PART III · PAPER III

III

Specialties & Therapeutics

Part III turns to the special populations, the treatments, and the place of psychiatry in law and society, where knowledge becomes practice.

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ONE-DAY REVISION · PAPER 3

Neurodevelopmental Disorders

A family of disorders that begin in the developmental period and shape the trajectory of learning, behaviour and social life. The exam rewards three things: knowing the classification, the severity grids for intellectual disability and autism, and the genetic-syndrome discriminators. Every comparison table here is a short-note answer in waiting.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. Severity bands and gene names are the high-yield core; hold the discriminators.

01 Overview & classification · **02** Intellectual disability · **03** Aetiology of intellectual disability · **04** Autism spectrum disorder · **05** Attention-deficit/hyperactivity disorder · **06** Specific learning & communication disorders · **07** Motor disorders

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. In DSM-5 intellectual disability needs deficits in **both intellectual and adaptive functioning** with developmental onset, and **severity is set by adaptive functioning, not the IQ band** (cut-off about IQ **70**, two SD below mean, range 65-75).
2. **Down syndrome (trisomy 21) is the commonest genetic ID; Fragile X (FMR1 CGG expansion >200) is the commonest inherited ID**; **do not confuse the two**.
3. At the 15q11-q13 locus, loss of the **paternal** copy gives Prader-Willi (hypotonia then hyperphagia/obesity) while loss of the **maternal** copy (UBE3A) gives Angelman (ataxia, paroxysmal laughter); Rett is almost only girls, **MECP2**, hand-wringing.
4. ADHD needs **>=6 of 9** symptoms (children) or **>=5 of 9** (age 17+), several present **before age 12** and impairing in **two or more settings**; stimulants (methylphenidate) are first-line by effect size, with atomoxetine (NRI) or alpha-2 agonists when tics, anxiety or diversion risk apply.
5. Autism is the dyad of social-communication deficits plus restricted/repetitive behaviour; **no drug treats the core social deficit**, risperidone and aripiprazole are licensed only for irritability/aggression, and **there is no link between MMR and autism**.

1 Overview & classification

The neurodevelopmental disorders are grouped together because they share a signature, not a single cause. They are defined by **onset in the developmental period** (often before school entry), an **early and persistent** course rather than a relapsing one, and very frequent **co-occurrence** (one rarely travels alone).

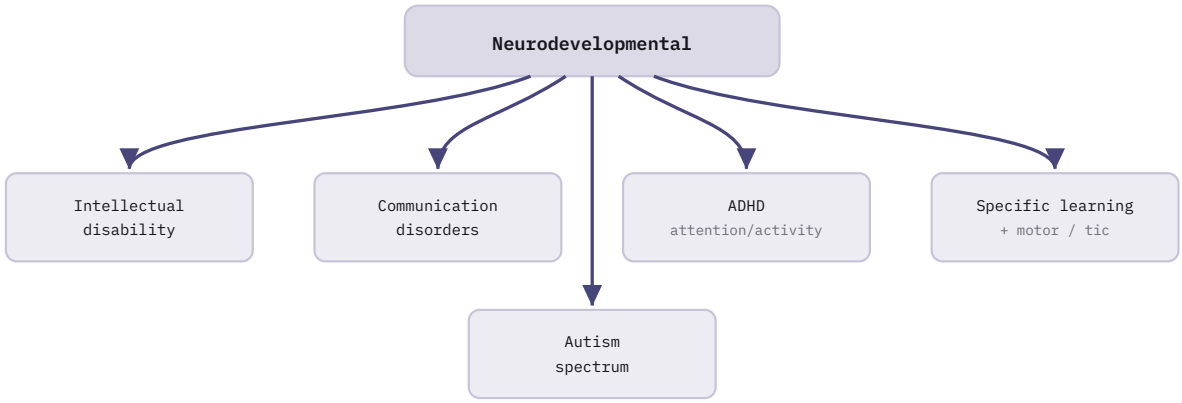
Shared features

- **Onset in development** – deficits emerge during the developmental period; many are apparent before the child starts formal schooling.
- **Early and persistent course** – typically lifelong traits with a developmental trajectory, not an episodic illness with discrete relapses.
- **Frequent co-occurrence** – high comorbidity within the group and with other disorders (for example autism with ID and ADHD, ADHD with learning disorder).

The categories

DSM-5-TR lists the umbrella as: **intellectual developmental disorders · communication disorders · autism spectrum disorder · attention-deficit/hyperactivity disorder · specific learning disorder · motor disorders (including tic disorders)**. ICD-11 groups the same conditions under disorders of intellectual development, developmental speech or language disorders, autism spectrum disorder, developmental learning disorder, developmental motor coordination disorder, ADHD, and stereotyped movement disorder; ICD-11 places primary tic disorders (including Tourette) in the nervous-system chapter but cross-lists them here.

THE NEURODEVELOPMENTAL UMBRELLA



One umbrella, six families: intellectual disability, communication disorders, autism spectrum disorder, ADHD, specific learning disorder, and the motor disorders (which include the tic disorders). All share developmental onset, an early persistent course, and high comorbidity.

2 Intellectual disability

Intellectual disability (intellectual developmental disorder) marks the central conceptual shift of DSM-5: away from **IQ alone** toward deficits in **both intellectual and adaptive functioning**, with **onset during the developmental period**. Three criteria must all be met.

- **Criterion A – intellectual** – deficits in reasoning, problem-solving, planning, abstract thinking, judgement, academic and experiential learning, confirmed by clinical assessment and standardised testing.
- **Criterion B – adaptive** – deficits in adaptive functioning across the conceptual, social and practical domains, limiting independence and participation. **Severity is set by adaptive functioning, not by the IQ score.**
- **Criterion C – onset** – both arise during the developmental period.

ID SEVERITY BY ADAPTIVE DOMAIN

		ADAPTIVE DOMAINS		
Severity	**IQ (approx.)**	**Conceptual**	**Social**	**Practical · support**
Mild	50–69 (~85% of ID)	academic skills lag; abstract thinking, planning weak	immature social judgement; risk of being manipulated	needs support for complex tasks (money, childcare); can live independently with help
Moderate	35–49	marked lag; basic academics slow, ongoing help needed	language simpler; relationships exist but social cues misread	self-care possible with prolonged teaching; supervised work; needs ongoing support

		ADAPTIVE DOMAINS		
Severity	IQ (approx.)	Conceptual	Social	Practical · support
Severe	20–34	little grasp of written language, number, time, money	spoken language limited; communicates in single words/phrases	needs support for all daily activities and supervision at all times
Profound	<20	conceptual skills mostly involve the physical world	very limited symbolic communication; may understand simple gestures	dependent for all care; co-occurring sensory/motor impairment common

● TRAP

In DSM-5 the **severity level is assigned by adaptive functioning**, because that is what determines the support needed, *not* by the IQ band. The IQ ranges above are the traditional ICD/textbook anchors and are still examined, but the conceptual point is the move from IQ-defined to adaptive-defined severity. By convention a test score about **two SD below the mean (IQ ~70, allowing measurement error 65–75)** is the threshold.

Related categories

- **Global developmental delay** – reserved for children **under 5** who fail to meet expected developmental milestones in several areas but cannot yet be reliably tested. A provisional label, re-assessed later.
- **Borderline intellectual functioning** – IQ roughly 71–84, above the ID cut-off; not a disorder but a coded condition that may need clinical attention (a "V/Z code" focus of attention).

3 Aetiology of intellectual disability

Group causes by **timing**. A cause is found in roughly half to two-thirds of severe cases and far fewer mild ones, where the contribution is more often polygenic and psychosocial.

Timing	Mechanisms	Examples
Prenatal	genetic / chromosomal, intrauterine infection, toxins, maternal/metabolic	Down, Fragile X and other syndromes; **TORCH** infections; **fetal alcohol** spectrum; congenital **hypothyroidism**; phenylketonuria; iodine deficiency
Perinatal	birth injury, hypoxia, prematurity, kernicterus	birth **asphyxia / hypoxic-ischaemic injury**, very low birth weight / prematurity, **kernicterus** (severe neonatal hyperbilirubinaemia), neonatal hypoglycaemia, neonatal sepsis
Postnatal	CNS infection, trauma, toxins, deprivation	meningitis / encephalitis, head injury, **lead** and other poisoning, severe malnutrition, profound **psychosocial deprivation**

● MNEMONIC

TORCH

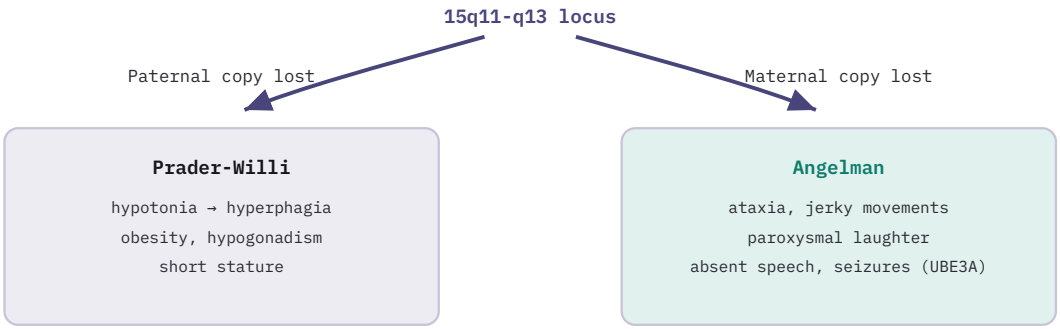
Toxoplasma, Other (syphilis, varicella, parvovirus B19, Zika), Rubella, Cytomegalovirus, Herpes. The classic intrauterine infections that injure the developing brain. CMV is the commonest infectious cause of congenital ID.

The genetic syndromes (high-yield)

Syndrome	Gene / mechanism	Discriminating phenotype
Down	trisomy 21 (95% non-disjunction; ~4% translocation; ~1% mosaic)	commonest genetic ID; up-slanting palpebral fissures, single palmar crease, hypotonia; AV septal defects, duodenal atresia; early **Alzheimer-type dementia**
Fragile X	**FMR1** CGG-repeat expansion (>200), X-linked	commonest *inherited* ID; long face, large ears, **macro-orchidism** (post-pubertal); gaze avoidance, hand-flapping; high autism overlap

Syndrome	Gene / mechanism	Discriminating phenotype
Prader-Willi	loss of **paternal** 15q11-q13 (deletion / maternal uniparental disomy)	neonatal hypotonia & poor feeding, then **hyperphagia and obesity**; hypogonadism, short stature, skin-picking
Angelman	loss of **maternal** 15q11-q13 (**UBE3A**)	"happy puppet": ataxic gait, jerky movements, **paroxysmal laughter**, near-absent speech, seizures
Williams	7q11.23 microdeletion (**elastin / ELN**)	"elfin" face, **supravalvular aortic stenosis**, hypercalcaemia; over-friendly "cocktail-party" speech with relatively spared expressive language
Rett	**MECP2** on Xq28 (X-linked dominant)	almost only **girls**; normal then regression at 6–18 months; loss of purposeful hand use with **hand-wringing stereotypy**, deceleration of head growth
Phenylketonuria	**PAH** deficiency, autosomal recessive	treatable; untreated → ID, seizures, eczema, musty odour, fair hair; prevented by newborn screening and a low-phenylalanine diet
Tuberous sclerosis	**TSC1 (hamartin) / TSC2 (tuberin)**, autosomal dominant	ash-leaf macules, facial angiofibromas, shagreen patch; cortical tubers → seizures and ID; strong autism link

15Q IMPRINTING: SAME LOCUS, TWO DISORDERS



Genomic imprinting at one locus gives two opposite syndromes. Lose the **paternal** 15q11-q13 and you get **Prader-Willi**; lose the **maternal** copy and you get **Angelman**. The parent of origin, not the gene, decides the disorder.

HOLD THIS

Down = **commonest genetic** ID. Fragile X = **commonest inherited** ID. Prader-Willi = **paternal** 15q loss; Angelman = **maternal**. Rett = girls, **MECP2**, hand-wringing. PKU and hypothyroidism = the **treatable**/preventable ones caught by newborn screening.

4 Autism spectrum disorder

DSM-5 collapsed the older subtypes (autistic disorder, Asperger, PDD-NOS) into a single **spectrum** defined by a two-part core, the **dyad**. Symptoms are present from early development, though they may be masked until social demands exceed capacity.

A · Social-communication deficits (all three)

Social-emotional reciprocity – reduced back-and-forth, sharing of interests and affect.
Nonverbal communication – poor eye contact, gesture, facial expression integration.
Relationships – difficulty making, keeping and understanding friendships; adjusting behaviour to context.

B · Restricted, repetitive behaviour (>=2 of 4)

Stereotyped movements, speech or object use (lining up, echolalia).
Insistence on sameness, rigid routines, ritualised patterns.
Restricted, fixated interests of abnormal intensity.
Sensory hyper- or hypo-reactivity (textures, sounds, lights).

Severity by support needs

Level	Label	Social communication	Restricted/repetitive behaviour
Level 1	requiring support	noticeable difficulty initiating; atypical/unsuccessful responses	inflexibility causes some interference; difficulty switching tasks
Level 2	requiring substantial support	marked deficits; limited initiation, reduced/odd responses	inflexibility obvious to the casual observer; distress on change
Level 3	requiring very substantial support	severe deficits; very limited initiation, minimal response	extreme inflexibility; great distress changing focus/action

Early red flags (by age)

◆ **By ~12 months** – no response to name, reduced eye contact, no babbling or pointing, limited social smiling.

◆ **By ~18 months** – no single words, no pointing to share interest (no **protodeclarative pointing**), little pretend play.

◆ **By ~24 months** – no two-word phrases; **any loss** of language or social skills at any age is a red flag for urgent assessment.

Epidemiology, sex ratio, comorbidity

● **Prevalence & sex** – estimates have risen toward roughly **1–2%** of children (broader criteria, better detection); male-to-female ratio about **3–4 : 1**, with likely under-diagnosis in girls who camouflage.

● **Comorbidity** – intellectual disability (a large minority), **ADHD**, **epilepsy**, anxiety, sleep problems, gastrointestinal complaints; the most heritable of the common neurodevelopmental disorders.

Management principles

■ **Early intensive behavioural intervention** – the evidence-based core: structured, developmental and behavioural programmes (for example applied behaviour analysis, naturalistic developmental behavioural interventions, speech and language therapy, social-skills work) started as early as possible.

■ **Family-centred and educational support** – parent training and mediated intervention, individualised education, occupational therapy for sensory needs; treat comorbidities.

▲ **Medication is adjunctive** – **no drug treats the core social deficit**. **Risperidone and aripiprazole** are licensed only for associated **irritability / aggression**; stimulants/atomoxetine for comorbid ADHD; SSRIs for anxiety. Medication never replaces behavioural therapy.

● TRAP

There is **no credible evidence linking the MMR vaccine to autism**; the original claim was retracted and the work found fraudulent. State this plainly. Autism is strongly heritable and neurodevelopmental in origin, with onset in early development.

5 Attention-deficit/hyperactivity disorder

ADHD is a persistent pattern of **inattention and/or hyperactivity-impulsivity** that interferes with functioning or development, beyond what fits the developmental level.

The three presentations

- **Predominantly inattentive** – careless errors, poor sustained attention, not listening, disorganised, loses things, distractible, forgetful. (The quiet, often-missed presentation, commoner in girls.)
- **Predominantly hyperactive-impulsive** – fidgets, leaves seat, runs/climbs, "on the go," talks excessively, blurts answers, cannot wait turn, interrupts.
- **Combined** – criteria met for both; the commonest clinic presentation.

The criteria that get tested

- **Symptom counts** – **>=6 of 9** symptoms in a dimension for children; **>=5 of 9** for older adolescents and adults (17+), persisting **>=6 months**.
- **Age of onset** – several symptoms present **before age 12** (DSM-5 raised this from the old age-7 threshold).
- **Cross-setting rule** – symptoms in **two or more settings** (home, school, work) and clear evidence they impair functioning. Not better explained by another disorder.
- **Adult ADHD & the Indian context** – ADHD persists into adulthood in a large proportion (hyperactivity fades, inattention and inner restlessness remain). It is markedly **under-recognised in India**, where presentation is often through academic underachievement or comorbidity, diagnosis is delayed, and stimulant access and awareness are limited.
- **Comorbidity** – oppositional defiant and conduct disorder, specific learning disorder, anxiety and mood disorders, tic disorders, and (in adults) substance use.

Pharmacology

Drug class	Mechanism	Key adverse effects	When to choose
Methylphenidate (stimulant)	blocks dopamine & noradrenaline reuptake	appetite loss, insomnia, growth slowing, raised HR/BP, irritability/rebound	first-line in most children; fast, largest effect size
Amphetamines (stimulant)	block reuptake *and* promote catecholamine release	as methylphenidate; higher misuse/diversion potential	alternative first-line stimulant if methylphenidate fails/poorly tolerated
Atomoxetine (non-stimulant)	selective **noradrenaline reuptake inhibitor**	GI upset, appetite/sleep changes, raised BP; monitor mood & suicidality; slow onset (weeks)	when stimulants fail, are not tolerated, with tics/anxiety, or **misuse/diversion** is a concern
Guanfacine / clonidine (non-stimulant)	**alpha-2 adrenergic agonists**	sedation, hypotension, dry mouth; taper to stop (rebound hypertension)	adjunct or alternative; useful with **tics**, marked impulsivity/aggression, or sleep onset problems

QUICK RULE

Stimulants first (largest, fastest effect). Switch to **non-stimulants** (atomoxetine, guanfacine/clonidine) when stimulants fail or are poorly tolerated, or where **tics, anxiety, or diversion risk** make a non-stimulant preferable. Always pair with behavioural/parent and school support.

● TRAP

Stimulants do **not** work paradoxically or sedate; they improve attention in everyone. They are first-line because of effect size, not because ADHD is the only condition they help. Screen cardiac history and growth before and during treatment, and remember the alpha-2 agonists must be **tapered**, never stopped abruptly.

6 Specific learning & communication disorders

These are **specific** deficits: skills well below the level expected for age and intelligence, not explained by intellectual disability, poor schooling, sensory deficit or another disorder.

Specific learning disorder

A single diagnosis with specifiers for the affected domain.

Specifier	Common name	Core difficulty
With impairment in reading	**dyslexia**	inaccurate/slow word reading, poor decoding and spelling
In written expression	dysgraphia (written-expression type)	spelling, grammar, punctuation, clarity and organisation of writing
In mathematics	**dyscalculia**	number sense, arithmetic facts, calculation, mathematical reasoning

● TRAP

Dyslexia and **dyscalculia** are recognised *alternative terms*, not separate DSM-5 categories. DSM-5 codes one specific learning disorder with the relevant academic-domain specifier(s). A child can have more than one specifier.

Communication disorders

- **Language disorder** – persistent difficulty acquiring and using language (vocabulary, sentence structure, discourse) across modalities; reduced quantity and quality.
- **Speech sound disorder** – difficulty producing speech sounds that impairs intelligibility (the old "phonological/articulation disorder"), not due to a structural or neurological cause.
- **Childhood-onset fluency disorder (stuttering)** – disturbance in fluency and time-patterning: sound/syllable repetitions, prolongations, blocks, with onset in the developmental period.
- ▲ **Social (pragmatic) communication disorder** – difficulty with the **social use** of verbal and nonverbal communication (greetings, context, turn-taking, inference) **without** the restricted/repetitive behaviours of autism. The discriminator: if RRBs are present, it is autism, not this.

7 Motor disorders

Coordination and stereotypy

- **Developmental coordination disorder** – motor skills (coordination, "clumsiness," catching, handwriting, self-care) markedly below age expectation, interfering with daily life; not due to a medical/neurological condition or ID.
- **Stereotypic movement disorder** – repetitive, purposeless, driven motor behaviour (hand-flapping, body-rocking, head-banging, self-biting), often beginning in the early years; specify if self-injurious. Common in ID and sensory impairment.

Tic disorders & Tourette syndrome

- **Tic** – a sudden, rapid, recurrent, non-rhythmic motor movement or vocalisation. Often preceded by a **premonitory urge** and briefly suppressible.

Disorder	Tics present	Duration
Tourette syndrome	**multiple motor + >=1 vocal** (not necessarily concurrently)	>**1 year**; onset before 18

Disorder	Tics present	Duration
Persistent (chronic) motor or vocal	motor *or* vocal, not both	>1 year; onset before 18
Provisional tic disorder	single or multiple, motor and/or vocal	<1 year

◆ **Typical course** – onset around **4–6 years**, peak severity near **10–12**, classic **waxing and waning**, and improvement in most by late adolescence. **Coprolalia is uncommon** (a minority), not a requirement.

● **Comorbidity** – **ADHD and OCD** are the two big companions; also anxiety and learning problems. The comorbidities often cause more impairment than the tics themselves.

Management

■ **First, psychoeducation** – many mild tics need only reassurance, explanation, and addressing the comorbidity. Treat tics when they cause distress, pain or impairment.

■ **Behavioural** – **habit reversal training** within **comprehensive behavioural intervention for tics (CBIT)** is first-line behavioural therapy.

■ **Pharmacological** – **alpha-2 agonists (clonidine, guanfacine)** first for milder tics, especially with comorbid ADHD; **antipsychotics** (for example risperidone, aripiprazole, or older agents like haloperidol/pimozide) for severe, disabling tics.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Umbrella:** developmental onset + early persistent course + high comorbidity. Six families: ID, communication, autism, ADHD, specific learning, motor (incl. tics).
- ★ **Intellectual disability:** needs deficits in intellectual *and* adaptive functioning with developmental onset. **Severity set by adaptive function, not IQ.** Cut-off ~IQ 70 (two SD below mean). GDD = under-5, untestable; borderline = IQ 71–84.
- **ID severity:** mild 50–69 (~85%) · moderate 35–49 · severe 20–34 · profound <20.
- **Aetiology:** prenatal (genetic, TORCH, fetal alcohol, hypothyroidism) · perinatal (hypoxia, kernicterus, prematurity) · postnatal (infection, trauma, lead, malnutrition, deprivation).
- **Syndromes:** Down = commonest genetic (trisomy 21) · Fragile X = commonest inherited (FMR1 CGG, macro-orchidism) · Prader-Willi = paternal 15q (hyperphagia) vs Angelman = maternal 15q/UBE3A (laughter, ataxia) · Williams = 7q elastin, supravalvular AS, over-friendly · Rett = girls, MECP2, hand-wringing · PKU + hypothyroidism = treatable/screened · tuberous sclerosis = TSC1/TSC2, ash-leaf macules, autism.
- **Autism:** dyad = social-communication deficits + restricted/repetitive behaviour (incl. sensory). Levels 1–3 by support needs. ~1–2%, M:F ~3–4:1. Risperidone/aripiprazole only for irritability; early behavioural intervention is the core. No MMR link.
- **ADHD:** inattentive / hyperactive-impulsive / combined. >=6/9 (children), >=5/9 (17+), onset before 12, >=2 settings. Stimulants (methylphenidate, amphetamine) first; non-stimulants (atomoxetine = NRI; guanfacine/clonidine = alpha-2) when stimulants fail or with tics/anxiety/diversion. Taper alpha-2 agonists.
- **Learning/communication:** one specific learning disorder with reading (dyslexia), written-expression, or mathematics (dyscalculia) specifiers. Social pragmatic communication disorder = autism's social deficits *without* the restricted/repetitive behaviours.
- **Tics:** Tourette = multiple motor + >=1 vocal, >1 year, onset <18; wax and wane; comorbid OCD + ADHD; coprolalia uncommon. Manage with psychoeducation → habit reversal/CBIT → alpha-2 agonists → antipsychotics for severe.

ONE-DAY REVISION · PAPER 3

Child & adolescent disorders, made retrievable

The developmental conditions: disruptive behaviour, childhood mood and anxiety, attachment, elimination, early feeding, maltreatment, and the principles of child assessment. Hold the discriminators – ODD vs CD, DMDD vs paediatric bipolar, RAD vs DSED – because the comparison is the answer. The family, not the child alone, is the unit of care.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. Forensic detail (POCSO, reporting) and eating disorders live in the companion packs.

01 Disruptive, impulse-control & conduct disorders · **02** Childhood mood & anxiety · **03** Attachment · **04** Elimination disorders · **05** Early feeding disorders · **06** Child maltreatment · **07** Principles of child & adolescent assessment

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. DMDD is chronic, non-episodic irritability (onset before age 10, diagnosed only ages 6-18) created to curb over-diagnosis of paediatric bipolar; a child irritable all the time has DMDD, not bipolar (bipolar mood is episodic), and DMDD longitudinally predicts unipolar depression and anxiety, not bipolar.
2. The line ODD → CD → IED is whose rights are violated: ODD = defiance/temper with no rights violation, CD = violation of others' basic rights, IED = impulsive outbursts only; the "with limited prosocial emotions" (callous-unemotional) specifier plus early (childhood) onset = worst prognosis and the strongest link to antisocial PD.
3. School refusal vs truancy: refusal is anxiety-driven, parents know, child is at *home*, somatic complaints worst on school mornings, usually a good student; truancy is conduct-driven, parents unaware, child is *out*, no distress; manage refusal with graded prompt return plus CBT.
4. Strange Situation classifies attachment on reunion behaviour, not separation distress: secure B (~60-65%, soothed), avoidant A (ignores), ambivalent/resistant C (seeks yet resists); disorganised D has no coherent strategy and is the one most linked to maltreatment and later psychopathology.
5. Enuresis is diagnosed at age ≥5 years; after excluding organic cause the ladder is behavioural → the enuresis alarm (most effective, lowest relapse, treatment of choice) → desmopressin (fast but relapses) → imipramine (last line, cardiotoxic in overdose).

1 Disruptive, impulse-control & conduct disorders

Three diagnoses sit on a spectrum of externalising behaviour. The line between them is **whose rights are violated**: ODD breaks rules and tempers, CD breaks the basic rights of others, IED is episodic aggression out of proportion to provocation. Get the centrepiece contrast and the prognosis trail right.

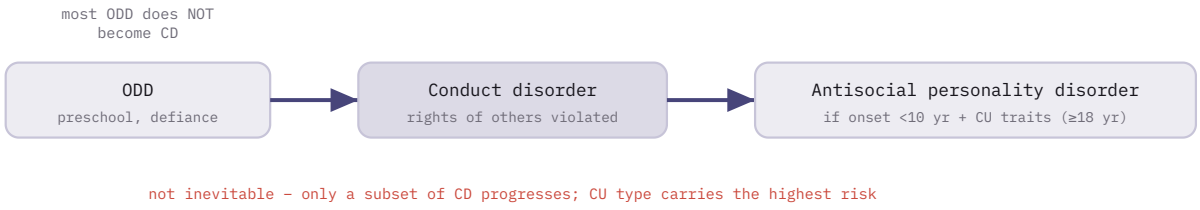
Feature	Oppositional defiant (ODD)	Conduct disorder (CD)	Intermittent explosive (IED)
Core	Angry/irritable mood, argumentative/defiant, vindictiveness toward authority. No major rights violation	Violation of others' basic rights and major age-norms	Recurrent impulsive aggressive outbursts grossly out of proportion to the trigger

Feature	Oppositional defiant (ODD)	Conduct disorder (CD)	Intermittent explosive (IED)
Four symptom clusters / domains	3 clusters: angry-irritable, argumentative-defiant, vindictive	4 groups: **aggression** to people/animals, **destruction** of property, **deceitfulness/theft**, **serious rule violations**	Verbal/non-destructive outbursts ≈2×/week ×3 months, *or* 3 destructive outbursts in 12 months
Typical onset	Preschool to early school years (often before CD)	Childhood-onset (<10 yr) or adolescent-onset (≥10 yr)	Rarely before age 6; usually late childhood / adolescence
Aggression premeditated?	Defiance, not predatory aggression	May be proactive/instrumental *and* reactive	Always **impulsive/reactive**, not premeditated or for gain
Key specifier	Severity by settings (1 / 2 / ≥3)	With limited prosocial emotions (callous-unemotional)	–
Prognosis	Many remit; a subset progress to CD	Childhood-onset & CU type → worst; can progress to antisocial PD	Chronic, fluctuating; high comorbidity

● CENTREPIECE TRAP

The **"with limited prosocial emotions"** specifier (the callous-unemotional, CU, subtype) needs ≥2 of: **lack of remorse/guilt, callous lack of empathy, unconcern about performance, shallow/deficient affect**, persistently over ≥12 months across relationships and settings. It marks the most severe, treatment-resistant CD and the strongest link to adult psychopathy. Examiners love it because it is the prognostic fork.

THE EXTERNALISING PROGRESSION (DEVELOPMENTAL TRAJECTORY)



A minority of ODD progresses to CD; a minority of CD (childhood-onset, callous-unemotional) progresses to antisocial personality disorder. The pathway is probabilistic, not fixed – the earlier the onset and the more callous the traits, the worse the trajectory.

Aetiology in one frame

- **Biological** – heritability, low resting autonomic arousal and reduced amygdala reactivity (especially in the CU subtype), prenatal substance exposure, difficult temperament.
- **Psychosocial** – harsh/inconsistent parenting, coercive family cycles (Patterson), maltreatment, deviant peers, poverty, neighbourhood disadvantage. The strongest *modifiable* driver is the parent-child interaction.

Management

- **Parent management training (PMT)** – first-line for ODD and younger CD. Teaches parents to reward prosocial behaviour and apply consistent, non-coercive limits; breaks the coercive cycle. Examples: Webster-Stratton Incredible Years, Parent-Child Interaction Therapy.
- **Multisystemic therapy (MST)** – intensive, home-based, family- and community-focused intervention for **older adolescents with severe CD/delinquency**. Works across all of the youth's systems (family, school, peers).

- **Adjuncts** – treat comorbidity (ADHD stimulants reduce aggression), CBT/anger management, functional family therapy. **No drug treats CD itself**; risperidone is used short-term for severe aggression. IED: SSRIs and CBT (relaxation, cognitive restructuring) have the best evidence.

QUICK RULE

ODD = **defiance, no rights violation**. CD = **rights of others violated**. IED = **impulsive outbursts only**. CU specifier + early onset = worst prognosis → antisocial PD. PMT for younger, MST for older.

2 Childhood mood & anxiety

Separation anxiety & selective mutism

- **Separation anxiety disorder** – developmentally excessive fear of separation from attachment figures: distress on separation, worry about losing them or harm befalling them, refusal to go out/sleep alone, nightmares of separation, somatic complaints. Duration **≥4 weeks** in children. The commonest childhood anxiety disorder and a frequent engine of **school refusal**.
- **Selective mutism** – consistent failure to speak in specific social situations (typically school) despite speaking normally elsewhere (usually home), for **≥1 month** (not the first month of school). Conceptually an **anxiety disorder**, strongly linked to social anxiety; not a language or defiance problem.

School refusal vs truancy

A favourite discriminator. Both are non-attendance, but the psychology is opposite: the refuser is anxious and at home; the truant is sensation-seeking and elsewhere.

Feature	School refusal (school phobia)	Truancy
Driver	Anxiety (separation, social, or performance)	Conduct – antisocial, sensation-seeking
Parents' knowledge	Parents **know**; child stays home with their awareness	Parents usually **unaware**; child conceals
Where is the child?	At **home**	Out – not at home, not at school
Emotional state	Distressed, somatic complaints (headache, abdominal pain) worst on school mornings, easing at weekends	No distress about non-attendance; little somatic complaint
Academics & behaviour	Often good student; behaviour otherwise compliant	Poor achievement; other rule-breaking, lying, stealing
Diagnostic home	Anxiety disorders	Conduct disorder (a serious rule violation)
Management	Graded **early return** to school + CBT/exposure; treat the anxiety	Behavioural/family + address conduct, peers, environment

RULE

School refusal is a *symptom*, not a diagnosis – always ask what it sits on (separation anxiety in younger children, social anxiety or depression in adolescents). The management principle is **graded, prompt return**: the longer the child is out, the harder re-entry becomes (avoidance is negatively reinforced).

Disruptive mood dysregulation disorder (DMDD)

- **DMDD core** – severe recurrent **temper outbursts** (verbal or behavioural), grossly out of proportion, ≥3×/week, with a **persistently irritable/angry mood between** outbursts. Present ≥12 months in ≥2 settings. Onset before age 10; diagnosed only between **ages 6 and 18**.

● THE DMDD VS PAEDIATRIC BIPOLAR TRAP

DMDD was created to curb the over-diagnosis of **paediatric bipolar disorder**. The key is the **course of mood**: bipolar is *episodic* (distinct, sustained periods of elevated/expansive mood, decreased need for sleep, grandiosity – a clear change from baseline). DMDD is **chronic, non-episodic irritability** with no discrete manic episodes. A child who is irritable *all the time* has DMDD, not bipolar. Longitudinally, DMDD predicts later **unipolar depression and anxiety**, not bipolar disorder. DMDD cannot coexist with ODD, IED, or bipolar (DMDD takes precedence over ODD/IED).

Childhood & adolescent depression

- **Presentation differs by age** – children may show **irritability rather than sadness** (irritable mood can substitute for depressed mood in the criteria), somatic complaints, social withdrawal, declining school performance, separation anxiety. Adolescents look closer to adult depression but with more irritability, hypersomnia, and reactivity.
- **Treatment** – **fluoxetine** is the best-evidenced and first-line antidepressant in paediatric depression (also escitalopram). CBT and IPT-A are first-line psychological treatments; combination (fluoxetine + CBT) is most effective (TADS study).

● SUICIDE-RISK TRAP

Suicide is a leading cause of death in adolescents. SSRIs carry a **black-box warning** for increased suicidal ideation/behaviour in under-25s, so start low, **monitor closely in early weeks**, and warn the family – but do not withhold treatment, as untreated depression is itself the larger risk. Always screen directly for suicidal ideation, plan, access to means, and prior attempts; ask about self-harm and substance use. Direct questioning does not plant the idea.

3 Attachment

Bowlby & Ainsworth

- **Bowlby's attachment theory** – the infant has an innate, evolved drive to seek proximity to a caregiver (a **secure base** from which to explore, a **safe haven** in distress). Repeated interactions build an **internal working model** of self and others. Phases: pre-attachment → attachment-in-the-making → clear-cut attachment → goal-corrected partnership. Prolonged early separation/deprivation harms development (the **monotropy** and maternal-deprivation hypotheses).
- **Ainsworth's Strange Situation** – a structured laboratory procedure (~20 min, 8 episodes) with repeated separations from and reunions with the caregiver, plus a stranger. The child's **behaviour at reunion** classifies the attachment style. The original three categories (A, B, C); Main & Solomon later added the fourth (D, disorganised).

Style	On separation	At reunion (key)	Caregiving linked
Secure (B) ≈60–65%	Distressed; explores using caregiver as a base	**Comforted & settles**, returns to play	Sensitive, responsive
Insecure-avoidant (A) ≈20%	Little distress	**Avoids/ignores** caregiver; minimal preference over stranger	Rejecting, intrusive, unavailable
Insecure-ambivalent/resistant (C) ≈10–15%	Intensely distressed; little exploration	**Seeks yet resists** contact; clingy then pushes away, hard to soothe	Inconsistent, unpredictable
Disorganised (D) ≈5–15%	Variable	**Contradictory, fearful, dazed**; no coherent strategy (freezing, approach-then-avoid)	Frightening/frightened, maltreatment, unresolved loss

MNEMONIC

"Secure Soothes, Avoidant Avoids, Ambivalent is Angry, Disorganised has no plan"

The classification turns on **reunion** behaviour, not separation distress. Disorganised (D) is the one most associated with maltreatment and the strongest predictor of later psychopathology.

Reactive attachment disorder vs disinhibited social engagement disorder

Both arise from the **same cause** – pathogenic care (social neglect, repeated changes of caregiver, institutional rearing) that prevents selective attachments. They diverge in the *direction* of the social behaviour: RAD is under-attached and inhibited; DSED is indiscriminately over-friendly.

Feature	Reactive attachment disorder (RAD)	Disinhibited social engagement (DSED)
Direction	Internalising – emotionally withdrawn, inhibited	Externalising – indiscriminately sociable, disinhibited
With caregivers	Rarely seeks or responds to comfort when distressed	Approaches and goes off with **unfamiliar adults** without checking back
Affect	Minimal social/emotional reciprocity, unexplained irritability, sadness, fearfulness	Overly familiar verbal/physical behaviour; reduced reticence with strangers
Resembles	Internalising disorders / depression; *not* autism (RAD improves with adequate care)	ADHD-like impulsivity, but the core is social disinhibition
Response to better care	Improves markedly with a stable, sensitive caregiver	May **persist** even after the caregiving environment improves
Minimum developmental age	Diagnosed only when developmental age ≥9 months (a selective attachment is normally expected)	

TRAP

Both require **documented pathogenic care**. RAD is the absence of attachment behaviour where it should appear; DSED is attachment behaviour aimed at everyone indiscriminately. The classic illustration is the post-institutional (e.g. Romanian orphanage) child. Distinguish RAD from **autism**: in autism the social deficit is pervasive and not explained by neglect, and does not reverse with caregiving change.

4 Elimination disorders

Enuresis

● **Definition** – repeated voiding of urine into clothes or bed, voluntary or involuntary, at a chronological (and developmental) age of ≥5 years, occurring ≥2×/week for ≥3 months (or causing distress/impairment), not due to a substance or medical condition.

Axis	Subtype	Meaning
By history	Primary	Continence **never** established (commoner; more often a maturational/genetic delay)
	Secondary	Relapse after ≥6–12 months dry (more often a **psychosocial stressor**: birth of a sibling, abuse, school stress)
By timing	Nocturnal	Night-time only (the commonest form)
	Diurnal	Daytime; raises suspicion of UTI, structural/urological cause, or psychological factors

■ **Management ladder** – reassurance and exclude organic cause first. Then: **(1) behavioural** (fluid timing, voiding schedule, star charts, lifting); **(2) the enuresis alarm** – the single most effective treatment, lowest relapse, treatment of choice for motivated families; **(3) desmopressin** (synthetic

ADH) – rapid effect, useful for sleepovers/short-term, but higher relapse; **(4) imipramine** (a TCA) – effective but reserved (relapse on stopping, cardiotoxicity in overdose).

Reassure and exclude an organic cause, then climb the ladder. The enuresis alarm is the most effective single treatment with the lowest relapse; desmopressin gives rapid short-term control; imipramine is last line.

Encopresis

- **Definition** – repeated passage of faeces in inappropriate places, at a chronological (and developmental) age of **≥4 years**, ≥1×/month for ≥3 months.
- **Retentive (with constipation & overflow)** – the commoner form (≈80–90%): chronic constipation, a distended rectum, and liquid **overflow incontinence** around the impacted mass. Managed with **disimpaction + laxatives**, toilet routine, dietary fibre/fluids, behavioural reinforcement.
- **Non-retentive (without constipation)** – no constipation; stool passed in inappropriate places, more often linked to **oppositional/emotional** factors. Managed behaviourally and by addressing the psychological driver; laxatives are not the answer.

5 Early feeding disorders

Three feeding/eating disorders of infancy and early childhood. ARFID overlaps the eating-disorders pack; cross-reference it there for the full contrast with anorexia nervosa.

Pica

Persistent eating of **non-nutritive, non-food** substances (clay, chalk, paint, hair, soil) for **≥1 month**, inappropriate to developmental level and not a culturally sanctioned practice. Minimum developmental age **≥2 years** (to exclude normal mouthing). Linked to iron/zinc deficiency, intellectual disability, autism, pregnancy. Risks: lead poisoning, obstruction, infection.

Rumination disorder

Repeated **regurgitation** of food (re-chewed, re-swallowed or spat out) for ≥1 month, not due to a GI/medical condition. Effortless, not nausea-driven. Seen in infants and in people with intellectual disability; risks weight loss/failure to thrive. Treated behaviourally (diaphragmatic breathing, aversive/differential reinforcement).

- **ARFID (avoidant/restrictive food intake disorder)** – restricted intake from **lack of interest in food, sensory aversion, or fear of aversive consequences** (choking, vomiting), leading to weight loss/nutritional deficiency/dependence on supplements/psychosocial impairment. The discriminator: **no body-image disturbance and no drive for thinness** – that is what separates it from anorexia nervosa. See the Eating Disorders pack.

TRAP

Pica and rumination both carry a **minimum developmental age** so that normal infant mouthing/regurgitation is not pathologised (pica ≥2 years of mental age; rumination is judged against the period after a normal feeding pattern). ARFID is **not** simple picky eating – it requires clinically significant nutritional or psychosocial consequences.

6 Child maltreatment

Four overlapping types, rarely occurring in isolation. The examiner wants the clinical indicators that should raise suspicion and the safeguarding sequence that follows.

Type	What it is	Clinical indicators (raise suspicion)
Physical abuse	Non-accidental physical injury	Injuries **inconsistent with the history** or developmental stage; injuries of different ages; patterned marks (bruises in non-bony/protected sites, immersion/cigarette burns); metaphyseal/posterior-rib fractures; delayed presentation; **changing or implausible story**
Sexual abuse	Involvement in sexual activity the child cannot consent to / does not understand	Often **no physical signs**; genital/anal injury or STI; age-inappropriate sexualised behaviour or knowledge; sudden behavioural change, regression, secrecy; recurrent UTIs, enuresis/encopresis relapse; the **disclosure** itself
Emotional abuse	Persistent ill-treatment harming emotional development (rejection, terrorising, denigration)	Low self-worth, fearfulness, developmental/speech delay, attachment problems, extremes of passivity or aggression
Neglect	Failure to meet basic physical/emotional/medical/educational needs	Failure to thrive, poor hygiene, untreated medical/dental problems, missed appointments, inadequate supervision/clothing, developmental delay

The safeguarding approach

- **Sequence** – ensure the child's **immediate safety** first; treat injuries; take a careful, non-leading history and document verbatim (the child's own words in quotes); examine and record (body maps, dated); do **not** conduct a forensic interrogation – that is the trained investigator's role; **report** to the statutory child-protection system; involve a multidisciplinary team; arrange follow-up and psychological support.
- **Stance** – the child's welfare is paramount; maintain a non-judgemental, believing stance toward the child; avoid confronting/accusing caregivers in a way that endangers the child; safety planning over confrontation.

INDIA: POCSO & MANDATORY REPORTING

The **Protection of Children from Sexual Offences (POCSO) Act, 2012** makes reporting of suspected child sexual abuse **mandatory** in India: failure to report by anyone who knows is itself an offence. It is child-friendly, gender-neutral, and defines a child as anyone under 18. The clinician's duty is to report; investigation and proof are not the clinician's burden. Full forensic and legal detail in the Forensic Psychiatry pack.

7 Principles of child & adolescent assessment

Child assessment is not adult psychiatry scaled down. It is multi-informant, developmental, play-based, and treats the family system as the patient.

- **Developmental history** – the spine of the assessment: pregnancy and birth, milestones (motor, language, social, toileting), temperament, attachment history, schooling, peer relationships, and the timeline of the presenting problem against the developmental baseline. A symptom is read **relative**

to developmental age, not chronological age alone.

- **Collaborative & multiple informants** – the child is rarely the sole or best historian. Gather **parent and school** reports (teachers see peer function and attention in a structured setting); standardised informant-rated scales (e.g. Conners, SDQ, CBCL). Discrepancies between settings are themselves data.
- **Play-based & observational assessment** – in younger children, play and drawing are the medium of communication and the window onto fears, relationships and inner life. Observe interaction, joint attention, separation/reunion behaviour, and the parent-child dyad directly.
- **The family as the unit of care** – the child is embedded in a family and school system; formulation and intervention target that system, not just the child. Parents are co-therapists (the logic of parent management training). Always consider the family's resources, stressors and the safeguarding context.
- **Developmentally appropriate communication** – match language, rapport and consent/assent to the child's stage; see the adolescent alone for part of the interview (confidentiality and its limits explained); be alert to risk (self-harm, abuse, exploitation).

HOLD THIS

Child assessment = **developmental history + multi-informant collateral (parents & school) + play-based observation + the family as the unit of care**. Read every symptom against developmental age, not chronology.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **ODD vs CD vs IED:** ODD = defiance/temper, no rights violation · CD = rights of others violated (aggression, destruction, deceit, rule-breaking) · IED = impulsive outbursts only. CU ("limited prosocial emotions") specifier + early onset = worst → antisocial PD. PMT younger, MST older adolescents.
- **School refusal vs truancy:** refusal = anxiety, parents know, child at *home*, somatic, good student · truancy = conduct, parents unaware, child *out*, no distress. Refusal → graded prompt return + CBT.
- ★ **DMDD vs paediatric bipolar:** DMDD = *chronic* non-episodic irritability (6–18 yr, onset <10), predicts later depression/anxiety · bipolar = *episodic* mania. Irritable all the time = DMDD, not bipolar.
- **Paediatric depression:** irritability can replace sadness; **fluoxetine** first-line; SSRI black-box for under-25 suicidality → monitor, don't withhold. Always screen suicide directly.
- **Attachment (Strange Situation, reunion behaviour):** secure (B, ≈60%, soothed) · avoidant (A, ignores) · ambivalent/resistant (C, seeks-yet-resists) · disorganised (D, no strategy, linked to maltreatment).
- **RAD vs DSED:** same cause (pathogenic care/neglect). RAD = withdrawn, won't seek comfort · DSED = indiscriminately friendly, goes off with strangers; DSED may persist after care improves. Both need developmental age ≥9 months.
- **Enuresis:** ≥5 yr. Primary = never dry (maturational) · secondary = relapse (stressor). Ladder: behavioural → **alarm (most effective, lowest relapse)** → desmopressin (fast, relapses) → imipramine (last line).
- **Encopresis:** ≥4 yr. Retentive (commonest) = constipation + overflow → disimpaction + laxatives · non-retentive = no constipation, oppositional/emotional → behavioural.
- **Early feeding:** pica = non-food, dev age ≥2 yr · rumination = effortless regurgitation · ARFID = restriction with *no* body-image disturbance (separates it from anorexia).
- **Maltreatment:** physical (injury inconsistent with history, different ages), sexual (often no signs; disclosure is key), emotional, neglect. Safety first → document verbatim → report. India: **POCSO 2012** makes reporting mandatory.
- **Assessment:** developmental history + multi-informant collateral (parents & school) + play-based observation + family as the unit of care. Symptom read against developmental age.

ONE-DAY REVISION · PAPER 3

Perinatal & Women's Mental Health

The hormone-sensitive states across a woman's reproductive life: premenstrual, perinatal, perimenopausal. The exam rewards crisp discrimination of the three postpartum syndromes, knowing postpartum psychosis as a true emergency, and prescribing safely in pregnancy and lactation. Every table here is a structured answer in waiting.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. Drug teratogenicity sits in the amber key cells; danger signs sit in coral.

01 The perinatal mood spectrum · **02** Postpartum psychosis: the emergency · **03** Premenstrual dysphoric disorder (PMDD) · **04** The reproductive life-cycle lens · **05** Prescribing in pregnancy & lactation · **06** Psychiatric disorders during pregnancy · **07** Women's MH epidemiology & special topics

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. Postpartum psychosis is a psychiatric emergency: abrupt onset within the first 2 weeks, a bipolar-spectrum illness carrying infanticide and maternal suicide risk, so admit (mother-baby unit) and treat with antipsychotic + lithium + ECT, never manage at home on first presentation.
2. Tell the three postpartum syndromes apart by onset and danger: blues (day 2-4, ~50-80%, self-limiting, no drugs), depression (first 4-6 weeks, ~10-15%, sertraline), psychosis (abrupt, first 2 weeks, ~1-2/1000, emergency).
3. Valproate is contraindicated in women of childbearing potential (neural tube defects + neurodevelopmental harm, a regulatory hard line); lamotrigine is the safer mood stabiliser and sertraline the preferred SSRI in breastfeeding.
4. Teratogen pairings to recall: lithium → Ebstein anomaly (1st trimester, cardiac), carbamazepine → neural tube defects, paroxetine → cardiac septal defects, benzodiazepines → floppy baby syndrome & neonatal withdrawal.
5. PMDD requires ≥5 luteal-phase symptoms with ≥1 core affective symptom and a symptom-free follicular week, confirmed by prospective daily ratings over 2 cycles; an SSRI dosed luteal-phase only is the distinctive treatment, and no symptom-free week means premenstrual exacerbation, not PMDD.

1 The perinatal mood spectrum

Three distinct entities, not a continuum. The whole topic turns on telling them apart by **onset, severity and danger**. Blues is normal and self-limiting; depression needs treatment; psychosis is an emergency. Get the timing and the discriminators right and the marks follow.

Feature	Postpartum (maternity) blues	Postpartum depression	Postpartum psychosis
Onset / timing	day 2–4, peaks ~day 5, resolves by day 10–14	within first 4–6 weeks (DSM-5 onset specifier: within 4 weeks; clinically up to 12 months)	abrupt, usually within first 2 weeks (often days 3–10)
Prevalence	very common, ~50–80% of mothers	~10–15%	rare, ~1–2 per 1000 births
Core features	tearfulness, lability, irritability, anxiety, mild insomnia; insight intact, function preserved	low mood, anhedonia, guilt, fatigue, sleep/appetite change, poor bonding, thoughts of inadequacy; can be anxious or with intrusive thoughts	rapidly fluctuating mood, confusion/perplexity, delusions (often about the baby), hallucinations, disorganisation; a delirium-like picture

Feature	Postpartum (maternity) blues	Postpartum depression	Postpartum psychosis
Danger signs	none specific; reassess if it persists >2 weeks (may herald depression)	active suicidal ideation, thoughts of harming the infant, total inability to care for self/baby	infanticide and maternal suicide risk; command hallucinations, delusions about the infant, neglect
Treatment	reassurance, support, sleep; no medication; watchful follow-up	psychotherapy (CBT/IPT) for mild; SSRI (sertraline first line, esp. if breastfeeding) for moderate–severe	psychiatric emergency: admit, ideally mother–baby unit; antipsychotic + lithium; ECT highly effective
Prognosis	excellent, self-limiting	good with treatment; risk of recurrence and chronicity if untreated	good for the acute episode but high recurrence in future pregnancies; strong bipolar link

Blues: days 2 to 4, self-limiting. Psychosis: abrupt within the first two weeks, an emergency. Depression: builds over the first four to six weeks and can persist for months.

HOLD THIS

Blues = **common, day 2–4, no drugs**. Depression = **~10–15%, weeks, SSRI**. Psychosis = **rare, abrupt, admit + ECT**. The onset and the danger signs separate them.

● CLASSIC TRAP

Do not call early tearfulness "depression." Blues peaks around day 5 and clears by two weeks with support alone. The screening discriminator is **persistence beyond 2 weeks** plus loss of function. Conversely, never dismiss abrupt confusion or odd beliefs in the first fortnight as "just blues" – that is psychosis until proven otherwise. The **Edinburgh Postnatal Depression Scale (EPDS)**, a 10-item self-report, is the standard screen; item 10 flags self-harm.

2 Postpartum psychosis: the emergency

The single most important obstetric–psychiatric emergency. Abrupt onset, fluctuating course, and a real risk to two lives. Treat it as you would any acute psychosis with delirium-like features: **admit, do not send home**.

Why it is an emergency

▲ Risk to the infant – **infanticide** risk, especially with delusions about the baby (it is evil, swapped, or better off dead) and command hallucinations. Altruistic motive is classic and dangerous.

▲ **Risk to the mother** – **maternal suicide**, a leading cause of maternal death in the year after birth. Rapid mood swings and perplexity make her unpredictable.

■ **Urgency of admission** – she should rarely be left alone with the infant. **Inpatient admission** is the default; never manage acute postpartum psychosis at home on the first presentation.

The bipolar link

● **Strong affective / bipolar spectrum** – postpartum psychosis is now understood largely as a **bipolar-spectrum** illness. Many cases are a bipolar manic or mixed episode triggered by childbirth; a personal or family history of bipolar disorder is the strongest risk factor.

◆ **Recurrence risk** – very high. A woman with a prior episode has roughly a **1 in 2 (up to ~50%)** risk of recurrence in a future pregnancy; higher still with established bipolar disorder. This drives prophylaxis planning before conception.

Treatment

Acute management

Admit, ideally to a **mother–baby unit** (keeps mother and infant together, protects bonding, allows supervised care).
Antipsychotic for psychotic and manic features.
Lithium as mood stabiliser (especially the bipolar phenotype).
ECT is highly effective and works fast – valuable when severe, when rapid response is needed, or when breastfeeding and avoiding drug load.

Watch-points

Rule out an **organic cause** (infection, eclampsia, thyroid, Sheehan, substance) – the perplexed, fluctuating presentation can mimic **delirium**.
Benzodiazepines for agitation as needed.
Weigh breastfeeding against drug choice; lithium passes into milk.
Plan **prophylaxis** for future pregnancies.

● MNEMONIC

"Postpartum psychosis = bipolar in disguise"

Treat the affective core: lithium and antipsychotic, ECT when severe. Mother–baby unit, not the postnatal ward. High recurrence means prophylaxis is planned *before* the next pregnancy.

● TRAP

ECT is among the most effective and fastest treatments in postpartum psychosis and severe postpartum depression, particularly where there is suicidality, refusal to eat or drink, or the need for rapid recovery to resume infant care. It is not a last resort here. Lithium prophylaxis started immediately after delivery markedly cuts recurrence in women with a bipolar diathesis.

3 Premenstrual dysphoric disorder (PMDD)

A DSM-5 depressive disorder, not a personality trait or "bad PMS." The marks live in the **criteria**, the **prospective rating requirement**, and the differential from premenstrual *exacerbation* of another disorder.

DSM-5 criteria (essentials)

■ **Timing** – symptoms in the **final week before menses (luteal phase)**, start to improve within a few days of onset of menses, and become minimal or absent in the week post-menses. Present in **most cycles of the past year**.

● **Symptom count** – at least **5 symptoms** total, including at least **one core affective** symptom: marked affective lability, marked irritability/anger, markedly depressed mood/hopelessness, or marked anxiety/tension.

- **Plus other symptoms to reach 5** – reduced interest, difficulty concentrating, lethargy, appetite change, hypersomnia/insomnia, feeling overwhelmed, and physical symptoms (breast tenderness, bloating, joint/muscle pain).

- **Impairment & exclusion** – symptoms cause clinically significant distress or interference, and are **not merely an exacerbation** of another disorder (MDD, panic, persistent depressive disorder, a personality disorder).

QUICK RULE

The diagnosis requires **prospective daily ratings over at least 2 symptomatic cycles** to confirm. Retrospective recall is unreliable. Until confirmed, code it as "provisional."

The key differential

PMDD

Symptoms are **confined to the luteal phase** and remit fully after menses begins. There is a clear **symptom-free week** follicular phase. Daily rating shows a clean cyclical pattern.

Premenstrual exacerbation (PME)

An underlying disorder (e.g. MDD, GAD, bipolar) is present **throughout the cycle** but *worsens* premenstrually. There is **no symptom-free week**. Treat the underlying disorder; the cyclical worsening is a clue, not a separate diagnosis.

Management

Tier	Option	Note
First line	SSRI (fluoxetine, sertraline, paroxetine, escitalopram)	can be **continuous** or **luteal-phase only** (dose from ovulation to menses); often works faster than in MDD
Hormonal	combined oral contraceptive (esp. drospirenone-containing); GnRH agonists in refractory cases	suppress ovulation; GnRH agonists need add-back to avoid hypo-oestrogenic effects
Lifestyle	exercise, sleep, reduced caffeine/alcohol/salt; CBT	adjuncts; calcium and possibly vitamin B6 have modest evidence
Refractory	GnRH agonist with add-back; rarely surgical oophorectomy	last resort, specialist setting

● RULE

PMDD is one of the few indications where an SSRI can be dosed **intermittently (luteal phase only)** with good effect, reflecting a rapid, partly serotonergic mechanism distinct from its slow antidepressant action. This intermittent dosing is a favourite viva point.

4 The reproductive life-cycle lens

Read women's mood disorders along the reproductive axis. Three states share a common thread: **rapid change in gonadal hormones** against a backdrop of individual vulnerability. The same woman is often vulnerable at each transition.

- **Hormone-sensitive states** – **premenstrual** (luteal drop in oestrogen/progesterone), **perinatal** (the steep post-delivery fall), and **perimenopausal** (fluctuating then falling oestrogen). It is the *change* in hormones, more than absolute levels, that matters.

- **Window-of-vulnerability concept** – certain women are **neuroendocrine-sensitive**: normal hormonal shifts that most tolerate destabilise their mood. A history of PMDD predicts perinatal and perimenopausal depression, and vice versa. This is the unifying exam idea.

- **Perimenopausal depression** – the **menopause transition** (perimenopause), not post-menopause, carries the peak risk. New-onset or recurrent depression rises 2–4 fold; vasomotor symptoms, sleep disruption and the "domino" effect contribute.

- **Perimenopausal treatment** – antidepressants (SSRI/SNRI) are first line for the depression; **oestrogen (HRT)** can have an antidepressant effect in perimenopausal (not post-menopausal) depression and treats vasomotor symptoms. SNRIs (venlafaxine, desvenlafaxine) also ease hot flushes.

QUICK RULE

Premenstrual, perinatal, perimenopausal: the **three P's** of reproductive-hormone-sensitive mood disorder. Vulnerability at one predicts vulnerability at the others.

5 Prescribing in pregnancy & lactation

The central tension: drug risk to the foetus versus the risk of **untreated maternal illness** (relapse, suicide, poor self-care, obstetric complications, impaired bonding). The exam wants the general principles and the specific teratogens.

General principles

- **Treat, do not under-treat** – untreated illness is itself a risk to mother and child. The decision is always risk–benefit, never "avoid all drugs."

- **Lowest effective dose, monotherapy** – prefer a single agent at the lowest dose that works; avoid polypharmacy; use an agent with the best evidence and longest track record.

- **Account for untreated-illness risk** – weigh teratogenic risk against relapse risk, especially in severe recurrent illness. Continue an effective agent if stopping it would likely cause relapse, after informed discussion.

- **Timing matters** – the **first trimester** (organogenesis) is the window for structural teratogenicity; late-pregnancy exposure risks neonatal adaptation/withdrawal and (for some drugs) specific syndromes.

Centrepiece: teratogens and lactation cautions to know

Drug	Pregnancy risk	Critical window	Lactation
Valproate	Neural tube defects (spina bifida), craniofacial & limb defects; neurodevelopmental harm (lowered IQ, autism risk)	1st trimester; neurodevelopment across pregnancy	relatively low milk levels, but the drug is so harmful it is broadly avoided
Lithium	Ebstein anomaly (atrialised right ventricle, tricuspid malformation); risk is small in absolute terms	1st trimester (cardiac)	excreted in milk; breastfeeding generally discouraged (neonatal toxicity, dehydration risk)
Carbamazepine	Neural tube defects, craniofacial defects, fingernail hypoplasia (fetal anticonvulsant syndrome)	1st trimester	usually considered compatible; monitor infant
Lamotrigine	Relatively safer mood stabiliser in pregnancy; preferred where one is needed	–	passes into milk in variable amounts; monitor infant (rash)
Paroxetine	Cardiac malformations (septal defects); the least-preferred SSRI in pregnancy	1st trimester	low milk levels; acceptable if already established
Benzodiazepines	Floppy baby syndrome & neonatal withdrawal (late); small cleft-palate risk (older data, disputed)	late pregnancy (neonatal); 1st trimester (cleft)	caution: sedation, poor feeding in infant

Drug	Pregnancy risk	Critical window	Lactation
Sertraline	among the better-studied SSRIs; no clear major teratogenicity	–	preferred in breastfeeding (low milk transfer, low infant serum levels)

HOLD THIS

Valproate = neural tube + neurodevelopment, **avoid in women of childbearing potential**. **Lithium** = Ebstein, 1st trimester. **Carbamazepine** = neural tube. **Lamotrigine** = safer. **Paroxetine** = cardiac. **Sertraline** = safest in lactation.

MNEMONIC

"Valproate & carbamazepine open the tube; lithium breaks the heart"

Both anticonvulsant mood stabilisers cause neural tube defects (folate-related); lithium causes Ebstein's cardiac anomaly in the first trimester. Lamotrigine is the safer stabiliser; sertraline the safer SSRI in lactation.

CLASSIC TRAP

Valproate is contraindicated in women of childbearing potential unless no alternative exists and a pregnancy-prevention programme is in place. This is a regulatory hard line, not a preference. If a woman of reproductive age needs a mood stabiliser, valproate is the wrong default answer. High-dose **folic acid (5 mg)** is given to any woman on an anticonvulsant who may conceive, though it does not fully abolish the valproate risk.

6 Psychiatric disorders during pregnancy

Pregnancy is not protective. Depression and anxiety are common antenatally and predict postpartum illness. The harder clinical problem is the woman with pre-existing serious mental illness who is, or wants to be, pregnant.

- **Depression & anxiety in pregnancy** – antenatal depression is common (around 1 in 10) and is the strongest predictor of postpartum depression. Untreated antenatal depression links to poor antenatal care, preterm birth and low birth weight. Manage with psychotherapy first for mild cases; SSRI (sertraline) for moderate–severe.
- **Pre-existing serious mental illness** – plan **preconception** where possible: review medication, switch off teratogens before conception, optimise the regimen, and arrange close perinatal follow-up. Bipolar and prior postpartum psychosis need a written perinatal plan and often prophylaxis around delivery.
- **Relapse risk of stopping medication** – abrupt discontinuation is dangerous. In **bipolar disorder**, stopping a mood stabiliser in pregnancy carries a high relapse rate (well over half), and abrupt lithium cessation risks rebound. The risk of relapse frequently outweighs the drug's foetal risk, especially in severe recurrent illness.

RULE

The commonest error is to **stop all medication the moment pregnancy is confirmed**. For serious recurrent illness this often precipitates relapse, with its own foetal and maternal harms. Decisions should be shared, documented, and made (ideally) before conception rather than reactively in the first trimester.

7 Women's MH epidemiology & special topics

The brief background that frames a long-answer introduction. Know the sex differences and the named special topics.

Epidemiology

◆ **Female preponderance** – depression and most anxiety disorders are roughly **twice** as common in women (the classic ~2:1 ratio). Women also have higher rates of PTSD, eating disorders and (in some data) bipolar II; men have higher rates of substance use and antisocial/completed-suicide patterns.

● **Drivers** – biological (hormonal sensitivity, the reproductive transitions above), psychological (rumination), and social (gender-based violence, caregiving load, poverty, discrimination). The exam wants the biopsychosocial framing, not a single cause.

Special topics in brief

▲ **Intimate partner violence (IPV)** – a major social determinant. Sequelae include depression, PTSD, anxiety, substance use and suicidality; IPV often **worsens in pregnancy**. Routine sensitive enquiry, safety planning and referral are part of perinatal care.

● **Infertility & pregnancy loss** – infertility, miscarriage, stillbirth and termination carry high rates of grief, depression and anxiety; perinatal loss is an under-recognised cause of complicated grief. Validate the loss; screen for depression.

● **PCOS & thyroid links** – **PCOS** is associated with higher rates of depression, anxiety and disordered eating (partly via hyperandrogenism, infertility and body image). **Thyroid disease** is commoner in women: hypothyroidism mimics depression, hyperthyroidism mimics anxiety, and **postpartum thyroiditis** is a key reversible mimic of postpartum mood disorder – always check thyroid function in perinatal mood presentations.

● TRAP

Before settling on a perinatal mood diagnosis, **exclude the organic mimics**: anaemia, thyroid dysfunction (postpartum thyroiditis), and sleep deprivation. A new depressive or anxious picture postpartum with a goitre, weight or pulse change should prompt thyroid function tests, not just an antidepressant.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Spectrum:** blues (day 2–4, 50–80%, self-limiting, no drugs) · depression (weeks, ~10–15%, SSRI) · psychosis (abrupt, first 2 wks, ~1–2/1000, emergency).
- ★ **Postpartum psychosis:** bipolar-spectrum illness; infanticide + maternal suicide risk; admit to a mother–baby unit; antipsychotic + lithium + ECT (highly effective); ~50% recurrence next pregnancy.
- **PMDD:** luteal-only, ≥5 symptoms with ≥1 core affective, symptom-free follicular week, confirmed by prospective daily rating over 2 cycles. PME = worsens premenstrually but no symptom-free week.
- **PMDD treatment:** SSRI continuous or luteal-phase only; COC; lifestyle. Intermittent SSRI dosing is the distinctive point.
- **Three P's:** premenstrual, perinatal, perimenopausal are hormone-sensitive states; window of vulnerability; perimenopausal (not post-menopausal) depression peaks; oestrogen helps then.
- **Prescribing principles:** treat don't under-treat · lowest effective dose · monotherapy · weigh untreated-illness risk · 1st trimester = organogenesis.
- **Teratogens:** valproate (neural tube + neurodevelopment, avoid in childbearing women) · lithium (Ebstein, 1st trimester) · carbamazepine (neural tube) · lamotrigine (safer) · paroxetine (cardiac) · benzodiazepines (floppy baby) · sertraline (safest in lactation).

- **In pregnancy:** antenatal depression predicts postpartum depression; plan preconception; never stop a mood stabiliser abruptly (high bipolar relapse).

- **Epidemiology:** depression and anxiety ~2:1 female. IPV worsens in pregnancy. Infertility/loss cause grief and depression. PCOS and thyroid (postpartum thyroiditis) are key links and reversible mimics.

ONE-DAY REVISION · PAPER 3

Measurements & rating scales, made retrievable

Psychometrics first, then the scales worth a name in an answer book. For every instrument hold three things: what it measures, who rates it (clinician or self), and the one feature or cutoff that makes it examinable. The master table is the centrepiece; the discriminators are the marks.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. IQ tests (WAIS, WISC, Binet, MISIC) live in the companion Assessment & Testing pack.

01 Psychometric foundations · **02** Depression & mania scales · **03** Psychosis scales · **04** Anxiety, OCD & trauma scales · **05** Cognition & dementia · **06** Global functioning, side effects & substance scales · **07** Child, developmental & intelligence instruments · **08** Master table: the scales worth a name

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. PANSS = 30 items as 7 positive + 7 negative + 16 general, each scored 1 to 7, so the minimum total is 30, not 0 (the single commonest factual slip).
2. Sensitivity rules out (SnNOUT) and specificity rules in (SpPIN); sensitivity and specificity are intrinsic to the test, but PPV and NPV move with prevalence, so a screen positive in a low-base-rate setting is often a false positive.
3. Reliability is necessary but not sufficient for validity: a consistently wrong scale is reliable yet invalid, but no scale is valid without first being reliable.
4. The recurring primary-care pair: PHQ-9 ≥10 = likely major depression (item 9 flags suicide) and GAD-7 ≥10 = likely GAD; EPDS screen positive is ≥13.
5. MMSE is out of /30 with impairment at <24; MoCA is the more sensitive screen for mild cognitive impairment, not MMSE, and CDR *stages* dementia (0/0.5/1/2/3) rather than screening.

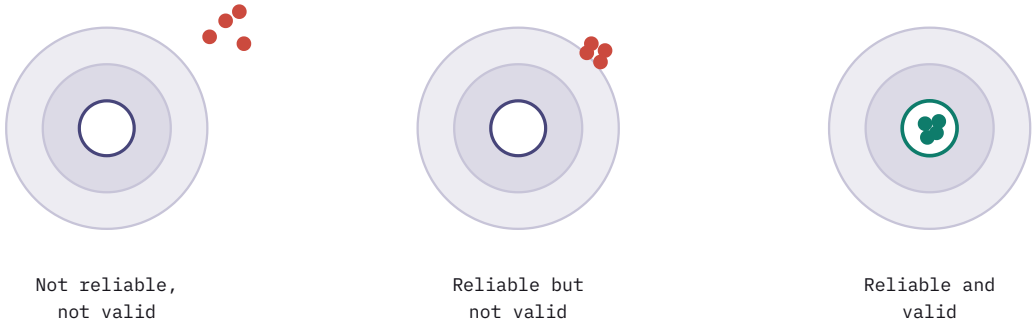
1 Psychometric foundations

Every scale lives or dies on two properties. **Reliability** = consistency (does it give the same answer twice?). **Validity** = accuracy (does it measure what it claims?). A scale can be reliable yet invalid, but it cannot be valid without first being reliable.

Reliability vs validity, side by side

Reliability (consistency)	What it checks	Validity (accuracy)	What it checks
Test-retest	same result on repeat administration over time (stability)	Face	looks like it measures the construct, on inspection (weakest)
Inter-rater	agreement between two raters (kappa for categorical)	Content	items cover the whole domain of the construct
Internal consistency (Cronbach α)	items correlate with each other; α > 0.7 acceptable, > 0.8 good	Construct	fits the theoretical construct (convergent + discriminant)
Split-half	two halves of the test agree (a form of internal consistency)	Criterion	agrees with a gold-standard criterion: **concurrent** (now) and **predictive** (future)

RELIABILITY VS VALIDITY: THE BULLSEYE IDEA



Scattered shots = unreliable. Tightly clustered but off-centre = reliable but invalid (consistent error). Tight and on-centre = both. Reliability is necessary but not sufficient for validity.

HOLD THIS

Reliability is necessary but not sufficient for validity. A consistently wrong scale is reliable and invalid. The reverse cannot happen: there is no valid-but-unreliable scale.

Diagnostic accuracy: the 2×2 that runs every screen

Once a scale gives a score, a cutoff turns it into a test. Sensitivity and specificity are properties of the *test*; predictive values depend on the *population* (prevalence).

	Disease present	Disease absent
Test positive	True positive (TP)	False positive (FP)
Test negative	False negative (FN)	True negative (TN)

- **Sensitivity** – TP / (TP + FN). Detects the diseased; a sensitive test rules **out** (SnNOUT: a negative on a Sensitive test rules out). Screening tools are built sensitive.
- **Specificity** – TN / (TN + FP). Identifies the well; a specific test rules **in** (SpPIN: a positive on a Specific test rules in). Diagnostic confirmation needs specificity.
- **PPV / NPV** – PPV = TP / (TP + FP), probability that a positive is truly diseased. NPV = TN / (TN + FN). Both swing with **prevalence**: in a low-prevalence population PPV falls even for an excellent test.

CLASSIC TRAP

Sensitivity and specificity are **intrinsic to the test** and do not change with prevalence. PPV and NPV **do** change with prevalence. This is why a screening positive (e.g. PHQ-9, EPDS) must be confirmed by clinical interview before any label: a positive screen in a low-base-rate setting is often a false positive.

Screening test

Applied to apparently well people to catch cases early. Built **high-sensitivity** (few missed cases). Tolerates false positives. Examples: PHQ-9, EPDS, AUDIT, M-CHAT, MoCA.

Diagnostic test

Applied to confirm or exclude in a suspected case. Built **high-specificity** (few false labels). Usually a structured clinical interview or a detailed instrument. Examples: CAPS for PTSD, full Y-BOCS interview.

2 Depression & mania scales

The most-tested cluster. Know the flagship clinician scale (HAM-D), the flagship self-report (BDI), and the brief primary-care screen (PHQ-9). For mania there is essentially one: YMRS.

Scale	Measures	Rater	Notable feature / cutoff
HAM-D / HDRS	depression severity	clinician	the classic; 17-item version standard; weighted to somatic items; trial gold standard
MADRS	depression severity, change	clinician	10 items; less somatic weighting than HAM-D; sensitive to treatment change
BDI / BDI-II	depression severity	self	21 items; the classic self-report (Beck); cognitive emphasis
PHQ-9	depression screen + severity	self	9 items mapped to DSM criteria; **≥10 = likely major depression**; item 9 flags suicide risk
EPDS	perinatal (postnatal) depression	self	10 items; omits somatic items (overlap with pregnancy); screen **≥13**
YMRS	mania severity	clinician	11 items; four items double-weighted (irritability, speech, thought content, disruptive behaviour)

■ **HAM-D vs MADRS** – both clinician-rated. HAM-D is older and weighted toward **somatic/sleep** items (can inflate scores in physical illness); MADRS is leaner, more sensitive to **change**, and a frequent trial outcome measure.

● **Self vs clinician** – self-rated: BDI, PHQ-9, EPDS. Clinician-rated: HAM-D, MADRS, YMRS. A common viva discriminator is to sort a list of scales into these two columns.

● MNEMONIC

"Mania has a Young rater"

YMRS = **Young** Mania Rating Scale, clinician-rated, 11 items, four double-weighted. There is no widely used self-report mania equivalent of comparable standing.

3 Psychosis scales

The examiner wants the PANSS three-subscale structure and the difference between BPRS (broad) and PANSS (positive plus negative plus general). CGI is the universal global add-on.

Scale	Measures	Rater	Notable feature
BPRS	broad psychopathology in psychosis	clinician	18 items; older, brief; covers psychotic + affective + somatic symptoms
PANSS	positive, negative & general symptoms	clinician	30 items; **3 subscales**: 7 positive + 7 negative + 16 general; the standard trial scale
SAPS	positive symptoms (Andreasen)	clinician	hallucinations, delusions, bizarre behaviour, formal thought disorder
SANS	negative symptoms (Andreasen)	clinician	affective flattening, alogia, avolition, anhedonia, attention
CGI	global severity & change	clinician	two parts: CGI-S (severity) & CGI-I (improvement); both **1–7**

◆ **PANSS structure** – **30 items = 7 positive + 7 negative + 16 general psychopathology**. Each item scored 1–7, so a floor of 30 (not 0). The positive and negative subscales map onto Crow's type I / type II distinction.

● **SANS / SAPS** – Andreasen's paired scales: **SANS = Negative, SAPS = Positive** (both have an S for symptoms). Predate PANSS; still used to characterise the negative syndrome.

■ **CGI two parts** – **CGI-S** rates current severity 1 (normal) to 7 (most severe). **CGI-I** rates change from baseline 1 (very much improved) to 7 (very much worse), with 4 = no change. Quick, global, transdiagnostic.

● TRAP

PANSS items are scored **1 to 7**, not 0 to 6. The minimum total is therefore 30, not zero, and the minimum for any subscale equals its item count. A common factual slip in vivas.

4 Anxiety, OCD & trauma scales

Scale	Measures	Rater	Notable feature / cutoff
HAM-A / HARS	anxiety severity	clinician	14 items; psychic + somatic anxiety subscores; the classic anxiety scale
GAD-7	generalised anxiety screen	self	7 items; ≥10 = likely GAD; pairs with PHQ-9 in primary care
Y-BOCS	OCD severity	clinician	the OCD standard; **10 items = 5 obsessions + 5 severity dimensions**; separate symptom checklist; rates severity independent of content
CAPS	PTSD diagnosis & severity	clinician	structured interview; the **gold standard** for PTSD; DSM-5 version CAPS-5
PCL-5	PTSD symptom screen	self	20 items mapped to DSM-5; self-report counterpart to CAPS

◆ **Y-BOCS** – the OCD reference scale. Ten items: **five rate obsessions, five rate compulsions**, across time spent, interference, distress, resistance and control. Severity is scored *independently of symptom content*, so an unusual obsession is not penalised. A symptom checklist catalogues content separately.

■ **CAPS vs PCL** – for PTSD, **CAPS = clinician interview, gold standard**; **PCL = self-report screen**. The pattern mirrors HAM-D/BDI: a clinician instrument for diagnosis, a self-report for screening and monitoring.

● MNEMONIC

"CAPS Confirms, PCL just Checks"

CAPS (Clinician-Administered PTSD Scale) confirms the diagnosis; PCL (PTSD Checklist) is the self-report screen. Y-BOCS = the OCD standard; GAD-7 ≥10 mirrors PHQ-9 ≥10.

5 Cognition & dementia

MMSE is the universal bedside screen but has a ceiling and an education bias; MoCA catches milder impairment; CDR *stages* rather than screens. Know the MMSE bands by heart.

Scale	Measures	Rater	Notable feature / cutoff
MMSE	global cognition screen	clinician	/30; quick; ceiling effect & education bias; misses mild & executive deficits
MoCA	cognition, MCI-sensitive	clinician	/30; more sensitive to **mild cognitive impairment**; tests executive & visuospatial; ≥26 normal; +1 point if ≤12 yrs education
ACE-III	5 cognitive domains	clinician	/100; attention, memory, fluency, language, visuospatial; includes the MMSE within it
Clock-drawing	executive / visuospatial	clinician	rapid; sensitive to executive dysfunction; often paired with MMSE (Mini-Cog)

Scale	Measures	Rater	Notable feature / cutoff
CDR	dementia **staging**	clinician	informant-based; rates 6 domains; 0 / 0.5 / 1 / 2 / 3 = none / questionable / mild / moderate / severe

◆ **MMSE bands (commonly quoted)** – out of 30: **24–30 normal, 18–23 (or 19–23) mild, 10–17 moderate, <10 severe**. Cutoff for cognitive impairment usually taken at **<24**. Adjust upward for high education, downward for low literacy.

● **MMSE limits** – **ceiling effect** (high-functioning people score full marks despite real decline), strong **education and literacy bias**, and poor at detecting executive or frontal dysfunction. MoCA was designed to fix the first and third.

■ **Screen vs stage** – MMSE, MoCA and ACE-III **screen** (is there impairment?). **CDR stages** (how advanced?), is informant-based, and is widely used in dementia trials and research.

● TRAP

MoCA is **more sensitive to mild cognitive impairment** than MMSE precisely because it taxes executive function, abstraction and delayed recall harder. Do not say MMSE is the more sensitive of the two: it is the quicker, more familiar, but blunter screen.

6 Global functioning, side effects & substance scales

Global functioning

● **GAF** – Global Assessment of Functioning, a single **0–100** score (higher = better). Was DSM-IV Axis V; **dropped in DSM-5** in favour of WHODAS 2.0.

● **WHODAS 2.0** – WHO Disability Assessment Schedule; assesses functioning across **6 domains** (cognition, mobility, self-care, getting along, life activities, participation). The DSM-5 replacement for GAF.

Movement & side-effect scales

Scale	Targets	Notable feature
AIMS	**tardive dyskinesia**	Abnormal Involuntary Movement Scale; 12 items; rates orofacial & limb-trunk dyskinesia
Simpson-Angus	**drug-induced parkinsonism**	rates rigidity, gait, tremor, glabellar tap, salivation
Barnes	**akathisia**	Barnes Akathisia Rating Scale; separates objective restlessness from subjective distress
UKU	broad side-effect burden	comprehensive: psychic, neurological, autonomic & other adverse effects

● MNEMONIC

"A-S-B for the three movements"

AIMS → tardive dyskinesia · **S**impson-Angus → parkinsonism · **B**arnes → akathisia. UKU = the broad catch-all side-effect scale.

Substance & withdrawal scales

Scale	Use	Rater	Notable feature / cutoff
CIWA-Ar	alcohol **withdrawal** severity	clinician	10 items; drives symptom-triggered benzodiazepine dosing; ≥8–10 treat

Scale	Use	Rater	Notable feature / cutoff
CAGE	alcohol problem screen	self / interview	4 questions (Cut down, Annoyed, Guilty, Eye-opener); **≥2 positive**
AUDIT	hazardous drinking screen	self	WHO 10-item; **≥8 hazardous**; AUDIT-C is the 3-item short form
Fagerstrom (FTND)	nicotine dependence	self	6 items; time-to-first-cigarette is the key item; grades dependence severity

● TRAP

CIWA-Ar rates withdrawal, not intoxication or dependence. It guides symptom-triggered detoxification (give a benzodiazepine when the score crosses threshold, typically 8–10). Do not confuse it with CAGE/AUDIT, which *screen for a drinking problem*, or with Fagerstrom, which grades *nicotine* dependence.

7 Child, developmental & intelligence instruments

A brief but examinable set: one adaptive-behaviour scale, two autism instruments, one ADHD scale, and a pointer to the IQ tests that live in the companion pack.

Scale	Measures	Rater / source	Notable feature
Vineland (VABS)	**adaptive behaviour**	caregiver interview	4 domains: communication, daily living, socialisation, motor; key in intellectual disability assessment
CARS	autism severity	clinician	Childhood Autism Rating Scale; 15 items; rates & grades autism severity
M-CHAT	autism **screen** in toddlers	parent	screening (not diagnostic) at 16–30 months; positive screen needs follow-up assessment
Conners	ADHD symptoms & behaviour	parent / teacher	multi-informant rating; tracks inattention, hyperactivity, conduct

■ **CARS vs M-CHAT** – **M-CHAT screens** toddlers (parent-completed, flags who needs assessment); **CARS rates severity** in those being evaluated. Vineland measures *adaptive function* and is essential alongside an IQ test to diagnose intellectual disability (both deficits required).

● **Cross-reference: IQ tests** – the intelligence instruments (**WAIS** adults, **WISC** children, **Stanford-Binet**, **Raven's Progressive Matrices** culture-fair, and the Indian adaptations **MISIC** and **Bhatia's BKT**) are covered in the companion *Assessment & Testing* pack. Deviation IQ: mean 100, SD 15.

8 Master table: the scales worth a name

One sheet to revise the lot. Sort first by who rates it, then by what it measures.

RATING SCALES MASTER LIST

Scale	Measures	Rater	One feature / cutoff
HAM-D	depression	clinician	17 items; trial classic; somatic-weighted
MADRS	depression, change	clinician	10 items; change-sensitive
BDI-II	depression	self	21 items; Beck; cognitive emphasis
PHQ-9	depression screen	self	≥10 likely MDD; item 9 = suicide
EPDS	perinatal depression	self	10 items; non-somatic; ≥13
YMRS	mania	clinician	11 items; 4 double-weighted

Scale	Measures	Rater	One feature / cutoff
BPRS	broad psychosis	clinician	18 items; brief, older
PANSS	psychosis (pos/neg/gen)	clinician	30 items; 7+7+16; scored 1–7
SANS / SAPS	negative / positive sx	clinician	Andreasen pair
CGI	global severity & change	clinician	S & I parts; both 1–7
HAM-A	anxiety	clinician	14 items; psychic + somatic
GAD-7	anxiety screen	self	≥10 likely GAD
Y-BOCS	OCD	clinician	10 items; severity, not content
CAPS-5	PTSD	clinician	structured interview; gold standard
PCL-5	PTSD screen	self	20 items; DSM-5
MMSE	cognition screen	clinician	/30; <24 impaired; education bias
MoCA	cognition, MCI	clinician	/30; ≥26 normal; more sensitive
CDR	dementia staging	clinician	0/0.5/1/2/3; informant-based
GAF	global functioning	clinician	0–100; dropped in DSM-5
WHODAS 2.0	disability / functioning	self / clinician	6 domains; DSM-5 GAF replacement
AIMS	tardive dyskinesia	clinician	12 items; orofacial + limb
Simpson-Angus	parkinsonism	clinician	rigidity, tremor, gait
Barnes	akathisia	clinician	objective + subjective
CIWA-Ar	alcohol withdrawal	clinician	10 items; treat ≥8–10
CAGE	alcohol screen	self	4 Q; ≥2 positive
AUDIT	hazardous drinking	self	10 items; ≥8 hazardous
Fagerstrom	nicotine dependence	self	6 items; time-to-first-cigarette
Vineland	adaptive behaviour	caregiver	4 domains; ID assessment
CARS / M-CHAT	autism rate / screen	clinician / parent	CARS rates, M-CHAT screens
Conners	ADHD	parent / teacher	multi-informant

HOLD THIS

Clinician-rated workhorses: **HAM-D, MADRS, YMRS, PANSS, HAM-A, Y-BOCS, CAPS, MMSE, MoCA, CDR**. Self-rated screens: **BDI, PHQ-9, EPDS, GAD-7, PCL, AUDIT, CAGE, Fagerstrom**. The PHQ-9 / GAD-7 pair and the ≥10 cutoff recur constantly.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Psychometrics:** reliability = consistency (test-retest, inter-rater, Cronbach α >0.7, split-half); validity = accuracy (face, content, construct, criterion = concurrent + predictive). Reliability is necessary but not sufficient for validity.
- **Accuracy:** Sensitivity rules **out** (SnNOUT), Specificity rules **in** (SpPIN). Sens/spec are test properties; PPV/NPV move with prevalence. Screens = high sensitivity; diagnostics = high specificity.
- **Depression/mania:** clinician = HAM-D (somatic-weighted), MADRS (change), YMRS (mania, 11 items). Self = BDI, PHQ-9 (≥10), EPDS (≥13, non-somatic perinatal).
- ★ **Psychosis:** PANSS = 30 items, 7 positive + 7 negative + 16 general, scored 1–7. SANS = negative, SAPS = positive (Andreasen). CGI = severity (S) + improvement (I), both 1–7.

- **Anxiety/OCD/trauma:** HAM-A (clinician, 14), GAD-7 (self, ≥10). Y-BOCS = OCD standard, 10 items, severity not content. CAPS = clinician gold standard for PTSD; PCL = self screen.
- **Cognition:** MMSE /30, <24 impaired, ceiling + education bias. MoCA /30, ≥26 normal, more sensitive to MCI. ACE-III /100. CDR *stages* (0/0.5/1/2/3), informant-based.
- **Function & side effects:** GAF 0–100 (dropped in DSM-5) → WHODAS 2.0 (6 domains). AIMS = tardive dyskinesia, Simpson-Angus = parkinsonism, Barnes = akathisia, UKU = broad.
- **Substance:** CIWA-Ar = alcohol withdrawal (treat ≥8–10), CAGE = 4 Q (≥2), AUDIT = 10 items (≥8), Fagerstrom = nicotine.
- **Child/dev:** Vineland = adaptive behaviour (ID work-up), CARS rates autism, M-CHAT screens toddlers, Conners = ADHD multi-informant. IQ tests (WAIS, WISC, Binet, Raven, MISIC, BKT) in the Assessment pack.

ONE-DAY REVISION · PAPER 3

Antipsychotics & mood stabilisers, made retrievable

The drugs that anchor Paper III: how dopamine blockade buys an antipsychotic effect and four side-effect bills in the same stroke, why the atypicals soften some of them, what makes clozapine both irreplaceable and dangerous, and how lithium, valproate, carbamazepine and lamotrigine divide the mood-stabiliser map. Hold the receptor logic, the monitoring numbers, and the one fact that distinguishes each agent.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. Receptor mechanisms, antidepressants & anxiolytics live in the companion pack (Psychopharmacology II).

01 Mechanism: the four dopamine pathways · **02** Typical (first-generation) antipsychotics · **03** Atypical (second-generation) antipsychotics · **04** Clozapine: the irreplaceable, dangerous one · **05** Adverse effects across the class · **06** Mood stabilisers · **07** Long-acting injectables & the prescribing baseline

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. The four dopamine tracts decode the whole class: **mesolimbic D2 block = antipsychotic effect**, mesocortical = worse negatives/cognition, nigrostriatal = EPS, tuberoinfundibular = **hyperprolactinaemia**.
2. What makes an antipsychotic "atypical" is added **5-HT2A antagonism** (plus faster D2 dissociation), which disinhibits striatal dopamine and lowers EPS; the therapeutic D2 occupancy window is **~65-80%**, above which EPS climbs steeply.
3. EPS sort by onset: acute dystonia (hours, give anticholinergic) → akathisia (days-weeks, give **propranolol** not anticholinergic) → parkinsonism (weeks-months) → tardive dyskinesia (months-years, may be irreversible); **anticholinergics can worsen tardive dyskinesia**.
4. Clozapine is reserved for treatment-resistant schizophrenia (two failed adequate trials) and is **never first-line**; mandatory ANC monitoring for agranulocytosis, watch myocarditis, dose-related seizures and constipation/ileus, but it causes **no EPS and no prolactin rise**.
5. Lithium range is **0.6-1.0 mmol/L** on a 12-hour trough; toxicity is precipitated by dehydration, NSAIDs, thiazides and ACE inhibitors, and first-trimester use risks **Ebstein's anomaly**.

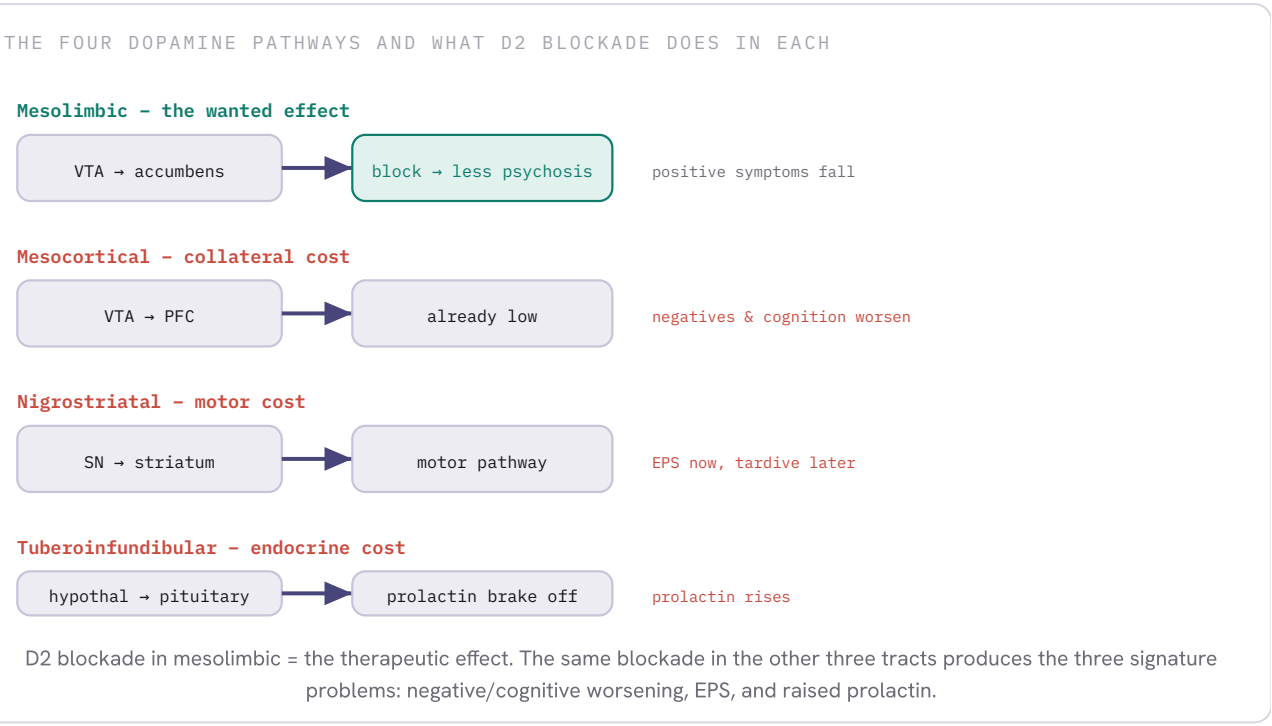
1 Mechanism: the four dopamine pathways

Every antipsychotic action and most of the side-effect burden trace back to **D2 blockade** in four dopamine tracts. The same drug helps in one pathway and harms in the other three. Get this map and the side-effect profile becomes deducible, not memorised.

Pathway	Runs from → to	Normal role	Consequence of D2 blockade
Mesolimbic	VTA → nucleus accumbens / limbic	reward, salience; overactive in psychosis	antipsychotic effect – dampens positive symptoms (delusions, hallucinations)
Mesocortical	VTA → prefrontal cortex	cognition, motivation; already underactive in schizophrenia	worsens negative symptoms & cognition (blocking an already low pathway)
Nigrostriatal	substantia nigra → striatum	motor control (the extrapyramidal system)	extrapyramidal side effects (EPS); long-term, tardive dyskinesia

III

Pathway	Runs from → to	Normal role	Consequence of D2 blockade
Tuberoinfundibular	hypothalamus → anterior pituitary	dopamine *tonically inhibits* prolactin release	hyperprolactinaemia (galactorrhoea, amenorrhoea, sexual dysfunction)



D2 blockade in mesolimbic = the therapeutic effect. The same blockade in the other three tracts produces the three signature problems: negative/cognitive worsening, EPS, and raised prolactin.

Typical versus atypical: what makes an antipsychotic "atypical"

● **Typical (first-generation)** – potent, relatively pure **D2 antagonists**. High D2 occupancy buys efficacy on positive symptoms but pays the full nigrostriatal and tuberoinfundibular bill: high EPS, high prolactin.

■ **Atypical (second-generation)** – defined by added **5-HT2A antagonism** alongside D2 blockade. Serotonin normally inhibits dopamine release in the nigrostriatal tract; blocking 5-HT2A *disinhibits* dopamine there, sparing the striatum and so reducing EPS. Atypicals also tend to have **looser, faster-dissociating D2 binding** (Kapur).

◆ **Fast dissociation hypothesis** – atypicals bind D2 loosely and come off quickly, allowing brief surges of endogenous dopamine to compete. Enough occupancy to block psychosis, too transient to lock the striatum into EPS. The **~65–80% D2 occupancy** window is the therapeutic sweet spot; above ~80% EPS climbs steeply.

● TRAP

"Atypical" does not mean "no EPS" or "no prolactin." **Risperidone** at higher doses behaves like a typical (dose-dependent EPS and the *highest* prolactin rise of the group). The atypical advantage is relative, and it is traded for a **metabolic** bill instead.

2 Typical (first-generation) antipsychotics

The first-generation drugs split on **potency** – how much drug is needed for the same D2 effect. Potency does not mean "stronger"; it predicts *which* side effects dominate. High-potency drugs hit D2 hard and little else; low-potency drugs need large doses and so drag in muscarinic, histaminic and alpha-1 blockade.

Group	Examples	Receptor reach
High-potency	haloperidol, fluphenazine, trifluoperazine, pimozide	tight, mostly D2
Low-potency	chlorpromazine, thioridazine	D2 plus muscarinic, H1, alpha-1

THE POTENCY / SIDE-EFFECT TRADE-OFF (CENTREPIECE)

Side effect	High-potency (e.g. haloperidol)	Low-potency (e.g. chlorpromazine)
EPS (acute dystonia, parkinsonism, akathisia)	high	low
Hyperprolactinaemia	high	lower
Sedation (H1 block)	low	high
Anticholinergic (dry mouth, constipation, blurred vision, urinary retention)	low	high
Orthostatic hypotension (alpha-1 block)	low	high

Read it as a see-saw: the EPS / prolactin burden runs *opposite* to the sedation / anticholinergic / hypotension burden. High-potency = EPS heavy, "clean" otherwise; low-potency = sedating and anticholinergic, fewer EPS.

QUICK RULE

High potency = high EPS & prolactin, low everything else. Low potency = low EPS, high sedation/anticholinergic/hypotension. The two columns mirror each other.

▲ Thioridazine – the standout for **QTc prolongation** (and pigmentary retinopathy at high dose); largely withdrawn or restricted for that reason. **Chlorpromazine** causes photosensitivity and, rarely, cholestatic jaundice and corneal/lens deposits.

● CLASSIC TRAP

Do not equate "high potency" with "more side effects." Potency is the dose needed for D2 effect, not toxicity. **Haloperidol** (high potency) gives florid EPS but is well tolerated cardiovascularly; **chlorpromazine** (low potency) is sedating and hypotensive but causes fewer movement problems.

3 Atypical (second-generation) antipsychotics

Each atypical is best remembered by its **one signature problem** – the fact a viva examiner is fishing for. The class shares the 5-HT2A/D2 logic; what differs is the secondary receptor profile and therefore the side-effect that dominates.

ATYPICAL PROFILES & SIGNATURE SIDE EFFECT

Risperidone

Receptor	potent D2 + 5-HT2A; dose-dependent
Signature / use	highest prolactin rise; dose-dependent EPS

Olanzapine

Receptor	broad D2/5-HT2A/H1/M block
Signature / use	heaviest metabolic burden (weight, lipids, glucose)

Quetiapine

Receptor	loose D2, strong H1; low D2 occupancy
Signature / use	sedation; very low EPS; useful in PD psychosis & bipolar depression

Aripiprazole

Receptor	D2 partial agonist ("dopamine stabiliser") + 5-HT1A partial agonist
Signature / use	low metabolic, low prolactin (can *lower* it); akathisia, activation

Ziprasidone	
Receptor	D2/5-HT2A; take with food (absorption)
Signature / use	QTc prolongation; weight-neutral

Lurasidone	
Receptor	D2/5-HT2A + 5-HT7; take with food
Signature / use	metabolically sparing; akathisia; licensed for bipolar depression

Amisulpride	
Receptor	selective D2/D3; low dose targets presynaptic autoreceptors
Signature / use	high prolactin; low-dose helps negative symptoms; renally cleared

Cariprazine	
Receptor	D3-preferring D2/D3 partial agonist
Signature / use	akathisia; evidence for negative symptoms & bipolar

One signature per drug: risperidone = prolactin, olanzapine = metabolic, quetiapine = sedation, aripiprazole = partial agonist, ziprasidone = QTc, lurasidone/aripiprazole = metabolic-sparing, amisulpride = prolactin + negative-symptom niche.

Metabolic burden ranking (the high-yield league table)

Tier	Agents	Weight / glucose / lipid risk
Highest	**clozapine, olanzapine**	marked
Intermediate	quetiapine, risperidone	moderate
Lowest	aripiprazole, ziprasidone, lurasidone	minimal

HOLD THIS

Clozapine and olanzapine top the metabolic table. **Aripiprazole, ziprasidone and lurasidone** are the metabolically sparing choices. Risperidone owns prolactin; ziprasidone owns QTc.

● MNEMONIC

"Clozapine & Olanzapine carry the most weight"

The two worst metabolic offenders both end in **-zapine**. The metabolically friendly trio (aripiprazole, ziprasidone, lurasidone) is the answer when a patient already has diabetes or obesity.

4 Clozapine: the irreplaceable, dangerous one

Clozapine is the most effective antipsychotic for the patients no other drug helps, and the only one with an evidence-backed anti-suicidal effect. It is fenced in by mandatory blood monitoring because its rare toxicities can kill. Examiners love the indications-versus-risks tension.

Indications (and the rule)

Treatment-resistant schizophrenia – failure of two adequate trials of different antipsychotics (one an atypical), each at adequate dose and duration.
Reduced suicidality in schizophrenia and schizoaffective disorder (InterSePT).
Also: persistent aggression, tardive dyskinesia, Parkinson-disease psychosis.
Never first-line. Its unique efficacy never offsets the monitoring burden until two drugs have failed.

Serious adverse effects & monitoring

Agranulocytosis – potentially fatal; mandatory **ANC monitoring** (weekly first, then less often). Stop if ANC falls below threshold.
Myocarditis / cardiomyopathy – early (first weeks), watch troponin, CRP, tachycardia.
Seizures – dose-related (risk rises above ~600 mg/day).
Constipation / paralytic ileus – can be fatal; the commonest lethal effect in practice.
Sialorrhoea – paradoxical hypersalivation, esp. nocturnal.
Metabolic – top of the weight/glucose/lipid table.
Also: sedation, myocarditis, fever in titration, orthostasis.

◆ **Haematological monitoring** – baseline FBC, then ANC checks (commonly weekly for the first 18 weeks, then fortnightly to a year, then monthly). A traffic-light system (green / amber / red) governs continuation; a red result mandates stopping clozapine. Granulocyte nadir typically occurs in the

first 3 months.

● TRAP

Clozapine itself *lowers* the seizure threshold and is markedly proconvulsant at high dose, yet it does **not** cause EPS or raise prolactin (negligible nigrostriatal/tuberoinfundibular effect) – the reason it helps tardive dyskinesia. Smoking **induces** CYP1A2 and lowers clozapine levels; stopping smoking (or an infection) can push levels into toxicity.

5 Adverse effects across the class

The extrapyramidal syndromes by time course (centrepiece)

Four movement syndromes, each with a characteristic **onset**. The time from starting the drug is the single best discriminator in a viva.

EPS TIME COURSE: HOURS TO YEARS

Earlier onset = the first three (reversible, dopaminergic imbalance). Tardive dyskinesia is late and may be irreversible (dopamine-receptor supersensitivity).

Syndrome	Onset	Features	Treatment
Acute dystonia	hours to days	sustained spasm: torticollis, oculogyric crisis, laryngospasm (emergency); young men at risk	anticholinergic (IM/IV benztropine or procyclidine); rapid relief
Akathisia	days to weeks	subjective inner restlessness + inability to sit still; linked to suicidality	reduce dose; **propranolol**, benzodiazepine; consider switch
Parkinsonism	weeks to months	bradykinesia, rigidity, resting tremor, masked facies (drug-induced)	reduce dose; anticholinergic; switch to lower-EPS agent
Tardive dyskinesia	months to years	choreoathetoid orofacial/lingual movements; may be irreversible	stop/reduce causal drug; switch to clozapine; VMAT2 inhibitors (valbenazine, tetrabenazine)

● CLASSIC TRAP

Anticholinergics treat acute dystonia and parkinsonism but can worsen tardive dyskinesia. Akathisia responds to **propranolol**, not anticholinergics. Distinguish akathisia (restlessness driven from inside) from agitation and from psychotic relapse – the management is opposite.

Neuroleptic malignant syndrome (NMS)

▲ Features – the tetrad: **hyperthermia, lead-pipe rigidity, autonomic instability, altered consciousness**. Labs: raised **CK**, leucocytosis, deranged LFTs; risk of rhabdomyolysis and renal failure. Onset over days, often early or after a dose increase; high-potency typicals are the classic trigger.

■ **Management** – **stop the antipsychotic**, supportive cooling and fluids (the priority), then **dantrolene** (muscle relaxant) and **bromocriptine** (dopamine agonist); benzodiazepines; ICU. Mortality if missed.

NMS

Cause: dopamine **blockade**. Onset over days. **Lead-pipe rigidity**, raised CK, bradyreflexia. Treat: dantrolene, bromocriptine.

Serotonin syndrome (contrast)

Cause: serotonin **excess**. Onset over hours. **Clonus, hyperreflexia**, agitation, dilated pupils. Treat: cyproheptadine.

Other class effects

● **Hyperprolactinaemia** – tuberoinfundibular D2 block: galactorrhoea, amenorrhoea, gynaecomastia, sexual dysfunction, and long-term reduced bone density. Worst with **risperidone, amisulpride** and high-potency typicals; least with **aripiprazole** (can lower prolactin).

◆ **Metabolic syndrome & monitoring** – weight gain, dyslipidaemia, impaired glucose tolerance / diabetes. Monitor **weight/BMI, waist, fasting glucose or HbA1c, lipids and blood pressure** at baseline, 12 weeks, then annually (more often early). Worst with clozapine and olanzapine.

◆ **QTc prolongation** – risk of torsades de pointes. Highest with **thioridazine, pimozide, ziprasidone** and IV haloperidol. Baseline and on-treatment **ECG** where risk factors exist; correct potassium and magnesium; caution with other QT-prolonging drugs.

6 Mood stabilisers

Four agents divide the territory: lithium (the reference standard), valproate, carbamazepine and lamotrigine. Each is pinned by its therapeutic niche, its monitoring number, and one signature toxicity.

Lithium

● **Proposed mechanism** – inhibits **inositol monophosphatase** (depleting the phosphatidyl-inositol second-messenger cycle) and **GSK-3β**; net effect is neuroprotective and modulates intracellular signalling. The precise mood-stabilising mechanism remains uncertain.

◆ **Therapeutic range** – **0.6 to 1.0 mmol/L** for maintenance (some target up to 1.2 in acute mania); measured as a **12-hour trough**. The narrow therapeutic index is why levels are mandatory.

▲ **Signs of toxicity** – early: coarse tremor, GI upset, ataxia, slurred speech; advancing (above ~1.5, severe above ~2.0 mmol/L): confusion, myoclonus, seizures, nystagmus, renal failure, coma. Precipitated by **dehydration, NSAIDs, thiazides, ACE inhibitors** and salt restriction.

■ **Monitoring** – **renal (U&E, eGFR) and thyroid (TSH)** function at baseline and roughly 6-monthly; lithium levels regularly (and after any dose or interacting-drug change); calcium (risk of hyperparathyroidism). Long-term risks: **nephrogenic diabetes insipidus, hypothyroidism, chronic kidney disease.**

▲ **Teratogenicity** – first-trimester lithium raises the risk of **Ebstein's anomaly** (tricuspid valve malformation). Counsel and weigh against relapse risk; do not stop abruptly.

● TRAP

Lithium is renally cleared and competes with sodium. Anything that drops sodium or reduces clearance (**dehydration, low-salt diet, NSAIDs, thiazides, ACE inhibitors/ARBs**) raises lithium and can tip a stable patient into toxicity. A lithium level "in range" can still be toxic if drawn too soon after a dose – always a 12-hour trough.

The anticonvulsant mood stabilisers

- **Valproate** – broad anticonvulsant; first-line for acute **mania**. Adverse: weight gain, tremor, hair loss, **hepatotoxicity, pancreatitis, thrombocytopenia**, and **highly teratogenic** (neural tube defects, neurodevelopmental harm). Avoid in women of childbearing potential. Level ~50–100 mcg/mL where measured.
- **Carbamazepine** – blocks voltage-gated sodium channels. Watch for **enzyme induction** (CYP3A4 autoinduction lowers its own and other drug levels, including oral contraceptives), **hyponatraemia (SIADH)**, and **blood dyscrasias** (agranulocytosis, aplastic anaemia). Risk of Stevens-Johnson, especially with **HLA-B*1502** (screen in at-risk Asian populations). Level ~4–12 mcg/mL.
- **Lamotrigine** – best for **bipolar depression maintenance** (prevents the depressive pole; weak against acute mania). Must be titrated **slowly** to minimise the risk of **Stevens-Johnson syndrome / toxic epidermal necrolysis**. Valproate raises lamotrigine levels, so the titration is even slower when combined. No routine level monitoring.

MOOD STABILISERS AT A GLANCE

MOOD STABILISERS AT A GLANCE (CENTREPIECE)

Agent	Main indication	Therapeutic level	Key adverse effects	Monitoring
Lithium	classic maintenance; anti-suicidal	0.6–1.0 mmol/L	tremor, hypothyroidism, nephrogenic DI, CKD; Ebstein's anomaly	levels, U&E/eGFR, TSH, Ca
Valproate	acute mania (first-line)	~50–100 mcg/mL	weight gain, hair loss, hepatotoxicity, pancreatitis; teratogen (NTD)	LFTs, FBC; pregnancy avoidance
Carbamazepine	mania / mixed	~4–12 mcg/mL	enzyme induction, hyponatraemia (SIADH), blood dyscrasias, SJS	FBC, Na, LFTs, levels; HLA-B*1502
Lamotrigine	bipolar depression maintenance	not routinely measured	Stevens-Johnson / TEN (slow titration)	clinical (rash); slow up-titration

Lithium = the reference, with the tightest monitoring. Valproate = acute mania (but teratogenic). Carbamazepine = the enzyme inducer with hyponatraemia. Lamotrigine = the depression-pole drug whose whole risk is the rash.

MNEMONIC

"Lamotrigine Lifts the Low"

Lamotrigine protects the **depressive** pole of bipolar; it is weak for mania. Mirror it: **valproate for the high (mania), lamotrigine for the low (depression).**

7 Long-acting injectables & the prescribing baseline

Long-acting injectable antipsychotics (LAIs, depots) deliver drug over weeks from a single injection, removing the day-to-day adherence question. They are a tool for relapse prevention, not a different class of drug.

When to use an LAI

- **Best indications** – **poor or uncertain adherence**, frequent relapse, patient preference, and as a relapse-prevention strategy in established psychotic illness. Steadier plasma levels and a clear record of what was actually delivered.

● **Available depots** – typicals: haloperidol decanoate, flupentixol/zuclopenthixol decanoate, fluphenazine decanoate. Atypicals: risperidone, paliperidone (monthly and 3-monthly), aripiprazole, olanzapine pamoate (caution: post-injection delirium/sedation syndrome). Establish tolerability with the oral form first.

▲ **Caveats** – slow to adjust (a side effect persists for weeks), injection-site reactions, and the same EPS/metabolic/prolactin profile as the oral drug. Not for acute titration or the first-ever, drug-naive presentation where the diagnosis is unsettled.

Baseline workup before starting any antipsychotic

◆ **Baseline metabolic & safety screen** – **weight/BMI and waist, blood pressure, fasting glucose or HbA1c, lipid profile**; **FBC, U&E, LFTs, TFTs, prolactin**; and an **ECG** (QTc) where cardiac risk factors or a QT-prolonging agent exist. Then repeat key measures at 12 weeks and annually.

HOLD THIS

Before the first antipsychotic: **weight, glucose/HbA1c, lipids, BP, ECG** at minimum. The patient is not the QA layer – the baseline catches the metabolic and cardiac risk before the drug adds to it.

QUICK-RECALL · DRILL THESE ONE-LINERS

★ **Four pathways:** mesolimbic D2 block = the antipsychotic effect; mesocortical = worse negatives/cognition; nigrostriatal = EPS; tuberoinfundibular = high prolactin.

- **Atypical = 5-HT2A antagonism** (disinhibits striatal dopamine) + faster D2 dissociation → less EPS, but a metabolic bill instead. Therapeutic D2 occupancy ~65–80%.
- **Typical potency:** high-potency (haloperidol) = high EPS & prolactin, low sedation/anticholinergic; low-potency (chlorpromazine) = the reverse. Potency is dose, not toxicity.
- **Atypical signatures:** risperidone = prolactin · olanzapine = metabolic · quetiapine = sedation/low EPS · aripiprazole = D2 partial agonist · ziprasidone = QTc · lurasidone = metabolic-sparing · amisulpride = prolactin + negative symptoms.
- **Metabolic league:** worst = clozapine + olanzapine; sparing = aripiprazole, ziprasidone, lurasidone.
- **Clozapine:** treatment-resistant schizophrenia (two failed trials) + anti-suicidal; **never first-line**. Watch agranulocytosis (ANC), myocarditis, seizures (dose-related), constipation/ileus, sialorrhoea, metabolic. No EPS, no prolactin.
- **EPS by clock:** dystonia (hours, give anticholinergic) · akathisia (days–weeks, give propranolol) · parkinsonism (weeks–months) · tardive dyskinesia (months–years, VMAT2 inhibitor, switch to clozapine).
- **NMS:** hyperthermia + lead-pipe rigidity + autonomic instability + altered consciousness, raised CK. Stop drug, cool, dantrolene + bromocriptine. Dopamine *blockade* (vs serotonin syndrome = clonus, hyperreflexia).
- **Lithium:** range 0.6–1.0 mmol/L (12-h trough); toxicity from dehydration/NSAIDs/thiazides/ACE-i; monitor renal + thyroid; Ebstein's anomaly in pregnancy.
- **Anticonvulsants:** valproate = acute mania but teratogen (NTD), hepatotoxic; carbamazepine = enzyme inducer + hyponatraemia (SIADH) + dyscrasias; lamotrigine = bipolar depression maintenance, slow titration for Stevens-Johnson.
- **LAIs:** for poor adherence / relapse prevention; establish tolerability orally first; not for acute titration. Baseline before any antipsychotic: weight, glucose/HbA1c, lipids, BP, ECG.

ONE-DAY REVISION · PAPER 3

Psychopharmacology II: Antidepressants & Anxiolytics

The mood and anxiety pharmacopoeia, made retrievable. Hold the class, the mechanism, the one signature adverse effect, and the two emergencies you must never confuse (serotonin syndrome vs neuroleptic malignant syndrome). Every comparison table here is a long-answer question already half-written.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. Antipsychotics, mood stabilisers & lithium live in the companion pack (Psychopharmacology I).

01 Antidepressants: the overview · **02** SSRIs: the agents and what separates them · **03** SNRIs, TCAs and MAOIs · **04** Serotonin syndrome vs neuroleptic malignant syndrome · **05** Anxiolytics & hypnotics · **06** Pharmacokinetics & drug interactions · **07** Special populations

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. Serotonin syndrome vs NMS: the splitting sign is neuromuscular, clonus and hyper-reflexia (hyperkinetic, onset in hours, serotonergic drug, treat with cyproheptadine) versus lead-pipe rigidity with markedly raised CK (hypokinetic, onset over days, dopamine D2 blockade, treat with dantrolene).
2. All antidepressant classes raise synaptic monoamines within hours, but the antidepressant effect needs 2 to 4 weeks (autoreceptor 5-HT_{1A} desensitisation, receptor down-regulation, neuroplasticity); the early activation window carries a transiently raised suicide risk, worst in the under-25s.
3. Half-life extremes: fluoxetine = longest (active metabolite norfluoxetine) → self-tapering, near-absent discontinuation, but a 5-week washout before an MAOI; paroxetine = short, no active metabolite → worst discontinuation (FINISH), most anticholinergic, avoid in pregnancy.
4. TCAs are cardiotoxic and lethal in modest overdose: fast-sodium-channel block → QRS widening, arrhythmia, seizures (the three C's: convulsions, coma, cardiotoxicity); treat QRS widening with sodium bicarbonate.
5. Two number-traps: citalopram/escitalopram prolong QTc so are dose-capped (citalopram 40 mg/day, 20 mg if >65 years); irreversible MAOIs cause the tyramine/cheese hypertensive crisis and need a 2-week washout, while benzodiazepines raise GABA-A channel-opening frequency (barbiturates raise duration).

1 Antidepressants: the overview

The unifying logic is the **monoamine hypothesis**: depression reflects a functional deficiency of monoamine neurotransmitters (serotonin / 5-HT, noradrenaline / NA, and dopamine) at the synapse. Every class raises monoamine availability, yet the antidepressant effect takes **2 to 4 weeks**. That delay is the hypothesis's biggest problem and its most important exam point.

The delay to therapeutic effect

◆ **The 2 to 4 week lag** – synaptic monoamine levels rise within *hours*, but mood lifts only after **2–4 weeks**. The gap is explained by slower downstream adaptations: **autoreceptor (5-HT_{1A}) desensitisation**, receptor down-regulation, and neuroplastic changes (raised BDNF, hippocampal neurogenesis). The simple "low amine" model alone cannot account for the lag.

III

● CLASSIC TRAP

In the first 1–2 weeks, side effects and **early activation/anxiety** appear before mood improves, and energy may return before the mood does. This window carries a transiently **raised suicide risk**, especially in the young. Counsel, monitor, do not abandon the drug for "no response" too early.

The classes at a glance

Class	Examples	Mechanism	Signature adverse effects
SSRI	fluoxetine, sertraline, paroxetine, citalopram, escitalopram, fluvoxamine	selective **serotonin reuptake** inhibition (block SERT)	GI upset, sexual dysfunction, hyponatraemia, bleeding, discontinuation syndrome
SNRI	venlafaxine, desvenlafaxine, duloxetine, milnacipran	serotonin **and** noradrenaline reuptake inhibition	as SSRI plus dose-dependent hypertension (venlafaxine), sweating
TCA	amitriptyline, imipramine, clomipramine (tertiary); nortriptyline, desipramine (secondary)	block 5-HT and NA reuptake; also block muscarinic, H_1, α_1 receptors	anticholinergic, sedation, postural drop, weight gain, cardiotoxic, lethal in overdose
MAOI	phenelzine, tranylcypromine, isocarboxazid (irreversible); moclobemide (RIMA)	inhibit monoamine oxidase → less amine breakdown	tyramine / cheese reaction (hypertensive crisis), serotonin syndrome on combination
Atypicals	mirtazapine, bupropion, trazodone, vortioxetine, agomelatine	varied: see note below	agent-specific (sedation, weight gain, seizure threshold, priapism)

● **The atypicals, decoded** – **Mirtazapine**: α_2 antagonist (NaSSA) → sedation, weight gain, useful for appetite/sleep. **Bupropion**: NA/dopamine reuptake inhibitor → activating, no sexual dysfunction, but lowers seizure threshold. **Trazodone**: 5-HT_2 antagonist + weak SRI → sedating hypnotic, risk of priapism. **Vortioxetine**: multimodal (SERT + 5-HT receptor modulation). **Agomelatine**: melatonergic ($MT_{1/2}$) agonist + 5-HT_{2C} antagonist, monitor LFTs.

HOLD THIS

Monoamine hypothesis = low 5-HT/NA/DA. All classes raise amines fast, but the **antidepressant effect needs 2–4 weeks** (receptor adaptation, neuroplasticity). SSRIs are first-line for safety; TCAs and MAOIs work but are dangerous.

2 SSRIs: the agents and what separates them

SSRIs are first-line because they are **safe in overdose** and broadly tolerable. They share a class profile; you also need the one feature that distinguishes each agent.

Shared (class) adverse effects

▲ **GI upset** – nausea, diarrhoea; commonest early effect, usually settles in 1–2 weeks.

▲ **Sexual dysfunction** – delayed orgasm, low libido, anorgasmia. Very common, often persistent, a leading cause of non-adherence.

▲ **Hyponatraemia / SIADH** – especially in the **elderly** and with diuretics; check sodium if confusion, lethargy or falls appear.

▲ **Increased bleeding risk** – serotonin is needed for platelet aggregation; raised GI bleeding risk, additive with NSAIDs/anticoagulants. Co-prescribe gastroprotection where indicated.

▲ **Discontinuation syndrome** – abrupt stop → **FINISH**: Flu-like, Insomnia, Nausea, Imbalance, Sensory disturbance ("brain zaps"), Hyperarousal. Worst with short half-life agents; taper to avoid.

▲ **Early activation / anxiety** – jitteriness and worsened anxiety in the first days; start low in anxious patients.

◆ **Suicidality warning** – a black-box-type warning of increased suicidal ideation/behaviour in **under-25s** in the first weeks. Monitor closely early; the warning is about the early window, not the drug being net harmful.

The distinguishing features

Agent	Half-life	Stands out for
Fluoxetine	long (1–4 days; active metabolite norfluoxetine 7–15 days)	**self-tapering** – the long half-life means almost no discontinuation syndrome; activating; potent **2D6 inhibitor**
Sertraline	~26 h	safe broad first-line; mild dopaminergic; preferred in cardiac patients and (relatively) breastfeeding
Paroxetine	short (~21 h, no active metabolite)	worst discontinuation syndrome; most **anticholinergic**; most sedating & weight-gaining; avoid in pregnancy (cardiac malformation signal)
Citalopram / Escitalopram	~35 h	QTc prolongation – dose-capped (citalopram 40 mg, 20 mg in elderly). Escitalopram = the active S-enantiomer, cleaner; fewest interactions
Fluvoxamine	~15 h	strong **1A2 inhibitor**; used in OCD; more nausea/sedation

The two extremes to memorise

Fluoxetine = longest half-life → activating, self-tapering, minimal discontinuation. The forgiving one if doses are missed.

The other end

Paroxetine = short half-life, no active metabolite → worst discontinuation, most anticholinergic and sedating. The unforgiving one.

● TRAP

Citalopram and escitalopram prolong QTc in a dose-dependent way, hence the maximum-dose caps (citalopram 40 mg/day; 20 mg/day if >65 years, hepatic impairment, or on 2C19 inhibitors). Check an ECG in cardiac patients and avoid combining with other QT-prolonging drugs.

● MNEMONIC

"FINISH" → SSRI discontinuation

Flu-like, **I**nsomnia, **N**ausea, **I**mbalance, **S**ensory (brain zaps), **H**yperarousal. Worst with **paroxetine**, near-absent with **fluoxetine**.

3 SNRIs, TCAs and MAOIs

SNRIs

- **Venlafaxine** – serotonergic at low dose; the **noradrenergic effect is dose-dependent**, emerging at higher doses, where it can cause **dose-related hypertension**. Short half-life → marked discontinuation syndrome. Desvenlafaxine is its active metabolite.
- **Duloxetine** – balanced 5-HT/NA reuptake inhibition across the dose range; strong evidence in **neuropathic and chronic pain** (diabetic neuropathy, fibromyalgia) and stress urinary incontinence. Avoid in significant hepatic impairment.

Tricyclic antidepressants (TCAs)

- **Mechanism** – block 5-HT and NA reuptake (the therapeutic action) but also **muscarinic (anticholinergic)**, **histamine H_1** (sedation, weight gain) and **α_1** (postural hypotension) receptors. The off-target blockade is the whole side-effect story.

▲ **Cardiotoxic and lethal in overdose** – TCAs block fast cardiac sodium channels → **QRS widening**, arrhythmia, hypotension, seizures. A modest overdose can be fatal; this is the key reason they are second-line. Manage overdose with **sodium bicarbonate** for QRS widening.

◆ **Secondary vs tertiary amines** – **tertiary** (amitriptyline, imipramine, clomipramine) are more serotonergic, more sedating, more anticholinergic. **Secondary** (nortriptyline, desipramine) are more noradrenergic, less anticholinergic, better tolerated. Clomipramine is the most serotonergic and is used in OCD.

● MNEMONIC

"Three C's" of TCA overdose

Convulsions, **C**oma, **C**ardiotoxicity (widened QRS). Plus anticholinergic toxidrome: hot, dry, blind, mad. Treat QRS widening with sodium **bicarbonate**.

Monoamine oxidase inhibitors (MAOIs)

	Irreversible MAOIs	RIMA (moclobemide)
Examples	phenelzine, tranylcypromine, isocarboxazid	moclobemide
Selectivity	non-selective (MAO-A & B), irreversible	reversible, selective for **MAO-A**
Tyramine reaction	high risk – strict tyramine-free diet	low risk – tyramine displaces it; few dietary limits
Recovery	needs new enzyme synthesis (~2 weeks)	rapid, on stopping the drug

▲ **Tyramine ("cheese") reaction** – with irreversible MAOIs, dietary **tyramine** (aged cheese, cured/fermented meats, soy, draught beer, broad beans, Marmite) is not broken down → surges of NA → **hypertensive crisis** (severe headache, palpitations). Treat with an α-blocker (phentolamine). This is why MAOIs need a tyramine-restricted diet.

▲ **Serotonin-syndrome risk & washout** – combining an MAOI with a serotonergic drug (SSRI, SNRI, TCA, tramadol, pethidine) can precipitate **serotonin syndrome**. Observe washout periods: **2 weeks** off most SSRIs before/after an MAOI, but **5 weeks** after fluoxetine (its long half-life), and 2 weeks after stopping an MAOI before a serotonergic drug.

QUICK RULE

Irreversible MAOI → **diet matters** (tyramine), 2-week washouts (5 for fluoxetine). RIMA (moclobemide) → reversible, MAO-A selective, much safer on diet. Never co-prescribe an MAOI with another serotonergic agent without a washout.

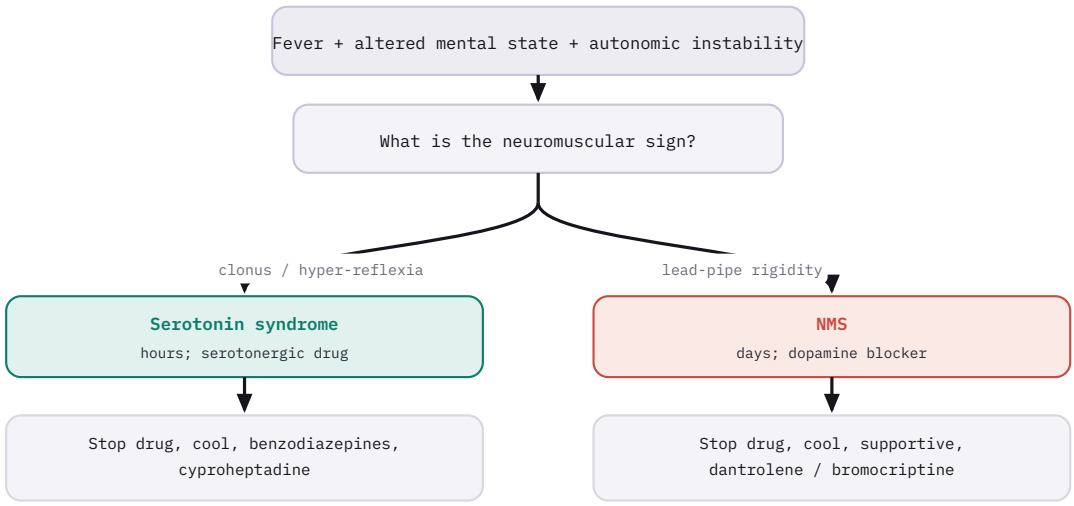
4 Serotonin syndrome vs neuroleptic malignant syndrome

The exam favourite. Both are hyperthermic, life-threatening, and look similar at a glance. The discriminators are the **cause**, the **speed of onset**, and above all the **neuromuscular sign**: clonus and hyper-reflexia (serotonin) versus lead-pipe rigidity (NMS).

Feature	Serotonin syndrome	Neuroleptic malignant syndrome
Cause	serotonergic excess (SSRI/SNRI + MAOI, tramadol, linezolid, triptans)	dopamine D_2 blockade (antipsychotics) or sudden dopamine-agonist withdrawal
Onset	fast – within **hours** (<24 h) of the trigger	slow – over **days to 1–2 weeks**
Neuromuscular	clonus, hyper-reflexia, myoclonus, tremor (hyperkinetic; worse in lower limbs)	lead-pipe rigidity, bradyreflexia (hypokinetic)
Mental state	agitation, anxiety, hypomania	stupor, mutism, fluctuating consciousness

Feature	Serotonin syndrome	Neuroleptic malignant syndrome
Autonomic	hyperthermia, tachycardia, diaphoresis, dilated pupils, diarrhoea, hyperactive bowel sounds	hyperthermia, tachycardia, labile BP, diaphoresis, pallor
Labs	mild CK rise; raised WCC	markedly raised CK, leucocytosis, myoglobinuria (risk of renal failure)
Time to resolve	usually **<24–72 h** once stopped	**days to weeks** (slow)
Specific drug	cyproheptadine (5-HT_{2A} antagonist)	dantrolene, bromocriptine, amantadine

HYPERTHERMIA TOXIDROME: WHICH WAY TO DECIDE



Both present with fever, altered mental state and autonomic instability. The neuromuscular sign splits them: clonus and hyper-reflexia (fast, serotonergic) point to serotonin syndrome; lead-pipe rigidity (slow, dopamine blockade) points to NMS.

● CLASSIC TRAP

The single most discriminating sign is the **neuromuscular exam**. Serotonin syndrome is *hyperkinetic* (clonus, hyper-reflexia, tremor, often worse in the legs); NMS is *hypokinetic* (generalised lead-pipe rigidity). Onset speed is the second clue: **hours** for serotonin syndrome, **days** for NMS. Markedly raised CK favours NMS.

5 Anxiolytics & hypnotics

Benzodiazepines

● **Mechanism** – positive allosteric modulators at the **GABA-A receptor**: they bind a site distinct from GABA and increase the **frequency of chloride-channel opening**, enhancing GABA's inhibitory effect (barbiturates, by contrast, increase channel-opening *duration*). The result is sedation, anxiolysis, hypnosis, muscle relaxation and anticonvulsant action.

Agent	Potency	Half-life	Typical use
Diazepam	low	long (active metabolites)	alcohol withdrawal, status, muscle spasm
Clonazepam	high	long	panic, epilepsy, mania adjunct
Lorazepam	high	intermediate	acute agitation, status; **safe in liver disease** (LOT)
Alprazolam	high	short	panic (high dependence/rebound risk)
Temazepam / Oxazepam	low	short–intermediate	hypnotic; **safe in liver disease** (LOT)

Agent	Potency	Half-life	Typical use
Midazolam	high	ultra-short	procedural sedation, anaesthesia

MNEMONIC

"LOT" → safe in liver disease

Lorazepam, **O**xazepam, **T**emazepam undergo only glucuronidation (no oxidative phase-I metabolism), so they are preferred in hepatic impairment and the elderly.

▲ **Tolerance, dependence, withdrawal** – tolerance to the hypnotic/anxiolytic effect builds within weeks; **physical dependence** follows. Abrupt withdrawal causes anxiety, insomnia, tremor, perceptual disturbance and, dangerously, **seizures and delirium**. Withdrawal is worst with **short half-life, high-potency** agents (alprazolam). Always taper.

■ **Flumazenil** – a competitive GABA-A benzodiazepine-site **antagonist**; reverses benzodiazepine sedation/overdose. Use cautiously: in the dependent or in mixed overdose it can precipitate **seizures**.

The non-benzodiazepine options

● **Z-drugs** – zolpidem, zopiclone, zaleplon. Act at the same GABA-A benzodiazepine site (selective for the α_1 subunit) → mainly hypnotic, shorter half-life. Marketed as lower-dependence but dependence and complex sleep behaviours (sleep-walking, sleep-driving) still occur.

◆ **Buspirone** – a **5-HT$_{1A}$ partial agonist**. **Non-sedating, non-dependence-forming**, no GABA action, no abuse potential and no interaction with alcohol. The price is a **delayed onset** (1–2 weeks), so it is useless for acute/PRN anxiety. Best for generalised anxiety where dependence must be avoided.

● **Pregabalin / gabapentin** – bind the **$\alpha_2\delta$ subunit of voltage-gated calcium channels**, reducing excitatory transmitter release. Pregabalin is licensed for generalised anxiety disorder and is used in neuropathic pain. Both carry misuse potential; taper on stopping.

● **Beta-blockers** – propranolol blunts the **somatic/peripheral** symptoms of anxiety (tremor, palpitations, sweating) without touching the psychological core. Useful for performance anxiety; no effect on the cognitive worry itself.

● **Hydroxyzine** – a sedating **H$_1$ antihistamine** with anxiolytic effect; non-dependence-forming, used short-term for anxiety and as an adjunct. Anticholinergic load and sedation are the limits.

RULE

For anxiety, benzodiazepines act fast but breed dependence, so they are for **short-term** or crisis use only. The **long-term** anxiolytics of choice are SSRIs/SNRIs (first-line) and, where dependence must be avoided, **buspirone** or **pregabalin**. Buspirone and SSRIs work slowly; benzodiazepines bridge the gap.

6 Pharmacokinetics & drug interactions

Most psychotropics are lipophilic, highly protein-bound, and hepatically metabolised through the **cytochrome P450 (CYP450)** system. The exam tests which antidepressants are powerful CYP **inhibitors** (they raise co-prescribed drug levels → toxicity) and which agents are **inducers** (they lower levels → failure).

CYP450 inhibitors and inducers to know

Drug	Effect on CYP	Enzyme	Why it matters
Fluoxetine	inhibitor	**2D6** (also 2C19, 3A4)	raises levels of TCAs, antipsychotics, beta-blockers; long half-life prolongs the effect
Paroxetine	inhibitor	**2D6**	potent 2D6 block; raises TCA, risperidone, tamoxifen activation lost
Fluvoxamine	inhibitor	**1A2** (also 2C19, 3A4)	raises clozapine, theophylline, caffeine, olanzapine levels
Carbamazepine	inducer	**3A4** (broad, autoinducer)	lowers levels of OCPs, many psychotropics, itself over time
Sertraline / Escitalopram / Citalopram	weak / minimal	–	fewest interactions; preferred when polypharmacy is a concern

MNEMONIC

"Fluoxetine & Paroxetine punch 2D6; Fluvoxamine fixes on 1A2"

The three SSRI inhibitors to recall by enzyme: **fluoxetine** and **paroxetine** at **2D6**; **fluvoxamine** at **1A2** (think clozapine and caffeine levels rising). Carbamazepine is the classic **inducer**.

- **Half-life & why it matters** – long half-life (fluoxetine) means self-tapering and forgiveness of missed doses but a long washout before an MAOI (5 weeks). Short half-life (paroxetine, venlafaxine) means brisk discontinuation symptoms. Steady state is reached in roughly **4–5 half-lives**.
- **Protein binding** – most antidepressants are highly protein-bound; only the free fraction is active. Displacement interactions (for example with warfarin) and altered binding in hypoalbuminaemia (the elderly, hepatic disease) raise free drug and toxicity risk.

7 Special populations

- **The elderly: start low, go slow** – reduced hepatic/renal clearance, lower albumin, more receptors sensitivity and polypharmacy. Begin at roughly **half** the adult starting dose and titrate slowly. Watch for **hyponatraemia** (SSRIs), **falls** (sedatives, postural drop), anticholinergic load (avoid TCAs and paroxetine), and **QTc** (citalopram cap 20 mg).
- **Hepatic impairment** – reduced first-pass and metabolism → accumulation. Lower doses; prefer agents needing less oxidative metabolism. For benzodiazepines, use **LOT** (lorazepam, oxazepam, temazepam: glucuronidation only). Avoid duloxetine and agomelatine in significant hepatic disease.
- **Renal impairment** – renally cleared drugs and active metabolites accumulate. Dose-reduce gabapentin and pregabalin (renally excreted), venlafaxine/desvenlafaxine and many others; monitor for toxicity. Watch electrolytes given the hyponatraemia risk.
- **Pregnancy & lactation** – covered in full in the companion **Perinatal & Women's Mental Health** pack. In brief: avoid **paroxetine** (cardiac malformation signal) and **valproate** absolutely; sertraline is a common SSRI choice in pregnancy and breastfeeding. See that pack for the detailed risk tables.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Monoamine hypothesis:** low 5-HT / NA / DA. Amines rise in hours but mood lifts in **2–4 weeks** (receptor desensitisation, neuroplasticity). Early window = raised suicide risk in the young.
- **SSRI class effects:** GI, sexual dysfunction, hyponatraemia/SIADH (elderly), bleeding (with NSAIDs), discontinuation (FINISH), early activation, under-25 suicidality warning.

- **SSRI agents:** fluoxetine = longest half-life, self-tapering, 2D6 inhibitor · paroxetine = shortest, worst discontinuation, most anticholinergic · citalopram/escitalopram = QTc (dose cap) · fluvoxamine = 1A2, OCD.
- **SNRIs:** venlafaxine = dose-dependent noradrenergic effect → hypertension; duloxetine = pain.
- **TCAs:** block 5-HT/NA reuptake + muscarinic/H_1/α_1; cardiotoxic, **lethal in overdose** (3 C's, bicarbonate for wide QRS). Tertiary (amitriptyline) > secondary (nortriptyline) for anticholinergic load.
- **MAOIs:** irreversible → tyramine/cheese reaction (hypertensive crisis), 2-week washouts (5 for fluoxetine). RIMA moclobemide = reversible, MAO-A, diet-safe.
- ★ **Serotonin syndrome vs NMS:** serotonergic + hours + **clonus/hyper-reflexia** + cyproheptadine, vs dopamine-blocker + days + **lead-pipe rigidity** + high CK + dantrolene.
- **Benzodiazepines:** GABA-A, increase channel-opening **frequency**. Withdrawal worst with short/high-potency (alprazolam). LOT safe in liver disease. Flumazenil = antagonist (can cause seizures).
- **Non-benzo anxiolytics:** Z-drugs (α_1), buspirone (5-HT_{1A} partial agonist, non-dependent, delayed onset), pregabalin ($\alpha_2\delta$), propranolol (somatic only), hydroxyzine (H_1).
- **CYP450:** fluoxetine & paroxetine inhibit **2D6**; fluvoxamine inhibits **1A2** (clozapine, caffeine up); carbamazepine = inducer (3A4). Sertraline/escitalopram = fewest interactions. Steady state ~4–5 half-lives.
- **Special populations:** elderly = start low go slow (hyponatraemia, falls, QTc cap 20 mg citalopram); hepatic = LOT benzodiazepines; renal = dose-reduce pregabalin/gabapentin; pregnancy → see Perinatal pack (avoid paroxetine & valproate).

ONE-DAY REVISION · PAPER 3

ECT, Neurostimulation & Psychosurgery

The therapeutic agent is the generalised seizure, not the current. Hold the indication list (where ECT is first-line vs reserved), the placement trade-off, the "no absolute contraindication" rule, and the ladder of neurostimulation from non-invasive (rTMS, tDCS) to implanted (VNS, DBS) to ablative psychosurgery. Each comparison table is a 10-mark answer in waiting.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. Real anatomy/EEG/imaging is flagged where a clinical figure belongs.

01 How ECT works · **02** Indications: first-line vs reserved · **03** The procedure · **04** Adverse effects & contraindications · **05** rTMS · **06** tDCS, VNS & DBS · **07** Psychosurgery

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. There is NO absolute contraindication to ECT; the key relative cautions are raised intracranial pressure / space-occupying lesion (the head) and recent myocardial infarction (the heart).
2. The therapeutic agent is the generalised tonic-clonic seizure, not the current; subconvulsive stimulation does not work and the precise mechanism remains unknown (anticonvulsant theory, rising seizure threshold across the course, is the favoured unifying account).
3. Placement trade-off: bilateral is most effective and fastest but most amnestic, while right unilateral spares memory but must be dosed high (around 5-6× threshold, vs 1.5× for bilateral) to match efficacy.
4. Modified ECT uses a short-acting induction agent plus the depolarising relaxant succinylcholine (suxamethonium) and an anticholinergic (atropine/glycopyrrolate); the relaxant prevents fractures, not the seizure.
5. Depression is the commonest indication; ECT is first-line when urgent (high suicide risk, food/fluid refusal, psychotic depression, catatonia after lorazepam fails, puerperal psychosis), and the standard rTMS protocol is high-frequency over the left DLPFC with a smaller effect size than ECT.

1 How ECT works

One claim anchors the topic: the **generalised tonic-clonic seizure is the therapeutic agent**, not the electrical stimulus itself. A seizure of adequate duration and generalisation is necessary for response; subconvulsive stimulation does not work. The precise mechanism remains **unknown**, but four families of theory each explain part of the effect.

Theory	Core claim	Supporting observation
Neuroendocrine	the seizure resets hypothalamic-pituitary output; a diencephalic "antidepressant" substance is released	surge of prolactin, ACTH, cortisol, oxytocin and vasopressin after each treatment
Neurotrophic / BDNF	ECT raises **BDNF** and drives hippocampal neurogenesis and synaptogenesis, reversing stress-related atrophy	increased hippocampal volume on MRI; raised serum BDNF tracks response
Monoamine / neurotransmitter	enhanced serotonergic, noradrenergic and dopaminergic transmission; up-regulated post-synaptic 5-HT and dopamine sensitivity	dopaminergic action underlies the antiparkinsonian benefit of ECT
Anticonvulsant	ECT raises the seizure threshold across a course (seizures shorten); this rise *is* the antidepressant action	enhanced GABA transmission and reduced cerebral blood flow; threshold rises treatment by treatment

HOLD THIS

The **seizure** is the treatment. Mechanism is **unknown**; the four theories are neuroendocrine, neurotrophic (BDNF), monoaminergic, and anticonvulsant. The anticonvulsant theory is the favoured unifying account.

TRAP

The **anticonvulsant hypothesis** sounds paradoxical: inducing seizures to gain an anticonvulsant effect. The point is that the brain's *own* seizure threshold rises over the course, and this endogenous anticonvulsant response correlates with clinical improvement. Do not confuse it with giving anticonvulsant drugs.

2 Indications: first-line vs reserved

ECT is reserved for severe, urgent or treatment-resistant presentations, and is occasionally first-line when speed matters (high suicide risk, food/fluid refusal, catatonia, pregnancy where drugs are best avoided). **Depression is the commonest indication.**

Indication	Position	Note
Severe / treatment-resistant depression	first-line when urgent; else reserved after drug failure	the most evidence-backed indication; remission rates high
Depression with psychotic features	strongly favoured	responds better to ECT than to antidepressant alone
Depression with high suicide risk	first-line (speed of action)	acts faster than drugs when life is at immediate risk
Depression with food / fluid refusal	first-line (medical urgency)	stupor, dehydration, severe weight loss
Catatonia	highly effective; first-line if lorazepam fails	dramatic response; benzodiazepine trial usually precedes
Treatment-resistant mania	reserved	effective when pharmacotherapy fails or in severe excitement
Treatment-resistant schizophrenia	reserved (adjunct to antipsychotic)	best for **catatonic** and affective subtypes; clozapine-augmentation
Neuroleptic malignant syndrome	reserved (when supportive care + drugs fail)	useful where NMS and lethal catatonia overlap
Puerperal (postpartum) psychosis	favoured; rapid response, breastfeeding-compatible	speed protects mother and infant

Where ECT shines (rapid / severe)

Psychotic depression, acutely suicidal depression, food/fluid refusal, **catatonia**, NMS, puerperal psychosis, and pregnancy where drugs are best avoided. The common thread is **urgency or drug intolerance**.

Where it is reserved

Non-urgent depression responding to drugs, treatment-resistant mania, and schizophrenia (as an antipsychotic adjunct). ECT is not first-line maintenance, though **continuation / maintenance ECT** prevents relapse in selected relapsing cases.

RULE

The single best predictor of a good ECT response is **severe depression, especially with psychotic or melancholic features or marked psychomotor change**. Catatonia of any cause responds dramatically. ECT treats the syndrome (catatonia, psychotic depression), not the diagnostic label.

3 The procedure

Work-up and consent

- **Pre-ECT work-up** – history and examination, especially cardiac and CNS; bloods, ECG, and anaesthetic fitness review. Review medications: stop or reduce drugs that raise the seizure threshold (benzodiazepines, anticonvulsants) where possible. Nil by mouth before anaesthesia.
- **Consent** – informed, written, and specific to ECT, covering benefits, the memory risk and alternatives. Where the patient lacks capacity, treatment proceeds under the relevant mental-health legislation with the required second opinion / board safeguard.

Anaesthesia (modified ECT)

Modern ECT is always **modified**: brief general anaesthesia plus a muscle relaxant abolish the violent motor convulsion (preventing fractures) while the brain seizure proceeds.

Drug class	Agent (typical)	Purpose
Induction agent	short-acting (e.g. thiopentone, methohexitone, propofol, etomidate)	brief anaesthesia for the stimulus and seizure
Muscle relaxant	succinylcholine (suxamethonium), depolarising, short-acting	relaxes muscles so the convulsion does not fracture bones
Anticholinergic	atropine or glycopyrrolate (premedication)	dries secretions; blocks the vagal bradycardia / asystole risk

CLASSIC TRAP

A **cuff is inflated on one limb before the relaxant circulates**, isolating it so the motor seizure can be timed visually in that limb while the rest of the body stays still. Hyperventilation (preoxygenation) *lowers* the seizure threshold and helps elicit an adequate seizure.

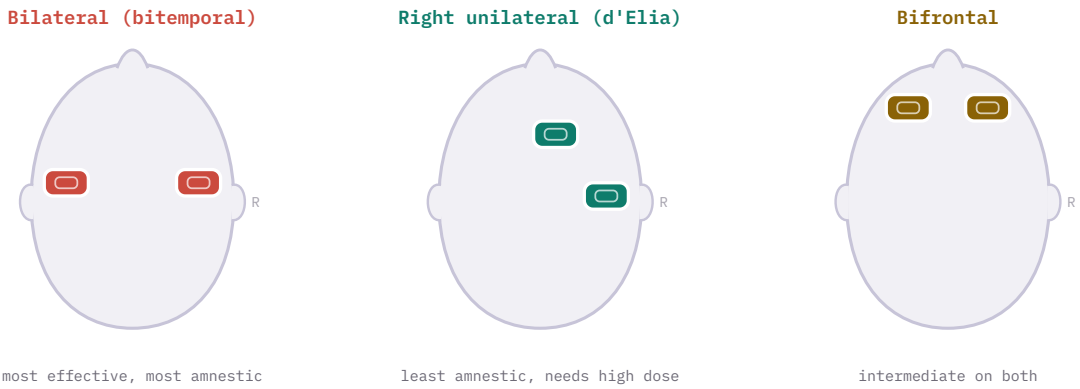
Electrode placement

The central trade-off of the whole topic: **bilateral is more effective but more amnestic; right unilateral is gentler on memory but needs a higher dose to match efficacy.**

Placement	Efficacy	Cognitive cost	Use
Bilateral (bitemporal)	highest, fastest	most memory impairment	when speed matters (high suicide risk, severe illness)
Right unilateral	equal *if* dosed well above threshold (high-dose)	least memory impairment	preferred when cognition must be protected
Bifrontal	intermediate	intermediate	an alternative balancing the two

ELECTRODE PLACEMENT – EFFICACY VS COGNITIVE COST

HEAD FROM ABOVE · NOSE = FRONT



Each head is viewed from above (nose forward, R = patient right). Bitemporal places both electrodes over the temples; right-unilateral (d'Elia) puts one at the right temple and one near the vertex; bifrontal sits high over both frontal poles. The trade-off runs left to right: efficacy against memory.

Dosing and the adequate seizure

◆ **Seizure threshold** – the minimum charge that triggers a generalised seizure. It varies widely between people (and rises with age, male sex, and across a course). Dose is set relative to this threshold: bilateral roughly **1.5×** threshold, right unilateral much higher, around **5–6×** (high-dose) to be effective.

● **Stimulus** – a **brief-pulse (or ultrabrief-pulse) constant-current** square wave is standard; it is far more efficient and less amnestic than the obsolete sine-wave stimulus.

■ **Adequate seizure** – bilateral generalised seizure of sufficient duration (a motor seizure of roughly **15–25 s**, EEG seizure longer) with the characteristic post-ictal EEG suppression. A missed or partial seizure should be re-stimulated. Quality matters more than raw duration.

◆ **Typical course** – usually **2–3 sessions per week** for a total of about **6–12** treatments, titrated to response. Twice-weekly is common in India; continuation/maintenance ECT then tapers the interval.

4 Adverse effects & contraindications

Domain	Effect	Course
Cognitive	acute post-ictal confusion / disorientation	minutes to an hour after each treatment
	anterograde amnesia (new learning impaired)	during the course; resolves within weeks of stopping
	retrograde amnesia (loss of memories before ECT)	greatest for events near the course; mostly recovers, some patchy permanent gaps possible
Somatic	headache, myalgia, jaw ache, nausea	transient, treat symptomatically
Cardiovascular	transient surge: initial vagal **bradycardia**, then sympathetic **tachycardia & hypertension**	seconds to minutes; the main intra-procedure risk
Anaesthetic	prolonged apnoea (pseudocholinesterase deficiency), aspiration, dental/tongue injury	rare, procedure-specific
Mortality	very low, comparable to brief general anaesthesia	roughly 1 per 10,000–100,000 treatments

QUICK RULE

The memory effect is real but mostly **transient**. Anterograde amnesia clears within weeks; retrograde amnesia is worst for the period around the course and largely recovers, though some autobiographical gaps can persist. Right unilateral and brief/ultrabrief pulse reduce it.

TRAP (MOST EXAMINED)

There is **NO absolute contraindication** to ECT. The key *relative* cautions are **raised intracranial pressure / space-occupying lesion** (highest concern) and **recent myocardial infarction**. Others: recent stroke, intracranial aneurysm, unstable arrhythmia, retinal detachment, phaeochromocytoma, and severe respiratory disease. Each raises the surge risk; none forbids ECT outright if the indication is compelling.

MNEMONIC

"No absolutes – mind the head and the heart"

The two cautions that lead every answer: raised **intracranial pressure** (the head) and recent **MI** (the heart). State first that there is no absolute contraindication, then list these.

5 rTMS

Repetitive transcranial magnetic stimulation uses a rapidly changing magnetic field over the scalp to induce a focal electric current that **depolarises cortical neurons** directly. No seizure, no anaesthesia, fully outpatient.

- **Mechanism** – a coil over the skull delivers brief magnetic pulses; high-frequency trains (≥5 Hz) are **excitatory**, low-frequency (≤1 Hz) is **inhibitory**. The dose is set relative to the individual **resting motor threshold**.
- **Standard depression protocol** – **high-frequency rTMS over the left dorsolateral prefrontal cortex (DLPFC)**, which is relatively hypoactive in depression. Right-DLPFC low-frequency is an alternative. The approved indication is **treatment-resistant major depression**; OCD protocols are also approved.

Advantages

No anaesthesia, **no induced seizure**, no muscle relaxant, outpatient, and minimal cognitive side effects (no amnesia). Main side effects are scalp discomfort and headache; the serious risk is a rare accidental seizure.

Limitations

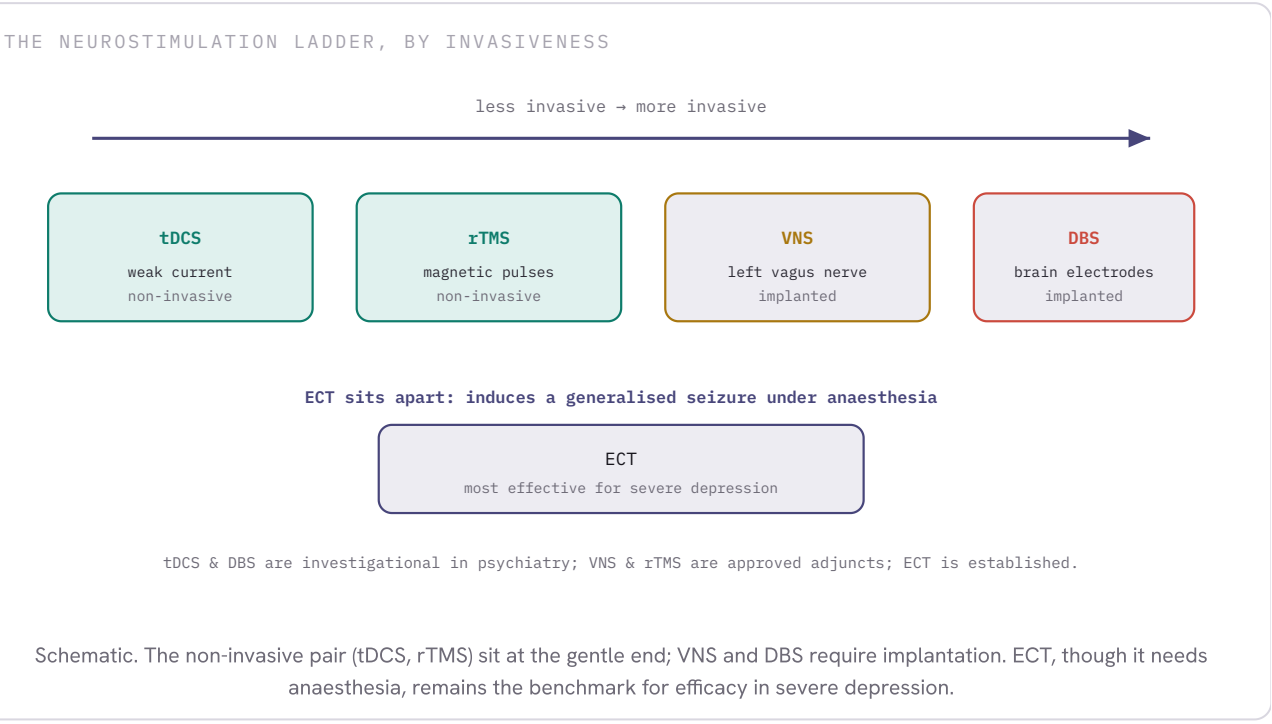
Smaller effect size than ECT (ECT remains the most effective treatment for severe depression). Needs **daily sessions, five days a week, over four to six weeks**. Less useful when speed is critical or the patient is acutely suicidal.

6 tDCS, VNS & DBS

A ladder by invasiveness: non-invasive scalp stimulation (tDCS), then implanted nerve stimulation (VNS), then implanted brain electrodes (DBS).

- **tDCS (transcranial direct current stimulation)** – a **weak constant current (~1–2 mA)** passed between scalp electrodes. The **anode** increases cortical excitability; the **cathode** decreases it. It modulates (does not directly trigger) neuronal firing. Cheap, portable, well tolerated, but still **investigational** for depression.
- **VNS (vagus nerve stimulation)** – an implanted pulse generator stimulates the **left vagus nerve** in the neck. Approved as an **adjunct for chronic / recurrent treatment-resistant depression** (and originally for refractory epilepsy). Benefit builds slowly over months.

● **DBS (deep brain stimulation)** – electrodes are **surgically implanted** into specific targets and driven by a pacemaker-like generator; reversible and adjustable. Investigational for **treatment-resistant depression** (targets: subgenual cingulate / Brodmann area 25, ventral capsule/ventral striatum) and **treatment-resistant OCD** (ventral capsule/ventral striatum). It is established for Parkinson's disease.



Schematic. The non-invasive pair (tDCS, rTMS) sit at the gentle end; VNS and DBS require implantation. ECT, though it needs anaesthesia, remains the benchmark for efficacy in severe depression.

ECT: INDICATIONS AND EVIDENCE

			CLINICAL USE	
Method	**Invasiveness**	**Mechanism**	**Indication**	**Evidence / approval**
ECT	anaesthesia, non-implanted	generalised seizure	severe / psychotic / resistant depression, catatonia, mania	established, most effective
rTMS	non-invasive	focal magnetic depolarisation (left DLPFC)	treatment-resistant depression, OCD	approved; smaller effect than ECT
tDCS	non-invasive	weak DC, anodal/cathodal modulation	depression (research)	investigational
VNS	implanted (neck)	left vagus nerve stimulation	chronic treatment-resistant depression	approved adjunct; slow onset
DBS	implanted (brain)	focal electrode stimulation of a target	resistant depression, resistant OCD	investigational in psychiatry

7 Psychosurgery

History and decline

▲ **Moniz (1935)** – introduced **prefrontal leucotomy** (frontal leucotomy); won the **Nobel Prize (1949)**. Freeman & Watts popularised the crude transorbital "ice-pick" lobotomy. The procedures were imprecise and caused frontal-lobe syndromes (apathy, disinhibition, blunting). They were **largely abandoned** after the arrival of chlorpromazine (1952) and on ethical grounds.

Modern stereotactic ablative procedures

Today's operations are **stereotactic** (image-guided, millimetre-precise), make small targeted lesions, and are reserved for the most **severe, chronic, refractory OCD and depression** that has failed all other treatment. Four named procedures:

Procedure	Target lesioned	Chiefly for
Anterior cingulotomy	anterior cingulate cortex	refractory OCD, depression, chronic pain
Anterior capsulotomy	anterior limb of the internal capsule	refractory OCD
Subcaudate tractotomy	substantia innominata (below caudate)	refractory depression, OCD
Limbic leucotomy	cingulotomy + subcaudate tractotomy combined	refractory OCD, depression

MNEMONIC

"Limbic leucotomy = Cingulate + Caudate-below"

Limbic leucotomy is the **combination** of anterior cingulotomy and subcaudate tractotomy. The four target sites: cingulate, internal-capsule anterior limb, substantia innominata, and the combined limbic lesion.

Ethical and legal safeguards

Strict safeguards – psychosurgery is a treatment of **last resort**: documented failure of all adequate alternatives, an **independent multidisciplinary review board**, fully **informed valid consent from a competent patient** (it cannot be done without genuine consent), and statutory oversight. It is **irreversible**, which is why DBS (reversible, adjustable) is increasingly preferred for the same targets.

RULE

Frame any psychosurgery answer around three pillars: **(1)** last resort after exhausting drugs, psychotherapy, ECT and (where available) neurostimulation; **(2)** precise small stereotactic lesions, not the old crude lobotomy; **(3)** rigorous consent and independent-board safeguards because it is irreversible.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Mechanism:** the **seizure** is the therapeutic agent; precise mechanism unknown. Theories: neuroendocrine, neurotrophic (BDNF up), monoamine, anticonvulsant (threshold rises across the course = the favoured account).
- **Indications:** commonest is depression. First-line when urgent – suicidal / psychotic / food-refusing depression, catatonia, NMS, puerperal psychosis. Reserved – resistant mania, resistant (catatonic/affective) schizophrenia.
- **Anaesthesia:** short-acting induction agent + **succinylcholine** (relaxant) + anticholinergic (atropine/glycopyrrolate). Modified ECT prevents fractures.
- **Placement: bilateral** = most effective, most amnestic; **right unilateral** = least amnestic but needs ~5–6× threshold; bifrontal intermediate. Brief/ultrabrief pulse beats sine wave.
- **Dosing:** bilateral ~1.5× threshold, RUL high-dose. Adequate seizure ~15–25 s motor with post-ictal suppression. Course ~6–12 treatments, 2–3/week.
- **Adverse:** confusion, anterograde + retrograde amnesia (mostly transient), headache, myalgia, transient cardiovascular surge. Mortality ~ that of brief anaesthesia.
- ★ **Contraindications: NO absolute contraindication**; key relative cautions are **raised ICP** (the head) and **recent MI** (the heart).
- **rTMS:** focal magnetic depolarisation; high-frequency over **left DLPFC** for resistant depression. No anaesthesia, no seizure, outpatient; smaller effect than ECT; daily, weeks.
- **Others:** tDCS = weak current, anodal/cathodal, investigational. VNS = left vagus, chronic resistant depression. DBS = implanted electrodes, resistant depression (BA25 / VC-VS) & resistant OCD, investigational.

- **Psychosurgery:** Moniz leucotomy (Nobel 1949), declined after chlorpromazine. Modern stereotactic: anterior cingulotomy, anterior capsulotomy, subcaudate tractotomy, limbic leucotomy (= cingulotomy + subcaudate tractotomy). Last resort, board review, valid consent, irreversible.

ONE-DAY REVISION · PAPER 3

Psychotherapies

The talking treatments, mapped so they stay mapped. For each school hold one thing: who founded it, the single mechanism it leans on, and the disorder it fits. The overview table and the named lists (cognitive distortions, Yalom's factors, IPT's four areas) are the marks. Know the contrasts, not just the contents.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. The ego defences live in the companion pack on defence mechanisms.

01 The map: schools & common factors · **02** Psychodynamic & psychoanalytic · **03** Behavioural therapies · **04** Cognitive & cognitive-behavioural · **05** Interpersonal, supportive & motivational · **06** Group, family & couples therapy · **07** Selection & combination

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. Therapeutic alliance is the strongest common factor and one of the most robust predictors of outcome; Bordin's three parts are the **bond**, agreement on **goals**, agreement on **tasks** (the Dodo bird verdict says bona fide therapies give roughly equal effects).
2. Classic behavioural trap: systematic desensitisation = graded hierarchy + relaxation + reciprocal inhibition (Wolpe), flooding = ungraded full-intensity exposure with no relaxation, and ERP = exposure plus blocking the compulsion (the first-line behavioural treatment for OCD).
3. Direction trap: transference is the patient's redirection of feelings onto the therapist, countertransference is the therapist's reaction back onto the patient, and the therapeutic alliance is a third, separate thing.
4. High expressed emotion (critical comments, hostility, emotional over-involvement) predicts relapse in schizophrenia, and lowering EE reduces relapse; this is the single most examined family-research finding.
5. First-line matching: OCD → ERP, borderline PD → DBT, PTSD → trauma-focused CBT or EMDR, depression → CBT or IPT, bulimia → CBT-E; in moderate-to-severe depression combined drug plus psychotherapy beats either alone.

1 The map: schools & common factors

Every therapy answers one question first: **where does the problem live?** In the unconscious past, in learned behaviour, in faulty thinking, in the thwarted self, or in the family system. The school is named by its answer.

Therapy	Founder(s)	Core mechanism	Typical indication
Psychoanalysis / psychodynamic	Freud	make the unconscious conscious; resolve conflict via insight, transference	chronic relational patterns, personality difficulties
Behaviour therapy	Wolpe, Skinner, Eysenck	relearning by conditioning; change the contingencies	phobias, OCD, habit disorders
Cognitive / CBT	Beck, Ellis	identify & restructure distorted thoughts and beliefs	depression, anxiety (first-line)
Humanistic-existential	Rogers, May, Frankl	growth through acceptance, meaning & authenticity	adjustment, self-concept, existential distress
Interpersonal (IPT)	Klerman & Weissman	link mood to current relationships & role events	depression, especially with a relational trigger

Therapy	Founder(s)	Core mechanism	Typical indication
Systemic / family	Minuchin, Haley, Bowen	change the system, not just the individual	child & adolescent problems, family conflict
Integrative / eclectic	–	draw techniques across schools to fit the case	complex, comorbid presentations

Common factors: what works across all of them

Outcome research (Lambert, Wampold) finds that specific techniques explain a minority of the variance; the shared, non-specific factors carry much of the change. The **Dodo bird verdict** (from Rosenzweig, after *Alice in Wonderland*): "everybody has won and all must have prizes" – bona fide therapies tend to show roughly comparable effects.

◆ **Therapeutic alliance** – the strongest common factor. Bordin's three parts: the **bond**, agreement on **goals**, agreement on **tasks**. Alliance quality is one of the most robust predictors of outcome.

● **Empathy & the relationship** – accurate understanding of the patient's experience; consistently linked to better outcomes regardless of modality.

● **Expectancy & hope** – the patient's belief that treatment will help; overlaps with the placebo response and the "remoralisation" of demoralised patients (Frank's *Persuasion and Healing*).

QUICK RULE

Across schools the active ingredients overlap: a strong **alliance**, **empathy**, and the patient's **expectancy**. Specific techniques add to, but do not replace, these common factors.

● MNEMONIC

"Bond, Goals, Tasks" → Bordin's alliance

The working alliance is the agreement on the **bond**, the **goals** and the **tasks** of therapy. Strain on any one weakens the lot.

2 Psychodynamic & psychoanalytic

Freud's claim: symptoms are the surface of unconscious conflict between drives and defences. Cure comes through **insight** – making the unconscious conscious – not advice.

The core techniques

● **Free association** – the patient says whatever comes to mind, uncensored. The "fundamental rule." Gaps and hesitations point to defended material.

● **Transference** – the patient unconsciously redirects feelings from early figures (often parents) onto the therapist. The **analysis of the transference** is the engine of classical work.

▲ **Countertransference** – the therapist's own emotional reaction to the patient. Once seen only as interference; now also used as **information** about what the patient evokes in others.

● **Resistance** – anything that blocks the work (lateness, silence, changing the subject). Not obstruction to be defeated but a signpost to defended conflict, to be interpreted.

● **Interpretation** – the therapist offers meaning, linking present feeling to unconscious conflict or past relationship. Timed for when the patient is near the insight.

● **Working through** – the repeated re-encounter of the same conflict across many contexts until insight becomes durable change. Insight once is not enough.

● TRAP

Transference is the patient's projection onto the therapist; **countertransference** is the therapist's reaction back. Do not swap them. The therapeutic **alliance** (the real, working relationship) is a third thing again, distinct from the transference.

Freud's structure, in one line

● **Topographic & structural** – the mind as conscious / preconscious / unconscious, and as **id** (drives, pleasure principle), **ego** (reality principle, mediator), **superego** (conscience). Symptoms = compromise formations. The **ego defences** are detailed in the companion pack.

Brief & modern dynamic therapy

Therapy	Author	Signature
Brief / focal dynamic	Malan, Sifneos, Davanloo	time-limited, one chosen focus; active therapist. Malan's **two triangles** (conflict; persons)
Mentalisation-based (MBT)	Bateman & Fonagy	build the capacity to read mental states in self & others; evidence base in **borderline PD**
Transference-focused (TFP)	Kernberg	uses the here-and-now transference to integrate split self/other representations; **borderline PD**
Interpersonal psychotherapy (IPT)	Klerman & Weissman	dynamic roots but manualised & present-focused (see section 5)

■ **Modern evidence base** – short-term psychodynamic psychotherapy has trial support in depression, anxiety and somatic disorders; MBT and TFP have randomised evidence in borderline personality disorder. The field has moved from open-ended analysis toward structured, manualised, time-limited forms.

3 Behavioural therapies

The behavioural premise: the symptom *is* the disorder, and it was learned, so it can be unlearned. No appeal to the unconscious. The two engines are classical and operant conditioning (detailed in the learning-theory pack).

Techniques built on classical conditioning

◆ **Systematic desensitisation (Wolpe)** – pairs a **graded fear hierarchy** with **relaxation**, by **reciprocal inhibition** (you cannot be relaxed and anxious at once). Classically imaginal and gradual. The template phobia treatment.

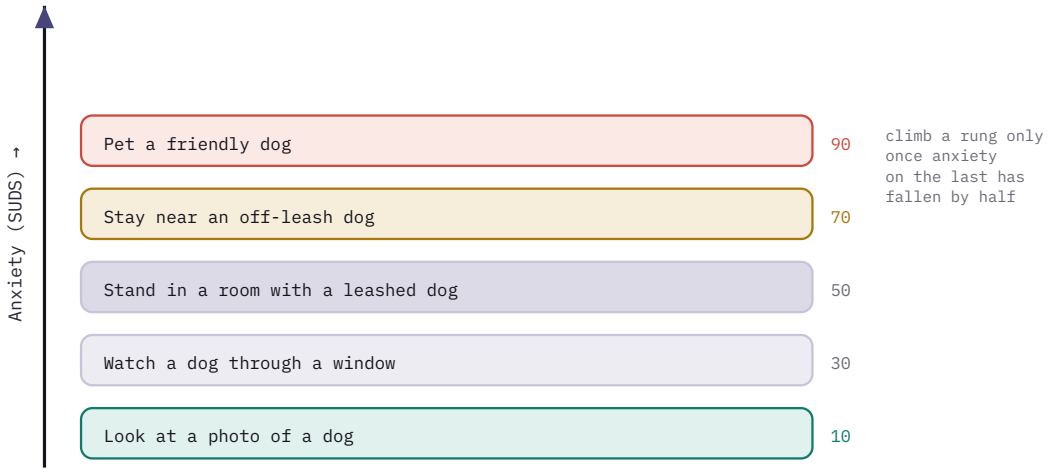
● **Graded (in vivo) exposure** – real-life confrontation of feared situations, low rung first, staying until anxiety falls (**habituation**). More effective than imaginal exposure for most phobias.

■ **Exposure with response prevention (ERP)** – the first-line behavioural treatment for **OCD**: expose to the trigger (touch the "contaminated" surface) and **prevent the compulsion** (no washing). Anxiety rises, then habituates, breaking the negative-reinforcement loop.

▲ **Flooding** – exposure to the *most* feared stimulus at full intensity from the start, no gradation, sustained until anxiety extinguishes. Faster but more distressing; needs consent and tolerance. **Implosion** = the imaginal version.

▲ **Aversion therapy** – pairs an unwanted behaviour with an unpleasant stimulus (e.g. disulfiram and alcohol, or covert sensitisation). Historically misused; now narrow indications and ethically constrained.

AN EXPOSURE HIERARCHY (FEAR LADDER)



The patient ranks feared steps by subjective units of distress (SUDS, 0 to 100), then works up the ladder. Each rung is repeated until anxiety habituates before moving higher.

Techniques built on operant conditioning

- **Token economy** – target behaviours earn tokens exchangeable for rewards; based on operant reinforcement and **shaping**. Historically used on inpatient/chronic wards and in intellectual disability and child settings.
- **Contingency management** – tangible reinforcers (often vouchers) for verified target behaviour, e.g. drug-negative urine samples. Strong evidence in **substance use disorders**.
- **Behavioural activation** – for **depression**: schedule pleasant and mastery activities to break the withdrawal-low mood spiral. Effective as a stand-alone treatment, not only a CBT component.
- **Relaxation training** – progressive muscle relaxation (Jacobson), controlled breathing, applied relaxation. A building block of desensitisation and a treatment in its own right for generalised anxiety.

● CLASSIC TRAP

Systematic desensitisation is graded + relaxation + reciprocal inhibition; **flooding** is ungraded, full-intensity, no relaxation. **ERP** is exposure *plus* blocking the compulsion (OCD). Do not call plain graded exposure "desensitisation" if no relaxation pairing is used.

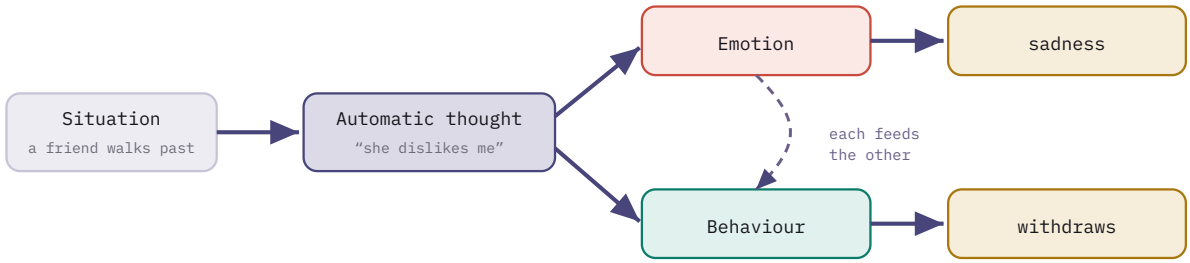
4 Cognitive & cognitive-behavioural

Beck's insight (working on depression): it is not events but the **meaning** we give them that drives feeling. Distorted, automatic thinking maintains the disorder; correcting it relieves it.

Beck's cognitive model

- **Negative cognitive triad** – the depressive's three negative views: of the **self** ("I am worthless"), the **world** ("nothing works out") and the **future** ("it will never change").
- **Three levels of cognition** – surface **automatic thoughts** (fast, situation-bound) rest on **intermediate beliefs** (rules & assumptions, "if I fail, I am nothing") which rest on deep **core beliefs / schemas** ("I am unlovable"). Therapy works the surface first, the core last.

THE COGNITIVE MODEL: SITUATION TO RESPONSE



The same situation yields different feelings depending on the thought it triggers. The thought, not the event, drives emotion and behaviour, and the two loop back to reinforce the thought.

Common cognitive distortions

Distortion	The faulty move
All-or-nothing (dichotomous)	seeing in black and white; anything short of perfect is failure
Overgeneralisation	one bad event becomes a never-ending pattern (“always,” “never”)
Mental filter	dwelling on a single negative, ignoring the positives
Disqualifying the positive	good events “don't count”
Jumping to conclusions	**mind-reading** (assuming others' thoughts) and **fortune-telling** (predicting the worst)
Magnification / minimisation	catastrophising bad, shrinking good (the “binocular trick”)
Emotional reasoning	“I feel it, so it must be true”
Should statements	rigid rules; guilt when self-imposed, anger when aimed at others
Labelling	extreme overgeneralisation: “I am a loser” not “I made a mistake”
Personalisation	taking blame for events one did not cause

● TRAP

Beck's **cognitive therapy** and Ellis's **Rational Emotive Behaviour Therapy (REBT)** are cousins, not the same. Ellis's **ABC** model: **A**ctivating event → **B**elief (often irrational) → **C**onsequence. REBT disputes irrational beliefs more confrontationally; Beck uses collaborative empiricism and Socratic questioning.

The structure of a CBT course

- **Collaborative & structured** – time-limited (often ~12 to 20 sessions), agenda-set each session, goal-directed, **present-focused**, with **homework** between sessions. The therapist and patient work as a team (collaborative empiricism).
- **Core methods** – **Socratic questioning** (guided discovery), **thought records**, **behavioural experiments** to test predictions, activity scheduling, and graded exposure. Treats thoughts as hypotheses, not facts.

The third wave, in brief

Therapy	Author	Distinctive move
DBT (dialectical behaviour)	Linehan	acceptance + change; four skill modules (mindfulness, distress tolerance, emotion regulation, interpersonal effectiveness). First-line for **borderline PD** & self-harm
ACT (acceptance & commitment)	Hayes	psychological flexibility: accept inner experience, defuse from thoughts, commit to **values**-based action. Not thought-content change
MBCT (mindfulness-based cognitive)	Segal, Williams, Teasdale	mindfulness + CBT to prevent **depressive relapse** (recurrent depression)
Schema therapy	Young	CBT + attachment + gestalt for entrenched **early maladaptive schemas** & modes; chronic / personality problems
CBASP	McCullough	cognitive-behavioural analysis system; designed specifically for **chronic depression**

MNEMONIC

"Mindful Distress, Regulated Relationships" → DBT's four modules

Mindfulness, **D**istress tolerance, **E**motion regulation, **I**nterpersonal effectiveness. The acceptance-versus-change dialectic runs through all four.

5 Interpersonal, supportive & motivational

Interpersonal psychotherapy (IPT)

Manualised, time-limited (~12 to 16 sessions), present-focused. It does not claim relationships caused the depression, only that depression plays out in current relationships, so the relationship is where to intervene. Strong evidence in **depression** and as a maintenance treatment.

Problem area	The focus of work
Grief (complicated bereavement)	facilitate mourning; rebuild interests & relationships
Role transition	adjust to a changed life role (parenthood, job loss, retirement, illness)
Role dispute (interpersonal)	resolve conflict with a significant other; renegotiate expectations
Interpersonal deficits	build social skills & a network when relationships are sparse or thin

MNEMONIC

"Grief, Transitions, Disputes, Deficits" → IPT's four areas

Pick the **one** problem area that best fits the patient and work it for the whole course. Most cases sit in grief, a role transition, or a role dispute.

Supportive psychotherapy

- Supportive psychotherapy – aims to **maintain or restore** function, not to restructure personality.

Bolsters existing defences, raises self-esteem, gives reassurance, advice and praise, and reduces anxiety. The widest applicability: useful for the medically ill, the acutely distressed, and patients too fragile for uncovering work.

Motivational interviewing (Miller & Rollnick)

A collaborative, person-centred style for resolving **ambivalence** about change. Born in the addictions field; now used across health behaviours.

- **The spirit** – **partnership, acceptance, compassion, evocation** (PACE). You evoke the patient's own reasons for change rather than installing them. The opposite of the "righting reflex" (the urge to fix and lecture).
- **OARS (the core skills)** – **O**pen questions, **A**ffirmations, **R**eflective listening, **S**ummaries.
- **Change talk vs sustain talk** – selectively elicit and reinforce the patient's own arguments *for* change (DARN-CAT: Desire, Ability, Reasons, Need; then Commitment, Activation, Taking steps). Roll with resistance rather than confronting it; arguing pushes the patient to defend the status quo.

MI spirit & method

Evoke, don't impose. Reflect more than you ask. Resist the righting reflex. Develop discrepancy between the patient's values and current behaviour. Support self-efficacy.

Stages of change (Prochaska & DiClemente)

Pre-contemplation → contemplation → preparation → action → maintenance (with relapse as part of the cycle). MI meets the patient at their stage; match the intervention to readiness.

Client-centred therapy (Rogers)

- **Rogers's three core conditions** – **unconditional positive regard** (non-judgemental acceptance), **empathy** (accurate understanding of the patient's frame), and **congruence / genuineness** (the therapist is real, not playing a role). Rogers held these to be **necessary and sufficient** for change.

TRAP

Motivational interviewing grows out of Rogers's client-centred soil (the same warmth and reflection) but adds a **direction**: it deliberately steers toward change talk. Pure client-centred therapy is non-directive; MI is directive about the goal, person-centred about the means.

6 Group, family & couples therapy

Yalom's therapeutic factors of group therapy

The change agents specific to the group format. The group becomes a **social microcosm**: members re-enact their outside relationship patterns in the room, where they can be seen and reworked.

Factor	What heals
Universality	"I am not the only one"; isolation breaks
Instillation of hope	seeing others improve makes recovery believable
Altruism	helping another member restores a sense of worth
Group cohesiveness	belonging & acceptance; the group's "alliance," a precondition for the rest
Catharsis	expressing strong feeling, held in a safe container
Interpersonal learning	feedback in the social microcosm reshapes relating
Imparting information	psychoeducation & advice
Imitative behaviour	modelling on the therapist or other members
Development of socialising technique	learning basic social skills
Corrective recapitulation of the primary family	the group re-enacts the family of origin, this time worked through
Existential factors	facing responsibility, mortality, isolation, meaning

HOLD THIS

The five most-cited: **universality, instillation of hope, altruism, group cohesiveness, catharsis, interpersonal learning**. Cohesiveness is the group analogue of the therapeutic alliance and underwrites the others.

Family & systems schools

School	Founder	Core idea & signature
Structural	Minuchin	treats family **structure**: boundaries, subsystems, hierarchies. Aims to fix **enmeshment** (over-close) and **disengagement** (too distant). Uses enactment & boundary-making
Strategic	Haley (& Madanes)	targets the presenting problem with **directives** & tasks, including **paradoxical injunction** (prescribing the symptom). Problem-focused, brief
Systemic (Milan)	Selvini Palazzoli & team	**circular questioning, hypothesising, neutrality**; positive connotation of the symptom's role in the system
Transgenerational	Bowen	**differentiation of self**, triangles, the multigenerational transmission of patterns; the **genogram**

● **Genogram** – a structured family tree (Bowen / McGoldrick) mapping relationships, illnesses and patterns across at least three generations. A standard assessment tool, not a school in itself.

◆ **Expressed emotion (EE)** – the family's emotional climate measured as **critical comments, hostility** and **emotional over-involvement**. **High EE** predicts relapse in **schizophrenia** (and in mood disorders, eating disorders). Family interventions that lower EE reduce relapse: a key exam fact.

● TRAP

Match founder to school: **Minuchin = structural** (boundaries, enmeshment), **Haley = strategic** (directives, paradox), **Milan team = systemic** (circular questioning, neutrality), **Bowen = transgenerational** (differentiation, genogram). High **expressed emotion** is the single most examined family-research finding.

7 Selection & combination

Matching therapy to disorder (first-line / strong evidence)

Disorder	Best-evidenced psychotherapy
Depression	CBT, IPT; behavioural activation; MBCT to prevent relapse
Panic / phobias / GAD	CBT with exposure (graded exposure, applied relaxation)
OCD	ERP (exposure with response prevention), within CBT
PTSD	Trauma-focused CBT, EMDR
Borderline PD	DBT; also MBT, TFP, schema therapy
Bulimia / binge eating	CBT-E (enhanced); IPT as an alternative
Substance use	MI, contingency management, CBT relapse prevention
Schizophrenia	family intervention (lower EE), CBT for psychosis as an adjunct to medication

Indications & contraindications

Points toward insight-oriented (dynamic) work

Psychological mindedness, capacity to tolerate frustration & affect, stable life circumstances, a curiosity about the self, and good ego strength. Wants understanding, not just symptom relief.

Points toward supportive / structured work

Acute crisis, fragile ego, active psychosis, severe cognitive impairment, low frustration tolerance, or poor motivation for uncovering work. Goal is to stabilise and maintain function.

▲ **General cautions** – no therapy substitutes for medication in acute mania, melancholic or psychotic depression, or acute psychosis; address active suicidality and substance intoxication first. Exposure and uncovering work can destabilise an unprepared or acutely unwell patient.

Combining drugs & psychotherapy

■ **Combination is often superior** – for moderate-to-severe depression, combined antidepressant + psychotherapy typically beats either alone; the same pattern holds across several disorders. The two work on different levers: medication on symptoms & biology, psychotherapy on skills, meaning & relapse prevention.

● **Sequencing & adherence** – psychotherapy improves treatment adherence and tackles the residual symptoms and relapse risk that drugs leave behind (e.g. MBCT after recovery from recurrent depression). Patient preference is itself a predictor of engagement and outcome.

● RULE

Three things select a therapy: the **disorder** (evidence base), the **patient** (psychological mindedness, ego strength, stage of change, preference), and the **setting** (time, resources, therapist training). Severe, acute or psychotic states need medication and stabilisation first; uncovering work waits.

QUICK-RECALL · DRILL THESE ONE-LINERS

★ **Common factors:** alliance (Bordin = bond + goals + tasks), empathy, expectancy. Dodo bird verdict = roughly equivalent outcomes across bona fide therapies.

- **Psychodynamic:** free association, transference (patient→therapist) vs countertransference (therapist→patient), resistance, interpretation, working through. Brief dynamic = Malan/Sifneos/Davanloo. MBT (Bateman-Fonagy) & TFP (Kernberg) for borderline PD.
- **Behavioural:** systematic desensitisation = graded + relaxation + reciprocal inhibition (Wolpe). Flooding = ungraded, full intensity. ERP = exposure + block the compulsion (OCD). Token economy & contingency management = operant. Behavioural activation for depression.
- **Cognitive:** Beck's negative triad = self, world, future. Automatic thoughts → intermediate beliefs → core schemas. Distortions: all-or-nothing, overgeneralisation, catastrophising, mind-reading, personalisation. Ellis REBT = ABC. CBT = structured, present-focused, homework, Socratic.
- **Third wave:** DBT (Linehan, borderline PD, 4 modules) · ACT (Hayes, values) · MBCT (relapse prevention) · schema therapy (Young) · CBASP (chronic depression).
- **IPT:** four areas = grief, role transition, role dispute, interpersonal deficits (Klerman & Weissman). Supportive therapy = maintain function, bolster defences.
- **MI:** spirit = PACE; skills = OARS; roll with resistance, evoke change talk. Stages of change: precontemplation → contemplation → preparation → action → maintenance.
- **Rogers:** unconditional positive regard + empathy + congruence = the core conditions (necessary & sufficient).
- **Group (Yalom):** universality, instillation of hope, altruism, cohesiveness, catharsis, interpersonal learning. Group = social microcosm.
- **Family:** Minuchin = structural · Haley = strategic (paradox) · Milan = systemic (circular questioning) · Bowen = transgenerational (genogram). High expressed emotion predicts relapse in schizophrenia.
- **Selection:** OCD = ERP · borderline PD = DBT · PTSD = TF-CBT/EMDR · bulimia = CBT-E. Combined drugs + psychotherapy beats either alone in moderate-severe depression; stabilise acute/psychotic states first.

ONE-DAY REVISION · PAPER 3

Rehabilitation Psychiatry

Rehabilitation is what happens after the acute illness is controlled and the symptoms are no longer the whole story. The targets shift from cure to function, from the ward to the community, from the patient alone to the patient-in-a-family. Hold the ICF triad, the high expressed-emotion concept, IDEAS, ACT, and the two Indian Acts – those are the marks.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. Disability law and certification detail sit in section 6.

01 Principles & the disablement framework · **02** Assessment in rehabilitation · **03** Psychosocial interventions · **04** Settings & services · **05** Rehabilitation in specific conditions · **06** The Indian context & the legal framework · **07** Outcomes & measurement

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. High expressed emotion is criticism, hostility & emotional over-involvement (mnemonic CHE); a high-EE home roughly doubles relapse in schizophrenia over 9-12 months. Trap: EE predicts relapse, it does not cause the illness or blame the family.
2. ICF (2001) replaced ICIDH (1980): impairment (body) → activity limitation (was disability, task level) → participation restriction (was handicap, social/role level), plus contextual (environmental + personal) factors; it is interactional, not a linear deficit chain.
3. IDEAS (Indian Disability Evaluation and Assessment Scale) is the notified instrument for psychiatric disability certification in India: four domains (self-care, interpersonal, communication/understanding, work), each scored 0-4, adjusted for duration of illness → percentage disability; the 40% benchmark under RPwD 2016 unlocks reservations and benefits.
4. Keep the two Acts distinct: RPwD Act 2016 is the disability-rights/certification law (21 disabilities incl. mental illness, ≥40% benchmark, reservations); MHCA 2017 is the treatment/admission law (advance directive, nominated representative, capacity-based admission, decriminalised suicide). Trap: reservations/certificates = RPwD; admission/advance directives/ECT = MHCA.
5. In chronic schizophrenia the rehabilitation target is the negative symptoms & cognitive/functional decline, not the positive symptoms; supported employment / IPS (place-then-train) beats sheltered work (train-then-place) for competitive employment, and ACT (Stein & Test) is the mobile team with a shared caseload, 24-hour cover & in-vivo outreach for the revolving-door severely ill.

1 Principles & the disablement framework

Rehabilitation answers a different question from treatment. Treatment asks “how do I remove the symptoms?” Rehabilitation asks “given the residual deficit, how do I restore function and role?” Two big shifts define the field: from the **medical/cure model to the recovery model**, and from the **institution to the community**.

● **Psychiatric rehabilitation** – the process of helping a person with a disabling mental illness to develop the emotional, social and intellectual skills needed to live, learn and work in the community with the least amount of professional support. The goal is **function and role recovery**, not symptom abolition.

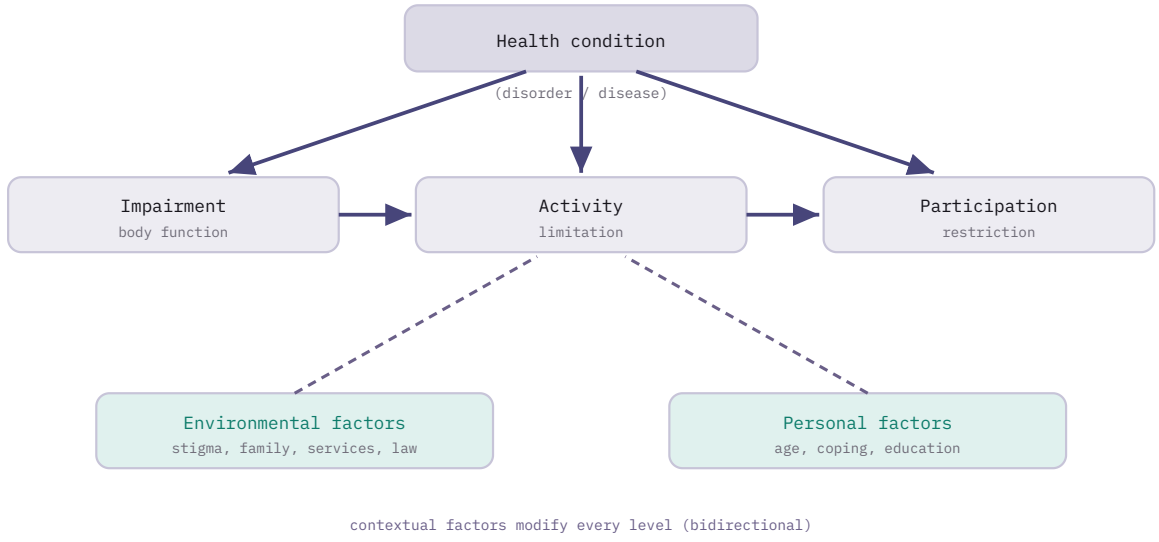
- **Recovery model** – recovery is defined by the person, not the clinician: living a meaningful, hopeful life *with or despite* ongoing symptoms. Pillars: **hope, agency/empowerment, identity beyond illness, meaning, and connection**. Contrast with the older "clinical recovery" (symptom remission) standard.
- **Institution → community shift** – deinstitutionalisation moved care from long-stay asylums to community settings, driven by chlorpromazine (1952), civil-rights critiques of asylums, and cost. The goal is the **least restrictive environment** and normalisation of daily life.

The WHO disablement model: ICF replaces ICIDH

This is the centrepiece. The 1980 **ICIDH** ran a linear, deficit-laden chain (impairment → disability → handicap). The 2001 **ICF** (International Classification of Functioning, Disability and Health) reframed it as an interaction between a health condition and contextual (environmental + personal) factors, with neutral, function-focused terms.

Old ICIDH term (1980)	New ICF term (2001)	Level affected	Psychiatric example
Impairment	Impairment (body function/structure)	the **organ / body** level	persistent auditory hallucinations and disordered thought (a deficit in mental function)
Disability	Activity limitation	the **person / task** level	cannot prepare a meal, manage money or maintain self-care
Handicap	Participation restriction	the **society / role** level	excluded from employment and from family/social roles by stigma and barriers

THE ICF MODEL OF FUNCTIONING AND DISABILITY (2001)



ICF is not a one-way chain. A health condition interacts with impairment, activity limitation and participation restriction, and all of these are shaped by environmental and personal (contextual) factors. The same impairment can produce very different participation depending on stigma, family support and services.

● CLASSIC TRAP

The old triad **impairment / disability / handicap (ICIDH 1980)** is examined alongside the new triad **impairment / activity limitation / participation restriction (ICF 2001)**. Map them in order: handicap became *participation restriction* (the social/role level), disability became *activity limitation* (the task level). The key conceptual change is that ICF is interactional and neutral, not linear and stigmatising, and it adds **contextual factors**.

2 Assessment in rehabilitation

Rehabilitation assessment is **function-led, not diagnosis-led**. You measure what the person can and cannot do, what they need, and what disability they carry – then you set graded goals.

Assessment	Question it answers	Tools / instruments
Functional assessment	what activities of daily living and skills are intact vs lost?	direct observation; **GAF**, social/occupational functioning scales, SOFAS
Needs assessment	what unmet needs (clinical and social) must services address?	**CAN** (Camberwell Assessment of Need)
Disability evaluation	how severe is the disability for benefits and certification?	WHO **WHODAS 2.0**; in India **IDEAS**

- **Functional assessment** – a structured look at activities of daily living (self-care, money, cooking, travel), social and occupational skills, and the supports available. It defines the **baseline and the goals**; reassessed to track change.
- **Needs assessment** – identifies **met and unmet needs** across domains (accommodation, food, daytime activity, company, money, physical health). The **CAN** is the standard structured instrument.

IDEAS – the Indian psychiatric disability instrument

- **IDEAS** – the **Indian Disability Evaluation and Assessment Scale**, developed by the Indian Psychiatric Society and notified by the Government of India for certifying **psychiatric disability**. It rates **four domains**: (1) self-care, (2) interpersonal activities/social relationships, (3) communication and understanding, (4) work. Each is scored **0–4**; the sum, adjusted for duration of illness, gives a global disability score converted to a **percentage** of disability.

RULE

IDEAS is the notified instrument for psychiatric disability certification in India, applied to the certifiable conditions (schizophrenia, bipolar affective disorder, severe recurrent depression, dementia, and obsessive-compulsive disorder under the disability framework). The **40% threshold** is the benchmark for "benchmark disability" under the RPwD Act 2016, which unlocks reservations and benefits. Certification is by a notified medical authority/board.

3 Psychosocial interventions

These are the working tools of rehabilitation. Learn what each one **targets** and the one fact that distinguishes it.

Intervention	Targets	Core method
Social skills training	deficits in conversation, assertiveness, daily-living skills	behavioural: modelling → role-play → feedback → homework
Psychoeducation	poor illness understanding, non-adherence, relapse	structured teaching of patient **and family** about illness, drugs, early warning signs
Family intervention	high expressed emotion, carer burden, relapse	education + communication + problem-solving to **lower EE**
Cognitive remediation	cognitive deficits (attention, memory, executive function)	drill and strategy training to improve cognition and function
Vocational rehabilitation	unemployment, loss of the work role	from sheltered work to **supported employment / IPS**
ACT	revolving-door admissions in the severely ill	a mobile multidisciplinary team delivering care **in vivo**

Family intervention and expressed emotion

This is the highest-yield single concept in the whole topic. **Expressed emotion (EE)** is the emotional climate of the household, measured from a relative's interview (the Camberwell Family Interview). A **high-EE** home roughly *doubles* the relapse rate in schizophrenia over 9–12 months compared with a low-EE home.

▲ **The three components of high EE** – **criticism** (critical comments), **hostility** (generalised rejection of the person), and **emotional over-involvement** (over-protective, intrusive, self-sacrificing). Hostility and over-involvement carry the most relapse risk.

● MNEMONIC

"CHE" → Criticism, Hostility, Emotional over-involvement

The three faces of high expressed emotion. Family intervention works by lowering EE and the relapse rate that rides on it.

● CLASSIC TRAP

High EE is a **predictor of relapse, not a cause of the illness** and not a sign that the family is to blame. It is transdiagnostic (also seen in mood disorders, eating disorders). Family interventions that reduce EE (psychoeducation + communication + problem-solving over several months) have a robust evidence base for cutting relapse in schizophrenia – one of the strongest in psychiatry.

Vocational rehabilitation and supported employment

Train-then-place (older)

Prevocational preparation first: **sheltered workshops**, vocational training, transitional employment, then graduation to open work. Critique: the "train" phase often never ends; few actually reach competitive jobs.

Place-then-train (modern, IPS)

Supported employment / Individual Placement and Support (IPS): rapid placement into a real, competitive job that matches the person's preference, with **on-the-job support**. Strongest evidence for competitive-employment outcomes in severe mental illness.

Assertive community treatment and case management

■ **Assertive community treatment (ACT)** – the **PACT/Madison model** (Stein & Test). A multidisciplinary team with a **shared (not individual) caseload**, low staff-to-patient ratio, **24-hour availability**, assertive outreach, and care delivered *in the person's environment* rather than the clinic. Targets the "revolving-door" severely ill; reduces admissions and homelessness.

● **Case management** – a single named worker coordinates and links a person's services. **Brokerage** (arranges services) vs **clinical/intensive** case management (also delivers care, smaller caseload). ACT is an intensive, team-based form of case management.

● RULE

ACT vs standard case management: the discriminators are the **shared team caseload**, the **low staff:patient ratio**, **24-hour cover**, and **in-vivo** assertive outreach. If a stem describes a mobile team that goes to the patient and holds a shared caseload, the answer is ACT.

4 Settings & services

The continuum runs from most to least supported. Match each setting to the level of support and the function it serves.

Setting	What it offers	Function
Day hospital	structured day treatment (therapy, medication, monitoring); patient sleeps at home	alternative to / step-down from admission
Day-care centre	occupation, social contact, structure; less clinical than a day hospital	ongoing support, combats isolation
Halfway home	residential, time-limited, supervised; rebuilds independent-living skills	bridge from hospital to independent living
Sheltered workshop	supervised, simplified, often subsidised work	vocational training / occupation for the more disabled
Supported housing	independent or shared housing with visiting support	least restrictive long-term accommodation
Self-help & user/carer groups	peer support, advocacy, lived-experience expertise	empowerment, destigmatisation, mutual aid

● TRAP

The lessons of deinstitutionalisation. Closing asylums without building adequate community services produced **transinstitutionalisation** (patients shifted to prisons, nursing homes and the street), homelessness, and the "revolving door." The lesson is that deinstitutionalisation only works when matched by funded community alternatives (ACT, supported housing, day services). It is not the same as simply discharging patients.

5 Rehabilitation in specific conditions

Condition	Main rehab target	Key interventions
Chronic schizophrenia	negative symptoms & functional decline (not the positive symptoms)	social skills training, family intervention (lower EE), supported employment, cognitive remediation
Intellectual disability	maximise adaptive skills and independence within the deficit	special education, life-skills training, vocational/sheltered work, family support
Substance use disorders	relapse prevention & reintegration after detoxification	relapse prevention, MI, contingency management, vocational rehab, peer/12-step support, sober housing
Dementia	maintain remaining function, dignity and safety	cognitive stimulation, environmental modification, carer support, ADL adaptation

◆ **Why schizophrenia is the prototype** – in chronic schizophrenia the long-term disability is driven mainly by **negative symptoms (avolition, blunting, social withdrawal)** and **cognitive impairment**, which respond poorly to antipsychotics. These functional deficits, not the hallucinations, are the principal rehabilitation target.

● RULE

For each condition, name the realistic goal: schizophrenia = role and function despite residual deficit; intellectual disability = habilitation (building skills never had) rather than rehabilitation; SUD = relapse prevention plus social reintegration; dementia = maintenance and carer support, since decline is progressive. Match the goal to the trajectory.

6 The Indian context & the legal framework

India's rehabilitation reality is shaped by a **service gap** (few rehabilitation facilities, large treatment gap), a **family-centred care model**, and two recent rights-based statutes.

- **District Mental Health Programme (DMHP)** – the delivery arm of the National Mental Health Programme (NMHP, 1982), launched **1996** on the Bellary model. It integrates mental-health care into general health services at the district level: training of primary-care staff, drug supply, community outreach, and increasingly rehabilitation and community resources.
- **The family as caregiver** – in India the family, not the state, is the primary provider of long-term care and supervision. This is a major resource (low institutionalisation, high involvement) but also a source of **carer burden**, and the reason family intervention and psychoeducation are central rather than optional.

The two Acts (highest-yield)

RPwD Act 2016

Rights of Persons with Disabilities Act 2016 (replaced PWD Act 1995; gives effect to the UNCRPD). Recognises **21 disabilities** including **mental illness** and intellectual disability. Defines **"benchmark disability" as ≥40%**, which unlocks reservation in education and jobs, and other benefits. Mandates non-discrimination, accessibility, and a certification system.

Mental Healthcare Act 2017

MHCA 2017 (replaced the Mental Health Act 1987; rights-based, UNCRPD-aligned). **Right to access mental healthcare**; **advance directive** and **nominated representative**; **capacity-based** admission; **decriminalises suicide** (presumed severe stress); bans unmodified ECT and ECT in minors (with safeguards). Establishes Mental Health Review Boards.

- **Certification & benefits** – psychiatric disability is certified using **IDEAS** by a notified authority; a score reaching the **40% benchmark** under the RPwD Act 2016 gives access to a **disability (UDID) certificate**, reservations, disability pension/allowance, travel concessions, and other welfare schemes. This links section 2 (IDEAS) to the legal framework here.

TRAP

Keep the two Acts distinct. **RPwD 2016** is the *disability rights / benefits / certification* law (40% benchmark, reservations, 21 conditions). **MHCA 2017** is the *treatment / admission / patient-rights* law (advance directive, nominated representative, capacity, decriminalised suicide). A stem about reservations and disability certificates is RPwD; a stem about admission, advance directives or ECT is MHCA.

7 Outcomes & measurement

Rehabilitation outcomes are **functional and personal**, not symptom counts. Symptom scales answer the treatment question; rehabilitation needs broader endpoints.

Outcome domain	What is measured	Example tools
Quality of life	subjective and objective wellbeing across life domains	**WHOQOL-BREF**, QLS
Social functioning	role performance, relationships, daily activities	SFS; **WHODAS 2.0**; GAF / SOFAS
Employment	competitive employment, hours, retention	employment status / days worked
Independent living	accommodation stability, self-care, autonomy	housing status, ADL measures
Recovery orientation	hope, empowerment, meaning (the person's own goals)	recovery / empowerment scales

- **Recovery orientation** – the modern frame: a good outcome is the person living a meaningful, self-defined life, judged by **their** goals (work, relationships, a home), not only by clinician-rated symptom remission. The hard outcomes that matter to patients are **employment, independent living, social connection and quality of life**.

HOLD THIS

Rehabilitation targets **function and role**, measured by quality of life, social functioning, employment and independent living – against a backdrop of **recovery** defined by the person, not symptom counts.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Principles:** rehab = restore function and role, not abolish symptoms. Recovery model = hope, agency, identity, meaning, connection. Shift = institution → community, least restrictive environment.
- **ICF (2001) replaced ICIDH (1980):** impairment (body) · activity limitation (was disability, task level) · participation restriction (was handicap, social/role level) · plus contextual (environmental + personal) factors. Interactional, not linear.
- **Assessment:** functional + needs (CAN) + disability (WHODAS / **IDEAS** in India). IDEAS = 4 domains (self-care, interpersonal, communication/understanding, work), each 0–4, adjusted for duration → % disability.
- ★ **Expressed emotion (high EE = CHE):** Criticism, Hostility, Emotional over-involvement; roughly doubles relapse in schizophrenia. A relapse predictor, not a cause or blame. Family intervention lowers EE.
- **Interventions:** social skills training (role-play) · psychoeducation · cognitive remediation · vocational rehab → **supported employment / IPS (place-then-train)** beats sheltered work (train-then-place).
- **ACT (Stein & Test, PACT):** mobile multidisciplinary team, shared caseload, low staff:patient ratio, 24-hour cover, in-vivo outreach; cuts admissions in the revolving-door severely ill.
- **Settings:** day hospital (clinical) vs day-care (social) · halfway home (residential bridge) · sheltered workshop · supported housing · user/carer groups. Deinstitutionalisation without services = transinstitutionalisation.
- **By condition:** chronic schizophrenia → target **negative symptoms & functional/cognitive decline**; ID → habilitation/life-skills; SUD → relapse prevention + reintegration; dementia → maintenance + carer support.
- **India:** DMHP (1996, Bellary model, under NMHP 1982) · family as primary caregiver · **RPwD Act 2016** (21 disabilities incl. mental illness, ≥40% benchmark, reservations, certification) · **MHCA 2017** (advance directive, nominated representative, capacity-based admission, decriminalised suicide).
- **Outcomes:** quality of life (WHOQOL-BREF), social functioning (WHODAS/SOFAS), employment, independent living · judged by recovery orientation (the person's own goals).

ONE-DAY REVISION · PAPER 3

Forensic Psychiatry & Mental Health Law

Where psychiatry meets the court and the statute. Learn the questions a psychiatrist is actually asked, the test that decides legal insanity, the criteria for capacity, and the architecture of the Mental Healthcare Act 2017. Note the recent renaming of the criminal codes, but hold the classic teaching points: those carry the marks.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. India-first throughout; where a law was renamed (IPC→BNS, CrPC→BNSS) the old name is kept in brackets.

01 The psychiatry-law interface · **02** Criminal responsibility · **03** Fitness and competence to face trial · **04** Civil capacity · **05** The Mental Healthcare Act 2017 · **06** Other relevant law · **07** Applied forensic topics

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. India's insanity defence is Section 84 IPC, now Section 22 BNS 2023: it codifies McNaughton as a purely cognitive test, the accused must, by unsoundness of mind, be incapable of knowing the nature of the act or that it was wrong or contrary to law.
2. Classic trap: under pure McNaughton/Section 84 an accused who knew the act was wrong but could not control the impulse is still responsible, the test asks only about knowing, not control, this gap is what the irresistible-impulse and ALI tests were written to fill.
3. Responsibility = then (mental state at the time of the act, a defence); fitness to stand trial = now (can the accused understand and instruct a defence); unfit does not mean acquitted, the trial waits while treatment is given.
4. MHCA 2017 is rights-based and capacity-based (replaced MHA 1987): advance directive + nominated representative, independent vs supported admission (Section 89 = up to 30 days), Review Boards, only modified ECT, and Section 115 decriminalising suicide by presuming severe stress.
5. Capacity is decision-specific, functional and presumed, the four abilities are understand, retain, use/weigh, communicate, and an unwise decision is not by itself incapacity.

1 The psychiatry-law interface

Forensic psychiatry is the application of psychiatry to legal questions. The law does not ask "what is the diagnosis?" It asks a specific legal question and wants the mental state mapped onto a legal standard. Keep the two roles separate.

The medico-legal questions a psychiatrist is asked

Question	Domain	The legal standard at stake
Responsibility	criminal	was the person legally insane at the time of the act? (Section 84)
Fitness	criminal	can the accused understand and participate in the trial now?
Capacity	civil	can this person make a will, consent to treatment, manage affairs?
Risk	civil / criminal	how likely is future violence or self-harm; what mitigates it?
Testimony	both	the expert opinion offered to the court, with its reasoning

The clinical role vs the legal role

The treating (clinical) role

Duty is to the **patient**. Goal is care and welfare. Therapeutic alliance, confidentiality, and the patient's best interests govern. The frame is diagnosis and treatment.

The forensic (legal) role

Duty is to the **court** and to the truth. Goal is an objective, impartial opinion. There is no therapeutic privilege; the assessee must be told the limits of confidentiality. The frame is the legal standard.

TRAP

Avoid the **dual-role conflict**: a psychiatrist should not ordinarily be both the treating clinician and the forensic evaluator for the same person in the same matter. The duties pull in opposite directions. The forensic evaluator gives a **warning of non-confidentiality** at the start of the assessment.

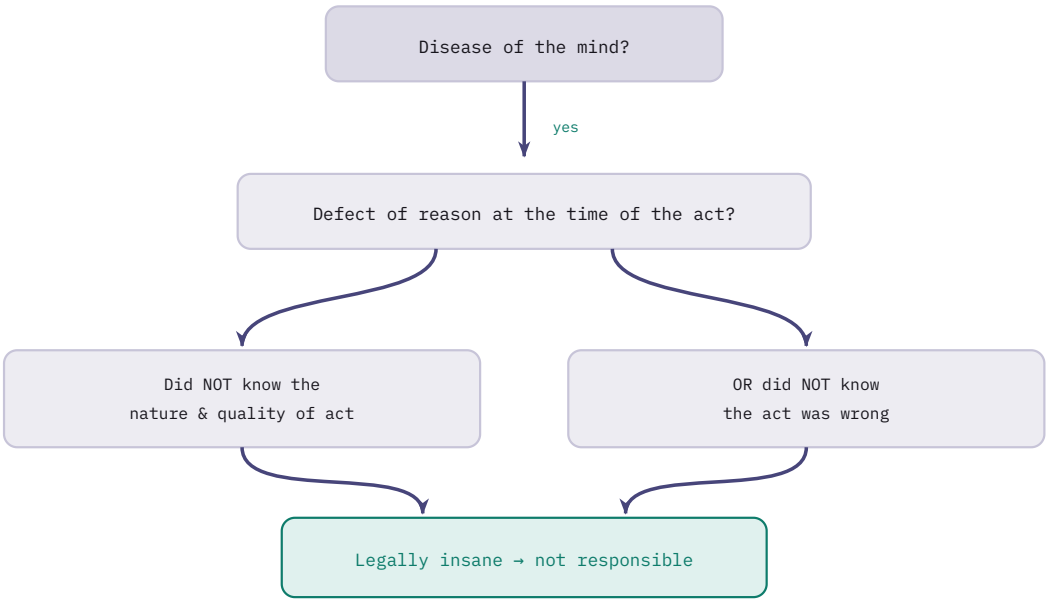
2 Criminal responsibility

A crime needs two parts: **actus reus** (the guilty act) and **mens rea** (the guilty mind). The insanity defence argues that mental illness removed the capacity for mens rea. The governing test in common-law systems, and in India, descends from the McNaughton rules.

The McNaughton rules (1843)

- **Origin** – Daniel McNaughton killed Edward Drummond (secretary to PM Robert Peel), believing he was persecuted. Acquitted on grounds of insanity; the outcry led the House of Lords to lay down the rules.
- **The test** – at the time of the act, because of a **defect of reason from disease of the mind**, the accused either (a) did not know the **nature and quality** of the act, or (b) did not know that it was **wrong**.
- **What it is** – a purely **cognitive** test. It asks only about *knowing*, not about control of impulse or emotion. This is its central limitation and its examinable feature.

MCNAUGHTON: THE RESPONSIBILITY LOGIC



A cognitive cascade: a disease of the mind that produces a defect of reason such that, at the time of the act, the accused did not know its nature and quality, or did not know it was wrong. Either limb suffices.

The Indian statutory basis

◆ **Section 84 (IPC)** – "Nothing is an offence which is done by a person who, at the time of doing it, by reason of **unsoundness of mind**, is incapable of knowing the **nature of the act**, or that he is doing what is either **wrong** or **contrary to law**." This is the McNaughton rule codified for India.

■ **The renaming** – the Indian Penal Code 1860 has been replaced by the **Bharatiya Nyaya Sanhita 2023** (in force July 2024). The insanity provision is carried over almost verbatim as **Section 22 BNS**. The wording "unsoundness of mind" is retained. The teaching point is unchanged.

● **Legal vs medical insanity** – Indian courts (e.g. *Surendra Mishra*, *Hari Singh Gond*) stress that **legal insanity**, not medical insanity, must be proved. Every mentally ill person is not exempt; the unsoundness must reach the Section 84 threshold **at the time of the act**. The burden is on the accused, on the balance of probabilities.

The centrepiece: the insanity tests compared

Test	Jurisdiction / origin	What it asks
McNaughton (1843)	England; the global template. India = Section 84 IPC / Section 22 BNS	cognitive: did the accused **know** the nature/quality of the act, or that it was wrong?
Irresistible impulse	added in some US states (19th c.)	volitional: even if he knew, was he **unable to control** the act?
Durham (product) rule (1954)	New Hampshire / DC; now abandoned	was the act the **product** of mental disease? (too broad, vague)
ALI / Model Penal Code (1962)	American Law Institute	did he lack **substantial capacity** to appreciate wrongfulness *or* to conform conduct to law? (cognitive + volitional)
Diminished responsibility	England (Homicide Act 1957); partial defence	abnormality of mind that **substantially impaired** responsibility; reduces murder to manslaughter, not a full acquittal

● MNEMONIC

"Know, Control, Product, Appreciate"

McNaughton = **Know** (cognitive). Irresistible impulse = **Control** (volitional). Durham = **Product** (and dead). ALI = **Appreciate + conform** (both). India follows the first: pure **Know**.

Two more concepts the court uses

● **Automatism** – an act done with no conscious control of the mind (during a seizure, sleepwalking, hypoglycaemia, concussion). **Sane automatism** (external cause, e.g. a blow) leads to a full acquittal; **insane automatism** (internal disease, e.g. epilepsy in some rulings) is treated under the insanity rules.

▲ **Intoxication** – **voluntary** intoxication is generally *no* defence to responsibility, though it may negate specific intent. **Involuntary** intoxication, or a disease of the mind caused by chronic alcohol use (e.g. alcoholic psychosis), may engage Section 84.

● CLASSIC TRAP

An accused may **know** the act is wrong yet feel unable to stop (the volitional deficit of, say, a command hallucination or a compulsion). Under pure McNaughton and Section 84, that person can still be held responsible, because the test asks only about **knowing**, not about **control**. This is the gap the irresistible-impulse and ALI tests were written to fill.

3 Fitness and competence to face trial

Responsibility looks **backward** (mental state at the time of the act). Fitness looks at the **present** (mental state now, during the proceedings). The two are independent: a person can be responsible for the act yet currently unfit, or vice versa.

● **Fitness to stand trial** – can the accused, *now*, understand the charge and the proceedings and instruct a defence? If unfit ("of unsound mind"), the trial is postponed and treatment given (in India under the procedure formerly in CrPC Sections 328–339, carried into the **BNSS 2023**, the new criminal procedure code).

● **Fitness to plead** – the narrower English formulation. Classically tested against the **Pritchard criteria**: the accused must be able to (1) understand the charge, (2) understand the plea (guilty / not guilty), (3) challenge a juror, (4) instruct counsel, (5) follow the evidence in court.

Structure of the fitness assessment

■ **What is assessed** – not the diagnosis but the **functional abilities**: comprehension of the situation, factual and rational understanding of the proceedings, ability to communicate with and assist the lawyer, and the capacity to make decisions about the defence. Decision-specific, like all capacity.

QUICK RULE

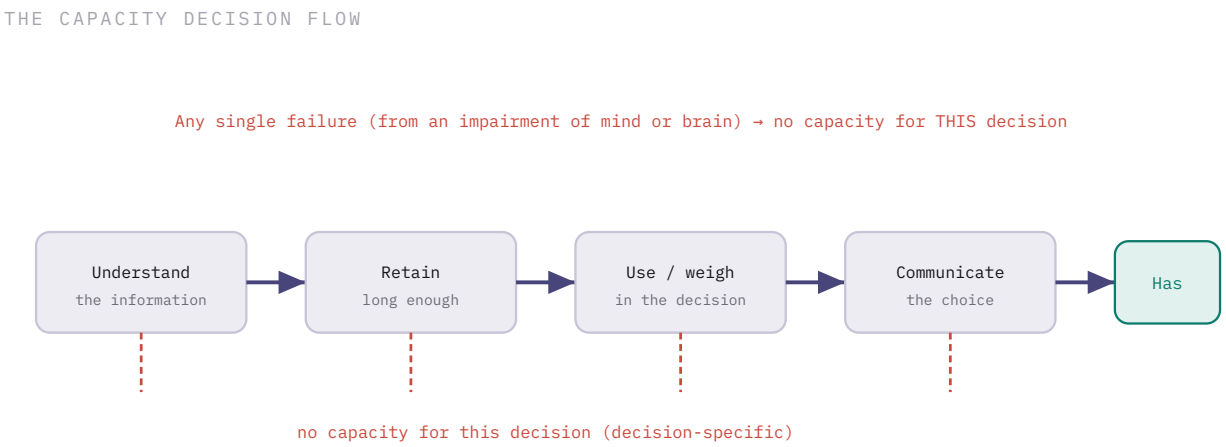
Responsibility = then. Fitness = now. Responsibility is a defence to the charge; fitness is about whether the trial can proceed at all. Unfit does not mean acquitted; it means the trial waits while the person is treated.

4 Civil capacity

Capacity is **decision-specific** and **functional**: a person may have capacity for one decision and not another, and may regain or lose it over time. It is presumed present in adults; the question is always about *this* decision, *now*. An unwise decision is not, by itself, evidence of incapacity.

The functional test of capacity

■ **The four abilities** – to have capacity for a decision a person must be able to: (1) **understand** the relevant information, (2) **retain** it long enough to use it, (3) **use or weigh** it in the decision, and (4) **communicate** the choice. Failure of any one, due to an impairment of mind or brain, means incapacity for that decision.



Capacity is a chain: understand, retain, weigh, communicate. Failure of any one link, caused by an impairment of the mind or brain, defeats capacity for that specific decision. Capacity is presumed and is decision- and time-specific.

Testamentary capacity (capacity to make a will)

◆ **The Banks v Goodfellow criteria** – to make a valid will the testator must, at the time, (1) understand the **nature of the act** of making a will and its effects, (2) understand the **extent of the property** being disposed of, (3) appreciate and weigh the **claims of those** who might expect to benefit, and (4) be free of any **disorder of mind or delusion** that perverts the disposition.

● MNEMONIC

"Act, Assets, Affections"

The three positive limbs of testamentary capacity: the **Act** of willing, the extent of the **Assets**, and the claims of the **Affections** (those who might benefit). The fourth limb is the absence of a distorting delusion.

Other civil capacities

● **Capacity to consent to treatment** – same functional four-ability test, applied to *this* treatment. Valid consent = capacity + adequate **information** + **voluntariness** (free of coercion). If the person lacks capacity, decisions follow the best-interests / substituted-judgement route, and in India the MHCA 2017 framework (advance directive, nominated representative) governs for mental healthcare.

● **Capacity to manage one's affairs** – the ability to handle property and finances. Where it is lacking, guardianship or a substitute decision-maker may be appointed (in India historically under the Mental Health Act, now read alongside the Rights of Persons with Disabilities Act 2016 and the National Trust Act for relevant groups).

● RULE

Capacity is **not** all-or-nothing and **not** diagnosis-based. A person with schizophrenia or early dementia may retain capacity for many decisions. Assess the specific decision, presume capacity, and remember that the right to make an *unwise* choice is part of having capacity.

5 The Mental Healthcare Act 2017

The MHCA 2017 replaced the Mental Health Act 1987. The shift is the headline: from a **custodial, guardianship** model to a **rights-based, capacity-based** one, aligned with the UN Convention on the Rights of Persons with Disabilities, which India ratified. The patient is now the centre of decision-making.

The shift from 1987 to 2017

Theme	MHA 1987 (old)	MHCA 2017 (current)
Frame	protection of society; custodial	rights-based; person-centred
Admission	reception orders; guardianship	independent vs supported; capacity-driven
Patient voice	limited	advance directive + nominated representative
Oversight	licensing of psychiatric hospitals	Mental Health Review Boards + Central/State Authorities
Suicide	attempt punishable (IPC 309)	decriminalised (Section 115)

The centrepiece: key MHCA 2017 provisions

Provision	What it means
Definition of mental illness (Section 2)	a substantial disorder of thinking, mood, perception, orientation or memory that grossly impairs judgement, behaviour, capacity to recognise reality or to meet ordinary demands of life. **Excludes** mental retardation (now called intellectual disability) except where it co-occurs.
Right to treatment (Section 18)	every person has a right to access mental healthcare run or funded by the government, with affordability and quality.
Advance directive (Sections 5–13)	a person may state, in advance, how they wish to be (or not be) treated, and may name a nominated representative. Made when the person has capacity; activated when they lose it.
Nominated representative (Section 14)	a person nominated to take decisions on the patient's behalf and to support their wishes; a legal substitute decision-supporter, not a guardian in the old sense.
Independent admission (Sections 86–88)	the person has capacity and admits/discharges **themselves**; can leave at will. The default, least-restrictive route.
Supported admission (Sections 89–90)	for a person who lacks capacity or poses risk; needs two independent assessments. Section 89 = up to 30 days; Section 90 = beyond 30 days, with Review Board oversight.
Restraint & seclusion (Section 97)	physical restraint and seclusion only to prevent imminent harm, as a last resort, least restrictive, recorded and reported; chaining is prohibited.
ECT (Section 95)	ECT only with anaesthesia and muscle relaxants (no **direct/unmodified** ECT). ECT is prohibited for minors except with Review Board permission. Psychosurgery needs informed consent + Board approval.
Mental Health Review Board (Sections 73–83)	a quasi-judicial body that reviews admissions and advance directives, hears complaints, protects rights. Sits under State Mental Health Authority; Central Authority above.
Decriminalisation of suicide (Section 115)	a person who attempts suicide is **presumed** to have severe stress and shall **not** be prosecuted; the government must provide care and rehabilitation.

HOLD THIS

MHCA 2017 = **rights-based, capacity-based**. Three pillars: **advance directive** + **nominated representative** (the patient's voice), two **admission** types (independent vs supported), and the **Review Boards** (oversight). Plus: no unmodified ECT, restraint as last resort, and Section 115 decriminalising suicide.

● TRAP

Section 115 does **not** repeal the old IPC 309 by itself; it creates a **presumption of severe stress** that bars prosecution, effectively decriminalising the attempt. Under the new criminal code (BNS), the offence of attempted suicide is not carried forward in the old form (one narrow exception relates to attempts to coerce or obstruct a public servant). The teaching point for psychiatry remains: the suicide attempt is a clinical emergency, not a crime.

6 Other relevant law

Statute	Year	What the psychiatrist must know
POCSO Act	2012	Protection of Children from Sexual Offences; child = under **18**. Mandatory reporting: any person (clinicians included) who knows of an offence must report it; failure is itself an offence. Child-friendly procedure; recent amendments add the death penalty for aggravated assault.
NDPS Act	1985	Narcotic Drugs and Psychotropic Substances. Controls manufacture, possession and trafficking; the legal frame for substance-use practice. Section 64A offers **immunity** to an addict who volunteers for treatment. Strict provisions; relevant to de-addiction and prescribing.
RPwD Act	2016	Rights of Persons with Disabilities; replaced the 1995 Act. Recognises **mental illness** and **intellectual disability** as disabilities (21 conditions listed); benchmark disability ≥40%; mandates non-discrimination, reasonable accommodation, certification.
Indian Lunacy Act	1912	colonial-era custodial law; superseded by the MHA 1987 (historical context only).
Mental Health Act	1987	the immediate predecessor to MHCA 2017; licensing-and-guardianship model; now repealed. Know it as the "before" of the rights-based shift.

● TRAP

Under **POCSO**, reporting is mandatory and overrides ordinary confidentiality: a clinician who learns of child sexual abuse *must* report it. The much-debated tension is with adolescent **consensual sexual activity** between minors, which POCSO technically criminalises; courts have urged a nuanced approach, but the reporting duty as written is strict.

7 Applied forensic topics

Violence risk assessment

● **Three approaches** – **unstructured clinical** judgement (intuitive, poor reliability); **actuarial** (statistical, fixed risk factors, e.g. VRAG); and **structured professional judgement** (a checklist guides but clinical judgement decides, e.g. the **HCR-20**). SPJ is the current standard.

◆ **HCR-20 structure** – **H**istorical (10 static items, the past), **C**linical (5 dynamic items, the present), **R**isk management (5 items, the future). The single best predictor of future violence is **past violence**.

Duty to warn and protect (Tarasoff)

■ **The Tarasoff principle** – from *Tarasoff v Regents of the University of California* (1976). When a therapist determines that a patient poses a **serious threat to an identifiable victim**, the therapist has a duty to take reasonable steps to protect that victim. "The protective privilege ends where the

public peril begins."

- **Warn vs protect** – the first Tarasoff ruling (1974) said duty to **warn**; the second (1976) broadened it to a duty to **protect** (which may mean warning the victim, notifying police, or hospitalising the patient). It is a US doctrine; in India there is no identical statutory duty, but the ethical logic of confidentiality yielding to prevention of serious harm to a third party is widely taught and applied.

Confidentiality and its limits

- **The rule** – confidentiality is a core duty, but it is **not absolute**. It yields when there is a risk of serious harm to the patient or an identifiable third party, where the law mandates disclosure (e.g. POCSO, notifiable conditions, court orders), or with valid consent.

Confidentiality holds

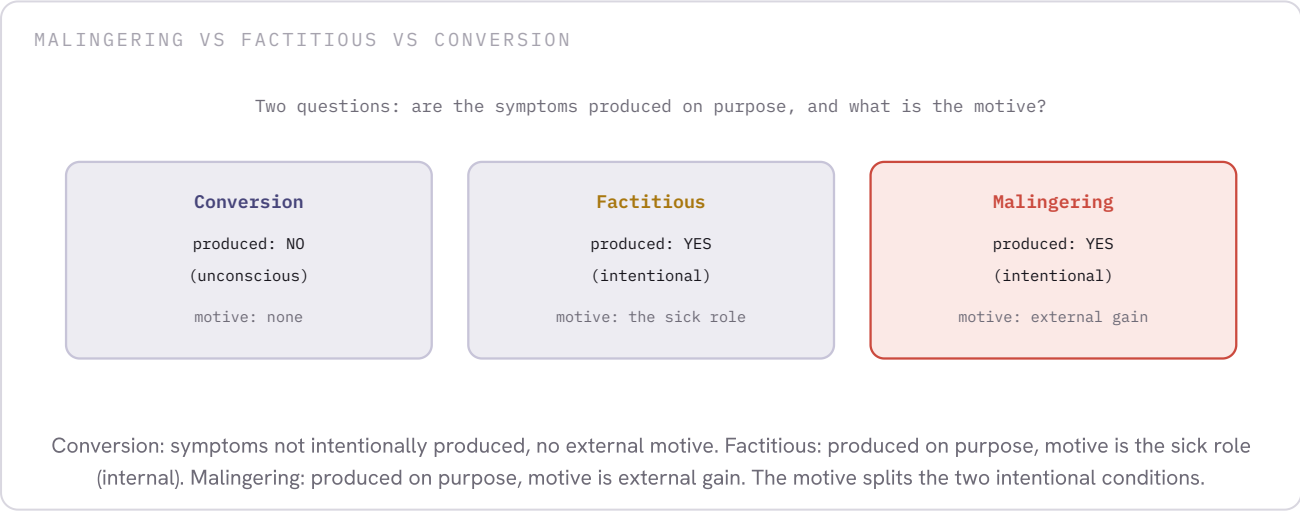
routine clinical information, the diagnosis, the content of sessions; disclosure only with consent or on a genuine need-to-know basis within the treating team.

Confidentiality yields

serious risk to self or others; mandatory reporting (POCSO, court order); a genuine public-interest or public-safety exception. Disclose the **minimum necessary**.

Malingering and its detection

- **Malingering** – the **intentional** production of false or grossly exaggerated symptoms, motivated by an **external incentive** (avoiding the army, prison or work; obtaining money or drugs). In DSM it is a V-code, not a disorder. Suspect it in a medico-legal context, with antisocial traits, and where the presentation does not fit any known syndrome.
- **The differential** – distinguish from **factitious disorder** (symptoms also intentionally produced, but the motive is the *sick role*, an internal incentive, not external gain) and from genuine **conversion/somatic** disorders (symptoms *not* intentionally produced).



Conversion: symptoms not intentionally produced, no external motive. Factitious: produced on purpose, motive is the sick role (internal). Malingering: produced on purpose, motive is external gain. The motive splits the two intentional conditions.

The psychiatric report and expert testimony

- **The report** – structured, factual, and addressed to the specific legal question. Standard sections: the instruction and the question, sources of information, the assessment, the mental state, the opinion with explicit **reasoning**, and the limits of the opinion. State facts and inferences separately; do not stray beyond expertise.
- **Giving testimony** – the psychiatrist gives an **opinion**; the court decides the **ultimate issue** (guilt, fitness). Be impartial, stay within competence, explain in plain language, concede uncertainty honestly. The expert serves the court, not the side that pays. In India the Indian Evidence Act (now the Bharatiya Sakshya Adhiniyam 2023) governs the admissibility of expert opinion.

● RULE

Never answer the **ultimate legal question** as if it were a medical one. The psychiatrist's job is to describe the mental state and map it onto the legal standard with reasons; whether the accused is "guilty," "insane in law," or "unfit" is for the court to rule. Offer the building blocks, not the verdict.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Roles:** clinical role = duty to patient; forensic role = duty to the court. Avoid the dual-role conflict; warn of non-confidentiality before a forensic assessment.
- ★ **Responsibility:** McNaughton = pure **cognitive** test (know the nature/quality, or that it was wrong). India = **Section 84 IPC**, now **Section 22 BNS**. Legal, not medical, insanity; at the time of the act; burden on the accused.
- **Insanity tests:** McNaughton = Know · Irresistible impulse = Control · Durham = Product (abandoned) · ALI = appreciate + conform · Diminished responsibility = partial defence (murder → manslaughter).
- **Fitness:** responsibility = *then*; fitness = *now*. Pritchard criteria for fitness to plead. Unfit ≠ acquitted; trial waits, treatment given (BNSS).
- **Capacity:** decision-specific, functional, presumed. Four abilities: **understand, retain, weigh, communicate**. Unwise ≠ incapable.
- **Testamentary capacity (Banks v Goodfellow):** nature of the **act**, extent of the **property**, claims of those who might **benefit**, free of a distorting delusion.
- **MHCA 2017:** rights-based, capacity-based (replaced MHA 1987). Advance directive + nominated representative; independent vs supported admission (Section 89 = up to 30 days); Review Boards; modified ECT only; restraint as last resort; Section 115 decriminalises suicide attempt.
- **Other law:** POCSO 2012 (child <18, mandatory reporting) · NDPS 1985 (substances; Section 64A immunity for volunteering addict) · RPwD 2016 (benchmark disability ≥40%) · ILA 1912 / MHA 1987 = history.
- **Risk:** unstructured < actuarial (VRAG) < structured professional judgement (**HCR-20**: Historical-Clinical-Risk). Best single predictor = past violence.
- **Tarasoff:** duty to warn (1974) → duty to protect (1976). "Protective privilege ends where public peril begins." US doctrine; ethical logic taught in India.
- **Malingering:** intentional symptoms for **external** gain (a V-code). Factitious = intentional, for the **sick role**. Conversion = not intentional. The motive splits the two intentional ones.
- **Testimony:** give the opinion with reasoning; the court decides the ultimate issue. Impartial, within competence, the expert serves the court.

ONE-DAY REVISION · PAPER 3

Cultural psychiatry, made retrievable

Culture shapes how distress is experienced, expressed, explained, brought to care and recovered from. Hold the DSM-5 framework (CFI and the Outline), the named cultural concepts of distress with their regions, the ethnopharmacology of CYP450 variation, and the migration findings. The centrepiece syndrome table is a 10-mark answer in waiting.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. The Indian context cross-references the companion Anthropology pack.

01 Why culture matters & the DSM-5 framework · **02** Cultural concepts of distress · **03** Culture & psychopathology · **04** Ethnopsychopharmacology · **05** The Indian context · **06** Migration, acculturation & minority mental health · **07** Culturally competent practice

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. DSM-5 framework: the CFI is a 16-question person-centred interview; the OCF is its 4-domain scaffold (cultural identity, conceptualisation of distress, psychosocial stressors/vulnerability, clinician-patient relationship) plus an overall assessment.
2. DSM-5 retired "culture-bound syndrome" and replaced it with cultural concepts of distress, in three types: cultural syndrome, idiom of distress, and explanation (perceived cause).
3. The centrepiece syndromes: dhat (South Asia, semen-loss anxiety), koro (epidemic genital retraction), amok (sudden homicidal outburst), taijin kyofusho (fear of offending others, the mirror image of Western social phobia); most map onto anxiety, depressive, somatic or dissociative disorders.
4. WHO IPSS (1973) and DOSMeD (1992): schizophrenia is universal, but course and outcome were better in the developing world (family support, low expressed emotion, social roles); a contested but classic result.
5. Ethnopharmacology: CYP2D6 poor-metabolisers are commoner in White Europeans and CYP2C19 poor-metabolisers in East Asians (so many Asian patients need lower doses); PM → toxic, UM → subtherapeutic, and HLA-B*1502 + carbamazepine causes Stevens-Johnson in Han Chinese.

1 Why culture matters & the DSM-5 framework

Culture is the shared system of meanings, norms and practices a person grows up in. It does not cause mental illness on its own, but it shapes every stage of it. Get these **five verbs** and you have the spine of the topic.

Culture shapes the…	Meaning	Example
Experience	what the inner distress feels like	depression felt as bodily heaviness vs guilt
Expression	how it is shown and described	somatic idioms vs psychological words
Explanation	what is believed to cause it	spirit, evil eye, karma, imbalance, biology
Help-seeking	who is consulted, and the pathway to care	faith healer → GP → psychiatrist
Outcome	course, recovery and social reintegration	IPSS better outcome in the developing world

The DSM-5 tools: CFI and the Outline for Cultural Formulation

- **Cultural Formulation Interview (CFI)** – a DSM-5 set of **16 questions** (a person-centred, semi-structured interview) the clinician asks to elicit the patient's own view of their problem. There are also an informant version and twelve supplementary modules.
- **Outline for Cultural Formulation (OCF)** – introduced in DSM-IV (appendix) and revised in DSM-5; the **conceptual scaffold** the CFI operationalises. Four domains plus an overall assessment.

OCF domain	What you assess
Cultural identity	ethnicity, language, religion, migration, generation; reference groups the person belongs to
Cultural conceptualisation of distress	the idioms, explanatory models and perceived causes the person uses to understand their symptoms
Psychosocial stressors & cultural features of vulnerability and resilience	stressors, supports, levels of functioning and disability, all read in cultural context (faith, family, kin networks)
Cultural features of the clinician-patient relationship	differences of culture, language and status between clinician and patient that may shape rapport, diagnosis and adherence
Overall cultural assessment	how the above feed diagnosis, prognosis and the management plan

MNEMONIC

"I·C·S·R" → Identity, Conceptualisation, Stressors, Relationship

The four OCF domains in order, then the overall assessment. The CFI is the interview; the OCF is the structure it fills in.

RULE

The CFI is **not a screening or diagnostic test**. It is a way of taking a cultural history so the clinician understands the problem in the patient's own terms, avoids misdiagnosis, and builds an acceptable plan. It can be used with any patient, not only ethnic minorities.

2 Cultural concepts of distress

DSM-5 retired the old label **"culture-bound syndrome"** (which wrongly implied tidy, discrete, exotic diseases sealed inside one culture) and replaced it with the broader, three-part **cultural concepts of distress**. Know the three categories first, then the named examples.

Category	Definition	Example
Cultural syndrome	a cluster of co-occurring symptoms recognised within a culture as a coherent pattern	ataque de nervios, amok
Cultural idiom of distress	a shared way of *expressing* or talking about suffering; not a discrete syndrome	"nerves", kufungisisa ("thinking too much")
Cultural explanation (perceived cause)	a culturally accepted causal model or label for symptoms	susto ("soul loss"), maladi moun (sent sickness)

The classic culture-related syndromes (centrepiece)

Syndrome	Region	Core features	Maps onto (DSM/ICD)
Dhat	South Asia (India, Sri Lanka)	anxious preoccupation with loss of semen in urine; fatigue, weakness, guilt over masturbation/"nightfall"	anxiety / depressive / somatic symptom disorder
Koro	South-East and East Asia (China, Malaysia, India outbreaks)	sudden terror that the penis (or, in women, breasts/vulva) is retracting into the body and that death will follow; can be epidemic	anxiety / acute panic; delusional in some cases

Syndrome	Region	Core features	Maps onto (DSM/ICD)
Amok	Malaysia, Indonesia, South-East Asia	a brooding period then a sudden outburst of violent, homicidal aggression, often ending in exhaustion, amnesia or suicide ("running amok")	dissociative / psychotic / mood episode; impulse-control
Latah	Malaysia, Indonesia (also Ainu, Siberia)	exaggerated startle followed by echolalia, echopraxia, command obedience and coprolalia; usually older women	a startle / dissociative reaction; no single DSM equivalent
Ataque de nervios	Latino / Caribbean cultures	uncontrollable shouting, crying, trembling, heat rising in the chest, aggression, faintness; often after a family stressor (a funeral, a fight)	panic / anxiety / dissociative / depressive presentations
Susto	Latin America	illness attributed to a frightening event causing the soul to leave the body ("soul loss"); appetite/sleep change, low mood, somatic complaints	depressive / anxiety / PTSD / somatic disorder
Brain fag	West Africa (Nigeria)	students under study pressure report "brain fatigue", head/neck pain, burning, blurred vision, poor concentration	anxiety / depressive / somatic symptom disorder
Taijin kyofusho	Japan, Korea	intense fear of *offending or embarrassing others* by one's gaze, blushing, body odour or appearance (an other-focused social anxiety)	social anxiety disorder (offensive subtype)
Hikikomori	Japan (spreading)	severe, prolonged (>6 months) social withdrawal: a young person confines to the home, avoiding work, school and contact	not a DSM diagnosis; linked to social anxiety, depression, ASD

● CLASSIC TRAP

Taijin kyofusho is the mirror image of Western social anxiety. Western social phobia fears *self*-embarrassment; the offensive (offending) subtype of taijin kyofusho fears *harming or embarrassing others* with one's body. Both are read as variants of social anxiety disorder, shaped by an individualist vs collectivist self.

QUICK RULE

Most "culture-bound" syndromes are **local expressions of recognised disorders** (anxiety, depression, panic, somatic, dissociative), not unique diseases. That is exactly why DSM-5 dropped the "bound" framing. Dhat = the Indian exam favourite; koro = epidemic genital retraction; amok = sudden homicidal outburst.

● MNEMONIC

"Dhat Kills All Latah's Ataques, Susto Brings Taijin Home"

Dhat, Koro, Amok, Latah, Ataque de nervios, Susto, Brain fag, Taijin kyofusho, Hikikomori – the nine to list.

3 Culture & psychopathology

Form vs content

◆ **The core principle** – culture shapes the **content** of symptoms, not the **form**. The *form* (that a person has delusions, hallucinations, low mood) is broadly universal; the *content* (what the delusion is about, what the voice says) is filled in by culture.

Form (universal)

The **structure** of the symptom: delusion, hallucination, depressive cognition, obsession. Schizophrenia, mood and anxiety disorders appear in every culture studied (the WHO finding of broadly similar core schizophrenia symptoms worldwide).

Content (culture-filled)

A persecutory delusion may centre on **spirits, witchcraft or the evil eye** in one setting and on the **CIA, microchips or the internet** in another. Religious and grandiose content tracks the dominant belief system. Guilt is more prominent in Western depression; somatic complaint more prominent elsewhere.

Somatisation across cultures

- **Somatisation** – the expression of psychological distress as bodily symptoms. It is **more prominent in many non-Western settings** (and in older and less psychologically minded patients), where physical complaints are a more acceptable idiom than naming sadness or anxiety. A key cause of *under-recognised depression* in primary care, including in India.

● TRAP

Somatisation is a matter of **idiom and emphasis, not a different disease**. The depression underneath is the same; the patient presents the body, not the mood. The WHO primary-care studies (Gureje and colleagues) found somatic presentation of depression is the rule worldwide, not a Western-vs-rest split.

Cross-cultural epidemiology: the IPSS schizophrenia-outcome finding

- **WHO IPSS & DOSMeD** – the **International Pilot Study of Schizophrenia** (IPSS, 1973, 9 countries) and the follow-up **Determinants of Outcome of Severe Mental Disorders** (DOSMeD, 1992, 10 countries) found schizophrenia is recognisable worldwide but, strikingly, that **outcome was better in the developing countries** (e.g. India, Nigeria, Colombia) than in the developed ones.

Study	Year	Headline finding
IPSS	1973	schizophrenia exists and is identifiable across 9 countries; first-rank symptoms found everywhere
DOSMeD	1992	broadly similar incidence of narrowly-defined schizophrenia, but better course and outcome in the developing world
ISoS	2001	long-term (15-25 yr) follow-up; partly confirmed the developing-world advantage, with caveats

● RULE & DEBATE

Explanations offered for the better developing-world outcome: stronger **extended-family and community support**, more available social/work roles, **lower expressed emotion**, less stigmatising attributions, and a higher share of acute/good-prognosis cases. The finding is *contested*: critics cite case-finding and diagnostic differences, higher mortality and loss to follow-up in low-income sites, and possible selection bias (Cohen and colleagues). Still examinable as the classic cross-cultural result.

4 Ethnopsychopharmacology

Ethnic groups differ, on average, in how they metabolise psychotropics. The driver is **genetic polymorphism of the cytochrome P450 (CYP) enzymes**, layered with diet, smoking and herbal use. The clinical upshot: standard Western doses can be too high for some groups.

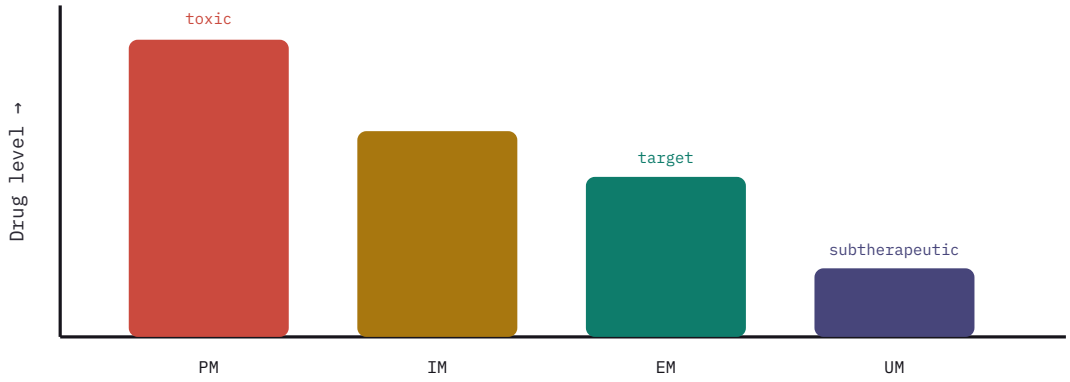
CYP450 metaboliser phenotypes

Phenotype	Enzyme activity	Effect on a standard dose
Poor metaboliser (PM)	little/no functional enzyme	high drug levels → more side-effects and toxicity at usual doses

Phenotype	Enzyme activity	Effect on a standard dose
Intermediate metaboliser (IM)	reduced activity	somewhat raised levels
Extensive metaboliser (EM)	normal (the reference)	expected levels and response
Ultra-rapid metaboliser (UM)	gene duplication, very high activity	subtherapeutic levels → apparent non-response at usual doses

◆ **The key enzymes & ethnic patterns** – **CYP2D6** (many antipsychotics, tricyclics, risperidone, some SSRIs): poor-metaboliser status is commoner in **White Europeans (~7-10%)**; ultra-rapid status is commoner in **North/East African and Middle Eastern** groups. **CYP2C19** (diazepam, citalopram, some TCAs): poor-metaboliser status is commoner in **East Asians (~15-20%)**. This underlies the observation that many **Asian patients need lower antipsychotic/antidepressant doses**.

CYP450 METABOLISER STATUS AND DRUG LEVEL



At one standard dose, poor metabolisers (PM) sit at toxic levels, extensive metabolisers (EM) at target, and ultra-rapid metabolisers (UM) below the therapeutic threshold and so appear to be non-responders.

Beyond genes: diet, smoking and herbal medicines

● **Diet & smoking** – chargrilled meat, cruciferous vegetables and especially **tobacco smoke induce CYP1A2**, lowering levels of clozapine and olanzapine (so smokers need higher doses and a dose review on quitting). High-protein diets speed, low-protein slow, oxidative metabolism.

● **Traditional & herbal remedies** – widely used alongside biomedicine and a real interaction risk. **St John's wort** (a CYP3A4/P-glycoprotein inducer) lowers levels of many drugs and can precipitate serotonin syndrome with SSRIs. **Grapefruit juice** inhibits CYP3A4, raising levels. Patients often do not volunteer herbal use; ask directly.

● **Adverse-effect susceptibility** – ethnic differences also appear in receptor and HLA pharmacogenetics: **HLA-B*1502** (carbamazepine-induced Stevens-Johnson/TEN) is commoner in **Han Chinese and South-East Asians** (screen before starting), and **HLA-A*3101** in Europeans/Japanese. Lithium and clozapine dosing also vary across populations.

5 The Indian context

India is the recurring exam frame: a pluralistic medical landscape where biomedicine, AYUSH systems and faith healing coexist, and where family and stigma dominate the pathway to care. Cross-reference the Anthropology pack for the wider model.

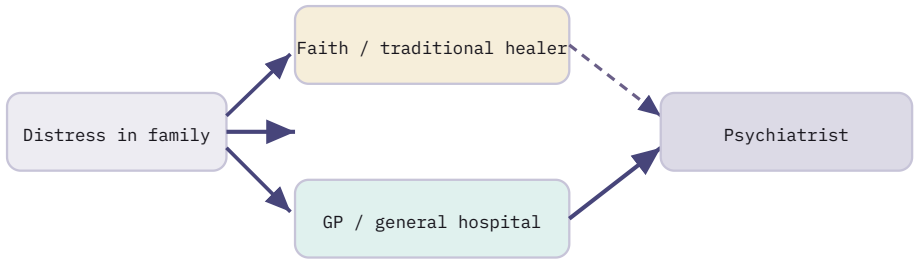
Explanatory models of illness

- **Explanatory model (Kleinman)** – the patient's own beliefs about **cause, mechanism, course, expected treatment and outcome** of their illness. In India these often invoke **supernatural causes** (spirit possession, black magic, evil eye, displeasure of a deity, "tension"), **karma and fate**, or humoral/Ayurvedic *dosha* imbalance, alongside or instead of a biomedical model. Multiple models are held at once.

Family, faith healers and the pathway to care

- **The family** – the **primary unit of care and decision-making**. Families bring the patient, hold the explanatory model, give treatment consent in practice, supervise medication and provide long-term support (a likely contributor to the better schizophrenia outcomes). Engaging the family is essential, not optional.
- **Traditional & faith healers** – temples (e.g. the dargahs and healing-temple traditions), *vaidyas*, *hakims*, astrologers and religious healers are usually the **first contact** for psychological distress, especially in rural areas. The classic Indian finding (Kapur, and the work at Vellore/NIMHANS) is a **multiple and parallel** pathway: patients consult healer and doctor at the same time, not in sequence.

A TYPICAL INDIAN PATHWAY TO PSYCHIATRIC CARE



healer and doctor consulted in parallel, often with long delay before specialist care

Care is typically sought first from family, then from faith healers and general doctors in parallel, with the psychiatrist reached late. Long help-seeking delays and treatment gaps are the norm.

Stigma and the treatment gap

- **Stigma & the treatment gap** – mental illness carries heavy stigma (fears about marriage prospects, employment and family reputation, especially for women), driving concealment and delay. The Indian National Mental Health Survey (NMHS 2015-16) reported a lifetime mental-morbidity prevalence near **13.7%** and a **treatment gap of roughly 70-92%** across disorders. The DALY burden is large and rising.
- **Integration of traditional and biomedical care** – rather than dismissing healers, the modern approach is **collaboration**: respecting the explanatory model, working with families, and engaging traditional sectors. The District Mental Health Programme (under the National Mental Health Programme, 1982) and the Mental Healthcare Act 2017 frame community-based, rights-based, integrated delivery.

6 Migration, acculturation & minority mental health

Acculturation and Berry's strategies

● **Acculturation** – the psychological and cultural change that follows sustained contact between two cultures. **Acculturative stress** is the distress this can generate (identity conflict, loss, discrimination, language barriers).

◆ **Berry's two-question model** – acculturation strategy depends on two choices: (1) *Do I keep my heritage culture?* and (2) *Do I take on the host culture?* The four cells are the named strategies.

	Maintain host culture: YES	Maintain host culture: NO
Keep heritage: YES	Integration (biculturalism; best mental-health outcome)	Separation (heritage only)
Keep heritage: NO	Assimilation (host only)	Marginalisation (neither; worst outcome)

QUICK RULE

Integration (keep both cultures) gives the **best** adjustment and mental health; **marginalisation** (lose both) gives the **worst**. The outcome also depends on whether the host society is multicultural or pressures assimilation.

Raised psychosis incidence in migrants and minorities

◆ **The migrant psychosis finding** – the incidence of schizophrenia and other psychoses is **raised in many migrant and minority groups**, classically reported as up to a **several-fold (around 4-5x) increase in African-Caribbean people in the UK**. The risk is higher in the **second generation** than the first, which points away from a purely genetic or selective-migration cause.

● RULE

Because the excess is greatest in the second generation and in those living in low-ethnic-density (more isolated) areas, the leading explanation is the **social defeat / chronic disadvantage hypothesis**: discrimination, exclusion, urban adversity and a sustained sense of outsider status, rather than ethnicity itself. Misdiagnosis (over-reading psychosis in unfamiliar idioms) contributes but does not fully explain it.

Refugee mental health

● **Refugees and asylum seekers** – a high-risk group facing trauma at three stages: **pre-migration** (war, torture, loss), **peri-migration** (a dangerous journey, camps), and **post-migration** (detention, uncertain status, unemployment, discrimination). Raised rates of **PTSD, depression and anxiety**; post-migration stressors are powerful predictors of poor outcome and are partly modifiable.

7 Culturally competent practice

Emic vs etic

Emic (insider)

Understanding distress **from within** the culture, in its own categories and meanings. The anthropologist's stance: how does *this* community frame the problem? Risks losing comparability across cultures.

Etic (outsider)

Understanding distress with **universal, cross-culture categories** imposed from outside (e.g. DSM diagnoses). Allows comparison and epidemiology, but risks imposing a Western frame where it does not fit.

MNEMONIC

"e-m-ic = from the middle (inside); e-t-ic = from the top (outside)"

From phon**EM**ic (specific to one language) and phon**ET**ic (universal). Good cross-cultural work holds both.

The category fallacy

◆ **Category fallacy (Kleinman)** – the error of taking a diagnostic category developed in one culture, assuming it is universal, and **applying it to another culture where it has no coherent local meaning**. The category is reified and then "found" everywhere, which manufactures false validity. The central caution against a purely etic psychiatry.

TRAP

The category fallacy is Kleinman's critique of imposing Western nosology (the etic stance) without checking whether the category means anything locally (the emic check). It is the conceptual engine behind DSM-5's shift to *cultural concepts of distress* and the CFI.

Working with interpreters

■ **Use a trained professional interpreter** – not family members (who edit, omit and create confidentiality and role conflicts) and not children. Brief the interpreter beforehand; speak in short segments directly **to the patient** ("How are you feeling?", not "Ask her how she feels"); allow extra time; watch for non-equivalent terms and for the interpreter normalising odd speech (which can mask thought disorder).

Cultural competence vs cultural humility

Cultural competence

A set of **knowledge, attitudes and skills** for working across cultures. Useful, but risks reducing a culture to a checklist of "facts about group X" and stereotyping.

Cultural humility

A **lifelong, self-reflective stance** (Tervalko and Murray-Garcia): the clinician treats the patient as the expert on their own culture, examines their own biases and the power imbalance, and stays curious rather than "competent."

RULE

The field has moved **from competence to humility**: not "I have mastered this culture" but "I keep learning from this patient." Avoid the stereotype trap (assuming every member of a group holds the group's typical beliefs); always check the individual's own model with the CFI.

QUICK-RECALL · DRILL THESE ONE-LINERS

★ **Why culture matters:** it shapes experience, expression, explanation, help-seeking and outcome. CFI = 16-question interview; OCF = 4 domains (identity, conceptualisation of distress, stressors/vulnerability, clinician-patient relationship) + overall assessment.

- **Cultural concepts of distress** replaced "culture-bound syndromes": three types = cultural syndrome, idiom of distress, explanation (perceived cause).
- **Syndromes:** dhat (S Asia, semen-loss anxiety) · koro (genital retraction, epidemic) · amok (sudden homicidal outburst) · latah (startle + echolalia) · ataque de nervios (Latino) · susto (soul loss) · brain fag (W Africa, students) · taijin kyofusho (fear of offending others) · hikikomori (withdrawal >6 mo). Most map onto anxiety/depression/somatic disorders.
- **Form vs content:** culture shapes content (what the delusion is about), not form (that there is a delusion). Somatisation is idiom, not a different disease.

- **IPSS/DOSMeD:** schizophrenia universal but **better outcome in the developing world** (family support, low EE, social roles). Contested (Cohen).
- **Ethnopharmacology:** CYP2D6 PMs commoner in Europeans, CYP2C19 PMs commoner in East Asians (lower doses). PM = toxic, UM = subtherapeutic. Smoking induces CYP1A2 (clozapine/olanzapine). HLA-B*1502 + carbamazepine SJS in Han Chinese.
- **India:** family-centred care, parallel faith-healer + doctor pathway, supernatural/karma explanatory models, heavy stigma, treatment gap ~70-92% (NMHS 13.7%).
- **Migration:** Berry = integration (best) / assimilation / separation / marginalisation (worst). Psychosis raised ~4-5x in African-Caribbeans, worse in 2nd generation → social defeat, not genes.
- **Competent practice:** emic (insider) vs etic (outsider); category fallacy (Kleinman) = imposing a Western category where it has no local meaning; trained interpreters not family; move from cultural competence to cultural humility.

ONE-DAY REVISION · PAPER 3

Community Psychiatry

The shift from the asylum wall to the doorstep: prevention before cure, the primary-care worker before the specialist, and the village before the institution. India's story runs NMHP → DMHP → the 2017 Act → Tele-MANAS. Hold the dates, the three objectives, the four DMHP components, and the treatment-gap number, and the long-answer writes itself.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. Dates and percentages are amber for a reason – examiners reward them.

01 Principles: from asylum to community · **02** The burden and the treatment gap · **03** National Mental Health Programme (NMHP, 1982) · **04** District Mental Health Programme (DMHP, 1996) · **05** Policy & evidence: mhGAP, the 2017 Act, NMHS · **06** Models & human resources for community care · **07** Special community topics

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. NMHS 2015-16 (NIMHANS, 12 states): current mental morbidity ~10%, lifetime ~13-14%, and a treatment gap of 70-90% across disorders → the single strongest exam justification for community psychiatry.
2. NMHP = 1982 national umbrella with three objectives (access to minimum care for all, apply mental-health knowledge in general health & social development, community participation/self-help); DMHP = 1996, its district field arm built on the Bellary model (NIMHANS, Karnataka). Trap: do not confuse the two.
3. DMHP four classic components: Training · Service delivery · IEC · Monitoring & evaluation; training PHC medical officers and non-medical staff to detect and manage common disorders is the task-shifting step.
4. Gordon/IOM (1994) tiers universal / selective / indicated all act before onset (they subdivide Leavell-Clark primary prevention); the IOM places early treatment and relapse-prevention outside "prevention" under treatment and maintenance.
5. Mental Healthcare Act 2017 is rights-based: legal right to access state-run/funded care, advance directive, nominated representative, least-restrictive care, and decriminalised suicide attempt (presumed severe stress, duty to treat).

1 Principles: from asylum to community

Community psychiatry treats the population, not just the patient in front of you. It moves care out of the institution, closer to where people live, and frames mental illness as a **public-health** problem to be prevented, detected early, and managed in the least restrictive setting.

The historical shift

- **Deinstitutionalisation** – the move from long-stay mental hospitals to community-based care, driven by the antipsychotic era (1950s), human-rights critique of the asylum, and cost. Done well it restores dignity; done badly (without funded community services) it produces homelessness, "trans-institutionalisation" into prisons, and the revolving door.
- **Public-health approach** – define the burden, identify determinants, deliver population-level interventions, monitor outcomes. Care is a *pyramid*: most needs met by self-care and primary care, fewer by specialist and hospital services (the WHO service-organisation pyramid).

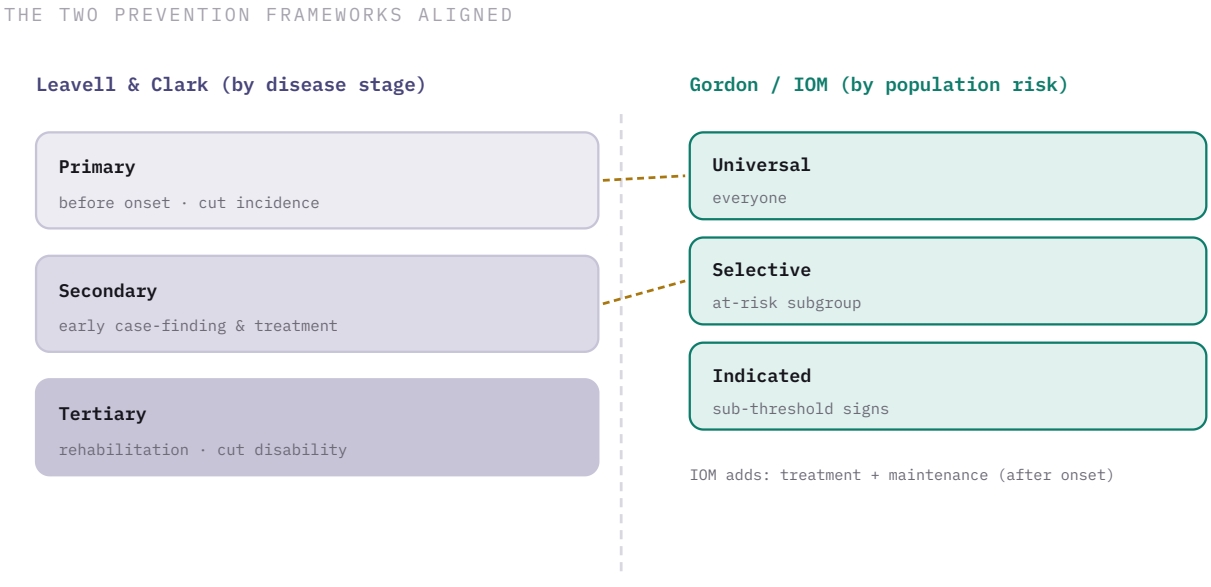
- **Least restrictive alternative** – treat in the setting that imposes the least limit on liberty consistent with safety and benefit. A founding ethic of community care, later a statutory right in the 2017 Act.

The two prevention frameworks (centrepiece)

Two classifications sit side by side and examiners love to compare them. The older **Leavell & Clark** public-health model keys prevention to the *stage of disease*; the newer **Gordon / IOM (Mrazek & Haggerty 1994)** model keys it to the *target population's risk*. The IOM model deliberately confines "prevention" to *before onset*.

Level	Timing vs disease	Aim	Mental-health example
Primary	before onset	reduce *incidence*: remove risk, build resilience	school life-skills programmes; reducing access to pesticides for suicide; perinatal support
Secondary	early disease	reduce *prevalence*: early detection & prompt treatment	screening for depression in primary care; early-intervention-in-psychosis services
Tertiary	established disease	reduce *disability* & relapse: rehabilitation	psychosocial rehabilitation, supported employment, relapse-prevention clinics

Gordon / IOM tier	Who is targeted	Mental-health example
Universal	the whole population, regardless of risk	nationwide anti-stigma / mental-health-literacy campaign; school SEL for all pupils
Selective	a subgroup at *above-average* risk	support for children of parents with mental illness; programmes for disaster survivors or bereaved
Indicated	high-risk individuals with *sub-threshold* signs	intervention for those with attenuated psychotic symptoms (CHR / ultra-high-risk clinics)



The classic three levels map roughly onto the IOM tiers, but the fit is loose: all three IOM prevention tiers sit *before* onset (so they are all closest to "primary"), while the IOM model handles established illness under separate "treatment" and "maintenance" headings.

● TRAP

Do not equate Gordon's tiers with the primary/secondary/tertiary triad. **Universal, selective and indicated prevention all act before the disorder begins** (they sub-divide what Leavell-Clark calls primary prevention). The IOM places early treatment and relapse-prevention *outside* "prevention" altogether, under treatment and maintenance.

HOLD THIS

Leavell-Clark: **primary / secondary / tertiary** = before / early / established. Gordon-IOM: **universal / selective / indicated** = everyone / at-risk group / sub-threshold individual.

2 The burden and the treatment gap

The case for community psychiatry rests on a simple mismatch: mental disorders are common and disabling, yet most people who need care never get it. Closing that gap means moving care to where people already go.

The burden

◆ **Global burden** – mental and substance-use disorders are among the leading causes of **years lived with disability (YLD)** worldwide. Depression is consistently a top-ranked contributor to global disability. Mental disorders carry high *disability* but relatively low direct mortality, so they dominate YLDs more than deaths.

● **The treatment gap** – the proportion of people with a disorder who do *not* receive treatment. It is driven by scarce specialists, geographic and financial barriers, and stigma. India's specialist workforce is a fraction of the need (roughly under 1 psychiatrist per 100,000 population, far short of recommended levels).

Three responses to the gap

■ **Task-shifting / task-sharing** – redistributing tasks from scarce specialists to trained non-specialist health workers (medical officers, nurses, community health workers), with specialist supervision. The core operational principle of community mental health in low-resource settings; the evidence base behind mhGAP.

■ **Integration into primary care** – embedding mental-health detection and basic treatment in general primary care rather than running parallel vertical services. Improves access, reduces stigma, addresses physical-mental comorbidity, and is affordable. A WHO core recommendation and the spine of the DMHP.

● **Social determinants of mental health** – the conditions in which people are born, grow, work and age that shape mental-health risk: **poverty, education, employment, housing, gender, violence and discrimination, social isolation, and adverse childhood experiences**. A vicious cycle runs both ways – poverty raises mental-illness risk, and mental illness deepens poverty (social drift / causation).

● RULE

"No health without mental health." Because determinants are largely social, the most powerful preventive levers lie *outside* the clinic – in schools, workplaces, welfare and law. This is why community psychiatry is intersectoral by design, not a branch of hospital medicine.

3 National Mental Health Programme (NMHP, 1982)

India launched the NMHP in **1982**, one of the earliest national mental-health programmes in the developing world. It grew out of the realisation that institution-based psychiatry could never reach a population of India's size, and was shaped by the ICMR / NIMHANS community-care experiments of the 1970s–80s.

The three core objectives

- **1. Availability & accessibility** – ensure the **availability and accessibility of minimum mental healthcare for all**, in the foreseeable future, particularly to the most vulnerable and underprivileged.
- **2. Knowledge into general care** – encourage the **application of mental-health knowledge in general healthcare and in social development**. Mental health is woven into general health services, not run as a separate silo.
- **3. Community participation** – promote **community participation in mental-health service development** and stimulate efforts toward *self-help* in the community.

MNEMONIC

"Access, Apply, Participate"

The three NMHP objectives: **Access** to minimum care for all · **Apply** mental-health knowledge in general health & social development · community **Participation** and self-help.

TRAP

The NMHP is the *national* umbrella programme (1982). Its flagship field arm is the **DMHP** (1996). Do not confuse the two: NMHP sets the objectives and adds non-DMHP components (upgrading psychiatric wings of medical colleges, modernising mental hospitals, manpower development through the Centres of Excellence scheme). The DMHP is the district-level delivery vehicle *within* the NMHP.

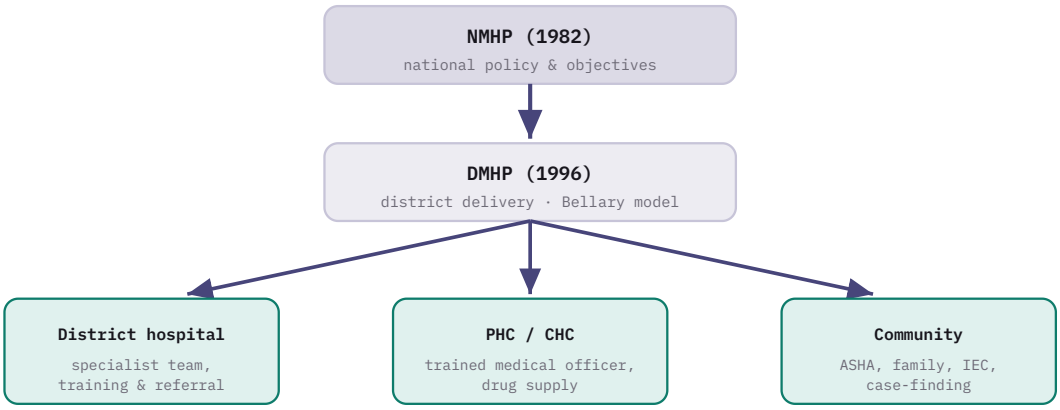
4 District Mental Health Programme (DMHP, 1996)

The DMHP was launched in **1996**, based on the **Bellary model** – a NIMHANS-led demonstration in Bellary district, Karnataka, showing that mental-health care could be delivered through the existing general-health system at the primary-care level. It is the operational engine that takes NMHP from policy to the PHC.

The four classic components

Component	What it means on the ground
Training	train primary-care **medical staff** (medical officers) and **non-medical staff** (health workers, nurses, ANMs) to detect and manage common mental disorders – the task-shifting step
Service delivery	identification, treatment and follow-up of cases at the **district hospital and PHC level**, with referral and outreach clinics; psychotropic drug supply to peripheral facilities
IEC / public awareness	Information, Education & Communication activities to raise awareness, reduce stigma and encourage help-seeking in the community
Monitoring & evaluation	record-keeping, reporting and supervision to track coverage and outcomes and to guide programme correction

HOW NMHP, DMHP AND THE PHC FIT TOGETHER



NMHP sets the national policy; DMHP carries it into each district, anchored at the district hospital and pushed down through PHCs into the community via trained staff, ASHAs and families.

Scale-up and evolution

- **Expansion** – the DMHP began as a pilot in a handful of districts and was progressively scaled up across hundreds of districts over successive Five-Year Plans, though coverage of all districts and consistent quality remain incomplete.
- **Broadened components** – later iterations (notably from the 12th Plan) added life-skills education and counselling in schools, college counselling, workplace stress management, suicide-prevention services, and linkages with other sectors – widening DMHP from clinical care toward promotion and prevention.

TRAP

Persistent implementation gaps are examinable as "challenges of the DMHP": shortage of trained manpower, irregular drug supply, weak monitoring, under-utilisation of funds, vertical (non-integrated) running, high attrition of trained staff, and uneven inter-state coverage. The programme's *design* is sound; *delivery* is the recurring weakness.

5 Policy & evidence: mhGAP, the 2017 Act, NMHS

WHO mhGAP

- **mhGAP (2008)** – the WHO **Mental Health Gap Action Programme**, launched to scale up services for mental, neurological and substance-use (MNS) conditions in low- and middle-income countries by closing the treatment gap.
- **mhGAP Intervention Guide (mhGAP-IG, 2010; v2.0 2016)** – a simplified, evidence-based decision-support tool that lets **non-specialist health workers** in primary and general care assess and manage priority MNS conditions (depression, psychoses, epilepsy, dementia, substance use, self-harm/suicide, child & adolescent and other significant emotional or medically unexplained complaints). The clinical embodiment of task-shifting.

Mental Healthcare Act, 2017

- **Rights-based, service mandate** – replaced the Mental Health Act 1987 and reframed care around the person's rights. It establishes a **legal right to access mental healthcare** run or funded by the government, and obliges the state to provide services that are *available, accessible, affordable and of good quality* – a statutory backing for community-level care.
- **Key community-relevant provisions** – the **advance directive** and **nominated representative**; decriminalisation of suicide (a person who attempts suicide is presumed to be under severe stress and is not punished, with a duty of care to provide treatment); a least-restrictive-care principle; and capacity-based admission procedures.

National Mental Health Survey of India 2015–16

- **NMHS 2015–16** – the landmark NIMHANS-led survey across 12 states. Headline figures: a **current prevalence of mental morbidity of about 10%** and a **lifetime prevalence of roughly 13–14%** (nearly 1 in 7–10 adults affected), with large urban-metro burden.
- **The treatment gap** – the survey's signature finding: a treatment gap of roughly **70 to 90 percent** across disorders (lowest for psychoses, around 75%, highest for common mental disorders and alcohol use, approaching or exceeding 85–90%). This single number is the strongest exam justification for community psychiatry.

QUICK RULE

NMHS 2015–16: current morbidity **~10%**, lifetime **~13–14%**, treatment gap **70–90%**. mhGAP-IG = the tool that lets non-specialists deliver the care that closes that gap.

6 Models & human resources for community care

Service models

Model	Core idea	Best for
Assertive community treatment (ACT)	a **multidisciplinary team** delivers care in the patient's own setting, with shared caseloads, 24-hour availability and assertive outreach	severe, persistent illness with frequent relapse & poor engagement; reduces hospitalisation
Crisis resolution / home treatment	rapid-response team manages acute crises at home as an **alternative to admission**	acute episodes that would otherwise need inpatient care
Case management	a single coordinator links the patient to needed services and follows up	complex needs spread across multiple agencies
Collaborative / stepped care	primary-care treatment with specialist supervision; intensity *stepped up* only if response is poor	common mental disorders integrated into primary care
Psychosocial rehabilitation	restores function and social role: skills training, supported employment, day care, halfway homes	tertiary prevention; long-term disability

● KEY NUMBER

ACT (the Madison / Stein & Test PACT model) is defined by a low staff-to-patient ratio (commonly around **1:10**), shared rather than individual caseloads, and an explicitly *assertive*, in-vivo style. Its strongest, most replicated effect is reduced inpatient bed-days.

Human resources: the people who deliver it

- **Primary-care integration** – the medical officer and PHC team, trained via DMHP / mhGAP, are the front line. Most common mental disorders can be managed here with specialist back-up.

- **The ASHA worker** – the **Accredited Social Health Activist**, India's village-level community health worker under the NHM. One ASHA per roughly 1,000 population. In mental health she is the key link for **case identification, awareness, follow-up, adherence support and reducing default** – the community anchor of task-shifting.
- **The family** – in India the family is the principal caregiver and the default unit of community care. **Psychoeducation, reducing expressed emotion (EE), and family support** are central; high EE (criticism, hostility, over-involvement) predicts relapse in schizophrenia.
- **NGOs & self-help** – non-governmental organisations (for example The Banyan, SCARF, Sangath, Basic Needs) pioneer models of rehabilitation, homeless care, lay-counsellor delivery and demonstration projects later absorbed into policy. User and carer self-help groups embody the NMHP's third objective.

Where India's model leans

Family-centred, primary-care-integrated, ASHA-anchored, NGO-innovated, low-cost task-shifting at population scale. Strength: reach and cultural fit.

Where high-income models lean

Specialist multidisciplinary teams (ACT, crisis teams), formal case management, statutory community treatment. Strength: intensity for severe illness; cost: workforce-heavy.

7 Special community topics

School & workplace mental health

- **School mental health** – schools reach children before disorders entrench, making them prime sites for *universal* prevention: life-skills education, social-emotional learning, anti-bullying and counselling. In India this runs through DMHP school components and the Ayushman Bharat / school-health initiatives.
- **Workplace mental health** – stress management, employee assistance programmes, and reasonable accommodation. Adults spend much of their lives at work, so the workplace is a major venue for both prevention and early detection.

Suicide prevention

- **National Suicide Prevention Strategy (2022)** – India's first such strategy, aligned with WHO's **LIVE LIFE** approach. Time-bound targets include reducing suicide mortality by **10% by 2030**. Pillars: effective surveillance, restricting access to means (notably **pesticide regulation**, the single most effective population lever in India), responsible media reporting, mental-health support in schools, and strengthening services.

KEY NUMBER

WHO LIVE LIFE = four evidence-based interventions: **L**imit access to means · **I**nteract with the media for responsible reporting · **V** = foster socio-emotional life skills in adolescents · **E**arly identify, assess, manage and follow up anyone affected by suicidal behaviour. Means restriction has the strongest evidence; pesticide bans have measurably cut national suicide rates in several countries.

Disaster mental health

- **Disaster mental health** – phased response: acute (**Psychological First Aid**: look, listen, link – *not* formal debriefing, which can harm), then screening and stepped care for those with persisting distress, PTSD, depression and grief. Build on community structures; integrate into general relief; the IASC guidelines stress a layered pyramid from basic services up to specialist care.

● TRAP

Single-session psychological **debriefing** (CISD) for all survivors is *not* recommended – trials show no benefit and possible harm. The endorsed early intervention is **Psychological First Aid**, which is supportive and non-intrusive, not a structured re-telling of the trauma.

Digital & tele-mental health

■ **Tele-MANAS (2022)** – India's national **tele-mental-health** programme (Mental Health Assistance – National Tele Mental Health Programme), launched under the 2022–23 budget. A 24x7 toll-free helpline (**14416** / 1-800-891-4416) with a two-tier structure: tier-1 state cells give counselling and triage; tier-2 links to specialists, DMHP and e-Sanjeevani for video consultation. Greatly extends reach beyond the scarce specialist workforce.

● **Digital mental health more broadly** – helplines, tele-psychiatry, apps and internet-delivered therapy expand access and lower stigma, but raise concerns over the digital divide, privacy, quality and the need for human escalation pathways.

HOLD THIS

Suicide: National Strategy 2022, target **–10% by 2030**, means restriction (pesticides) strongest lever. Disaster: **PFA, not debriefing**. Digital: **Tele-MANAS**, helpline **14416**, since 2022.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Prevention (Leavell-Clark):** primary = before onset (cut incidence) · secondary = early detection · tertiary = rehabilitation (cut disability).
- **Prevention (Gordon / IOM 1994):** universal = everyone · selective = at-risk subgroup · indicated = sub-threshold individual. All three are *pre-onset*.
- **Gap responses:** task-shifting · integration into primary care · act on social determinants (poverty, gender, ACEs, isolation).
- **NMHP 1982 – three objectives:** access to minimum care for all · apply mental-health knowledge in general health & social development · community participation & self-help.
- **DMHP 1996 (Bellary model) – four components:** training · service delivery · IEC · monitoring. DMHP is the field arm of NMHP.
- **mhGAP (2008) & mhGAP-IG (2010/2016):** WHO tool letting non-specialists manage priority MNS disorders. The clinical face of task-shifting.
- **MHCA 2017:** rights-based; legal right to access state-funded care; advance directive, nominated representative, decriminalised suicide attempt, least-restrictive care.
- ★ **NMHS 2015–16:** current morbidity ~10%, lifetime ~13–14%, treatment gap **70–90%**.
- **Models:** ACT (assertive outreach, ~1:10, cuts bed-days) · crisis/home treatment · stepped/collaborative care · psychosocial rehabilitation.
- **People:** trained PHC medical officer · ASHA (case-finding, follow-up, adherence) · family (psychoeducation, lower EE) · NGOs (Banyan, SCARF, Sangath).
- **Special topics:** school SEL · workplace EAPs · National Suicide Prevention Strategy 2022 (means restriction, –10% by 2030, WHO LIVE LIFE) · disaster = PFA not debriefing · Tele-MANAS 2022 (14416).

ONE-DAY REVISION · PAPER 3

Ethics in psychiatry, made retrievable

Psychiatry sits where autonomy is most contested and coercion is most available, so its ethics are examined harder than any other specialty. Hold the four principles, the limits of confidentiality, the elements of valid consent, and the research codes with the abuse that produced each. Every comparison here is a written-answer in waiting.

CONCEPT · RULE · TRAP · KEY FACT · MNEMONIC

The colour tells you what kind of fact it is. Indian statute (MHCA 2017) and the capacity test cross-link to the Forensic pack.

01 The four principles & the theories beneath · **02** Confidentiality & its limits · **03** Informed consent · **04** Capacity & competence · **05** Boundaries & the therapeutic relationship · **06** Coercion & the ethics of treatment · **07** Research ethics

★ HIGH YIELD TOP 5 IF NOTHING ELSE

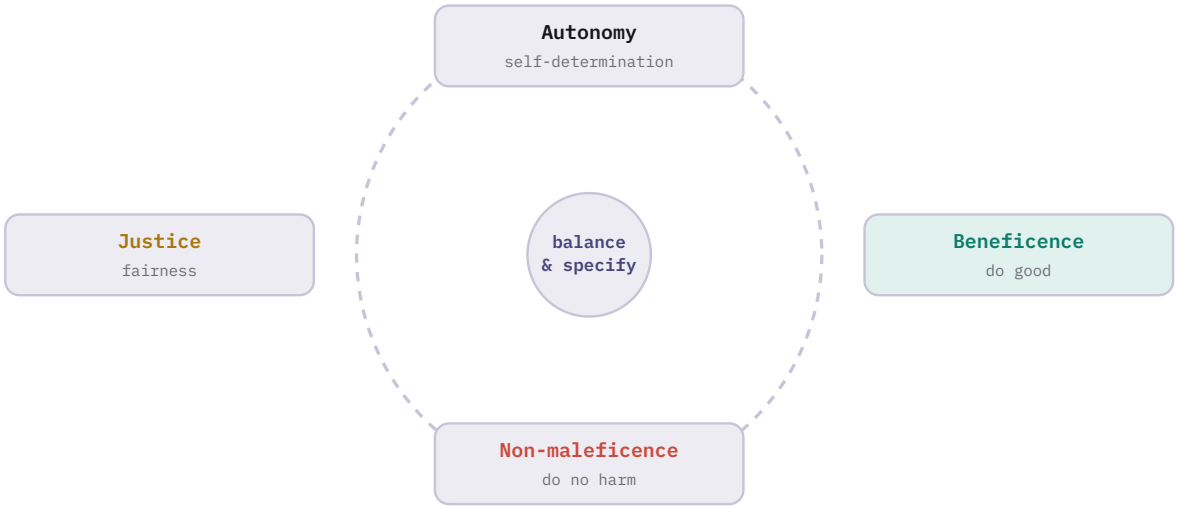
1. Capacity is decision- and time-specific, tested by four abilities (URWC: Understand, Retain, Weigh, Communicate); failing any one owing to impairment of mind or brain means incapacity, while an unwise choice with all four intact is still binding.
2. The four principles (autonomy, beneficence, non-maleficence, justice) are prima facie and balanced not ranked; the recurring exam tension is autonomy versus beneficence, resolved through capacity, not by overriding refusal.
3. Tarasoff (1976) created a duty to protect an identifiable victim from a serious threat (1974 warn → 1976 protect); vague non-specific anger does not trigger it.
4. Valid consent needs all three of CIV (Capacity, Information, Voluntariness); disclosure follows the Montgomery (2015) material-risk standard, not the older Bolam professional standard, and consent is withdrawable at any time.
5. Each research code answers a specific abuse: Nazi experiments → Nuremberg Code (1947, voluntary consent absolutely essential); Tuskegee → Belmont Report (1979); WMA → Declaration of Helsinki (1964).

1 The four principles & the theories beneath

Beauchamp and Childress reduced clinical ethics to four mid-level principles. They are **prima facie** binding (each holds unless it conflicts with a stronger one); none is absolute, and the work of an ethics answer is showing how they are **balanced and specified** in the case at hand.

Principle	What it demands	Psychiatric example
Autonomy	respect the patient's right to make their own informed choice; the duty to seek consent and tell the truth	a patient with capacity declines an antipsychotic; their refusal is honoured even if unwise
Beneficence	act in the patient's best interest; promote their welfare	starting clozapine for treatment-resistant schizophrenia to relieve suffering and risk
Non-maleficence	*primum non nocere*; do not inflict avoidable harm; weigh risk against benefit	monitoring lithium levels and renal function to avoid toxicity; avoiding polypharmacy
Justice	fairness in distributing benefits, risks and resources; treat like cases alike	equitable access to scarce inpatient beds; not de-prioritising care for the mentally ill

THE FOUR PRINCIPLES, BALANCED NOT RANKED



No principle outranks the others by default. In a real case they are weighed against one another (balanced) and made concrete for the situation (specified). Conflict between autonomy and beneficence is the engine of most psychiatric ethics questions.

◆ **The recurring tension** – **autonomy versus beneficence**. The paternalistic instinct to act for a patient's good collides with the duty to respect their refusal. Psychiatry resolves it through **capacity**: a capacitous refusal is binding; an incapacitous one is overridden in the patient's best interests.

The major ethical theories in brief

Theory	Right action is judged by…	Tag
Deontology (Kant)	duties and rules; some acts are wrong in themselves whatever the outcome. The **categorical imperative**: act only on a rule you could will to be universal; treat persons as ends, never merely as means	duty-based
Consequentialism / utilitarianism (Bentham, Mill)	outcomes alone; the right act maximises overall good (the greatest good for the greatest number)	outcome-based
Virtue ethics (Aristotle)	the character of the agent, not the act or its result; what would a person of practical wisdom (*phronesis*) do?	character-based
Principlism (Beauchamp & Childress)	balancing the four prima facie principles; the working framework of clinical ethics	framework

● MNEMONIC

"Duties, Outcomes, Character"

Deontology = the act and its duty · consequentialism = the **O**utcome · virtue ethics = the agent's **C**haracter. Principlism is the bridge clinicians actually use.

● CLASSIC TRAP

A **deontologist** will not lie even to produce a better outcome (truth-telling is a duty); a **utilitarian** may, if the consequences are better on balance. The questioner often hides this in a vignette about breaking confidentiality "for the greater good" – name the theory each stance reflects.

2 Confidentiality & its limits

Confidentiality is a **prima facie** duty rooted in autonomy and trust, not an absolute. It is the precondition for honest disclosure in psychiatry. It yields only to a stronger competing duty, and only as far as necessary.

When confidentiality may or must be breached

Ground	Trigger	Note
Patient consent	the patient agrees to disclosure	not a true breach; the cleanest route, always preferred
Serious risk to others	credible threat of serious harm to an identifiable person	the **Tarasoff** situation; duty to protect
Serious risk to self	imminent suicide or grave self-neglect	disclose to enable protection (family, crisis team)
Statutory / mandatory reporting	child abuse, certain notifiable diseases, some firearm/violence laws	the law compels disclosure; consent is not required
Court order	a judge orders the record or testimony	disclose only what the order specifies; assert privilege first where it exists
Public interest	preventing or detecting a serious crime; fitness to drive (impaired)	proportionate disclosure to the proper authority

QUICK RULE

A breach is justified only when it is **necessary**, **proportionate** (disclose the minimum), and directed to **someone who can act**. Tell the patient you are disclosing, and why, unless doing so would itself cause harm.

Tarasoff: the duty to warn and protect

◆ **Tarasoff v. Regents** (California, 1976) – a therapist whose patient voices a serious threat against an **identifiable victim** has a duty to take reasonable steps to protect that person. The court's phrase: *"the protective privilege ends where the public peril begins."*

■ **Warn vs protect** – the 1974 ruling was a duty to **warn**; the 1976 re-hearing broadened it to a duty to **protect**, which may be discharged by warning the victim, notifying police, or hospitalising the patient. Persuasive in many jurisdictions but not binding everywhere.

● TRAP

Tarasoff requires a **serious threat to an identifiable person**. Vague, non-specific anger does not trigger it. And it is a *permission and duty* to act, not a licence to broadcast: disclose only what protects the target, to those who can protect them.

Confidentiality with minors

● **The general rule** – a minor is owed confidentiality, but parents/guardians ordinarily share in decisions and information. A mature minor's wish for privacy is weighed against welfare and statute.

▲ **The override** – confidentiality is breached to a parent or authority when the minor faces **serious risk** (suicidality, abuse, exploitation). Safeguarding outranks the adolescent's request for secrecy.

3 Informed consent

Valid consent is not a signature; it is a process. Three elements must all be present, and the absence of any one invalidates it.

Element	Means	Fails when…
Capacity	the patient can understand, retain, weigh and communicate the decision	delirium, severe psychosis, advanced dementia removes one of these abilities
Information / disclosure	the nature, purpose, material risks, benefits and alternatives (including no treatment) are explained	risks are withheld or the explanation is not understood
Voluntariness	the choice is free of coercion or undue influence	pressure from staff, family, or the leverage of detention distorts it

MNEMONIC

"Consent needs a CIV mind"

Capacity · **I**nformation · **V**oluntariness. Drop any one and the consent is void.

Standard of disclosure – the modern test is patient-centred: disclose the **material risks** a reasonable patient in this position would want to know, plus any this particular patient flags (the *Montgomery* standard, 2015), not merely what a body of doctors would disclose (the older *Bolam* professional standard).

When the patient lacks capacity

- **Best-interests standard** – where the patient cannot consent, treatment proceeds on their **best interests**, weighing their past wishes, values, beliefs and the views of those close to them. Use the **least restrictive** option that meets the need.
- **Advance directive** – a decision made *while capacitous* about future care. An **advance refusal** of a specified treatment, if valid and applicable, binds; an **advance statement** of wishes guides but does not bind. Under the MHCA 2017 a person may also nominate a **nominated representative**.
- **Surrogate / substituted decision-making** – a proxy decides for the incapacitated patient. Two logics: **substituted judgement** (decide as the patient would have) and, where their prior wishes are unknown, **best interests**.

TRAP

Consent is **decision- and time-specific** and may be **withdrawn at any time**. A signed form for one procedure does not consent to another, and consent given last week can be revoked today. Emergencies permit life-saving treatment without consent under the doctrine of necessity, limited to what the emergency requires.

4 Capacity & competence

Capacity is the gatekeeper of autonomy. Get the functional test and its four limbs exact – this is examined verbatim and cross-links to the Forensic pack.

The functional test: four abilities

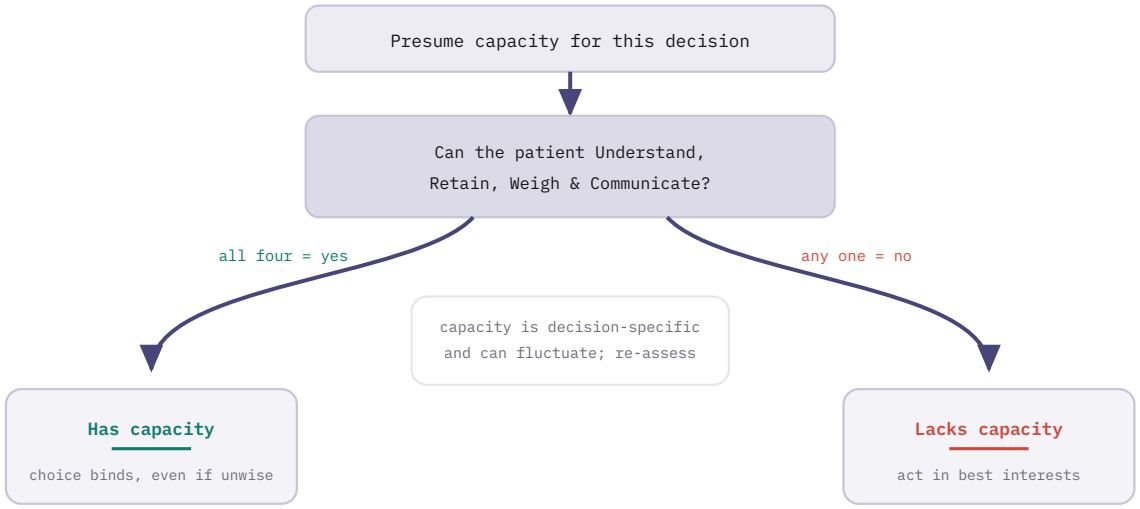
Ability	The patient can…
Understand	take in and comprehend the relevant information
Retain	hold it long enough to use it (even briefly is enough)
Weigh / use	use and weigh the information in arriving at a choice
Communicate	express the decision (by any means)

● MNEMONIC

"URWC" → Understand, Retain, Weigh, Communicate

All four are needed. Failing **any one**, because of an impairment of mind or brain, means the patient lacks capacity for *that* decision.

ASSESSING CAPACITY FOR A SPECIFIC DECISION



Start from the presumption of capacity. Test the four abilities for this specific decision at this time. All four present means the choice stands, however unwise. Any one absent, owing to an impairment of mind or brain, means treat on best interests.

The three anchoring rules

- **Presumption of capacity** – every adult is **presumed to have capacity** until shown otherwise. The burden is on the assessor to prove its absence, not on the patient to prove its presence.
- **Decision-specific** – capacity is judged **for a particular decision at a particular time**, not as a global on/off trait. A person may have capacity to choose a meal but not to consent to surgery, and capacity may **fluctuate** (delirium, mania).

▲ **An unwise decision is not incapacity** – a capacitous patient may make a choice others think foolish. The test is the *process* of deciding, never the wisdom of the outcome.

● CAPACITY VS COMPETENCE

Capacity is the *clinical* judgement a doctor makes at the bedside about a specific decision now. **Competence** is the *legal* status a court determines (for example, fitness to stand trial, to manage affairs). A clinician assesses capacity; only a court declares incompetence. In everyday use the words are blurred, but the exam wants the clinical/legal split.

5 Boundaries & the therapeutic relationship

The clinical relationship carries an inherent **power asymmetry** and intense transference. Boundaries protect the patient from exploitation of that asymmetry. The exam wants the crossing/violation distinction and the bright line on sexual contact.

Boundary crossing

A minor, often benign deviation from the usual frame that may even serve the therapy: a self-disclosure to normalise, accepting a token gift, a home visit for a housebound patient. Judged by **context and intent**; reversible; discussable in supervision.

Boundary violation

A transgression that **exploits** the patient and serves the clinician's needs: a sexual or financial relationship, a business deal, progressive erosion of the frame. Harmful, non-discussable, and a disciplinary matter.

▲ **Sexual contact with a patient** – an **absolute prohibition**. It is never therapeutic, never consensual in any meaningful sense given the power gradient, and is grounds for deregistration. It applies regardless of who initiated it and persists after the formal end of treatment (especially in psychotherapy, where transference endures).

● **Dual relationships** – holding a second role with the patient (treating a friend, relative, employee, or trading services). They blur judgement and are avoided where possible; unavoidable in small communities, where they are managed with transparency and supervision, never denied.

● **Gifts** – a small token may be accepted graciously; a valuable gift raises questions of meaning, influence and the patient's expectations, and is explored rather than simply accepted or refused.

● **Self-disclosure** – brief, purposeful disclosure can build alliance; disclosure that meets the *clinician's* needs (unburdening, seeking comfort) inverts the relationship and is a warning sign.

◆ **Transference & countertransference as ethical risk** – the patient's transferred feelings and the clinician's **countertransference** are clinical data, but unexamined countertransference (rescue fantasies, attraction, hostility) is how violations begin. The ethical duty is to **recognise, supervise and contain** these feelings, never to enact them.

● TRAP

A crossing is not automatically a violation, and a violation rarely begins as one. The danger sign is a **pattern**: secrecy from colleagues, exceptions made for one patient, sessions that run long or move out of hours, and the clinician's own needs creeping into the room. Supervision exists precisely to catch this slide early.

6 Coercion & the ethics of treatment

Psychiatry alone can detain and treat against a person's will. That power is what makes its ethics distinctive, and why a rights-based legal frame governs it.

Involuntary admission & treatment

● **Justification** – involuntary care is ethically defensible only when a **mental disorder** causes a serious **risk to self or others** (or grave health deterioration) *and* the person cannot or will not accept care voluntarily. It rests on beneficence and protection, constrained by due process.

◆ **Least-restrictive-alternative principle** – use the **least restrictive** setting and intervention that will meet the need: community before ward, voluntary before involuntary, the shortest duration, the smallest necessary limit on liberty. The MHCA 2017 makes this a statutory duty.

Seclusion, restraint & ECT

▲ **Seclusion & restraint** – emergency measures of **last resort** to prevent imminent harm, never punishment, convenience or a substitute for staffing. Time-limited, documented, monitored, and reviewed. The MHCA 2017 **prohibits chaining** and restricts seclusion/restraint to immediate prevention of harm.

◆ **Consent for ECT** – informed consent is the rule; capacity to consent to ECT is assessed specifically. The MHCA 2017 **bans unmodified ECT** (always with anaesthesia and muscle relaxant), **bans ECT in minors** save with extra safeguards (Board permission), and requires safeguards for emergency ECT without consent.

The MHCA 2017: a rights-based frame

● **The shift** – the Mental Healthcare Act 2017 (India) moved from a custodial model to a **rights-based** one, aligned with the UN CRPD. It grants a **right to access mental healthcare**, advance directives, a nominated representative, confidentiality, community living, and protection from cruel or degrading treatment.

◆ **Decriminalising suicide** – the Act presumes a person who attempts suicide is under **severe stress** and is **not punished** under the penal code, mandating care and rehabilitation instead. Capacity to make mental-health decisions is presumed.

● **Stigma** – ethically, stigma is a justice and non-maleficence issue: it deters help-seeking, justifies neglect, and shapes coercive over-reach. Reducing it (parity of esteem, anti-discrimination, dignified language) is part of the psychiatrist's duty, not an optional extra.

● RULE

Coercion is ethical only when it is **necessary, proportionate, least-restrictive, time-limited and reviewable**. The moment any of these fails, the intervention shifts from protection to a rights violation. The MHCA 2017 encodes each of these as an enforceable safeguard.

7 Research ethics

Every modern research safeguard was written in response to a specific abuse. Pair the code with the scandal that produced it – that pairing is the high-yield answer.

Abuse	What happened	Produced
Nazi experiments	lethal, non-consensual experiments on prisoners; exposed at the Nuremberg doctors' trial	Nuremberg Code (1947) – voluntary consent is "absolutely essential"
Tuskegee (1932–72)	Black men with syphilis left untreated for decades to study the disease, even after penicillin existed; deceived about their care	Belmont Report (1979) & the US common rule; IRBs
Evolving consensus	the medical profession's own standard for human research	Declaration of Helsinki (WMA, 1964, revised)

The Belmont Report: three principles

● **Respect for persons** – treat people as autonomous agents and protect those with **diminished autonomy**. Operationalised as **informed consent**.

● **Beneficence** – maximise benefits, minimise harms. Operationalised as **risk-benefit assessment**.

● **Justice** – distribute the burdens and benefits of research fairly; do not exploit the vulnerable for others' gain. Operationalised as fair **subject selection**.

● MNEMONIC

"Persons, Beneficence, Justice" map to "Consent, Risk, Selection"

Belmont's three principles each have a procedural twin: respect → informed consent · beneficence → risk-benefit · justice → fair selection.

Consent, oversight & the vulnerable

■ **Informed consent in research** – voluntary, informed, documented, and revocable. It must distinguish research from treatment (countering the **therapeutic misconception**, the patient's belief that a trial is individualised care).

● **Ethics committee (IEC / IRB)** – an independent **institutional ethics committee** (India) or institutional review board reviews protocols before they begin and monitors them, weighing scientific merit, risk-benefit, consent and the protection of participants. In India, registration with the relevant national authority is required.

▲ **Vulnerable populations** – groups with limited autonomy or liable to coercion: people with severe mental illness, children, prisoners, the cognitively impaired. They need **extra safeguards** (surrogate consent, independent oversight, justification that the research could not be done in a less vulnerable group), not exclusion that would deny them research benefits.

Placebo controls in psychiatric trials

◆ **The placebo problem** – a placebo arm yields the cleanest evidence but withholds active treatment. The Declaration of Helsinki permits placebo only when there is **no proven effective treatment**, or when its use is **methodologically necessary** and patients on placebo face **no serious or irreversible harm**.

● **In psychiatry specifically** – the sharp issue is risk: trials in suicidal depression or acute psychosis raise the stakes of assigning placebo. Mitigations include rescue criteria, close monitoring, brief placebo exposure, add-on rather than monotherapy designs, and active-comparator (non-inferiority) trials where an effective treatment already exists.

● TRAP

Placebo is not automatically unethical; blanket use of it where an effective treatment exists is. The test is the Helsinki one: **no proven therapy, or compelling methodology with no risk of serious harm.** A placebo arm in mild illness with rescue rules can be ethical; one in acutely suicidal patients usually is not.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Four principles:** autonomy, beneficence, non-maleficence, justice – all prima facie, balanced not ranked (Beauchamp & Childress). The core tension is autonomy vs beneficence, resolved through capacity.
- **Theories:** deontology = duties (Kant, categorical imperative) · consequentialism/utilitarianism = outcomes (Mill) · virtue ethics = character (Aristotle) · principlism = the four-principle framework.
- **Confidentiality:** a prima facie duty, breached only when necessary, proportionate, and to someone who can act – consent, risk to others (Tarasoff), risk to self, mandatory reporting, court order, public interest.
- **Tarasoff (1976):** duty to protect an identifiable victim from a serious threat. "The protective privilege ends where the public peril begins." 1974 warn → 1976 protect.
- **Consent = CIV:** Capacity + Information (material risks, Montgomery) + Voluntariness. Decision- and time-specific; withdrawable anytime; emergencies use the doctrine of necessity.
- ★ **Capacity = URWC:** Understand, Retain, Weigh, Communicate. Presumed; decision-specific; can fluctuate; an unwise choice is not incapacity. Capacity = clinical, competence = legal (court).
- **Boundaries:** crossing (minor, may help) vs violation (exploitative, harmful). Sexual contact with a patient = absolute prohibition. Watch unexamined countertransference.
- **Coercion:** involuntary care needs disorder + risk + refusal; least-restrictive alternative; seclusion/restraint as last resort. MHCA 2017 bans unmodified ECT, chaining, and decriminalises suicide.

- **Research codes:** Nazi experiments → Nuremberg Code (1947, voluntary consent essential) · Tuskegee → Belmont (1979) · WMA → Declaration of Helsinki (1964).
- **Belmont:** respect for persons → consent · beneficence → risk-benefit · justice → fair selection. IEC/IRB reviews; vulnerable groups need extra safeguards; placebo only if no proven therapy or no serious harm.