidence-based treatment:

- **Dialectical Behaviour Therapy for Adolescents (DBT-A):** strongest evidence for NSSI and suicidality in adolescents; family component; skills (TIPP, PLEASE, opposite action, distress tolerance)
- CBT for depression/anxiety if comorbid
- Family therapy where family conflict is a precipitant

EXAM PEARL

The therapeutic response to NSSI must be non-punitive. Punitive responses (restriction, withdrawal of care, dismissal) worsen outcomes and increase concealment. The function of the behaviour (emotional regulation) must be addressed, not just the behaviour itself.

VIGNETTE 10: Complex PTSD

Clinical Scenario:

Kavitha, 34, is referred from a women's shelter. She was in an abusive marriage for 9 years; her husband was physically and sexually violent throughout. She left 8 months ago. She presents with intrusive memories of abuse (flashbacks), avoidance of any reminders of her husband (refuses to enter the kitchen where most abuse occurred), and constant hypervigilance ("I can never relax"). She is also described as having "explosive" episodes of rage, followed by deep shame and self-loathing. She finds it impossible to trust anyone, including her therapist. She describes herself as "broken and worthless, no one would want someone like me." On Beck Depression Inventory, she scores 32 (severe depression).

Questions for Active Recall:

1. What is the diagnosis? Why does standard PTSD not capture this presentation fully?
2. Apply the ICD-11 Complex PTSD criteria.
3. How does Complex PTSD differ from Borderline Personality Disorder?
4. What is the phase-based treatment approach?
5. What role does Schema Therapy play?

Model Answer Points:

Diagnosis: Complex PTSD (ICD-11: 6B41). Standard PTSD (6B40) does not fully capture this presentation because in addition to the three core PTSD clusters (re-experiencing,

avoidance, hyperarousal), she has three additional symptom domains that constitute Complex PTSD.

ICD-11 Complex PTSD Criteria:

Core PTSD criteria (6B40, all met):

1. Re-experiencing: flashbacks of abuse ✓
2. Avoidance: refuses reminders (kitchen) ✓
3. Sense of current threat (hypervigilance): constant ✓

Duration >1 month ✓; significant impairment ✓

Additional DSO (Disturbances in Self-Organisation) criteria (all three met):

1. **Affect dysregulation:** explosive rage episodes, difficulty modulating emotion ✓
2. **Negative self-concept:** "broken and worthless, no one would want me", persistent shame, guilt, worthlessness ✓
3. **Disturbances in relationships:** inability to trust, including therapist ✓

Complex PTSD vs BPD Differential:

FEATURE	COMPLEX PTSD	BORDERLINE PERSONALITY DISORDER
Traumatic aetiology	Central (required)	Common but not required
Trauma type	Chronic interpersonal / developmental	Variable
Affect dysregulation	Present	Present (more severe, chronic)
Identity disturbance	Present (shame / worthlessness)	Present (more pervasive identity diffusion)
Impulsivity	Not core	Core criterion
Self-harm	Secondary	Often primary and persistent
Frantic abandonment fears	Less prominent	Central
Paranoid ideation under stress	Possible	Present (transient)
Relationship instability	Present	More severe; idealization / devaluation

C-PTSD and BPD frequently co-occur; the distinction is not always clean clinically.

Phase-based Treatment (ICD-11 treatment guidelines):

Phase 1, Safety and Stabilisation (often longest phase in C-PTSD):

- Establish safety (physical: shelter, legal protection; psychological: therapeutic alliance)
- Skills for affect regulation (DBT-informed: TIPP, temperature, distress tolerance)

- Grounding techniques for flashbacks
- Psychoeducation about trauma and C-PTSD
- Build trust slowly, therapeutic relationship IS the intervention at this stage

Phase 2, Trauma Processing:

- EMDR: evidence-based for PTSD; requires adaptation and caution in C-PTSD (dissociative windows)
- Trauma-focused CBT
- Do NOT rush, destabilisation in C-PTSD is severe and set-backs are costly

Phase 3, Integration and Reconnection:

- Identity reconstruction: who is she outside the trauma narrative?
- Relational work: trust, intimacy, healthy relationships
- Schema Therapy's reconnection with "Healthy Adult" mode

Schema Therapy role:

Schema Therapy (Jeffrey Young) is particularly well-suited to C-PTSD because:

- It explicitly addresses Early Maladaptive Schemas activated by developmental trauma (Defectiveness/Shame, Mistrust/Abuse, Emotional Deprivation, all evident here)
- Mode work addresses the protective/avoidant responses (Detached Protector, Punitive Critic) that maintain the negative self-concept
- The Healthy Adult mode is built collaboratively, directly addressing Kavitha's belief that she is "broken"
- Limited reparenting within the therapeutic relationship directly addresses the attachment wounds underlying C-PTSD

CLINICAL ANCHOR

In C-PTSD, the therapeutic relationship is not merely a vessel for delivering techniques, it IS part of the corrective experience. Kavitha's inability to trust her therapist is not resistance; it is her trauma speaking. The therapist's consistent, boundaried, non-abandoning presence over time is itself curative.

VIGNETTE 11: Insomnia and Depression: Differential Sleep Features

Clinical Scenario:

Mohan, 52, presents to the psychiatry OPD with a 3-month history of disturbed sleep and low mood. He goes to bed at 10 pm, lies awake for 1.5 hours, wakes at 2 am, and cannot return to sleep. He gets up feeling unrefreshed at 5:30 am. He reports persistent low mood, anhedonia, fatigue, poor concentration, and a sense of hopelessness about the future. He

has lost 4 kg without dieting. He is a retired schoolteacher living alone following his wife's death 18 months ago. He has two adult children who live in other cities. His past history includes a depressive episode 12 years ago treated successfully with escitalopram.

Questions for Active Recall:

1. What is the sleep disturbance pattern and what does it suggest?
2. What specific sleep architecture changes occur in major depression?
3. What is the diagnosis and what criteria are met?
4. How would you approach treatment of both the depression and the insomnia?
5. What specific medication choice is advantageous here and why?

Model Answer Points:

Sleep disturbance pattern:

- Sleep onset insomnia (1.5 hour latency)
- Sleep maintenance insomnia (wakes at 2 am)
- Early morning awakening (EMA): wakes at 2 am and cannot return to sleep, classic EMA pattern
- EMA is the most characteristic sleep symptom of major depression (vs anxiety which tends to cause sleep onset insomnia)

Sleep architecture changes in major depression:

- Increased sleep latency
- Reduced sleep efficiency
- Reduced N3 (slow wave sleep)
- **Shortened REM latency (<65 minutes)**, most characteristic biological finding
- Increased total REM sleep
- Increased REM density (more eye movements per unit of REM)
- Frequent awakenings
- Early morning awakening

Diagnosis: Major Depressive Disorder, single episode (recurrence, prior episode 12 years ago).

Criteria met:

1. Depressed mood ✓
2. Anhedonia ✓
3. Fatigue ✓
4. Insomnia (sleep maintenance + EMA) ✓
5. Psychomotor (not described but concentration impaired) ✓

- 6. Poor concentration ✓
- 7. Hopelessness (→ worthlessness) ✓
- 8. Weight loss 4 kg ✓

Duration ≥ 2 weeks (3 months) ✓; significant impairment ✓

Treatment approach:

Depression:

- Antidepressant: restart escitalopram (previously effective, good prior response) 10–20 mg/day
- OR consider mirtazapine given insomnia comorbidity (see below)
- 8–12 weeks for full antidepressant response
- Psychotherapy: CBT for depression; IPT (particularly appropriate given bereavement/loss context)

Insomnia:

- CBT-I: appropriate as standalone or alongside antidepressant
- Short-term hypnotic if severe insomnia impairing daily function while waiting for antidepressant to work (zopiclone/melatonin; not long-term)

Medication choice advantage:

Mirtazapine 15–30 mg at bedtime is advantageous here because:

- H1 and 5-HT_{2A/2C} antagonism provides sedation and improves sleep architecture
- Antidepressant efficacy (NaSSA mechanism)
- Weight gain effect may be beneficial (4 kg loss already)
- Appetite stimulation helpful
- No sexual side effects (relevant in older patients)
- Does not suppress REM at low doses (unlike SSRIs)

Alternatively: escitalopram (good prior response) + short-term zopiclone while awaiting efficacy, then taper.

EXAM PEARL

When both depression and insomnia are present, first treat the depression, insomnia often resolves with remission. However, persistent insomnia predicts depressive relapse, so it must be specifically addressed. CBT-I + antidepressant outperforms either alone for comorbid insomnia/depression.

VIGNETTE 12: Depersonalisation/Derealization Disorder

Clinical Scenario:

Rishi, 22, a final-year medical student, presents to the psychiatry clinic. For the past 8 months he has been experiencing episodes, now nearly continuous, of feeling "detached from myself, like I'm watching myself from outside." He describes his surroundings as looking "foggy, artificial, like a film set." He knows these perceptions are not real and finds this frightening. He remains fully oriented and performs well academically despite the symptoms. The symptoms began after a period of heavy cannabis use 9 months ago, which he has since stopped. He was investigated by neurology (normal MRI brain, normal EEG) and cardiology (normal ECG, 24-hour Holter). He scores 28 on the Cambridge Depersonalisation Scale (moderate-severe).

Questions for Active Recall:

1. What is the diagnosis? What distinguishes it from psychosis?
2. What is the prevalence of transient vs persistent depersonalisation?
3. What is the role of cannabis in the aetiology?
4. What is the first-line psychological treatment?
5. What pharmacological options are available?

Model Answer Points:

Diagnosis: Depersonalisation/Derealization Disorder (DSM-5: 300.6; ICD-11: 6B66).

Distinguishing from psychosis:

The critical differentiating feature is **intact reality testing**, Rishi KNOWS his perceptions are not real ("he knows these perceptions are not real"). In psychosis, the patient believes the altered perception IS reality. This is the defining boundary.

Additional distinguishing features:

- No hallucinations (which are perceived as real external stimuli)
- No delusions
- No thought disorder
- Normal neurological and cardiac workup (excluded organic causes)

Prevalence:

- Transient depersonalisation episodes: up to 74% of the general population (stress, sleep deprivation, meditation, drugs)
- Persistent DPDR disorder: 1–2% of the general population
- Often under-diagnosed because patients fear being told they are "mad"

Cannabis and DPDR:

Cannabis (particularly high-THC preparations) can precipitate depersonalisation episodes, especially in vulnerable individuals. Cannabis may trigger a persistent DPDR disorder in susceptible individuals, the episodes persist beyond cannabis cessation. This is not withdrawal, it appears to represent a triggered dysfunction of the prefrontal-limbic regulatory system (reduced amygdala response + increased prefrontal inhibition → emotional numbing + dissociation). Rishi's timeline (DPDR persists 8 months post-cessation) is consistent with cannabis-triggered persistent DPDR.

First-line psychological treatment:

CBT for DPDR using the Hunter and Phillips model ("Feeling Real" therapy, developed at the Institute of Psychiatry, London). Components:

- Psychoeducation: normalise the experience; reduce secondary anxiety about the symptoms
- Identify and reduce safety behaviours (checking whether surroundings are real, avoidance)
- Reduce selective attention to symptoms (attention training)
- Behavioural activation (counteracts emotional numbing)
- Cognitive techniques: challenge catastrophic interpretations

Pharmacological options:

DRUG	EVIDENCE	MECHANISM
Lamotrigine	Open-label trials; some benefit	Glutamate modulation
Naltrexone	Open-label data	Mu-opioid antagonism; emotional numbing may be opioid-mediated
SSRIs	Treat comorbid anxiety / depression; modest direct DPDR effect	5-HT reuptake inhibition
SSRIs + lamotrigine	Combination may be superior	Complementary mechanisms

EXAM PEARL

DPDR is frequently misdiagnosed as anxiety disorder, psychosis, or "nothing" (normal investigation results leading to dismissal). The key diagnostic feature that should prompt the diagnosis: "I feel detached from myself / surroundings BUT I know it's not real." Reality testing intact = DPDR, not psychosis.

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