PART II · PAPER II

II

Clinical Psychiatry

Part II covers clinical psychiatry itself: how the disorders are classified, recognised, and worked up across the patients met on the ward.

ONE-DAY REVISION · PAPER 2

History of psychiatry, made retrievable

From temple incantation to the Mental Healthcare Act. The story is a single argument fought over and over: is madness a moral, supernatural matter, or a natural, treatable one? Hold the names, the one contribution attached to each, and the dates – especially the Indian legislative arc, which examiners love.

● ERA / FIGURE ● REFORM / RULE ● TRAP ● DATE TO MEMORISE ● MNEMONIC

The colour tells you what kind of fact it is. The somatic-therapy eponyms (Cade, Moniz, Delay-Deniker, Cerletti-Bini) live in fuller form in the companion Eponyms pack.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. Trap: Kraepelin coined **dementia praecox** (vs manic-depressive insanity); Bleuler renamed it "schizophrenia" in 1908 and gave the 4 A's; Schneider gave the first-rank symptoms. The most-swapped trio in vivas.
2. India's legislative arc: Lunacy Acts 1858 → **Indian Lunacy Act 1912** (governed ~75 yrs) → Mental Health Act 1987 (in force 1993) → Mental Healthcare Act 2017 (in force 2018); the 2017 Act decriminalised suicide attempts and brought advance directives plus a nominated representative.
3. The drug revolution dates: **Cade lithium 1949**, Delay & Deniker chlorpromazine 1952 (the date that ended the asylum era), Kuhn imipramine 1957.
4. Moral treatment: **Chiarugi** earliest (Florence 1788), **Pinel** "unchained the insane" (Paris 1793, with Pussin), **Tuke** York Retreat (1796), **Dix** US asylum campaign (1840s). Trap: Pinel is famous, but Chiarugi acted earlier and Pussin did much of the hands-on work.
5. Two Nobel prizes in somatic psychiatry: **Wagner-Jauregg** (malaria therapy for GPI, 1917; Nobel 1927) and **Moniz** (prefrontal leucotomy, 1935; Nobel 1949).

1 Ancient & classical roots

Two competing models run the whole length of the history. The **supernatural**: madness is possession, divine punishment or sin, treated by priests and exorcism. The **naturalistic**: madness is bodily disease, treated by physicians. The naturalistic strand begins, for the West, with Hippocrates.

Source	Model	Contribution
Mesopotamia / Egypt	supernatural	madness as demonic possession; incantation, exorcism, trephination
Hippocrates (c. 460–370 BC)	naturalistic	brain is the seat of thought and madness; coined **mania, melancholia, phrenitis, hysteria, epilepsy** ("the sacred disease" is no more sacred than any other)
Galen (129–c. 216 AD)	naturalistic	systematised humoral theory; Greco-Roman authority for ~1500 years
Asclepiades, Soranus, Aretaeus	naturalistic	humane Roman care; Aretaeus saw mania and melancholia as one illness (an early bipolar intuition)

Humoral theory: the four humours and temperaments

Health is the **balance** of four bodily fluids; disease, including madness, is their imbalance (dyscrasia). Each humour maps to an element, a quality pair, and a temperament.

Humour	Element / quality	Excess produces	Temperament
Blood	air · warm + moist	over-optimism, restlessness	Sanguine (cheerful, sociable)
Yellow bile	fire · warm + dry	irritability, mania	Choleric (irritable, driven)
Black bile	earth · cold + dry	sadness, fixed gloom	Melancholic (despondent)
Phlegm	water · cold + moist	apathy, sluggishness	Phlegmatic (calm, slow)

◆ **Melancholia** – literally *melan* (black) + *chole* (bile). An **excess of black bile** was held to cause depression. The word, and the humoral idea behind it, survived into the twentieth century and still names a depressive subtype.

● MNEMONIC

"Blood, bile, bile, phlegm → Sanguine, Choleric, Melancholic, Phlegmatic"

Yellow bile = choleric (hot temper). Black bile = melancholic (gloom). The four temperaments are the distant ancestor of trait and type theories of personality.

● TRAP

Hippocrates is the father of the **naturalistic** tradition, not of any single diagnosis. The humoral theory is usually credited to him but was **systematised by Galen**, whose authority froze Western medicine for over a millennium. Both the four humours and the "wandering womb" account of hysteria are Greek, pre-Galenic ideas.

2 Medieval to the asylum era

In the European Middle Ages the supernatural model returned with force. Madness was read as possession, witchcraft or divine wrath. Care, where it existed, was custodial. The Islamic world, meanwhile, kept the naturalistic tradition alive.

Demonology and the witch-hunts

● **Demonological model** – mental disturbance as possession or pact with the devil. Treatment was exorcism, prayer, sometimes torture or execution.

▲ **Malleus Maleficarum** (1487) – the "Hammer of Witches" by Kramer and Sprenger: a manual for identifying and prosecuting witches. Many of the accused were almost certainly mentally ill. The dissenting voice was **Johann Weyer** (1515–1588), who argued witches were sick women needing care, not punishment – often called the first to take a clinical stance.

Credit where due: the Islamic bimaristans

■ **Bimaristans** – the medieval Islamic world ran dedicated hospitals (*bimaristan*) with wards for the mentally ill from the eighth and ninth centuries, in **Baghdad, Cairo, Damascus and Fez**, centuries before Europe. **Al-Razi (Rhazes)** and **Ibn Sina (Avicenna)** wrote on mental illness as bodily disease. Humane care, music and occupation were used while Europe still burned the afflicted.

The first asylums

Asylum	Note	Date
Bethlem ("Bedlam"), London	founded as the Priory of St Mary of Bethlehem by Simon FitzMary; housed the insane by the late 14th-early 15th century; England's first and most infamous asylum ("bedlam" entered the language as a word for uproar)	1247
Valencia, Spain	an early dedicated asylum, founded by Father Jofre	1409

Asylum	Note	Date
Hopital General, Paris	"the great confinement": mad, poor and criminal warehoused together (Foucault's pivotal date)	1656

● TRAP

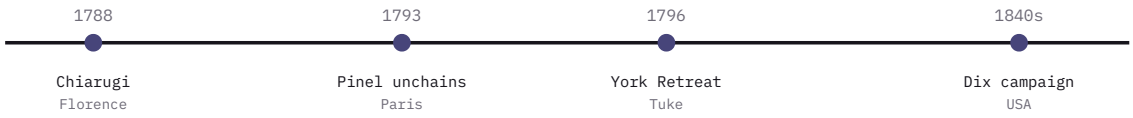
Bethlem was founded in 1247 as a priory; it did not begin as a psychiatric hospital. It became associated with the insane only over the following century, and only later a byword ("Bedlam") for chaos and the spectacle of paying visitors. Do not state that a purpose-built mental asylum existed in 1247.

3 Moral treatment & reform

The late eighteenth century turns the corner. Independently, in France, England and Italy, reformers replaced chains and cruelty with kindness, routine, work and dignity. This is **moral treatment** (*traitement moral*): the belief that humane environment and self-respect can restore reason.

Reformer	Where	Contribution	Date
Vincenzo Chiarugi	Florence, Italy	banned chains and championed humane regulations at the Bonifazio hospital; often cited as the *earliest* reformer	1788–89
Philippe Pinel	Paris (Bicetre, then Salpetriere)	"unchained the insane"; the founding act of moral treatment; with **Jean-Baptiste Pussin**, the lay superintendent who did much of the work	1793–95
William Tuke	York, England	Quaker; founded the **York Retreat**: a homelike, non-restraint regimen of work, kindness and routine	1796
Dorothea Dix	USA	campaigner; her surveys of jails and almshouses drove the building of dozens of state asylums	1840s–50s

MORAL TREATMENT AND THE REFORMERS



Moral treatment arose almost simultaneously: Chiarugi in Florence (1788), Pinel in Paris (1793), Tuke's York Retreat (1796), then Dix's American asylum-reform campaign (1840s).

■ Non-restraint movement – the next British step. **John Conolly** (Hanwell Asylum) and **Robert Gardiner Hill** (Lincoln) abolished mechanical restraint entirely in the 1830s–40s, extending Tuke's principle.

● MNEMONIC

"Chiarugi first, Pinel famous, Tuke gentle, Dix loud"

Chiarugi (Italy) is earliest; Pinel (France) gets the iconic image of striking off chains; Tuke (England) builds the homelike Retreat; Dix (USA) drives the asylum-building campaign by lobbying legislatures.

● CLASSIC TRAP

Pinel is the textbook "liberator," but **Chiarugi acted earlier** (Florence, 1788) and much of Pinel's hands-on reform was carried out by the lay superintendent **Jean-Baptiste Pussin** and his wife. The famous image of Pinel personally striking off the chains is partly later myth. Tuke's **York Retreat** (1796) is the English parallel, run by Quakers, not physicians.

4 Clinical psychiatry & the biological era

The nineteenth century turns madness into a **medical specialty**. German university psychiatry leads: careful observation, classification by course, and the conviction that mental disease is brain disease. (Fuller one-liners on each figure are in the Eponyms pack.)

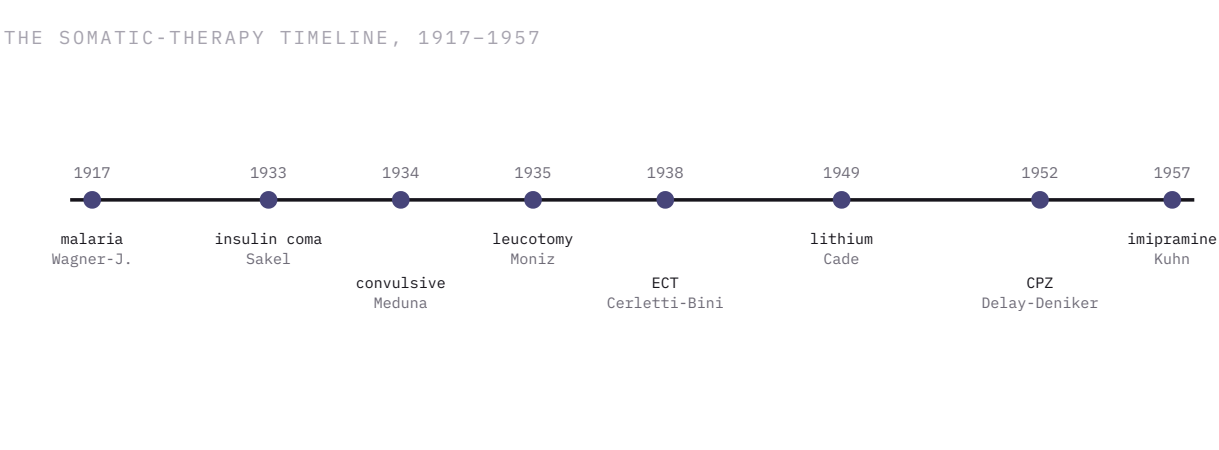
Figure	One-line claim to fame	Era
Wilhelm Griesinger	**"mental diseases are brain diseases"**; the slogan of biological psychiatry	1845
Karl Kahlbaum	described **catatonia**; classification by symptom clusters and course (with Hecker on hebephrenia)	1860s–70s
Emil Kraepelin	the great dichotomy: **dementia praecox vs manic-depressive insanity**; classify by course and outcome – the father of modern nosology	1890s–1900s
Eugen Bleuler	renamed dementia praecox **"schizophrenia"** (1908); the **4 A's**; coined *autism* and *ambivalence*	1908–11
Kurt Schneider	**first-rank symptoms** of schizophrenia; emphasis on form over content	1930s–50s
Sigmund Freud	founded **psychoanalysis**: the unconscious, defence, transference; the psychodynamic stream alongside the biological	1890s–1930s

● TRAP

Do **not** credit the word *schizophrenia* to Kraepelin. Kraepelin's term was **dementia praecox**; **Eugen Bleuler** renamed it. The 4 A's are Bleuler's; the first-rank symptoms are Schneider's. The most-swapped pair in vivas.

The somatic-therapy & psychopharmacology revolution

From the 1910s, psychiatry gained its first physical treatments, then in 1949–57 the drugs that emptied the asylums. Hold the order and the names.



The somatic era: fever (malaria), insulin and convulsive therapies (1917–1938), then the pharmacology revolution: lithium (1949), chlorpromazine (1952), imipramine (1957).

Treatment	Introduced by	Year
Malaria (fever) therapy for GPI	**Julius Wagner-Jauregg** – Nobel Prize 1927	1917
Insulin coma therapy	**Manfred Sakel**	1933
Convulsive therapy (cardiazol, chemical)	**Ladislas Meduna**	1934
Prefrontal leucotomy (lobotomy)	**Egas Moniz** – Nobel Prize 1949	1935–36
Electroconvulsive therapy (ECT)	**Ugo Cerletti & Lucio Bini** (electrical)	1938
Lithium for mania	**John Cade** (Australia)	1949
Chlorpromazine (first antipsychotic)	**Jean Delay & Pierre Deniker**; surgeon **Henri Laborit** noted its calming effect	1952
Imipramine (first tricyclic)	**Roland Kuhn**	1957

HOLD THIS

Two Nobel laureates: **Wagner-Jauregg** (malaria, 1927) and **Moniz** (leucotomy, 1949). The drug revolution: **lithium 1949** (Cade), **chlorpromazine 1952** (Delay & Deniker), **imipramine 1957** (Kuhn). Chlorpromazine 1952 is *the* date that ended the asylum era.

Deinstitutionalisation & community psychiatry

● **Deinstitutionalisation** – from the 1950s–60s, asylum populations fell sharply. Drivers: effective antipsychotics (after 1952), the civil-rights and antipsychiatry critique, cost, and a shift toward care in the community. In the USA the **Community Mental Health Centers Act (1963)**, signed by Kennedy, was the legislative turning point.

▲ **The trans-institutionalisation critique** – deinstitutionalisation without adequate community services produced homelessness, the "revolving door," and a drift of the mentally ill into prisons. A genuine reform with a well-documented dark side.

5 Antipsychiatry & the critics

In the 1960s a wave of thinkers attacked psychiatry's very premises: that mental illness is a disease, that diagnosis is objective, that the asylum heals. Know each name, their one book or experiment, and their central claim.

Critic	Key work	Central claim
Thomas Szasz	*The Myth of Mental Illness* (1961)	mental illness is a **metaphor**, not a literal disease; "problems in living" medicalised. Psychiatry as social control
R. D. Laing	*The Divided Self* (1960)	psychosis is an **intelligible response** to an unbearable situation/family; existential, not merely biological
Erving Goffman	*Asylums* (1961)	the asylum is a **total institution** that strips identity ("mortification of the self"); much patient behaviour is an artefact of confinement
Michel Foucault	*Madness and Civilization* (1961)	madness is a **social construction**; the "great confinement" (1656) silenced the mad rather than curing them
David Rosenhan	"On Being Sane in Insane Places" (1973)	the famous **pseudopatient experiment**: diagnosis cannot reliably tell the sane from the insane in context

CLASSIC TRAP

The **Rosenhan experiment** (1973): eight healthy "pseudopatients" reported a single symptom (hearing a voice saying "thud"), were admitted, then behaved normally. All were diagnosed (mostly schizophrenia) and discharged "in remission" – the label, once applied, coloured everything. A second study, where staff were warned to expect pseudopatients, generated false positives among genuine patients. It drove the move toward the **operationalised criteria** of DSM-III (1980). Note: the study's data integrity has since been seriously questioned.

MNEMONIC

"Szasz says Myth, Goffman builds the Total Institution, Foucault writes Civilization, Rosenhan goes undercover"

Szasz = the myth (illness isn't real disease). Laing = psychosis is meaningful. Goffman = the institution as the problem. Foucault = history/power. Rosenhan = the diagnostic experiment. 1960–61 is the cluster year for the books; 1973 for Rosenhan.

6 History of psychiatry in India

India's story has its own deep roots and its own legislative arc. Examiners reward the ancient sources, the right colonial dates, Vidya Sagar's family model, the two flagship institutions, and the 1987 → 2017 legislative shift.

Ancient roots

- **Atharvaveda** – the earliest references to mental disturbance and its supernatural causes (possession by spirits, *grahas*), with incantatory remedies. The supernatural strand of Indian tradition.
- **Ayurveda & the Charaka Samhita** – the naturalistic strand. **Unmada** (insanity) and **apasmara** (epilepsy/fainting) are described and classified. Mind governed by the three *doshas* (vata, pitta, kapha) and the mental qualities *sattva, rajas, tamas*. Treatment combined diet, herbs, lifestyle and *daivavyapashraya* (spiritual) measures. **Charaka** and **Sushruta** are the key names.

RULE

Pair them: **Atharvaveda** = supernatural/spirit-possession model; **Ayurveda / Charaka** = naturalistic, humoral (tridosha) model with **unmada** as the term for insanity. The same supernatural-vs-naturalistic split that runs through Western history runs through Indian history too.

Colonial asylums & the Lunacy Acts

Milestone	Note	Date
Early colonial asylums	established for Europeans and Indians (Bombay, Calcutta, Madras); custodial, segregative	late 18th–19th c.
Lunacy Acts of 1858	the first consolidated colonial legislation governing asylums and the certification of "lunatics"	1858
Indian Lunacy Act	the consolidating Act (modelled on the English Lunacy Act 1890); governed Indian psychiatry for ~75 years until 1987	1912

Vidya Sagar's family-participation model

- **Dr Vidya Sagar** (1909–1978) – at the **Amritsar Mental Hospital** in the 1950s, he housed families in **tents** on the hospital grounds and involved them directly in patient care. A pioneering, culturally rooted model of **family participation** and community psychiatry, decades ahead of its time and

distinctly Indian.

The flagship institutions

Institution	Note	Date
CIP Ranchi	Central Institute of Psychiatry; opened by the British as the Ranchi European Lunatic Asylum / European Mental Hospital; one of only two purpose-built asylums in British India; renamed CIP in 1977	1918
NIMHANS, Bangalore	roots in the 1847 Bangalore asylum; **All India Institute of Mental Health (AIIMH)** set up 1954; **NIMHANS formed by merging the State Mental Hospital and AIIMH** on 27 Dec 1974; later an Institute of National Importance	1954 / 1974

The Indian Psychiatric Society & the legislative arc

◆ **Indian Psychiatric Society (IPS)** – founded on **7 January 1947** in New Delhi (during the Indian Science Congress) with 15 founder members; later registered at Patna. The professional home of Indian psychiatry; publishes the *Indian Journal of Psychiatry*.

THE INDIAN LEGISLATIVE ARC

1858 Lunacy Acts → 1912 Indian Lunacy Act → 1987 Mental Health Act (in force 1993) → 2017 Mental Healthcare Act (in force 2018). Each step shifts from custody toward rights.

Act	What it did	Date
Mental Health Act	repealed the 1912 Lunacy Act; replaced "lunatic" with "mentally ill person"; introduced licensing of psychiatric hospitals and voluntary admission. Passed **22 May 1987**, brought into force **1993**	1987
Mental Healthcare Act	rights-based, aligned with the UN CRPD; **decriminalised suicide attempts**, gave the **right to mental healthcare**, **advance directives** and a **nominated representative**; created Mental Health Review Boards. Passed **7 April 2017**, in force **29 May 2018**	2017

● TRAP

Mind the gap between *passed* and *in force*. The **Mental Health Act 1987** was passed in 1987 but only implemented in **1993**. The **Mental Healthcare Act 2017** was passed in 2017 and came into force on **29 May 2018**. The 2017 Act is the one that decriminalised suicide attempts and introduced advance directives.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Two models, all eras:** supernatural (possession, exorcism, witch-hunts) vs naturalistic (Hippocrates → Galen → moral treatment → biological psychiatry). The whole history is this contest.
- **Humours:** blood = sanguine · yellow bile = choleric · **black bile = melancholic** (melan + chole) · phlegm = phlegmatic. Hippocrates names them, **Galen** systematises.
- **Medieval:** *Malleus Maleficarum* (1487) for witch-hunts · **Johann Weyer** the dissenter · Islamic **bimaristans** kept humane, naturalistic care alive (Razi, Avicenna).

- **First asylums: Bethlem / Bedlam, 1247** (priory, only later for the insane) · Hopital General Paris 1656 (the "great confinement," Foucault).
- **Moral treatment: Chiarugi** earliest (Florence 1788) · **Pinel** unchains (Paris 1793, with Pussin) · **Tuke** York Retreat (1796) · **Dix** US asylum campaign (1840s). Conolly = non-restraint.
- **Biological founders:** Griesinger ("mind diseases = brain diseases") · Kahlbaum (catatonia) · **Kraepelin** (dementia praecox vs manic-depressive) · **Bleuler** coined schizophrenia + 4 A's · Schneider FRS · Freud psychoanalysis.
- **Somatic / pharma:** Wagner-Jauregg malaria 1917 (Nobel) · Sakel insulin 1933 · Meduna convulsive 1934 · Moniz leucotomy 1935 (Nobel) · Cerletti-Bini ECT 1938 · **Cade lithium 1949** · **Delay-Deniker chlorpromazine 1952** · Kuhn imipramine 1957.
- **Antipsychiatry:** Szasz (*Myth of Mental Illness*) · Laing (*Divided Self*) · Goffman (*Asylums*, total institution) · Foucault (*Madness and Civilization*) · **Rosenhan 1973** (pseudopatients) → pushed DSM-III operational criteria.
- **India, ancient: Atharvaveda** = supernatural · **Ayurveda / Charaka** = naturalistic, **unmada** = insanity, tridosha model.
- **India, modern:** Lunacy Acts 1858 → **Indian Lunacy Act 1912** · **IPS 1947** · **CIP Ranchi 1918** · **NIMHANS** (AIIMH 1954, formed 1974) · **Vidya Sagar** family-tent model (Amritsar, 1950s).
- ★ **India, law: Mental Health Act 1987** (in force 1993) → **Mental Healthcare Act 2017** (in force 2018): rights-based, decriminalised suicide attempts, advance directives, nominated representative.

ONE-DAY REVISION · PAPER 2

Classification, nosology & history

How psychiatry learned to agree on what it is looking at. The story runs from Kraepelin's dichotomy through a reliability crisis to operational criteria, and into the two systems you actually use: ICD and DSM. Hold the dates, the one idea that distinguishes each milestone, and the comparison table – that table is a 10-mark answer in waiting.

● CONCEPT ● RULE ● TRAP ● KEY DATE ● MNEMONIC

The colour tells you what kind of fact it is. Every edition year is exam-critical: get the dates exactly right.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. The operational spine: Kraepelin dichotomy (1899) → 1960s reliability crisis (US/UK project, Rosenhan 1973) → Feighner 1972 → RDC 1975 → DSM-III 1980 (operational, atheoretical, multiaxial).
2. High reliability does NOT guarantee validity: reliability = agreement (kappa); validity = truth, with Robins & Guze (1970) five validators (clinical description, lab study, exclusion of other disorders, follow-up, family study).
3. ICD = WHO: ICD-10 in use 1992, ICD-11 adopted 2019 (World Health Assembly) and in force 1 Jan 2022; mental disorders are Chapter 06, guidance is the CDDR; trap: do not say "published 2018."
4. ICD-11 fully dimensionalised personality disorder (severity mild/moderate/severe plus five trait domains, borderline pattern qualifier kept), whereas DSM-5-TR keeps categorical PD types in Section II with the dimensional AMPD parked in Section III.
5. RDoC (NIMH, Insel, launched 2010) is a transdiagnostic research framework of functional domains × units of analysis, not a clinical replacement for ICD/DSM; HiTOP is the empirical companion.

1 Why classify, and how

A classification is a map, not the territory. It exists to serve practical ends, and every design choice (categorical vs dimensional, aetiological vs descriptive) is a trade-off. The deepest tension is between a diagnosis being **reliable** (consistently applied) and **valid** (carving nature at its joints).

The purposes of a classification

Purpose	What it buys
Communication	a shared shorthand: “schizophrenia” means the same to two clinicians
Prediction / prognosis	diagnosis carries an expected course and outcome
Treatment selection	links the case to an evidence base and a management plan
Research	defines comparable groups so studies can replicate and aggregate
Administration	statistics, billing, service planning, medico-legal and disability decisions

The design choices

Axis of choice	One pole	The other pole
Form	**Categorical**: present or absent, discrete boxes (a clinical tradition)	**Dimensional**: a position on continua of severity / traits
Basis	**Aetiological**: grouped by cause (e.g. older organic categories)	**Descriptive / atheoretical**: grouped by observed phenomenology, cause set aside
Structure	**Monothetic**: every feature required	**Polythetic**: any m of n criteria (the modern norm)

- **Categorical approach** – matches clinical intuition (you treat or you don't) and eases communication, but imposes arbitrary thresholds, hides severity, and forces a yes/no on what may be continuous.
- **Dimensional approach** – preserves severity and sub-threshold information and handles overlap well, but is clumsier at the bedside and resists a clean treat/no-treat decision. Modern systems are **hybrid**: categories with dimensional add-ons.

Reliability vs validity

- **Reliability** – agreement: do different raters (inter-rater) or the same rater over time (test-retest) reach the same diagnosis? Measured by **kappa**. The achievable win of operational criteria.
- **Validity** – truth: does the category correspond to a real entity? **Robins & Guze (1970)** gave five validators: clinical description, laboratory study, exclusion of other disorders, follow-up (course), and family (genetic) study. Reliability is necessary but not sufficient for validity.

CLASSIC TRAP

High reliability does **not** guarantee validity. Two clinicians can reliably apply a label that names nothing real. Operational criteria fixed reliability (the post-war crisis) but the *validity* of many categories remains contested – the core of the modern critique and the motive for RDoC.

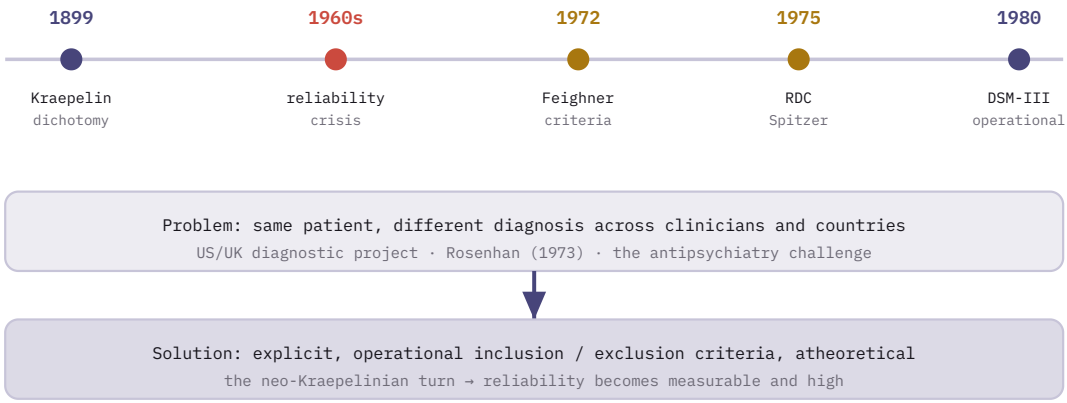
The hierarchy and comorbidity problem

- **Diagnostic hierarchy** – older systems ranked disorders so a "higher" one (e.g. schizophrenia) pre-empted "lower" ones (e.g. an anxiety diagnosis), forcing a single label (Jaspers' hierarchy; Foulds).
- **Comorbidity** – relaxing the hierarchy and using polythetic criteria let one patient meet several diagnoses at once. This is partly real co-occurrence and partly an **artefact** of splitting one underlying problem into overlapping categories. The high comorbidity of common disorders is a key argument against the categorical model.

2 The road to operational criteria

Modern classification is the answer to one embarrassment: in the 1960s psychiatrists could not agree on diagnoses. The fix was to write the criteria down explicitly. Hold this chain of events – it is the most reliably examined part of the topic.

FROM KRAEPELIN TO DSM-III: THE OPERATIONAL TURN



Kraepelin's descriptive dichotomy seeded the approach; the 1960s reliability crisis exposed the problem; Feighner (1972) and the RDC (1975) wrote criteria down; DSM-III (1980) made them the standard.

Kraepelin's dichotomy

● **Emil Kraepelin** (textbook, 1899) – separated the major psychoses by **course and outcome**, not just cross-section: **dementia praecox** (deteriorating; later renamed schizophrenia by Bleuler, 1911) vs **manic-depressive insanity** (episodic, with recovery between episodes). The descriptive, longitudinal method is the root of modern nosology.

The post-war reliability crisis

▲ **US / UK Diagnostic Project** (Cooper et al., 1972) – New York diagnosed **schizophrenia** far more often, London diagnosed **affective disorder / mania**, for the *same* filmed patients. The disorder was in the diagnostic habits, not the patients. Concept-level evidence that diagnosis was unreliable.

▲ **Rosenhan "On being sane in insane places"** (1973) – pseudopatients reporting a single symptom ("empty, hollow, thud") were admitted, mostly labelled schizophrenia, and only discharged "in remission." A famous (and contested) attack on the reliability and the stickiness of psychiatric labels. Sharpened the pressure for operational criteria.

The operational criteria

◆ **Feighner criteria** (1972) – Washington University, St. Louis. The first widely used **operational** diagnostic criteria, for 14 conditions; explicit inclusion / exclusion rules. The single most-cited paper in psychiatry for years. Laid the groundwork for everything after.

◆ **Research Diagnostic Criteria (RDC)** (Spitzer, Endicott & Robins, 1975) – extended Feighner for research use; the immediate template for DSM-III.

● **Neo-Kraepelinian turn** – the movement (Robins, Guze, Spitzer; Washington University "St. Louis school") that returned psychiatry to Kraepelin's descriptive, biological, research-led stance and away from psychoanalytic aetiology.

◆ **DSM-III** (APA, **1980**) – the watershed. Operational diagnostic criteria, explicitly **atheoretical** (described, did not explain), and **multiaxial** (five axes). Reliability rose sharply. Robert Spitzer chaired the task force.

HOLD THIS CHAIN

Kraepelin (course-based dichotomy) → **1960s reliability crisis** (US/UK, Rosenhan) → **Feighner 1972** → **RDC 1975** → **DSM-III 1980** (operational, atheoretical, multiaxial). This is the spine of the whole answer.

MNEMONIC

"Feighner First, RDC Refines, DSM-III Delivers"

1972 Feighner (first operational set) → 1975 RDC (research refinement) → 1980 DSM-III (the operational standard). Three steps, three dates.

3 ICD-11 (WHO)

The International Classification of Diseases is the **WHO** system, the one Indian practice uses. ICD-10 (1992) ran for a generation; ICD-11 is its successor, designed around global use and clinical utility.

Fact	Detail
Publisher	World Health Organization (WHO)
ICD-10 dates	endorsed 1990, came into use 1992
ICD-11 adopted	at the World Health Assembly in 2019
ICD-11 in effect	came into force 1 January 2022
Mental disorders chapter	Chapter 06: "Mental, behavioural or neurodevelopmental disorders"

Key changes from ICD-10

- **Reorganised groupings** – new and regrouped blocks: a single **schizophrenia and other primary psychotic disorders** group (the ICD-10 subtypes such as paranoid / hebephrenic are dropped, replaced by **symptom-rating qualifiers**); a dedicated **bipolar and related** grouping; a separate grouping for **obsessive-compulsive and related disorders**; a separate grouping for **disorders specifically associated with stress** (now including **complex PTSD** and prolonged grief disorder).
- **Neurodevelopmental disorders** – gathered into one grouping (intellectual developmental disorders, autism spectrum disorder, ADHD), aligning with a lifespan view.
- **Dimensional elements** – ICD-11 brings in dimensions where ICD-10 used categories: psychotic symptoms are rated on severity qualifiers, and **personality disorder is fully dimensionalised** – the named types are replaced by a single PD diagnosis graded by **severity** (mild / moderate / severe) plus **trait domains** (negative affectivity, detachment, dissociality, disinhibition, anankastia), with a borderline pattern qualifier retained.
- **New entities** – **gaming disorder** added; **gender incongruence** moved *out* of the mental disorders chapter into a new sexual-health chapter (de-psychiatrising it); complex PTSD and prolonged grief disorder formally recognised.

Clinical descriptions vs diagnostic requirements

- **CDDR** – ICD-11's clinical guidance is the **Clinical Descriptions and Diagnostic Requirements** (the successor to ICD-10's "Clinical Descriptions and Diagnostic Guidelines", the blue book). It deliberately avoids rigid arbitrary counts and cut-offs in favour of prototypic descriptions plus essential features, to fit varied global and primary-care settings.
- **Global / clinical-utility emphasis** – ICD-11 was field-tested for ease of use across countries and resource levels; it is fully digital (a foundation linearised into the tabular list) and designed for **clinical utility** as much as for statistical reporting.

● DATES TO LOCK

ICD-10 in use **1992**. ICD-11 **adopted 2019** (World Health Assembly), **in effect from 2022**. Do not say "published 2018" in the exam: the foundation was released for preview in 2018, but the examinable milestones are 2019 (adopted) and 2022 (in force).

4 DSM-5 / DSM-5-TR (APA)

The Diagnostic and Statistical Manual is the **American Psychiatric Association** system. DSM-5 was the big structural break from DSM-IV; DSM-5-TR is a text revision that updated text and added one disorder.

Edition	Year	Signature change
DSM-III	1980	operational criteria, atheoretical, multiaxial
DSM-IV	1994	(text revision DSM-IV-TR in 2000); added the clinical-significance criterion
DSM-5	2013	multiaxial system abolished; spectrum / dimensional additions; Section III
DSM-5-TR	2022	text revision; added **prolonged grief disorder**; updated terminology and prevalence text

The headline changes in DSM-5

◆ **Multiaxial system abolished** – the DSM-III/IV five axes are gone. Former Axes I–III are combined into a single list of diagnoses; psychosocial factors (old Axis IV) move to Z-codes; the GAF (old Axis V) is dropped in favour of measures such as the **WHODAS 2.0**.

● **Spectrum / dimensional additions** – **autism spectrum disorder** merges the old PDD subtypes; schizophrenia subtypes dropped; **substance use disorder** collapses abuse + dependence into one graded (mild/moderate/severe) diagnosis; cross-cutting symptom measures and severity ratings introduced; chapters reordered on a developmental / lifespan logic.

● **Section III** – "Emerging measures and models": the home for proposals not yet in the main system. It holds the **Alternative DSM-5 Model for Personality Disorders (AMPD)**, assessment measures, the cultural formulation interview, and Conditions for Further Study. (Section I = basics; Section II = the criteria in use.)

● TRAP

It is "DSM-**5**" in Arabic numerals (a deliberate break from the Roman "DSM-IV"), chosen so the manual could be updated incrementally (5.1, 5.2). The 2022 update is the **text revision, DSM-5-TR** – it added prolonged grief disorder and a suicidal-behaviour code, not a wholesale criteria overhaul.

5 ICD vs DSM, compared

The most examinable single table in the topic. Know the publisher, scope and structural differences, and where they part ways on specific disorders.

Feature	ICD-11	DSM-5-TR
Publisher	WHO (a UN body)	American Psychiatric Association
Scope	all of medicine; mental disorders are Chapter 06	mental disorders only
Audience / use	global, all settings; official mortality & morbidity statistics	chiefly research and specialist clinical, US-centred
Cost / access	free to use (public-health mandate)	copyrighted, purchased

Feature	ICD-11	DSM-5-TR
Diagnostic style	prototypic descriptions, "essential features" (CDDR), fewer rigid counts	explicit polythetic criteria with counts and durations
Latest dates	adopted 2019, in effect 2022	DSM-5 2013, DSM-5-TR 2022

Where they differ on key disorders

Area	ICD-11	DSM-5-TR
Personality disorder	fully dimensional: severity + trait domains (borderline pattern qualifier kept)	categorical types in Section II; dimensional AMPD sits in Section III
Stress disorders	recognises complex PTSD as a distinct diagnosis	no separate complex PTSD; a dissociative PTSD subtype instead
Bereavement / grief	prolonged grief disorder	prolonged grief disorder (added in the TR, 2022)
Bipolar / mixed	mixed episode retained as an episode type	mixed episode dropped; a "with mixed features" specifier instead
Gender incongruence	moved out of mental disorders (sexual-health chapter)	retained as "gender dysphoria" within the manual

INDIA SITS HERE

Indian psychiatric practice, official statistics and most training are **ICD-based** (currently transitioning ICD-10 → ICD-11). DSM is used heavily in **research** and reading the international literature. Know both, default to ICD for clinical answers.

● RULE

The two systems have been progressively **harmonised** (shared revision committees, aligned codes) but deliberately retain differences. The headline distinction for a viva: ICD = WHO, whole-of-medicine, free, global; DSM = APA, psychiatry-only, copyrighted, research-leaning.

6 Key concepts & critiques

The conceptual vocabulary and the standing objections. Examiners love the definitions (syndrome vs disorder vs disease) and the antipsychiatry names. Hold the precise distinctions.

The conceptual ladder

- **Syndrome** – a recurring *cluster* of symptoms and signs that occur together, with no implication about cause or mechanism. Most psychiatric diagnoses remain at this level.
- **Disorder** – the deliberately neutral term both systems use: a clinically significant syndrome associated with distress or impairment, chosen *precisely to sidestep* the stronger claim of "disease."
- **Disease** – a syndrome with a known pathology, mechanism or aetiology (e.g. a demonstrable lesion). Few psychiatric conditions yet qualify; this gap fuels the validity critique.
- **Caseness** – whether a person crosses the threshold to "count" as a case, the dividing line between normality and disorder. Threshold choice is partly arbitrary and shifts prevalence dramatically.
- ▲ **Reification** – the error of treating a useful descriptive *construct* as if it were a proven natural *thing*. Forgetting that "major depressive disorder" is a label of convenience, not a confirmed disease entity.

- **Spectrum** – the idea that related conditions lie on a continuum rather than in discrete boxes (e.g. the autism spectrum, the schizophrenia spectrum). A dimensional concept inside a categorical system.
- **NOS / unspecified** – the residual categories for presentations that don't fit a defined type. DSM-IV's **"not otherwise specified (NOS)"** became DSM-5's **"other specified" / "unspecified."** Heavy use of these flags weak coverage of the defined categories.

The critiques

Antipsychiatry

Thomas Szasz – *The Myth of Mental Illness* (1961): mental illness is a metaphor; without bodily pathology these are "problems in living," and diagnosis is a tool of social control.
R.D. Laing – psychosis as a meaningful response to an unliveable situation, not a brute disease.
Goffman – the institution and the "moral career" of the labelled patient.

Validity critiques (from within)

Diagnoses are **reliable but of uncertain validity**; no biomarkers confirm them. High **comorbidity** and heavy NOS use suggest the categories carve nature badly. Thresholds are arbitrary (caseness); labels risk **reification** and stigma. These are mainstream, not fringe, concerns.

The dimensional alternative for research

◆ **RDoC (Research Domain Criteria)** – the **NIMH** framework (Insel, launched **2010**). Not a clinical classification: a *research* framework that abandons DSM/ICD categories and instead studies functional **domains** (e.g. negative valence, positive valence, cognitive, social, arousal/regulatory systems) across **units of analysis** (genes → molecules → cells → circuits → physiology → behaviour → self-report). Dimensional, transdiagnostic, neuroscience-anchored. The explicit response to the validity critique.

● TRAP

RDoC is **not** a replacement for ICD or DSM in the clinic – it is a research scaffold meant to seed a future, more valid classification. Pair it with the **HiTOP** consortium (Hierarchical Taxonomy of Psychopathology) as the other major dimensional/empirical alternative if you want a bonus mark.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Why classify:** communication, prognosis, treatment selection, research, administration. Categorical (boxes) vs dimensional (continua); aetiological vs descriptive/atheoretical; monothetic vs polythetic.
- **Reliability vs validity:** reliability = agreement (kappa); validity = truth (Robins & Guze 1970, five validators). High reliability does not guarantee validity.
- **Hierarchy / comorbidity:** dropping the diagnostic hierarchy + polythetic criteria → comorbidity, partly real, partly artefact of over-splitting.
- ★ **The operational chain:** Kraepelin dichotomy (1899: dementia praecox vs manic-depressive) → reliability crisis (US/UK project 1972; Rosenhan 1973) → **Feighner 1972** → **RDC 1975** → **DSM-III 1980** (operational, atheoretical, multiaxial).
- **ICD:** WHO. ICD-10 in use **1992**; ICD-11 **adopted 2019**, **in effect 2022**. Chapter 06; CDDR guidance; PD dimensionalised; gaming disorder added; gender incongruence de-psychiatrised; complex PTSD recognised.
- **DSM:** APA. DSM-III **1980**, DSM-IV **1994**, DSM-5 **2013**, DSM-5-TR **2022**. DSM-5 abolished the multiaxial system; Arabic numeral; Section III holds the AMPD and emerging models.
- **ICD vs DSM:** ICD = WHO, all medicine, free, global, official stats; DSM = APA, psychiatry-only, copyrighted, research-leaning. India = ICD-based.
- **Concepts:** syndrome (cluster) → disorder (neutral, distress/impairment) → disease (known pathology). Caseness = threshold; reification = construct mistaken for a thing; spectrum, NOS/unspecified.

- **Critiques:** Szasz (*Myth of Mental Illness* 1961, "problems in living"), Laing, Goffman. Mainstream worry: reliable but uncertain validity, no biomarkers, comorbidity, arbitrary thresholds.

- **Dimensional alternative: RDoC** (NIMH, 2010) = research domains × units of analysis, transdiagnostic, not for the clinic. HiTOP as the empirical companion.

ONE-DAY REVISION · PAPER 2

Schizophrenia, the high-yield map

The single most examined psychosis. Hold the five symptom domains, the diagnostic durations (DSM-5 vs ICD-11), the dopamine story in its revised form, and the prognosis lists. Every comparison table here is a structured-answer in waiting.

CONCEPT · RULE · TRAP · KEY NUMBER · MNEMONIC

The colour tells you what kind of fact it is. The eminent contributors (Kraepelin, Bleuler, Schneider) and landmark trials (Kane 1988) live in their companion packs – one pointer line each here.

★ HIGH YIELD — TOP 5 IF NOTHING ELSE

1. DSM-5 = 6 months total disturbance with ≥1 month active phase plus a functional decline; ICD-11 = ≥1 month and no strict functioning criterion – the single highest-yield discriminator in the topic.
2. Risk tracks shared genes: ~1% general population → ~10% first-degree relative → ~50% with both parents affected or an MZ co-twin; heritability ~80%, polygenic, strongest single CNV = 22q11.2 deletion.
3. Revised dopamine hypothesis (Howes & Kapur): the primary lesion is presynaptic striatal dopamine dysregulation, not a postsynaptic receptor fault, assigning aberrant salience; mesolimbic hyper → positive, mesocortical hypo → negative symptoms.
4. Treatment-resistant schizophrenia = failure of ≥2 adequate antipsychotic trials (at least one atypical); clozapine is the only superior agent (Kane 1988) but carries agranulocytosis ~0.8% needing mandatory FBC monitoring.
5. Trap: Bleuler's 4 A's (association loosening, affect blunting, ambivalence, autism) are the fundamental symptoms and are NOT the negative-symptom 5 A's (affective flattening, alogia, avolition, anhedonia, asociality); both ICD-11 and DSM-5 have abolished the classical subtypes for symptom dimensions.

1 Concept & epidemiology

Schizophrenia is a chronic disorder of thought, perception, affect and volition, with relative preservation of clear consciousness and intellect. The label has moved through three hands.

- **The naming, in one line** – Kraepelin grouped it as **dementia praecox** (early, deteriorating course); Bleuler renamed it **schizophrenia** (split of psychic functions) and gave the **4 A's**; Schneider sharpened diagnosis with the **first-rank symptoms**. Detail lives in the eminent-contributors pack.

The numbers worth memorising

Measure	Figure	Note
Lifetime risk	~1% (roughly 0.7–1%)	broadly uniform across populations
Sex ratio	~equal lifetime	men onset earlier & often more severe
Age of onset	men 15–25, women 25–35	women show a second peak after 40
Mortality	life expectancy reduced ~15–20 yr	cardiometabolic disease + suicide
Suicide	~5–10% lifetime	highest risk early, post-discharge

Risk-factor headline figures (relative risk)

Factor	Approx. relative risk
Both parents affected	~40–50% offspring risk
Monozygotic co-twin affected	~40–50% concordance
One parent / dizygotic twin / sibling	~10–15%
Heavy cannabis use (adolescent)	~2–4×
Migration	~2–3× (highest in 2nd generation)
Urban birth / upbringing	~2×

QUICK RULE

Genetic loading is the strongest single predictor: risk tracks the **proportion of shared genes**. ~1% general population → ~10% first-degree relative → ~50% with both parents affected or an MZ co-twin.

2 Clinical features by domain

Modern teaching splits the syndrome into **five symptom domains** rather than discrete subtypes. Knowing the domain a symptom belongs to is the examinable skill.

Domain	Core features
Positive	delusions, hallucinations (auditory most common), passivity phenomena
Negative	the **5 A's** (below) – loss or reduction of normal function
Disorganisation	formal thought disorder (loosening, tangentiality, neologisms), disorganised/bizarre behaviour
Cognitive	deficits in attention, working memory, processing speed, executive function
Affective	depression, anxiety, suicidality; affective flattening overlaps negative domain

Schneider's first-rank symptoms (FRS)

Once central to diagnosis, now **not specific** to schizophrenia (also in mania, organic states) and downgraded in ICD-11/DSM-5, but heavily examined.

◆ **The FRS list** – (1) **audible thoughts** (thought echo); (2) **voices arguing / discussing** in the third person; (3) **running commentary** voices; (4) **thought withdrawal, insertion, broadcast**; (5) **made (passivity) feelings, impulses, acts**; (6) **somatic passivity**; (7) **delusional perception** (a real percept given a sudden delusional meaning).

MNEMONIC

"ABCD" for FRS clusters

Auditory hallucinations (3 kinds) · **B**roadcasting / thought alienation · **C**ontrolled (passivity, made acts) · **D**elusional perception. Bleuler's 4 A's are different – see below.

The negative-symptom cluster: the 5 A's

- **Affective flattening** – reduced range and intensity of emotional expression (blunted face, monotone voice).
- **Alogia** – poverty of speech / poverty of content of speech.
- **Avolition** – loss of drive and goal-directed behaviour; apathy.
- **Anhedonia** – reduced capacity for pleasure.

- **Asociality** (with **attentional** impairment often added) – social withdrawal, reduced interest in interaction.

Positive vs negative: the contrast that carries marks

	Positive symptoms	Negative symptoms
Nature	excess / distortion of function	loss / reduction of function
Examples	delusions, hallucinations, FRS	the 5 A's
Course	fluctuate; respond well to antipsychotics	persistent; respond poorly to medication
Dopamine link	mesolimbic hyperdopaminergia	mesocortical hypodopaminergia (one model)
Prognosis	better outcome predictor	predict worse functional outcome

CLASSIC TRAP

Do not confuse **Bleuler's 4 A's** (the fundamental symptoms: **A**ssociation loosening, **A**ffect blunting, **A**mbivalence, **A**utism) with the **negative-symptom 5 A's** (affective flattening, alogia, avolition, anhedonia, asociality). Bleuler also named accessory symptoms (hallucinations, delusions). Schneider's FRS are a third, separate list.

3 Diagnosis

The two systems agree on the symptom picture but differ on **duration**. That single difference is the most common one-mark question.

	DSM-5 / DSM-5-TR	ICD-11
Symptoms required	≥2 of 5, at least one being a **core** symptom (delusions, hallucinations, or disorganised speech)	≥2 symptoms, at least one a **core** positive symptom; FRS / thought alienation / passivity weighted
Total duration	≥6 months of disturbance	≥1 month
Active-phase duration	≥1 month of active symptoms (less if treated)	the ≥1 month *is* the requirement
Functioning	marked decline in work, relationships or self-care	not a strict criterion
Exclusions	schizoaffective, mood-with-psychosis, substance/organic causes ruled out	same exclusions

HOLD THIS

DSM-5 = 6 months total, 1 month active. ICD-11 = 1 month. DSM also demands a functional decline; ICD-11 does not. This is the highest-yield single fact in the topic.

- **Subtypes abolished** – both **ICD-11 and DSM-5 dropped the classical subtypes** (paranoid, hebephrenic/disorganised, catatonic, simple, residual, undifferentiated). Poor reliability and stability. Replaced by **symptom-dimension specifiers / course specifiers** rated on severity. Catatonia is now a transdiagnostic specifier, not a subtype.

Differential diagnosis

Disorder	Distinguishing feature
Schizophreniform	full picture but duration **1–6 months** (DSM-5); functional decline not required
Brief psychotic disorder	<1 month, full recovery; often a stressor
Schizoaffective	mood episode + psychosis concurrently, **plus ≥2 weeks of psychosis without prominent mood symptoms**

Disorder	Distinguishing feature
Mood disorder with psychotic features	psychosis occurs **only** within mood episodes (mood-congruent typically)
Delusional disorder	non-bizarre delusion(s) ≥1 month, function otherwise preserved, no other active-phase symptoms
Substance / medication-induced	temporal link to use/withdrawal (e.g. amphetamine, cannabis, steroids)
Organic / secondary psychosis	delirium, temporal-lobe epilepsy, encephalitis (e.g. anti-NMDA-R), tumour, dementia

● RULE

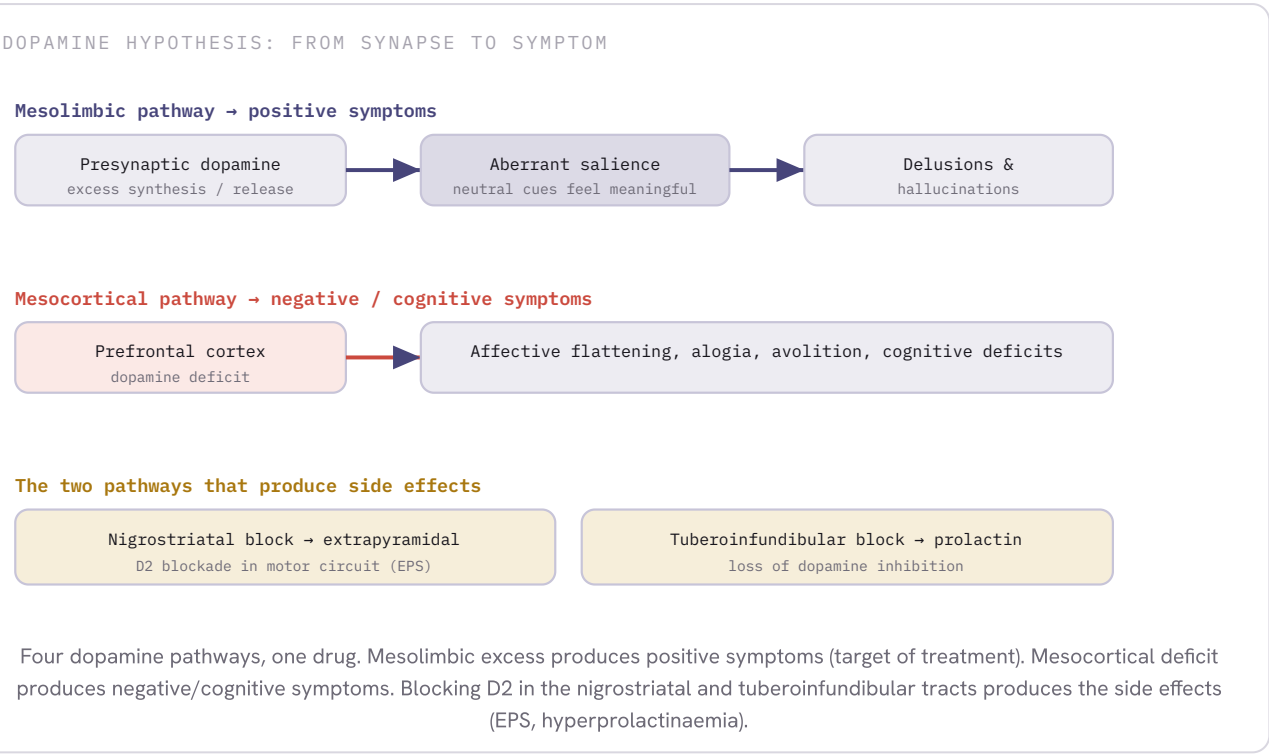
The schizoaffective vs mood-with-psychosis split turns on **timing**: schizoaffective needs a stretch of psychosis (≥2 weeks) *in the absence* of a prominent mood episode. If psychosis appears *only* during mood episodes, it is a mood disorder with psychotic features. Always exclude organic and substance causes first with history, examination and investigations.

4 Aetiology

The integrating frame is the **neurodevelopmental hypothesis**: schizophrenia is the late expression of an early disturbance of brain development, decompensating in adolescence when synaptic pruning and frontal maturation peak. Genes, environment and neurochemistry feed into it.

The dopamine hypothesis

- **Original (classic) form** – psychosis results from **excess dopamine** / D2-receptor hyperactivity. Two pillars: antipsychotic potency correlates with **D2 affinity**; dopaminergic drugs (amphetamine, L-DOPA) can induce psychosis.
- **Pathway-specific refinement** – **mesolimbic hyperdopaminergia** drives positive symptoms; **mesocortical hypodopaminergia** underlies negative and cognitive symptoms.
- **Revised hypothesis (Howes & Kapur)** – the primary lesion is **presynaptic striatal dopamine dysregulation** (raised synthesis and release), not a postsynaptic receptor fault. Excess dopamine assigns **aberrant salience** to neutral stimuli; delusions are the mind's attempt to explain that abnormal salience. The “final common pathway” through which diverse risk factors act.



Four dopamine pathways, one drug. Mesolimbic excess produces positive symptoms (target of treatment). Mesocortical deficit produces negative/cognitive symptoms. Blocking D2 in the nigrostriatal and tuberoinfundibular tracts produces the side effects (EPS, hyperprolactinaemia).

Glutamate hypothesis

- **NMDA-receptor hypofunction** – **NMDA antagonists** (ketamine, phencyclidine/PCP) reproduce positive, negative *and* cognitive symptoms in healthy people, better mimicking the full syndrome than amphetamine. Hypofunction of NMDA receptors on cortical interneurons is proposed to disinhibit downstream dopamine, linking the two systems.

Genetics

- **Heritability ~80%** – one of the most heritable psychiatric disorders, yet **polygenic**: many common variants of small effect (GWAS) plus rare, high-impact **copy-number variants (CNVs)**. The strongest single CNV is **22q11.2 deletion** (velocardiofacial syndrome; ~25% develop psychosis). **Neuregulin-1, dysbindin and DISC1** are historically prominent candidate genes.

Environmental risk factors

Prenatal / perinatal

Obstetric complications (hypoxia), maternal infection & malnutrition (Dutch Hunger Winter), **winter / late-spring birth**, and **advanced paternal age** (de novo mutations).

Later-life exposures

Adolescent cannabis (dose-dependent, high-THC), **urbanicity**, **migration / minority status**, childhood trauma and social adversity. These shift risk on a developmentally primed brain.

Neuroimaging

- **Structural findings** – the most replicated is **lateral and third ventricular enlargement** with reduced overall **grey-matter volume** (especially frontal and medial temporal, including hippocampus). Findings are subtle, present at first episode, and not diagnostic individually.

● TRAP

The dopamine hypothesis is **not** simply "too much dopamine everywhere." The revised account locates the fault **presynaptically in the striatum** and is **pathway-specific**: hyperdopaminergic mesolimbically (positive symptoms), hypodopaminergic mesocortically (negative symptoms). This is why D2 blockade treats positive symptoms but barely touches negative ones.

5 Course & prognosis

Prognostic factors

Good prognosis

Acute / sudden onset, late onset, clear **precipitating stressor**, prominent **mood (affective)** symptoms, predominantly **positive** symptoms, good premorbid function, married / good support, female sex, no family history, short **DUP**, good treatment adherence.

Poor prognosis

Insidious onset, early onset, no precipitant, prominent **negative** symptoms, poor premorbid adjustment, social withdrawal, single / poor support, male sex, positive family history, long DUP, substance misuse, poor adherence.

◆ **The rule of thirds (approximate)** – of patients followed long-term, roughly **one-third** recover or have a good outcome, **one-third** have an intermediate course with relapses but some function, and **one-third** have a chronic, deteriorating course. A useful exam heuristic, not a precise law.

■ **DUP matters** – a longer **duration of untreated psychosis (DUP)** predicts worse symptomatic and functional outcome. The rationale for **early-intervention services** and prompt treatment of the first episode.

QUICK RULE

The single most reliable prognostic axis: **acute onset + mood symptoms + positive symptoms + good premorbid function = good outcome**. Its mirror (insidious, negative-symptom, poor premorbid) is the poor-outcome profile.

6 Management overview

Management is biopsychosocial: antipsychotics are the backbone, but psychosocial work and family interventions change relapse rates as much as drug choice does.

Antipsychotics: typical vs atypical

	Typical (first-generation)	Atypical (second-generation)
Examples	haloperidol, chlorpromazine, fluphenazine	risperidone, olanzapine, quetiapine, aripiprazole, clozapine
Mechanism	strong **D2** blockade	D2 + **5-HT2A** blockade (looser, faster-dissociating D2)
Strength	effective on positive symptoms	positive symptoms; possibly better tolerated; modest negative-symptom benefit
Main adverse effects	EPS (dystonia, parkinsonism, akathisia, tardive dyskinesia), hyperprolactinaemia	metabolic (weight gain, dyslipidaemia, diabetes), esp. olanzapine & clozapine

◆ **Clozapine & treatment resistance** – **treatment-resistant schizophrenia (TRS)** = failure of **≥2 adequate antipsychotic trials** (adequate dose & duration), at least one atypical. **Clozapine** is the only agent with superior efficacy in TRS. Landmark evidence: **Kane et al. 1988** (clozapine vs

chlorpromazine in resistant patients) – cross-ref the studies pack.

● CLASSIC TRAP

Clozapine (spelled with a **z**) is reserved for TRS, not first-line, because of **agranulocytosis** (~0.8%), requiring mandatory **FBC monitoring**. Other serious effects: myocarditis, seizures (dose-related), severe constipation (ileus), weight gain, hypersalivation. It also uniquely reduces suicidality (InterSePT). Never confuse with clonazepam.

Psychosocial interventions

● **Evidence-based options** – **CBT for psychosis (CBTp)** for residual positive symptoms; **family intervention** (reduces relapse); **psychoeducation**; **social-skills training**; **supported employment** / vocational rehab; **cognitive remediation** for cognitive deficits; **assertive community treatment** for frequent relapsers.

◆ **Expressed emotion (EE) & relapse** – high **EE** in the family (critical comments, hostility, emotional over-involvement) strongly predicts **relapse**. Family interventions that lower EE and improve communication cut relapse rates – one of the best-replicated psychosocial findings.

ECT: the niche

■ **When ECT is indicated** – not first-line for schizophrenia, but useful in **catatonia** (after a benzodiazepine trial), **treatment-resistant illness** as an adjunct to clozapine, severe risk requiring rapid response, and prominent affective symptoms. Catatonia is the strongest schizophrenia-spectrum indication.

HOLD THIS

First-line = an atypical antipsychotic + psychosocial support. Fail **two** adequate trials → **clozapine** (the TRS drug, FBC-monitored). High family EE → relapse → family intervention. ECT → catatonia and resistance.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Concept:** Kraepelin (dementia praecox) → Bleuler (schizophrenia, 4 A's) → Schneider (first-rank symptoms). Lifetime risk **~1%**; men onset 15–25, women 25–35; suicide ~5–10%.
- **Genetics of risk:** ~1% general → ~10% first-degree → ~50% both parents or MZ co-twin. Heritability **~80%**, polygenic; strongest CNV = **22q11.2**.
- **Domains:** positive, negative, disorganisation, cognitive, affective. Negative = the **5 A's** (affective flattening, alogia, avolition, anhedonia, asociality). Bleuler's 4 A's ≠ the negative 5 A's.
- **FRS (Schneider):** three auditory hallucination types, thought alienation (withdrawal/insertion/broadcast), passivity/made acts, somatic passivity, delusional perception. Not specific to schizophrenia.
- ★ **Diagnosis:** DSM-5 = **6 months total, 1 month active** + functional decline. ICD-11 = **1 month**. Both **abolished the classical subtypes**; use dimensions. Catatonia = transdiagnostic specifier.
- **Differentials:** schizophreniform 1–6 mo; brief psychotic <1 mo; schizoaffective = ≥2 wk psychosis without mood; mood-with-psychosis = psychosis only in mood episodes; delusional disorder; substance/organic.
- **Dopamine:** mesolimbic **hyper** → positive; mesocortical **hypo** → negative. Revised = **presynaptic striatal** dysregulation → **aberrant salience** (Howes & Kapur). Nigrostriatal block → EPS; tuberoinfundibular → prolactin.
- **Other aetiology:** NMDA hypofunction (ketamine/PCP mimic the full syndrome); environment = cannabis, urbanicity, migration, obstetric complications, paternal age, winter birth; imaging = ventricular enlargement + reduced grey matter.
- **Prognosis:** good = acute onset + mood + positive symptoms + good premorbid + short DUP; poor = insidious + negative + poor premorbid + long DUP. Rule of thirds. DUP matters.

- **Management:** atypical + psychosocial first-line; fail **2 trials** → **clozapine** (TRS, Kane 1988; agranulocytosis, FBC). High **EE** → relapse → family intervention. ECT for catatonia / resistance.

ONE-DAY REVISION · PAPER 2

Mood disorders, the high-yield map

Affective illness sorted for the exam: the depressive-versus-bipolar split, the clinical syndromes and their specifiers, the duration cut-offs that separate mania from hypomania and BP-I from BP-II, and a management spine built on mood stabilisers, antidepressant caution and ECT. Every contrast table here is a structured-answer in waiting.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. Heritability detail lives in the genetics pack; trials and scales are cross-referenced by name.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. One lifetime **manic or hypomanic episode flips the label to bipolar**; depressions only = unipolar, and a single full manic episode = bipolar I permanently.
2. Duration cut-offs: **mania ≥ 1 week** (or any duration if hospitalisation needed), **hypomania ≥ 4 days**; **psychosis or hospitalisation makes it mania regardless of duration**, and hypomania is never psychotic.
3. **Lithium is first-line bipolar maintenance**, works on both poles, and is the only agent with consistent **anti-suicidal** evidence; level **0.6-1.0 mmol/L**, toxic above ~1.5.
4. **Antidepressant monotherapy in bipolar depression risks a switch into mania** and rapid cycling; cover with a mood stabiliser and prefer quetiapine, lamotrigine or lurasidone for the depressive pole.
5. Suicide: the single strongest predictor is a **previous attempt** and the key cognition is **hopelessness**; risk peaks in early recovery as retardation lifts before mood does.

1 Classification

The first cut in mood disorders is **depressive (unipolar)** versus **bipolar**. A bipolar diagnosis needs at least one manic or hypomanic episode ever; without one, the illness is unipolar however many depressions there have been.

Category	Defining episode(s)	ICD-11 / DSM-5 note
Major depressive disorder	one or more depressive episodes, **no** mania/hypomania ever	ICD-11: single episode vs recurrent depressive disorder
Bipolar I	at least one **manic** episode (depression usual but not required)	full mania defines it; psychosis may occur
Bipolar II	at least one **hypomanic** + at least one **major depressive** episode, **never** full mania	one true manic episode reclassifies to BP-I for life
Persistent depressive disorder (dysthymia)	chronic low-grade depression ≥ **2 years** (1 year in youth)	DSM-5 merges chronic MDD + dysthymia; ICD-11 keeps dysthymic disorder
Cyclothymia	≥ **2 years** of fluctuating sub-threshold hypomanic and depressive symptoms	never meets full hypomanic or depressive episode criteria

● **Unipolar vs bipolar** – unipolar = depressive episodes only. Bipolar = at least one episode of pathologically **elevated** mood (manic or hypomanic) in the lifetime course. The single elevated episode is what flips the label.

● **The bipolar spectrum** – BP-I (mania) → BP-II (hypomania + depression) → cyclothymia (sub-threshold, chronic). Severity and episode "height" fall as you move down; chronicity defines the bottom.

◆ **The two-year rule** – both **persistent depressive disorder** and **cyclothymia** need symptoms for at least **2 years** in adults (1 year in children/adolescents). Chronic but sub-syndromal is the shared theme.

● CLASSIC TRAP

A single **manic** episode is sufficient for **bipolar I**: a preceding depressive episode is *not* required. By contrast, **bipolar II** needs both a hypomanic **and** a major depressive episode. If a patient labelled BP-II ever has one full manic episode, the diagnosis becomes BP-I permanently.

2 Depressive disorder: clinical features

Group the symptoms into three clusters: **core**, **biological/somatic** and **cognitive**. The ICD core triad (low mood, anhedonia, low energy) anchors the diagnosis; severity is graded by symptom count and impairment.

Cluster	Features
Core	persistent **low mood**, **anhedonia** (loss of interest/pleasure), **reduced energy** / fatigue
Biological / somatic	**early-morning waking** (terminal insomnia), **diurnal variation** (mood worst in the morning), appetite and weight loss, **psychomotor retardation** or agitation, loss of libido, constipation
Cognitive	Beck's **negative triad** (negative view of self, world, future), **guilt** and worthlessness, **hopelessness**, poor concentration, **suicidal ideation**

◆ **Beck's negative cognitive triad** – the depressed mind holds a negative view of the **self** ("I am worthless"), the **world** ("everything is against me") and the **future** ("it will never improve"). Hopelessness about the future is the cognition most tied to suicide risk.

Severity grading

Grade	Rough marker	Function
Mild	few symptoms beyond the threshold	minor functional impairment, distress with effort to continue
Moderate	symptoms or impairment between mild and severe	considerable difficulty continuing activities
Severe	most symptoms present, marked distress	unable to continue most activities; with or without psychotic features

Specifiers (the high-yield list)

Specifier	Hallmark
Melancholic	profound anhedonia, **non-reactive mood**, early waking, diurnal variation (worse a.m.), marked psychomotor change, weight loss, excessive guilt
Atypical	**mood reactivity** preserved, **reversed** biological features: hypersomnia, hyperphagia/weight gain, **leaden paralysis**, rejection sensitivity
Seasonal	regular onset in a season (typically autumn/winter), remitting in spring; often atypical-flavoured
Peripartum	onset in pregnancy or within **4 weeks** postpartum (DSM-5); examine for postpartum psychosis
With psychotic features	delusions/hallucinations; **mood-congruent** (guilt, nihilism, poverty, deserved punishment) vs **mood-incongruent**

Specifier	Hallmark
With mixed features	depressive episode plus ≥ 3 concurrent manic/hypomanic symptoms (or vice versa)
With catatonia	stupor, mutism, posturing, negativism, waxy flexibility; responds to benzodiazepines and ECT

Melancholic depression

Mood non-reactive (no lift even to good news).
Early-morning waking, worse in the morning.
Anorexia and **weight loss**.
Marked psychomotor retardation or agitation.
The classic "biological" / endogenous picture; more ECT-responsive.

Atypical depression

Mood reactive (brightens to positives).
Hypersomnia and **hyperphagia** / weight gain.
Leaden paralysis (heavy limbs).
Long-standing **rejection sensitivity**.
Historically the MAOI-responsive subtype.

TRAP

Atypical depression is not "mild" or "rare." The label means the *reverse* of the melancholic somatic pattern: mood that still **reacts** to circumstances, plus hypersomnia and hyperphagia rather than insomnia and anorexia. Mood reactivity is the cardinal feature.

RULE

In psychotic depression, label the **congruence**: mood-congruent delusions fit the depressed affect (guilt, nihilism, poverty, somatic decay, Cotard's nihilistic delusion). Mood-incongruent content (e.g. persecution, thought insertion) carries a worse prognosis and prompts a wider differential.

3 Bipolar disorder: mania, hypomania & subtypes

The whole bipolar viva turns on two contrasts: **mania vs hypomania** (a matter of duration, impairment and psychosis) and **BP-I vs BP-II** (a matter of which elevated episode has ever occurred).

Feature	Mania	Hypomania
Duration	≥ 1 week (or any duration if hospitalisation needed)	≥ 4 days consecutively
Functional impairment	marked impairment in work/social function	no marked impairment; change is observable but not disabling
Psychosis	may have delusions/hallucinations	never psychotic (psychosis = mania by definition)
Hospitalisation	may be required (and itself qualifies the episode)	not required
Defines	Bipolar I	Bipolar II (with a depressive episode)

◆ **The duration cut-offs** – **mania ≥ 1 week** (or any duration if hospital admission is needed), **hypomania ≥ 4 days**. Both need elevated/irritable mood *plus* increased activity/energy (the energy criterion is mandatory in DSM-5 and ICD-11).

● **Core manic symptoms (DIGFAST)** – **D**istractibility, **I**ndiscretion (risk-taking), **G**randiosity, **F**light of ideas, **A**ctivity increase, **S**leep decreased (reduced *need* for sleep), **T**alkativeness (pressured speech).

MNEMONIC

"DIG FAST" → the seven manic symptoms

Distractibility, Indiscretion, Grandiosity, Flight of ideas, Activity, Sleep (decreased need), Talkativeness. Three of seven (four if mood only irritable) plus the duration cut-off makes the episode.

BP-I vs BP-II at a glance

	Bipolar I	Bipolar II
Required episode	≥ 1 manic episode ever	≥ 1 hypomanic + ≥ 1 major depressive
Full mania	yes	never (one episode reclassifies to BP-I)
Depression	usual, not required for the diagnosis	required and often the dominant, disabling pole
Psychosis	possible (in mania)	not in hypomania; possible in depressive episodes

■ **Rapid cycling** – **≥ 4 mood episodes in 12 months** (each meeting full criteria, with remission or switch of polarity between). More common in **women** and in BP-II; antidepressants and hypothyroidism can drive it; carries a poorer prognosis.

● **Mixed features** – manic and depressive symptoms coexist in one episode (e.g. depressed mood with racing thoughts and agitation). High suicide risk; antidepressant monotherapy is hazardous; prefer a mood stabiliser or atypical antipsychotic.

TRAP

The line between mania and hypomania is **not** just duration. Even a brief episode counts as full **mania** if it causes **psychosis** or needs **hospitalisation**. Conversely, four days of clear elevation with preserved function and no psychosis is **hypomania**, however striking it looks.

4 Epidemiology & aetiology

	Major depression	Bipolar disorder
Lifetime prevalence	~10–15% (point prevalence lower)	~1% BP-I; ~2–4% the broader spectrum
Sex ratio (F:M)	~2:1 (female preponderance)	~1:1 (BP-II commoner in women)
Typical onset	late 20s (any age)	late teens to early 20s (earlier than unipolar)
Heritability	moderate (~35–40%)	high (~60–85%); see genetics pack

Biological models

● **Monoamine hypothesis** – depression reflects a functional **deficit of serotonin, noradrenaline and dopamine** at the synapse; antidepressants raise monoamine availability. Limited by the **therapeutic lag** (weeks to work though synaptic levels rise within hours), pointing to downstream receptor and neuroplastic adaptation.

● **HPA-axis dysregulation** – depression shows **hypercortisolaemia** and a blunted feedback. On the **dexamethasone suppression test**, many melancholic/psychotic patients fail to suppress cortisol (**non-suppression**): sensitive but not specific, so not a diagnostic test.

◆ **Genetics / heritability** – bipolar disorder is among the most heritable psychiatric conditions (twin estimates ~**60–85%**); unipolar depression is more modestly heritable (~35–40%). First-degree relatives of bipolar probands carry raised risk of both bipolar and unipolar illness. Detail in the genetics pack.

■ **Kindling (Post)** – early episodes are often precipitated by life stress; with each recurrence episodes become **more autonomous, more frequent and less stress-dependent**. The rationale for **early and continued prophylaxis** in bipolar disorder.

Psychosocial models

● **Learned helplessness (Seligman)** – uncontrollable aversive events produce passivity and a depressive picture; reformulated as a **depressive attributional style** (internal, stable, global attributions for bad events).

● **Cognitive model (Beck)** – depression is driven by negative **schemas** activated by stress, the **negative triad**, and systematic **cognitive distortions** (overgeneralisation, catastrophising, selective abstraction). The theoretical basis of CBT.

● **Life events** – **loss** events (Brown & Harris) precede many first depressive episodes; vulnerability factors (early maternal loss, lack of a confidant, several young children, unemployment) raise risk. Stressors matter most for *early* episodes (cf. kindling).

5 Course, prognosis & suicide risk

● **Course of depression** – episodes are typically self-limiting (median untreated ~6–9 months) but **recurrent**: risk of a further episode rises with each one. Roughly a fifth run a chronic course; residual symptoms predict relapse.

● **Course of bipolar** – recurrent and lifelong; most patients have multiple episodes, with depressive episodes outnumbering manic ones over time and accounting for most of the disability. Inter-episode subsyndromal symptoms are common.

◆ **Suicide burden** – lifetime suicide risk in mood disorders is high; bipolar disorder carries one of the highest rates of any psychiatric illness. Suicide risk is greatest in **severe depression**, **mixed states**, and early in recovery as retardation lifts before mood does.

Suicide risk factors

Domain	Higher-risk markers
Demographic	male sex, older age, single/divorced/widowed, social isolation, unemployment
Clinical	**hopelessness**, severe depression, **mixed states**, psychotic features, comorbid anxiety, insomnia, agitation
Behavioural	**previous attempt** (the strongest single predictor), a clear plan, access to means, recent discharge
Comorbid	alcohol and substance misuse, chronic physical illness, chronic pain

QUICK RULE

The single strongest predictor of completed suicide is a **previous attempt**; the cognition most linked to intent is **hopelessness**. Watch the early-recovery window when energy returns before mood.

6 Management overview

Match the drug class to the pole and the phase. The cardinal bipolar principle: an **antidepressant alone can destabilise** bipolar illness, so cover with a mood stabiliser and treat the depressive pole with agents proven in bipolar depression.

Antidepressants in depression

Class	Examples	Note
SSRI	fluoxetine, sertraline, escitalopram	first-line; best tolerated

Class	Examples	Note
SNRI	venlafaxine, duloxetine	serotonin + noradrenaline; venlafaxine raises BP at high dose
TCA	amitriptyline, imipramine, clomipramine	effective but lethal in overdose (cardiotoxic)
MAOI	phenelzine, tranylcypromine	atypical/refractory depression; tyramine ("cheese") reaction
Atypical / other	mirtazapine, bupropion, agomelatine, vortioxetine	mirtazapine aids sleep/appetite; bupropion lowest switch risk

● CLASSIC TRAP

Antidepressants take **2–4 weeks** to act and should continue **at least 6–9 months after remission** of a first episode to prevent relapse. In **bipolar depression**, antidepressant monotherapy risks a **switch into mania** and rapid cycling: never give one without a mood-stabiliser umbrella, and prefer quetiapine, lamotrigine or lurasidone for the depressive pole.

Mood stabilisers

Agent	Best for	Key caution
Lithium	first-line maintenance; mania; **anti-suicidal**	narrow therapeutic index; thyroid/renal monitoring; level ~0.6–1.0 mmol/L
Valproate	acute mania; maintenance	teratogenic – avoid in women of childbearing potential
Lamotrigine	bipolar depression & depression-predominant maintenance	titrate slowly – **Stevens-Johnson** rash; weak against acute mania
Carbamazepine	mania, esp. rapid cycling / mixed	enzyme inducer; rash, hyponatraemia, blood dyscrasias

◆ **Lithium, the anchor** – **first-line for long-term maintenance** in bipolar disorder, effective against both poles, and the only agent with consistent **anti-suicidal** evidence. Narrow therapeutic window (maintenance ~**0.6–1.0 mmol/L**); monitor renal and thyroid function. Toxicity above ~1.5 mmol/L (tremor, ataxia, confusion, seizures).

■ **Lamotrigine, the depressive-pole agent** – protects best against the **depressive** pole; poor for acute mania. Titrate slowly to avoid a serious rash (Stevens-Johnson / TEN). A practical answer to bipolar depression where antidepressants are risky.

Antipsychotics, ECT & psychotherapy

● **Antipsychotics** – atypicals (olanzapine, quetiapine, risperidone, aripiprazole, lurasidone, cariprazine) treat **acute mania** and serve in maintenance; **quetiapine** and **lurasidone** are evidenced in bipolar depression. Used as adjuncts in psychotic and treatment-resistant unipolar depression.

◆ **ECT** – the most rapidly effective treatment for **severe**, **psychotic**, **catatonic** or **treatment-resistant** depression, where suicide risk is high, food/fluid refusal threatens life, or in severe mania and the puerperium. First-line when speed of response is life-saving.

● **Psychotherapy** – **CBT** (targets the negative triad and distortions) and **IPT** (grief, role transitions, disputes, deficits) have the strongest evidence in depression; behavioural activation and MBCT (relapse prevention) also feature. In bipolar disorder, psychoeducation and adherence work complement pharmacotherapy.

LANDMARK TRIALS & SCALES

Trials: STAR*D (sequenced next-step strategies in unipolar depression) · STEP-BD (bipolar: adjunctive antidepressants added little over mood stabiliser alone) · BALANCE (lithium, alone or with valproate, beat valproate monotherapy for maintenance). **Scales:** clinician-rated depression HAM-D and MADRS; self-rated BDI; mania YMRS (Young Mania Rating Scale).

QUICK-RECALL · DRILL THESE ONE-LINERS

★ **The split:** one manic/hypomanic episode ever → bipolar; depressions only → unipolar. A single full manic episode = bipolar I.

- **Chronic sub-threshold:** persistent depressive disorder (dysthymia) and cyclothymia both need **2 years** (1 year in youth).
- **Depression clusters:** core (low mood, anhedonia, low energy) + biological (early waking, diurnal variation, weight loss, psychomotor change) + cognitive (Beck's negative triad, guilt, hopelessness, suicidality).
- **Specifiers:** melancholic = non-reactive, early waking, weight loss; atypical = reactive mood, hypersomnia, hyperphagia, leaden paralysis. Psychotic = mood-congruent vs incongruent.
- **Mania vs hypomania:** mania ≥ 1 week (or hospitalisation), impaired, may be psychotic; hypomania ≥ 4 days, not impaired, never psychotic.
- **BP-I vs BP-II:** BP-I needs mania; BP-II needs hypomania + a major depressive episode and never full mania.
- **Rapid cycling:** ≥ 4 episodes in 12 months; commoner in women and BP-II. Mixed features = both poles at once, high suicide risk.
- **Epidemiology:** MDD ~10–15% lifetime, F:M ~2:1; bipolar ~1%, F:M ~1:1, earlier onset, higher heritability.
- **Aetiology:** monoamine deficit + therapeutic lag · HPA / dexamethasone non-suppression · kindling (Post) · learned helplessness (Seligman) · cognitive model (Beck) · loss life events (Brown & Harris).
- **Suicide:** strongest predictor = previous attempt; key cognition = hopelessness; watch early recovery.
- **Management:** SSRI first-line for depression; never antidepressant monotherapy in bipolar (switch risk). Lithium = first-line maintenance + anti-suicidal; valproate teratogenic; lamotrigine for the depressive pole; carbamazepine for rapid cycling. ECT for severe/psychotic/refractory.
- **Cross-refs:** STAR*D, STEP-BD, BALANCE; scales HAM-D, MADRS, BDI (self), YMRS (mania).

ONE-DAY REVISION · PAPER 2

Anxiety Disorders

The clinical anxiety group, sorted by what the fear is about and how it is maintained. Fear answers a present threat; anxiety anticipates a future one. Hold the disorder-comparison table, the panic cycle, and the first-line drug order – those carry the marks.

CONCEPT · RULE · TRAP · KEY NUMBER · MNEMONIC

The colour tells you what kind of fact it is. ICD-11 and DSM-5 labels are flagged where they diverge.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. SSRIs and SNRIs are first-line drug for the entire anxiety group; benzodiazepines are a short-term bridge only (dependence, tolerance, rebound), buspirone and pregabalin are GAD options, and beta-blockers cover performance anxiety alone (somatic, not cognitive).
2. Panic attack = abrupt fear peaking within minutes with ≥4 symptoms; panic disorder = recurrent unexpected attacks **plus ≥1 month** of anticipatory worry, and Clark's model = catastrophic misinterpretation of benign bodily sensations.
3. Classification trap: DSM-5 and ICD-11 moved OCD and PTSD *out* of the anxiety group, while separation anxiety and selective mutism moved *in* (no longer childhood-only).
4. GAD = uncontrollable free-floating worry across multiple domains with motor tension and autonomic arousal for at least 6 months (DSM-5); CBT plus graded exposure is first-line psychological, and Mowrer's two-factor model = classical acquisition maintained by operant avoidance.
5. Trap: blood-injection-injury phobia gives a biphasic *vasovagal faint* (not a sympathetic surge), so management adds applied tension, not relaxation.

1 The anxiety grouping & the core concept

Both **ICD-11** and **DSM-5** pulled the anxiety disorders into a coherent block, sorted by the *object* of the fear. The organising contrast underneath all of it is fear versus anxiety.

◆ **Fear vs anxiety** – **fear** is the response to a *present, identifiable* threat (sympathetic surge, fight-or-flight, escape). **Anxiety** is anticipation of a *future, often vague* threat (muscle tension, vigilance, avoidance). Fear is the alarm; anxiety is the smoke detector left on.

The grouping (ICD-11 “Anxiety or fear-related disorders”)

Disorder	The fear is about…
Generalised anxiety disorder	everything – free-floating, multiple domains
Panic disorder	the next panic attack (fear of the fear)
Agoraphobia	situations where escape is hard if panic strikes
Specific phobia	one circumscribed object or situation
Social anxiety disorder	scrutiny and negative evaluation by others
Separation anxiety disorder	separation from attachment figures
Selective mutism	speaking in specific social settings

● CLASSIFICATION TRAP

In **DSM-5**, obsessive-compulsive disorder and PTSD were *removed* from the anxiety chapter into their own groups (OCD-related disorders; trauma-related disorders). ICD-11 mirrors this. So "anxiety disorders" no longer includes OCD or PTSD. Separation anxiety and selective mutism moved *in* (no longer childhood-only).

Normal vs pathological anxiety

Anxiety is adaptive: it sharpens attention and primes the body. It becomes a disorder when it crosses thresholds.

Dimension	Normal anxiety	Pathological anxiety
Proportion	matched to a real stressor	out of proportion or no trigger
Duration	resolves when stressor passes	persistent, self-sustaining
Control	can be set aside	uncontrollable worry
Function	mobilises performance	impairs work, relationships, self-care

The symptom set

Somatic (autonomic + motor)

Cardiac: palpitations, chest tightness. **Respiratory:** dyspnoea, choking, hyperventilation. **Autonomic:** sweating, dry mouth, flushing, tremor, dizziness. **GI:** nausea, "butterflies", urgency. **Motor:** muscle tension, restlessness, startle, fatigue.

Psychological (cognitive + affective)

Apprehension, dread, worry, feeling on edge. **Hypervigilance** and poor concentration. **Derealisation / depersonalisation** in acute surges. Fear of losing control, going mad, or dying. Irritability and initial insomnia.

2 Generalised anxiety disorder

The fear has no single object. Worry floats from topic to topic and is the defining feature.

- **Free-floating worry** – excessive, **uncontrollable** apprehension about multiple everyday domains (health, money, family, work), shifting from one to the next. The patient describes themselves as "a worrier."
- **The tension picture** – the somatic load is **motor tension** (restlessness, muscle ache, trembling, fatigue) and **autonomic over-arousal** with poor concentration, irritability and disturbed sleep. Less paroxysmal than panic, more grinding.
- **Duration** – symptoms most days for **at least 6 months** (DSM-5). ICD-11 emphasises *several months* of general apprehension or worry not restricted to any particular circumstance.

● WORRY MODEL (BORKOVEC)

Worry is conceptualised as a **verbal-linguistic avoidance** of more distressing imagery and somatic arousal: it feels productive and superstitiously "prevents" bad outcomes, so it is negatively reinforced and self-perpetuating. **Intolerance of uncertainty** and **positive and negative beliefs about worry** (metacognition, Wells) maintain GAD. This is what CBT for GAD targets.

3 Panic disorder & agoraphobia

A panic attack is a symptom, not a diagnosis. The disorder is defined by what the attacks *become*.

◆ **Panic attack** – an abrupt surge of intense fear that **peaks within minutes** (typically ~10 min), with ≥4 of a cluster: palpitations, sweating, trembling, dyspnoea, choking, chest pain, nausea, dizziness, chills/heat, paraesthesiae, **derealisation/depersonalisation**, fear of losing control or going mad, and **fear of dying**. Attacks can be *cued* or come out of the blue (*uncued*).

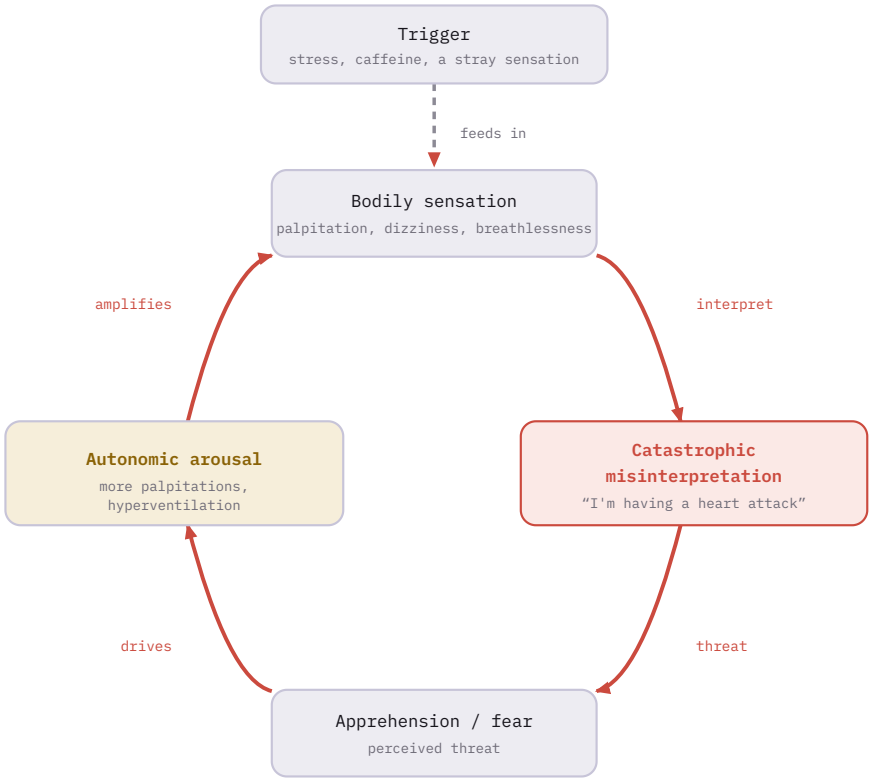
● **Panic disorder** – recurrent *unexpected* panic attacks **plus** ≥1 month of persistent worry about further attacks or a maladaptive change in behaviour to avoid them (**anticipatory anxiety**). The fear is of the panic itself – "fear of fear."

● **Agoraphobia** – marked fear of ≥2 of: public transport, open spaces, enclosed spaces, queues/crowds, being outside the home alone – situations where **escape is difficult or help unavailable** if panic-like symptoms occur. In DSM-5 it is a *standalone* diagnosis, coded separately from panic disorder.

Clark's cognitive model of panic

Clark (1986) explains the spiral: a benign bodily sensation is read as a sign of imminent catastrophe, which amplifies the very sensation. The loop is the high-yield piece.

CLARK'S VICIOUS CYCLE OF PANIC



A trigger feeds in a bodily sensation; the sensation is misread as catastrophic (heart attack, suffocation, madness); fear drives autonomic arousal, which intensifies the sensation, closing the loop. Treatment breaks it at the **misinterpretation** step.

HOLD THIS

Panic **attack** = the symptom cluster, peaks in minutes. Panic **disorder** = recurrent unexpected attacks + a month of anticipatory worry. Clark's model: **catastrophic misinterpretation of benign bodily sensations** is the engine.

4 Phobic disorders

A phobia is fear out of proportion to actual danger, leading to avoidance. They differ by the object of fear.

Phobia	Feared object	Note
Specific phobia	a circumscribed object/situation (animal, blood-injection-injury, natural environment, situational)	commonest anxiety disorder; earliest onset
Social anxiety disorder (social phobia)	scrutiny, embarrassment, negative evaluation	fear of **humiliation**; often onset in adolescence
Agoraphobia	situations where escape/help is hard	see Section 3; the *most disabling* phobia

TRAP

Blood-injection-injury phobia is the exception to the sympathetic surge: it shows a **biphasic vasovagal** response (brief rise then a drop in heart rate and blood pressure), so the patient *faints*. Management adds **applied tension** (tensing large muscles to raise blood pressure) rather than relaxation alone.

◆ **Preparedness theory (Seligman)** – we are **biologically prepared** to acquire fears of evolutionarily dangerous stimuli (snakes, spiders, heights, the dark) far more readily than of modern hazards (cars, electrical sockets). Explains why phobias cluster around ancestral threats and resist extinction.

The childhood-onset pair

● **Separation anxiety disorder** – developmentally excessive fear of separation from major attachment figures: distress on parting, fear that harm will befall them, school refusal, nightmares of separation, somatic complaints on school mornings. Most common anxiety disorder of childhood; can persist or arise in adults.

● **Selective mutism** – consistent failure to speak in *specific* social situations (typically school) despite speaking normally elsewhere (usually home). Lasts ≥1 month (not the first month of school) and is best understood as an **anxiety** phenomenon, strongly linked to social anxiety, not a primary speech or language disorder.

5 Epidemiology & aetiology

Epidemiology

◆ **Frequency & sex** – anxiety disorders are the **commonest psychiatric disorders**, with a 12-month prevalence around 10–18% in community surveys. Marked **female preponderance** (roughly 2:1) across most subtypes.

● **Onset** – generally **early**: specific phobia and separation anxiety in childhood, social anxiety in adolescence, panic disorder and GAD in late adolescence to early/mid adulthood. High comorbidity with depression and with each other.

Genetics

● **Heritability** – moderate, around **30–50%**. Twin studies show what is inherited is a broad **trait of neuroticism / negative affectivity** rather than a specific disorder; the environment shapes which disorder appears. Panic disorder runs in families more tightly than the others.

Neurobiology

● **The fear circuit** – centred on the **amygdala**, which appraises threat and drives autonomic and behavioural fear output via the hypothalamus and brainstem. Normally restrained by the **ventromedial prefrontal cortex**; in anxiety this top-down control is weak and the amygdala is hyper-reactive. The **hippocampus** supplies context; the **BNST** (bed nucleus of stria terminalis) mediates sustained anticipatory anxiety.

System	Role in anxiety	Therapeutic hook
GABA	main inhibitory transmitter; **under**-active tone is anxiogenic	benzodiazepines enhance GABA-A; pregabalin
Serotonin (5-HT)	modulates the amygdala-prefrontal circuit	SSRIs (first-line)
Noradrenaline	drives autonomic arousal	SNRIs; beta-blockers for somatic signs
Locus coeruleus	the brainstem **noradrenergic** hub; stimulation evokes panic-like states	the "alarm" nucleus in panic

● CLASSIC TRAP

The **locus coeruleus** is the panic-relevant nucleus: it is the brain's main noradrenergic source, and yohimbine (an alpha-2 antagonist that fires it) provokes panic, while clonidine (an alpha-2 agonist that quietens it) reduces arousal. **Lactate, CO2 inhalation and caffeine** are reliable panic *provocation* agents, supporting a hypersensitive "suffocation alarm" (Klein).

Cognitive & behavioural models

● **Classical conditioning** – a neutral stimulus paired with fear becomes a conditioned cue (Watson's **Little Albert**). Explains how a phobia is *acquired*.

◆ **Mowrer's two-factor model** – fear is **acquired by classical conditioning** and then **maintained by operant conditioning**: avoidance removes the anxiety and is *negatively reinforced*, so the patient never learns the feared outcome would not occur. **Avoidance is the engine that keeps anxiety alive** – and the target of exposure therapy.

● **Cognitive maintenance** – attentional bias toward threat, catastrophic interpretation (Clark, Section 3), intolerance of uncertainty (GAD), and **safety behaviours** that prevent disconfirmation (carrying pills, sitting near the exit). These keep the belief intact even when no harm occurs.

6 Management

The exam answer is structured: psychological and pharmacological, with first-line agreed and the cautions named.

Psychological

■ **CBT** – the **first-line psychological treatment** across the group. Cognitive restructuring of catastrophic appraisals plus behavioural experiments; for panic, drops safety behaviours and tests the catastrophe directly (interoceptive exposure to induce sensations).

■ **Exposure (& response prevention)** – graded, repeated confrontation of the feared stimulus while **preventing avoidance/escape**, allowing extinction and new (safety) learning. *Systematic desensitisation* (Wolpe): hierarchy + relaxation, reciprocal inhibition. The core treatment of phobias; ERP is the OCD form.

- **Applied relaxation** – trained progressive muscle relaxation deployed at the first cue of anxiety; an evidence-based option especially in GAD. **Applied tension** is its counterpart for blood-injection-injury phobia.

Pharmacological

Drug class	Place in therapy	Caution
SSRIs	first-line for GAD, panic, social anxiety, all phobias	initial jitteriness/worsening – start low, go slow; full effect in weeks
SNRIs (venlafaxine, duloxetine)	first-line alternative, esp. GAD	blood pressure rise at higher venlafaxine doses
Benzodiazepines	**short-term** only – acute/crisis cover while an SSRI takes effect	dependence, tolerance, sedation, rebound; avoid in substance misuse; not for long-term use
Buspirone	5-HT1A partial agonist; option in GAD	slow onset; **not** for panic or PRN use; no dependence
Pregabalin	licensed in GAD; alpha-2-delta calcium-channel ligand	sedation, dependence/misuse potential
Beta-blockers (propranolol)	**performance anxiety**; blunt somatic signs (tremor, palpitations)	do not touch the cognitive/worry component; avoid in asthma

QUICK RULE

SSRIs/SNRIs first-line for the anxiety group. Benzodiazepines only short-term (dependence). Buspirone and pregabalin are GAD options. Beta-blockers are for performance anxiety only – somatic, not cognitive.

MNEMONIC

"Same drugs, slower start, no quick fix"

The same SSRIs treat anxiety and depression; in anxiety you start lower (early jitteriness) and the benzodiazepine is a bridge, never the destination. Avoidance is what exposure must dismantle.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Core split:** fear = present threat (alarm, escape) · anxiety = anticipated future threat (tension, vigilance, avoidance). DSM-5/ICD-11 moved OCD and PTSD *out* of the anxiety group.
- **GAD:** free-floating uncontrollable worry across domains + motor tension/autonomic arousal, ≥6 months. Worry = verbal avoidance (Borkovec); intolerance of uncertainty maintains it.
- **Panic:** attack = abrupt fear, peaks in minutes, ≥4 symptoms · disorder = recurrent unexpected attacks + ≥1 month anticipatory worry. Clark = catastrophic misinterpretation of bodily sensations.
- **Agoraphobia:** fear of situations where escape is hard; standalone in DSM-5; most disabling phobia.
- **Phobias:** specific (commonest, earliest) · social (fear of humiliation/evaluation) · blood-injection-injury = vasovagal faint, treat with applied tension. Preparedness (Seligman) = primed to fear ancestral threats.
- **Childhood pair:** separation anxiety (commonest childhood anxiety) · selective mutism (speaks at home, not at school; an anxiety, not a speech disorder).
- **Epidemiology:** commonest psychiatric disorders · female > male ~2:1 · early onset · heritability ~30–50% (inherits neuroticism, not the specific disorder).
- **Neurobiology:** amygdala-centred fear circuit, weak vmPFC restraint · GABA (anxiolytic, benzodiazepine target), serotonin (SSRI), noradrenaline (LC). Locus coeruleus = panic; lactate/CO2/caffeine provoke panic.
- **Models:** Mowrer two-factor = classical acquisition + operant (avoidance, negatively reinforced) maintenance. Avoidance and safety behaviours keep anxiety alive.
- **Management:** CBT + exposure/ERP first-line psychological · SSRIs/SNRIs first-line drug · benzodiazepines short-term only (dependence) · buspirone & pregabalin for GAD · beta-blockers for performance anxiety (somatic only).

ONE-DAY REVISION · PAPER 2

OCD & Related Disorders

An ego-dystonic engine: an intrusive thought drives anxiety, a compulsion buys relief, and the relief feeds the next thought. Hold the obsession-anxiety-compulsion-relief loop, the DSM-5 related cluster, the cortico-striato-thalamo-cortical circuit, and the two pillars of treatment (SSRI at high dose, and ERP). Every comparison row here is a viva answer in waiting.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. The clean trap is ego-dystonic OCD vs ego-syntonic OCPD; keep them apart from the first line.

II

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. Obsession = ego-dystonic, recurrent, intrusive, resisted thought/image/urge; compulsion = the repetitive overt or covert mental act done to neutralise it; the classic discriminator is ego-dystonic OCD vs ego-syntonic OCPD (and vs depressive rumination, and vs ego-syntonic GAD worry).
2. The maintenance loop: obsession → anxiety → compulsion → relief, where the brief relief negatively reinforces the compulsion; ERP works by blocking the compulsion so anxiety habituates and the loop breaks.
3. Two first-line treatment pillars are an SSRI and ERP; OCD pharmacotherapy differs from depression on two axes, a higher dose and a longer trial of 10-12 weeks, with clomipramine the most serotonergic second-line and antipsychotic augmentation best when tics coexist.
4. Neurobiology is a hyperactive cortico-striato-thalamo-cortical (OFC-caudate-thalamus) loop with direct over indirect pathway imbalance; only serotonergic drugs work, and caudate hypermetabolism normalises with response to either SSRI or ERP.
5. Y-BOCS is the standard severity measure and trial endpoint: 10 items (5 obsessions, 5 compulsions) each scored 0-4 for a total of 0-40; epidemiology is lifetime ~2-3%, equal sexes in adults but male-predominant in childhood, with bimodal onset ~10 yr and ~20 yr.

1 OCD: the core

Two halves, locked together. **Obsessions** are the intrusive thought; **compulsions** are the act that neutralises it. Get the definitional pair exact, because the marks live in the contrast.

	Obsession	Compulsion
What it is	Recurrent, intrusive, unwanted thoughts, images or urges	Repetitive behaviours or mental acts the person feels driven to perform
Felt as	ego-dystonic – alien, against the self, distressing	done to **neutralise** the obsession or prevent a feared event
Relation to reality	recognised (with insight) as one's own mind, not inserted	not realistically connected to what they aim to prevent, or clearly excessive
Response	actively **resisted**, at least early on	aimed at relief, but the relief is brief
Examples	contamination, doubt, "blasphemous" or aggressive images	washing, checking, counting, ordering, silent praying

● RULE

DSM-5 keeps the criterion that the acts are time-consuming (more than **1 hour/day**) or cause marked distress or impairment. ICD-11 drops the "own thoughts" and resistance emphasis but keeps the obsession-compulsion pairing. A compulsion can be **overt** (washing) or a **covert mental act** (counting, neutralising phrases) – both are compulsions.

Common symptom dimensions

OCD content is heterogeneous but clusters into a few stable, factor-analytic **dimensions**. Each obsession theme has its signature compulsion.

Dimension	Obsession	Compulsion
Contamination	dirt, germs, disease, disgust	washing, cleaning, avoidance
Doubt / harm	"did I lock it? turn off the gas?", fear of causing harm	checking, reassurance-seeking
Symmetry / order	need for "just right", incompleteness	ordering, arranging, counting, repeating
Forbidden / taboo thoughts	aggressive, sexual, blasphemous intrusions	mostly covert: mental neutralising, praying, avoidance
Hoarding (now separate)	fear of discarding	saving, acquiring – reclassified as its own disorder in DSM-5

The insight specifier

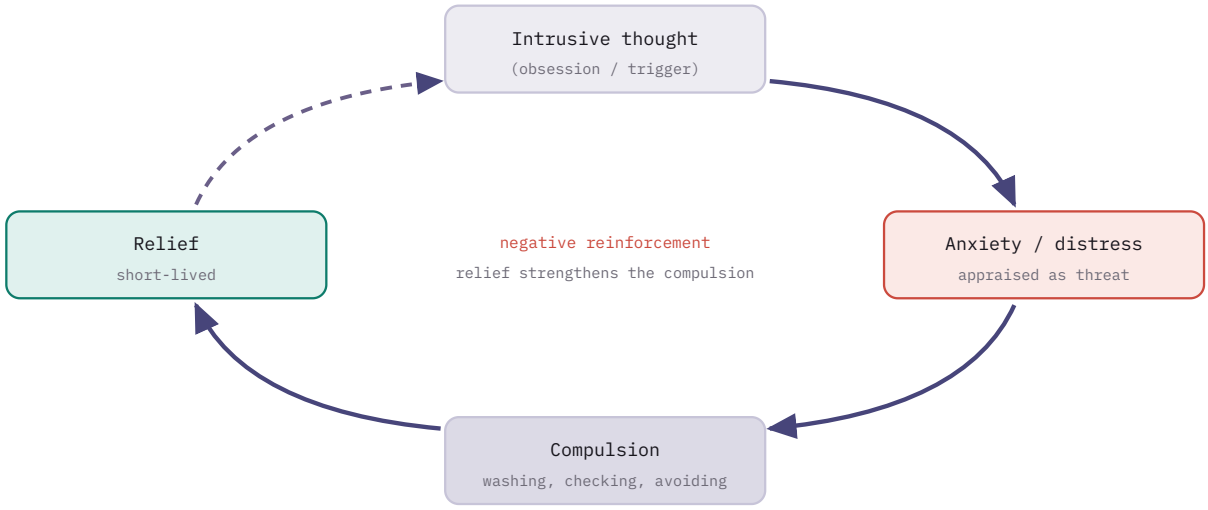
- **Good or fair insight** – recognises the beliefs are probably or definitely not true. The commonest presentation.
- **Poor insight** – thinks the beliefs are probably true.

▲ **Absent insight / delusional beliefs** – completely convinced the beliefs are true. DSM-5 keeps this *within* OCD (with a specifier) rather than coding it as a psychotic disorder. A tic-related specifier also exists.

The maintenance loop

This is the schematic every examiner wants and every therapy targets. The obsession sparks anxiety; the compulsion drops it; the drop in anxiety **negatively reinforces** the compulsion, so the cycle tightens. Avoiding the trigger does the same job and prevents disconfirmation.

THE OBSESSION-ANXIETY-COMPULSION-RELIEF CYCLE



The obsession raises anxiety; the compulsion lowers it; the relief is short-lived and negatively reinforces the compulsion, returning the person to the next intrusion. ERP breaks the loop by blocking the compulsion so anxiety habituates on its own.

Measuring it

◆ **Y-BOCS** – the Yale-Brown Obsessive Compulsive Scale: clinician-rated, the standard severity measure and trial endpoint. A symptom **checklist** maps content, then **10 items** (5 for obsessions, 5 for compulsions) each scored **0–4** on time, interference, distress, resistance and control, for a total of **0–40**. A child version (CY-BOCS) exists.

HOLD THIS

Obsession = the intrusive **thought** (ego-dystonic, resisted). Compulsion = the neutralising **act** (overt or mental). The bridge between them is **anxiety**; the trap that keeps it going is **relief**.

● CLASSIC TRAP

Distinguish three intrusive experiences. An **obsession** is ego-dystonic, repetitive, and drives a compulsion ("my hands are contaminated"). **Rumination** is brooding over a theme, often depressive, without a neutralising act. **Worry** (as in GAD) is about real-life concerns, is more ego-syntonic and verbal, and is not resisted as alien. Obsession content is intrusive and senseless to the patient; worry content feels reasonable to them.

2 The OCD-related cluster (DSM-5)

DSM-5 broke OCD out of the anxiety disorders and gave it a new chapter, **Obsessive-Compulsive and Related Disorders**, grouping conditions that share repetitive thoughts or behaviours and respond to similar treatment. Know each member and exactly how it relates to OCD.

Disorder	Core feature	The repetitive act	How it relates to OCD
Body dysmorphic disorder	preoccupation with a perceived defect in appearance, unnoticeable to others	mirror-checking, comparing, grooming, reassurance-seeking	obsession + compulsion structure; poorer insight; high suicide risk
Hoarding disorder	persistent difficulty discarding, distress at parting, clutter that disables living space	saving and excessive acquisition	split out from OCD; usually *not* ego-dystonic; responds less to SSRI

Disorder	Core feature	The repetitive act	How it relates to OCD
Trichotillomania	recurrent hair-pulling with hair loss	pulling (scalp, brows, lashes); a body-focused repetitive behaviour	tension-relief, not anxiety-neutralising; no true obsession
Excoriation (skin-picking)	recurrent skin-picking causing lesions	picking, often of perceived blemishes	body-focused repetitive behaviour; pairs with trichotillomania
Substance / medication-induced and due to another medical condition	OC symptoms secondary to a substance or illness	varies	aetiological subtypes within the chapter

The body-focused repetitive behaviours

- **Trichotillomania & excoriation** – grouped as **body-focused repetitive behaviours (BFRBs)**. The pull or pick is preceded by rising tension and followed by gratification or relief, but there is **no preceding obsession**. This is the key separation from classic OCD: a habit driven by tension-relief, not an idea that must be neutralised.

The other-specified members

- **Olfactory reference disorder** – preoccupation that one emits a foul body odour others can smell, with checking and camouflage. ICD-11 lists it; DSM-5 places it under other-specified OCRD. Structurally a BDD-like obsession about smell.
- **Hypochondriasis links** – classic hypochondriasis (illness preoccupation with checking and reassurance-seeking) sits at the OCD-anxiety border. DSM-5 split it into **somatic symptom disorder** and **illness anxiety disorder** (in the somatic chapter), but ICD-11 places **hypochondriasis within the OCRD group**, recognising the obsessive checking phenomenology.

TRAP

Not every repetitive behaviour is a compulsion. In OCD the act **neutralises an obsession** and is ego-dystonic. In BFRBs (hair-pulling, skin-picking) and in tics there is **no obsessional driver** – tics are sudden, semi-voluntary movements with a premonitory urge. Naming the driver (obsession vs urge vs none) is what separates these on a viva.

3 Epidemiology & course

Parameter	OCD
Lifetime prevalence	~2–3% (about 1 in 40); 12-month ~1.2%
Sex ratio	roughly equal in adults; **male-predominant** in childhood-onset
Mean onset	late adolescence / early adulthood; **bimodal** – an early peak ~10 yr (more male, more tics) and a later peak ~20 yr
Onset after 35	uncommon; new late onset should prompt a search for organic cause
Course	chronic, **waxing and waning** with stress; complete spontaneous remission is uncommon

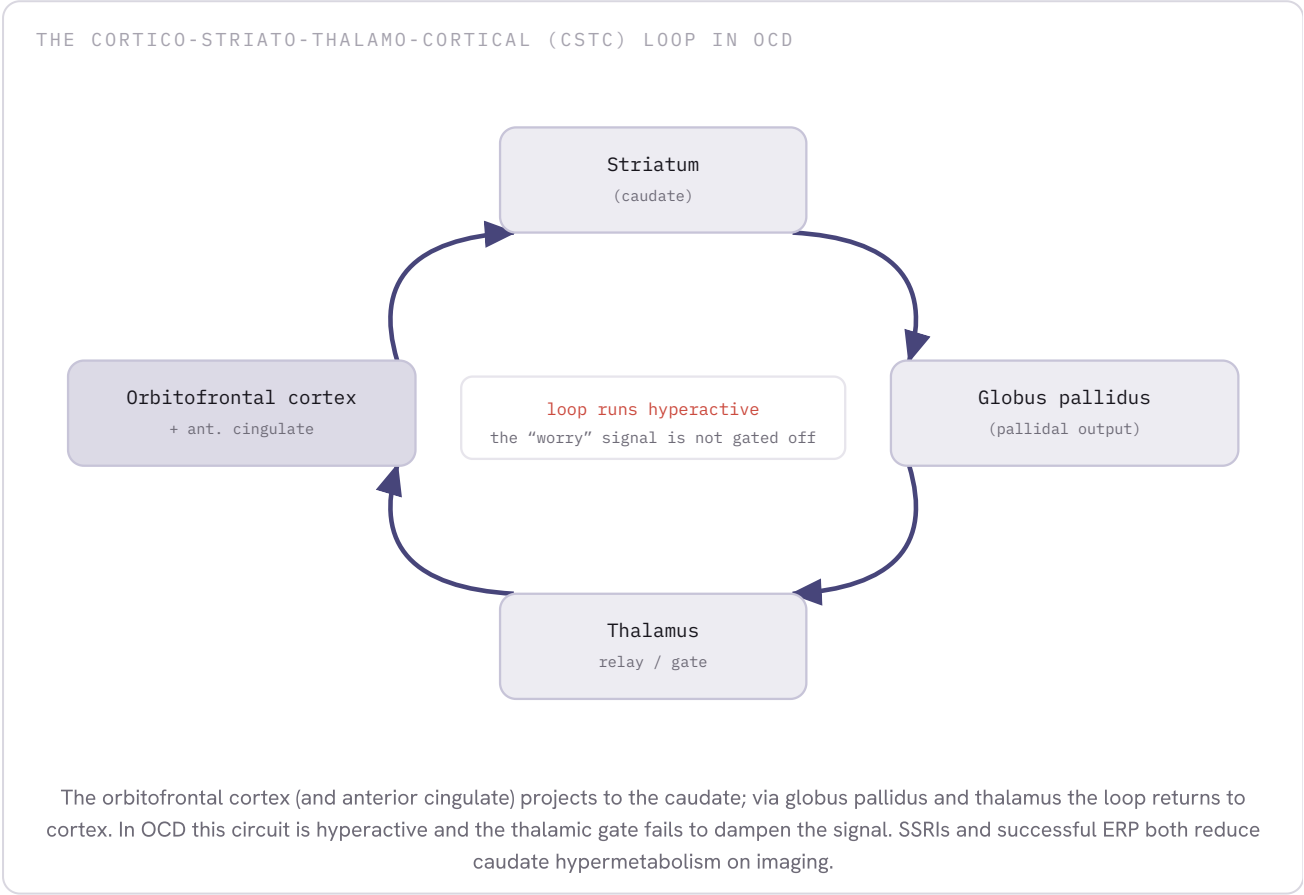
- **Comorbidity** – high. **Major depression** is the commonest comorbidity (mood often follows the OCD, not the reverse). Also anxiety disorders, **tic disorders / Tourette** (especially early-onset, male), BDD, eating disorders, and OCPD traits. Comorbid depression worsens prognosis and raises suicide risk.

- **Prognostic pointers** – better: later onset, good insight, episodic course, absence of compulsions or of comorbidity. Worse: early onset, poor insight, hoarding symptoms, comorbid tics or depression, and a need for hospitalisation.

4 Aetiology

The circuit (cross-ref: neuroanatomy)

The dominant neurobiological model is a hyperactive **cortico-striato-thalamo-cortical (CSTC)** loop, specifically the **orbitofrontal cortex – caudate – thalamus** circuit. The model says the brake on the loop fails, so an alarm signal cycles unchecked.



The orbitofrontal cortex (and anterior cingulate) projects to the caudate; via globus pallidus and thalamus the loop returns to cortex. In OCD this circuit is hyperactive and the thalamic gate fails to dampen the signal. SSRIs and successful ERP both reduce caudate hypermetabolism on imaging.

- **Direct vs indirect pathway imbalance** – the model frames OCD as relative overactivity of the **direct (excitatory)** pathway over the **indirect (inhibitory)** pathway, so the loop disinhibits and a fixed thought-action pattern repeats. Functional imaging shows raised metabolism in OFC, caudate and anterior cingulate that **normalises** with response to SSRI or to ERP.

Neurochemistry

- **Serotonin hypothesis** – the core pharmacological clue: only **serotonergic** agents (SSRIs, clomipramine) work; noradrenergic antidepressants (desipramine) do not. Response to serotonergic drugs implicates 5-HT dysregulation, though it is a downstream marker more than a proven primary lesion. **Glutamate** and dopamine (especially in tic-related OCD) are also implicated.

Genetics & immune

- **Genetics** – familial: first-degree relatives have higher rates, more so for early-onset and tic-related OCD. Twin studies support moderate heritability. Shares genetic loading with Tourette syndrome.

◆ **PANDAS / PANS** – Paediatric Autoimmune Neuropsychiatric Disorders Associated with **Streptococcus**: abrupt-onset OCD and/or tics in a child after group A streptococcal infection, thought to involve cross-reactive antibodies against the **basal ganglia** (molecular mimicry, as in Sydenham chorea). PANS is the broader, non-strep label for the same acute-onset picture.

Psychological model

● **Cognitive model (Salkovskis)** – intrusive thoughts are *normal* and near-universal; OCD arises from how they are **appraised**. An **inflated sense of responsibility** turns a neutral intrusion into a threat ("if I do not check, the harm is my fault"), driving neutralising behaviour.

◆ **Thought-action fusion** – the belief that having a thought is morally or causally equivalent to acting on it ("thinking it makes it more likely / as bad as doing it"). This appraisal bias makes taboo intrusions intolerable and fuels neutralising. Other targets: overestimation of threat, intolerance of uncertainty, perfectionism, need to control thoughts.

■ **Behavioural model (Mowrer two-factor)** – the obsession acquires anxiety by classical conditioning; the compulsion is maintained by operant **negative reinforcement** (it removes the anxiety). This is the theoretical basis of ERP – block the reinforcing escape and the conditioned anxiety extinguishes.

5 Management

Two first-line pillars, used alone or together: a high-dose serotonergic drug and **exposure and response prevention**. Match intensity to severity (a stepped-care logic).

Pharmacotherapy

Agent	Place	The exam points
SSRIs	first-line (fluoxetine, sertraline, fluvoxamine, paroxetine, escitalopram)	need higher doses than in depression and a longer trial (10–12 weeks) to judge response
Clomipramine	most serotonergic TCA; strong evidence, often second-line	used when SSRIs fail; anticholinergic burden, cardiac and seizure caution, ECG/levels
Antipsychotic augmentation	added to an SSRI in resistant cases	low-dose **risperidone, aripiprazole, haloperidol**; best evidence with comorbid tics
Others / experimental	refractory adjuncts	glutamatergic agents, IV clomipramine, high-dose SSRI; weaker evidence

QUICK RULE

OCD pharmacology differs from depression on two axes: **higher dose** and **longer trial**. Continue an effective drug for at least **1–2 years**; relapse on withdrawal is common, so taper slowly.

Psychotherapy: ERP is the core

■ **Exposure and response prevention (ERP)** – the gold-standard psychotherapy and the most specific. The patient is **exposed** to the feared trigger (touching a "contaminated" surface) and **prevented from the compulsion** (not washing). Anxiety rises then **habituates** on its own, disconfirming the belief that the compulsion was necessary. Done graded, up an anxiety hierarchy. A form of CBT.

● **Cognitive therapy** – targets the appraisals (inflated responsibility, thought-action fusion, overestimated threat). Usually combined with exposure; ERP plus cognitive work is standard CBT for OCD.

● STEPPED CARE

Mild OCD: low-intensity CBT/ERP or guided self-help first. Moderate: an SSRI *or* CBT with ERP. Severe: combined SSRI + CBT. ERP works directly on the maintenance loop by removing the negatively reinforcing escape, so anxiety extinguishes – the behavioural mirror image of the serotonin route.

Refractory OCD

● **Defining resistance** – failure of adequate trials (right dose, ≥10–12 weeks) of at least two SSRIs and of clomipramine, plus an adequate course of ERP, before escalating.

◆ **Neurosurgery & neuromodulation** – for severe, truly refractory cases. Ablative procedures target the loop: **anterior cingulotomy**, **anterior capsulotomy**, subcaudate tractotomy, limbic leucotomy. **Deep brain stimulation (DBS)** is the reversible modern option (targets include the ventral capsule/ventral striatum and the STN). **TMS** is an emerging non-invasive add-on. All last-resort, specialist, multidisciplinary.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Core pair:** obsession = recurrent, intrusive, unwanted, **ego-dystonic**, resisted thought/image/urge. Compulsion = repetitive behaviour or **mental act** to neutralise, not realistically connected. Time criterion > 1 hr/day.
- **Dimensions:** contamination/washing · doubt/checking · symmetry/ordering · forbidden thoughts (covert neutralising). Hoarding now separate.
- **Insight specifier:** good/fair → poor → absent/delusional – all stay within OCD. Tic-related specifier too.
- ★ **Loop:** obsession → anxiety → compulsion → relief → (negative reinforcement) → back to obsession. ERP blocks the escape so anxiety habituates.
- **Y-BOCS:** 10 items, 0–4 each, total **0–40**; standard severity measure and trial endpoint.
- **Related cluster (DSM-5):** BDD · hoarding · trichotillomania · excoriation · substance/medical-induced. BFRBs = tension-relief, no obsession. ICD-11 adds olfactory reference & hypochondriasis to the group.
- **Epidemiology:** lifetime ~2–3%; equal sexes in adults, male in childhood; **bimodal onset** (~10 yr and ~20 yr); chronic **waxing-waning**. Commonest comorbidity = depression; also tics/Tourette.
- **Circuit:** hyperactive **CSTC / OFC-caudate-thalamus** loop; direct > indirect pathway; metabolism normalises with SSRI or ERP. Serotonin hypothesis (only serotonergic drugs work). PANDAS = post-strep, anti-basal-ganglia antibodies.
- **Cognitive model:** normal intrusions + **inflated responsibility** + **thought-action fusion** → OCD. Mowrer two-factor underpins ERP.
- **Treatment:** SSRI at **higher dose, longer trial (10–12 wk)**; clomipramine; **ERP** is the core psychotherapy; antipsychotic augmentation (best with tics); refractory → cingulotomy/capsulotomy, DBS.
- **Trap:** OCD **ego-dystonic** (alien, resisted) vs OCPD **ego-syntonic** (the rigid, perfectionistic traits feel right to the person, no true obsessions/compulsions). Obsession vs rumination (depressive brooding, no neutralising) vs worry (GAD, real-life, ego-syntonic).

ONE-DAY REVISION · PAPER 2

Trauma & Stress Disorders

One family, defined not by symptoms but by a cause: an identifiable stressor sits at the head of every diagnosis here. The work is to place each disorder on a timeline, hold the PTSD symptom clusters cold, and keep ICD-11 and DSM-5 apart where they diverge. The cluster table is the centrepiece – learn it as a 10-mark answer.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. The defining feature of this group is a known stressor; everything else is timing and form.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

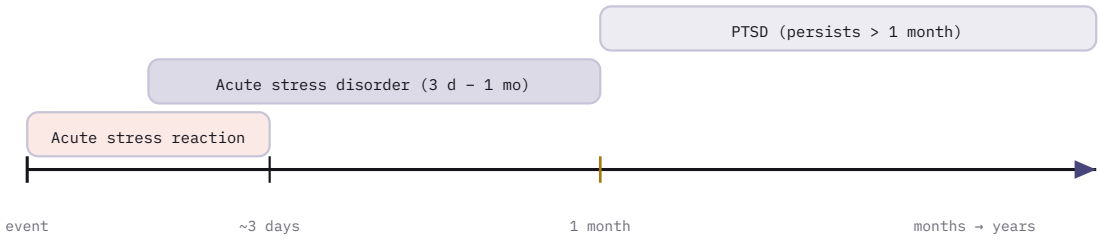
1. The 1-month rule is the single most testable number: trauma-linked symptoms lasting under a month are acute stress disorder (DSM-5), over 1 month they become PTSD; acute stress reaction settles in days and is a normal response, not a disorder.
2. PTSD has four DSM-5 symptom clusters (intrusion, avoidance, negative cognition & mood, hyperarousal) but ICD-11 streamlines to three (re-experiencing in the here-and-now, avoidance, persistent sense of current threat), dropping the negative-cognition cluster.
3. Complex PTSD is an ICD-11-only diagnosis = core PTSD (3) plus the disturbances-in-self-organisation triad (affect dysregulation, negative self-concept, relationship difficulty) after prolonged or repeated trauma; DSM-5 has no Complex PTSD category and instead uses a dissociative subtype.
4. First-line management is trauma-focused CBT and EMDR (NICE / WHO) ahead of drugs (SSRIs / SNRIs, prazosin for nightmares), and routine single-session debriefing is ineffective and may be harmful, so it is advised against.
5. The HPA paradox of PTSD is low cortisol with enhanced negative feedback (cortisol supersuppression on dexamethasone), the opposite of the high-cortisol, blunted-feedback pattern of melancholic depression.

1 The grouping & the stress-response spectrum

What unites this chapter is causation: a traumatic or stressful event is a **required criterion**, not an incidental finding. The disorders then separate along a single axis – **time since the event**. Hold the timeline and most of the topic falls into place.

Condition	Onset after stressor	Duration / course	Status
Acute stress reaction	within minutes	begins to settle in hours, gone in 2–3 days	ICD-11: a normal reaction, not a mental disorder (coded in "factors")
Acute stress disorder	within ~1 month	lasts 3 days to **1 month**	DSM-5 diagnosis; ICD-11 does not list it as a disorder
PTSD	usually within 6 months	symptoms persist **beyond 1 month**	full disorder in both ICD-11 and DSM-5
Complex PTSD	after prolonged / repeated trauma	persistent, pervasive	distinct ICD-11 diagnosis (DSM-5 folds it into PTSD)

THE STRESS-RESPONSE SPECTRUM, BY TIMELINE



The same event can read as a transient reaction, an acute disorder, or PTSD depending only on how long symptoms run. The 1-month line is the watershed: cross it and the diagnosis becomes PTSD.

◆ **The 1-month rule** – the single most testable number here. Trauma-linked symptoms **under a month** are acute stress disorder (DSM-5); **over a month** they are PTSD. Acute stress reaction settles within days and is treated as a normal response.

● CLASSIC TRAP

Acute stress reaction and **acute stress disorder** are not the same entity. The *reaction* (ICD-11) is an immediate, transient, normal stress response lasting hours to a few days, explicitly *not* a mental disorder. The *disorder* (DSM-5) is a defined diagnosis running 3 days to 1 month. ICD-11 dropped acute stress disorder as a diagnostic category.

■ **Delayed onset** – PTSD is labelled **delayed-onset (delayed expression)** when the full criteria are not met until at least **6 months** after the event, even though some symptoms may appear earlier.

2 PTSD: exposure, clusters & the ICD-11 streamline

The exposure (Criterion A)

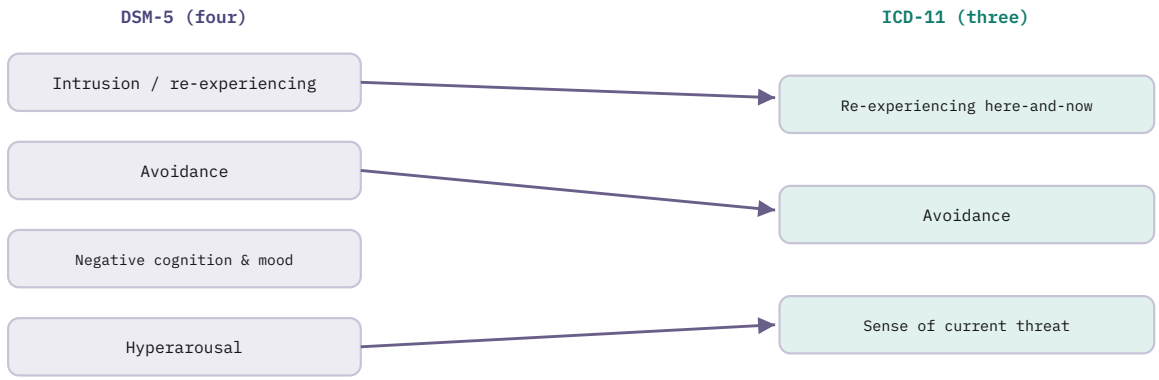
PTSD requires a qualifying event: **actual or threatened death, serious injury, or sexual violence**. DSM-5 allows exposure by directly experiencing it, witnessing it, learning a close other was affected, or repeated occupational exposure to aversive detail (police, first responders). It excludes exposure through media unless work-related.

The four symptom clusters (DSM-5)

This table is the heart of the topic. Learn the four headings, then one or two exemplars each. The ICD-11 model compresses these into three (right column).

Cluster (DSM-5)	Core feature	Examples	ICD-11 equivalent
Intrusion / re-experiencing (B)	the trauma forces its way back into the present	**flashbacks**, nightmares, intrusive memories, psychological & physiological distress to reminders	Re-experiencing in the here-and-now
Avoidance (C)	effortful avoidance of trauma reminders	avoiding thoughts, feelings, people, places, conversations that recall the event	Avoidance
Negative alterations in cognition & mood (D)	persistent negative shift in beliefs and affect	amnesia for part of the event, blame, persistent negative beliefs, anhedonia, detachment, inability to feel positive emotion	(folded into the disorder, not a named cluster)
Hyperarousal / reactivity (E)	a body kept on alert	hypervigilance, exaggerated startle, irritability / anger, reckless behaviour, sleep & concentration problems	Persistent sense of current threat

THE FOUR DSM-5 CLUSTERS COLLAPSE INTO THREE IN ICD-11



ICD-11 keeps PTSD deliberately narrow: re-experiencing in the here-and-now, avoidance, and a persistent sense of current threat (which carries the hyperarousal load). DSM-5's negative-cognition cluster has no separate ICD-11 heading.

◆ **ICD-11 streamlined PTSD** – only **three** required elements: (1) **re-experiencing** in the here-and-now (not just remembering, but reliving with a sense of present reality), (2) **avoidance** of reminders, (3) a persistent **sense of current threat** (hypervigilance, startle). Plus functional impairment, lasting weeks. A tighter, higher-threshold construct than DSM-5.

● MNEMONIC

"I Avoid Negative Hyper-states" → Intrusion, Avoidance, Negative cognition/mood, Hyperarousal

The four DSM-5 clusters in criterion order (B, C, D, E). For ICD-11, drop the Negative cluster and rename the rest: re-experiencing, avoidance, current threat.

● **Flashbacks vs intrusive memory** – a **flashback** is re-experiencing the event as if it were happening *now*, with loss of present-moment awareness; an intrusive memory is recalled as past. ICD-11 hinges its re-experiencing criterion on this here-and-now quality.

● RULE

Onset is usually within 6 months of the trauma, and most cases remit within the first year. A substantial minority run a chronic course (> 3 years). Symptoms present for **more than a month** with functional impairment are required – under a month it cannot be PTSD.

3 Complex PTSD (ICD-11)

An ICD-11 innovation. Complex PTSD = **all three core PTSD features** *plus* three additional "disturbances in self-organisation" (DSO). It maps onto trauma that is **prolonged or repeated** and hard to escape: childhood abuse, domestic violence, torture, captivity, trafficking.

Component	What it covers
Core PTSD (all three required)	
Re-experiencing	flashbacks, nightmares in the here-and-now
Avoidance	of trauma reminders
Sense of current threat	hypervigilance, exaggerated startle
Disturbances in self-organisation (DSO – all three)	

Component	What it covers
Affect dysregulation	heightened reactivity, violent outbursts, or emotional numbing / dissociation; difficulty calming down
Negative self-concept	persistent beliefs of being diminished, defeated, worthless, with deep shame or guilt about the trauma
Disturbed relationships	difficulty feeling close to others, avoiding or disengaging from relationships

HOLD THIS

Complex PTSD = **PTSD core (3) + disturbances in self-organisation (3)**. The DSO triad – **affect dysregulation, negative self-concept, relationship difficulty** – is what separates it from ordinary PTSD, and it follows **prolonged, repeated, inescapable** trauma.

● TRAP

Complex PTSD is an **ICD-11** diagnosis. DSM-5 has *no* separate Complex PTSD category – it captures some of the same presentations under a PTSD **dissociative subtype** (depersonalisation / derealisation). Do not credit DSM-5 with Complex PTSD. Also distinguish it from **borderline personality disorder**: C-PTSD shares affect dysregulation and relational difficulty but lacks the unstable identity, fear of abandonment, and frantic efforts to avoid it; its self-concept is stably negative rather than oscillating.

4 Adjustment disorders & prolonged grief

Adjustment disorder

A maladaptive reaction to an identifiable, usually **non-catastrophic** life stressor (job loss, divorce, illness, migration). The distress is *out of proportion* to the stressor or impairs function, yet does **not** meet criteria for another disorder – it is the residual, sub-threshold category.

Feature	Adjustment disorder
Stressor	identifiable, often everyday; need **not** be life-threatening (unlike PTSD)
Onset	within **1 month** (ICD-11) / 3 months (DSM-5) of the stressor
Response	disproportionate distress or functional impairment, but sub-threshold for any other diagnosis
Duration	resolves within **6 months** of the stressor (or its consequences) ending

● RULE

Adjustment disorder is a **diagnosis of exclusion**: if the picture meets criteria for depression, an anxiety disorder, or PTSD, diagnose that instead. It also requires more than ordinary, expectable distress (which would be a normal reaction, not a disorder).

Prolonged grief disorder

New to both ICD-11 and DSM-5-TR. Grief that is **persistent, pervasive and disabling** well beyond the expected social and cultural norm. The stressor is bereavement; the disorder is the failure of grief to integrate.

◆ **Time threshold** – the grief reaction must persist beyond **6 months** (ICD-11) or at least **12 months** in adults (DSM-5-TR; 6 months in children/adolescents) after the death, and exceed expected cultural norms.

● **Core symptoms** – intense **yearning / longing** for the deceased or persistent preoccupation, plus emotional pain (sorrow, guilt, anger, denial, difficulty accepting the death, feeling part of oneself is lost, emotional numbness, difficulty re-engaging with life).

	Normal grief	Prolonged grief disorder
Course	eases and integrates over time, in waves	persistent and pervasive, does not integrate
Function	recovers; able to re-engage with life	marked, lasting impairment
Timing	most intense early, settles over months	still disabling beyond 6–12 months

TRAP

Grief is **not** the same as a major depressive episode. In grief, dysphoria comes in waves tied to reminders, self-esteem is usually preserved, and any wish to die centres on *joining* the deceased; in depression, mood is pervasively low, worthlessness is prominent, and suicidality is self-directed. DSM-5 removed the old bereavement exclusion, so the two can also co-occur.

5 Aetiology & neurobiology

PTSD is the rare disorder where the necessary cause is known. The interesting question is therefore *why most exposed people do not develop it* – a story of dose, peritraumatic factors, biology and resilience.

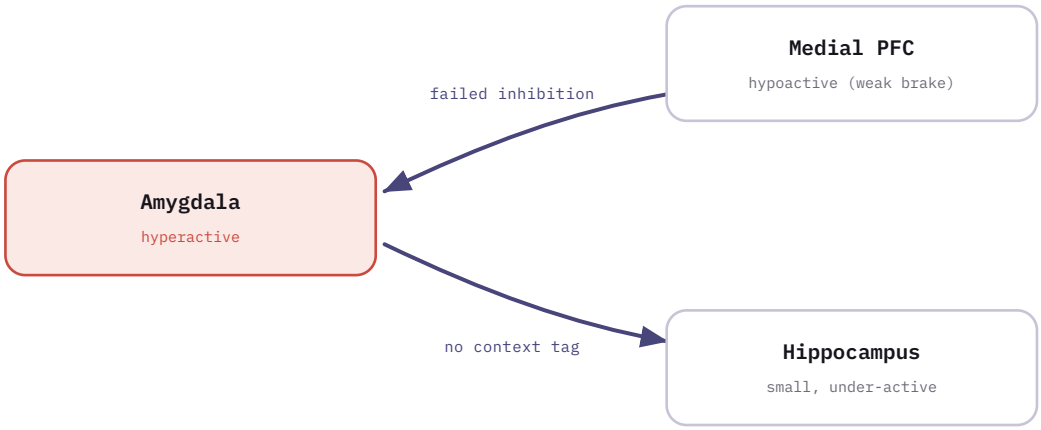
The event and the moment

- **Trauma type & dose** – risk rises with **severity, proximity and duration** of exposure (dose-response). **Interpersonal** trauma (assault, rape, torture) is more pathogenic than impersonal disasters; repeated trauma drives the complex form.
- **Peritraumatic dissociation** – dissociation *during or shortly after* the event (feeling unreal, detached, time distorted) is one of the strongest single predictors of later PTSD. The mind's emergency disconnection foreshadows poor integration of the memory.

The fear circuit

Structure	Finding in PTSD	Consequence
Amygdala	**hyperactive**, hyper-responsive to threat cues	exaggerated fear, startle, vigilance
Medial prefrontal cortex (incl. vmPFC, ACC)	**hypoactive**; weak top-down control	fails to dampen the amygdala → poor fear inhibition
Hippocampus	reduced volume & activity	poor contextualisation; memory feels timeless, ever-present

THE AMYGDALA – PREFRONTAL – HIPPOCAMPUS CIRCUIT



A hyperactive amygdala drives the fear response; a hypoactive medial prefrontal cortex fails to inhibit it (impaired extinction); a small, under-active hippocampus fails to tag the memory as past and place-bound, so it intrudes as ever-present.

■ **Fear conditioning & failed extinction** – PTSD is read as **over-conditioned fear** (the trauma = US; reminders = CS) plus a **failure of extinction**: the safe new learning that should overwrite the fear does not consolidate, because the mPFC cannot recruit it. This is the mechanistic rationale for **exposure therapy** (it builds extinction learning).

The stress axis

◆ **HPA-axis paradox** – counter-intuitively, PTSD is associated with **low circulating cortisol** alongside **enhanced negative feedback** (cortisol supersuppression on the dexamethasone suppression test) and up-regulated glucocorticoid receptors. This is the opposite of the high-cortisol, blunted-feedback pattern of melancholic depression – a favourite distinguishing fact.

● **Noradrenaline** – heightened central noradrenergic tone underpins hyperarousal, startle and trauma nightmares; the rationale for **prazosin** (an alpha-1 antagonist).

Risk & resilience

Raises risk

Pre-trauma: female sex, prior trauma / abuse, prior psychiatric history, low education / SES, family history.
Peri-trauma: severity & proximity, interpersonal trauma, peritraumatic dissociation.
Post-trauma: lack of social support, further life stress, maladaptive appraisals.

Protects (resilience)

Strong **social support** after the event is the most robust protective factor. Also: higher cognitive ability, secure attachment, effective coping, and a sense of mastery / meaning. Early support buffers; debriefing does not.

● RULE

The single best-replicated **protective** factor is post-trauma social support; the single strongest peritraumatic **predictor** is peritraumatic dissociation. If a viva asks “why doesn't everyone exposed get PTSD?”, these two anchor the answer.

6 Management

The evidence is unusually clear: **trauma-focused psychotherapy first**, medication second, and one well-known intervention is to be actively avoided.

First-line: trauma-focused psychotherapy

Therapy	How it works	Note
Trauma-focused CBT (incl. prolonged exposure, cognitive processing therapy)	exposure to the memory / reminders builds **extinction**; cognitive work reappraises trauma-related beliefs	first-line, strongest evidence base
EMDR (eye-movement desensitisation & reprocessing)	recall the trauma while making guided saccades / bilateral stimulation; aids reprocessing (taxing working memory dampens vividness)	first-line (NICE, WHO)

■ **Guideline stance** – NICE and WHO place **trauma-focused CBT and EMDR as first-line** for PTSD, ahead of medication. Psychological treatment is preferred to drugs as the initial step in adults.

Pharmacotherapy

● **SSRIs / SNRIs** – first-line drug treatment. **Sertraline** and **paroxetine** are the SSRIs FDA-approved for PTSD; **venlafaxine** (SNRI) is also evidence-supported. Used when psychotherapy is unavailable, declined, or insufficient.

◆ **Prazosin for nightmares** – an **alpha-1 adrenergic antagonist** that specifically targets **trauma-related nightmares and sleep disturbance**. The reflex answer when a stem mentions PTSD nightmares. (Trial evidence is mixed, but it remains the classic exam choice.)

● **Avoid as a rule** – benzodiazepines are **not recommended** (no benefit on core symptoms, risk of dependence, may impair extinction). Antipsychotics are adjuncts only, for refractory cases.

The intervention to avoid

HIGH-YIELD

Single-session psychological **debriefing** (critical incident stress debriefing) given routinely to everyone after a trauma is **ineffective and may be harmful** – it can increase the risk of later PTSD. NICE and WHO advise **against** it. The correct early step is watchful waiting plus practical and social support.

Children: two attachment disorders

● **Reactive attachment disorder** – following **severe neglect / inadequate care**, the child is **emotionally withdrawn and inhibited**, rarely seeks or responds to comfort. Think the "under-attached," shut-down child.

● **Disinhibited social engagement disorder** – same root of neglect, but the child is **over-familiar and indiscriminate** with strangers, wandering off without checking back. Think the "indiscriminately friendly" child.

● TRAP

Both require a history of pathogenic care (neglect, repeated caregiver changes, institutional rearing). The split is the behavioural direction: **RAD = inhibited / withdrawn**; **DSED = disinhibited / over-friendly**. DSED can persist even after attachment improves; RAD tends to respond to a stable caregiving environment.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Defining feature:** every disorder in this group requires an identifiable **stressor / trauma** as a criterion. They separate by **timeline**.
- **The spectrum:** acute stress reaction (hours–days, a normal response) · acute stress disorder (3 days–**1 month**, DSM-5 only) · PTSD (persists **> 1 month**) · delayed onset if full criteria not met until **6 months**.

- **PTSD Criterion A:** actual / threatened death, serious injury or sexual violence – experienced, witnessed, learned of, or repeated occupational exposure.
- **Four DSM-5 clusters:** Intrusion (flashbacks, nightmares) · Avoidance · Negative cognition & mood · Hyperarousal. Mnemonic: I-A-N-H.
- **ICD-11 PTSD (three):** re-experiencing in the **here-and-now** · avoidance · persistent **sense of current threat**. Narrower than DSM-5; no separate negative-cognition cluster.
- **Complex PTSD (ICD-11 only):** PTSD core **+** disturbances in self-organisation = **affect dysregulation, negative self-concept, relationship difficulty**. Follows prolonged / repeated / inescapable trauma.
- **Adjustment disorder:** non-catastrophic stressor, disproportionate but **sub-threshold** response, onset within 1 month (ICD-11), resolves within 6 months; a diagnosis of exclusion.
- **Prolonged grief disorder:** persistent disabling yearning beyond **6 months (ICD-11) / 12 months (DSM-5-TR)**. Grief ≠ depression (waves vs pervasive; esteem preserved; wish to *join* the deceased).
- **Neurobiology:** amygdala **up**, medial PFC **down** (failed extinction), hippocampus **small**. HPA paradox: **low cortisol + enhanced feedback** (cortisol supersuppression). Noradrenaline up. Peritraumatic dissociation = key predictor; social support = key protector.
- ★ **Management:** first-line = **trauma-focused CBT & EMDR** (NICE / WHO) · drugs = **SSRIs / SNRIs** (sertraline, paroxetine, venlafaxine) · **prazosin** for nightmares · **avoid** routine single-session debriefing (ineffective / harmful) and benzodiazepines.
- **Children: RAD** = inhibited, withdrawn, won't seek comfort · **DSED** = disinhibited, over-familiar with strangers. Both from pathogenic care.

ONE-DAY REVISION · PAPER 2

Dissociative Disorders

A disruption in the normally integrated functions of consciousness, memory, identity, emotion, perception and behaviour. The unifying thread is trauma and the unifying defence is detachment. Hold the continuum, the four core disorders, and the two contrasts that examiners love: depersonalisation vs derealisation, and dissociative vs organic.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. Conversion (dissociative neurological symptom disorder) and Ganser are cross-referenced to the somatic and eponyms packs.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. **Reality testing stays intact** is the single line that separates depersonalisation-derealisation disorder from psychosis: the patient says the world "seems" unreal, the psychotic patient holds the delusion that it *is*.
2. Dissociative amnesia is **retrograde, psychogenic and reversible with new learning intact**; the classic **trap** is confusing it with organic amnesia, which is **anterograde** (cannot form new memories) with confabulation (Korsakoff).
3. Always exclude organic causes first: the cardinal mimic is **temporal lobe epilepsy**, so any first amnesia, fugue or depersonalisation needs an organic screen (EEG, neuroimaging, bloods, toxicology) before a dissociative label.
4. Depersonalisation = detachment from the **self** ("I feel unreal, like a robot"); derealisation = detachment from the **surroundings** (the world looks foggy, dreamlike, two-dimensional).
5. Conversion is the **classification trap**: ICD-11 classes it as **dissociative neurological symptom disorder**, whereas DSM-5 keeps it under somatic symptom and related disorders; DSM-5 also makes fugue a *specifier* of dissociative amnesia, not a separate disorder.

1 The concept of dissociation

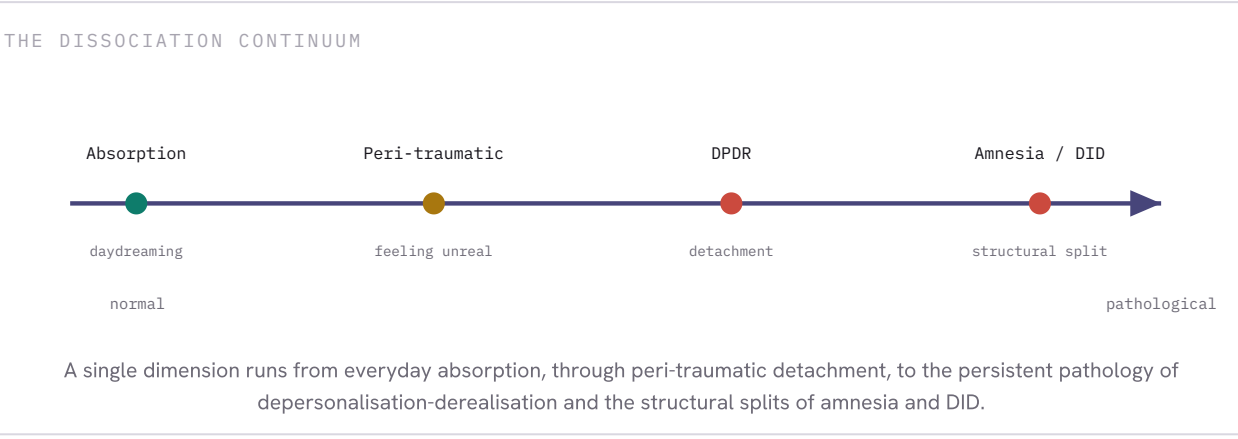
Normally, mind and experience are bound into one integrated stream. In dissociation that binding fails: a piece of consciousness, memory, identity or perception splits off and runs separately. It sits on a **continuum**, from everyday absorption to the severe, chronic pathology of dissociative identity disorder.

- **Dissociation (defining it)** – a disruption of, and discontinuity in, the normal integration of consciousness, memory, identity, emotion, perception, body representation and behaviour. It can be a normal experience, a transient defence, or a disorder.
- **The trauma link** – dissociative disorders are strongly associated with **psychological trauma**, especially severe, early, repeated childhood abuse or neglect. Detachment serves as an escape when no physical escape is possible ("flight when flight is impossible").

The dissociation continuum

Level	Example	Pathological?
Normal absorption	highway hypnosis, getting lost in a film or book, daydreaming	no
Transient / peri-traumatic	feeling unreal or detached during an accident or assault	usually no

Level	Example	Pathological?
Mild pathological	depersonalisation, derealisation episodes	yes, if persistent and distressing
Severe pathological	dissociative amnesia, fugue, identity disorder	yes



A single dimension runs from everyday absorption, through peri-traumatic detachment, to the persistent pathology of depersonalisation-derealisation and the structural splits of amnesia and DID.

Primary, secondary, tertiary dissociation

- **Primary dissociation** – intrusion of a single, undivided traumatic memory (re-experiencing); the classic PTSD pattern, one unintegrated traumatic experience.
- **Secondary dissociation** – peri-traumatic detachment from the experience: out-of-body sensations, emotional numbing, derealisation during or just after the trauma.
- **Tertiary dissociation** – distinct identity states that hold different aspects of the trauma; the structural dissociation seen in **DID**. The split deepens from one memory, to detachment, to separate identities.

FRAME

A useful split: **detachment** phenomena (depersonalisation, derealisation, emotional numbing) versus **compartmentalisation** phenomena (amnesia, conversion/functional neurological symptoms, identity division). Holmes and colleagues proposed this; it maps onto why DPDR sits apart from amnesia/DID.

2 Dissociative amnesia & fugue

An inability to recall important **autobiographical** information, usually of a traumatic or stressful nature, that is too extensive to be ordinary forgetting. The memory is encoded and intact but blocked from retrieval; it is reversible.

- **Core feature** – loss of *personal, autobiographical* memory, not general knowledge or skills. Procedural memory and semantic memory are preserved. This is **retrograde**, psychogenic, and potentially reversible, which separates it from organic amnesia.

Types of dissociative amnesia

Type	What is lost
Localised	all events of a specific period (commonest); the hours around a trauma are blank
Selective	some, not all, events of a period (recalls the accident but not pulling the child out)
Generalised	entire life history and identity lost (rare, dramatic; prompts organic work-up)
Continuous	each new event forgotten as it occurs, from a point in time onward
Systematised	loss confined to one category (e.g. all memory of one person)

Dissociative fugue

■ **Dissociative fugue** – dissociative amnesia *plus* apparently **purposeful travel** away from home or work, with **confusion about personal identity** or assumption of a new identity. The journey looks organised and goal-directed; afterwards the fugue period itself is amnesic.

◆ **Classification note** – in **DSM-5** fugue is a *specifier* of dissociative amnesia, not a separate disorder. In **ICD-11** dissociative amnesia has an optional "with dissociative fugue" qualifier. Either way, fugue = amnesia + travel + identity confusion.

● TRAP

Dissociative amnesia is almost always **retrograde** (loss of past events), preserves the ability to learn new information, and the "lost" memories often return. Organic amnesia (e.g. Korsakoff, head injury) is typically **anterograde** (cannot form new memories), with islands of recall and confabulation. The pattern of loss is the clue.

3 Dissociative identity disorder (DID)

Formerly **multiple personality disorder**. The most severe, chronic dissociative disorder: identity itself is fragmented into two or more distinct personality states, each with its own pattern of relating to the world.

Diagnostic essentials

● **Two or more distinct personality states** – ("alters"), each a relatively enduring pattern of perceiving, relating and thinking. In some cultures described as an experience of possession. There is a marked discontinuity in sense of self and agency.

● **Recurrent gaps in recall** – of everyday events, personal information or trauma, beyond ordinary forgetting. One state may have no access to what another did (**inter-identity amnesia**).

● **Distress or impairment** – the symptoms are not a normal part of a broadly accepted cultural or religious practice, and not due to substances or another medical condition (e.g. not the imaginary play of a child).

● MNEMONIC

"Switching, Splitting, Gaps"

Switching between distinct identity states · the split of one identity into many · amnesic gaps for time and events. Plus near-universal childhood trauma. That quartet is the DID answer.

The trauma model vs the controversy

Trauma / post-traumatic model

DID arises from **severe, repeated childhood trauma** (typically before age 5-9) during the window when a unified self is consolidating. The personality fails to integrate and stays compartmentalised. The dominant clinical view; supported by high rates of reported early abuse.

Sociocognitive / iatrogenic model

DID is shaped (or created) by **suggestion, media and therapy**: leading questions, hypnosis and expectation generate alters in suggestible patients. Points to a culture-bound rise after high-profile cases. The two models remain genuinely debated.

Differential: the three D's to exclude

Mimic	How it differs from DID
Malingering	conscious feigning for an **external incentive** (avoiding court, gain). Symptoms appear under observation, worsen when watched, serve a clear goal

Mimic	How it differs from DID
Factitious disorder	conscious feigning for the **sick role** itself, no external reward
Iatrogenesis	alters elaborated by suggestion/hypnosis in therapy; consider when history emerges only after such techniques
Borderline PD	identity disturbance and dissociation occur, but without distinct, amnesic personality states; the two often coexist
Psychosis	"voices" in DID are experienced as internal (the alters), with intact reality testing, not true hallucinations or formal thought disorder

● RULE

DID voices are typically **inside the head** and ego-dystonic alters communicating; schizophrenic voices are usually experienced as **external** with delusional elaboration. First-rank-like symptoms can occur in DID, so the *context* (trauma history, amnesia, switching) decides, not the symptom in isolation.

4 Depersonalisation-derealisation disorder

Persistent or recurrent detachment, either from the **self** (depersonalisation) or from the **surroundings** (derealisation), with one feature that defines the whole condition: **reality testing stays intact**. The person knows the experience is subjective, not literally true.

Depersonalisation vs derealisation

	Depersonalisation	Derealisation
Detached from	the self – body, mind, feelings, actions	the surroundings – people, objects, world
Typical phrase	"I feel unreal," "like a robot," watching myself from outside	"the world looks unreal," foggy, dreamlike, flat, two-dimensional
Quality	emotional numbing, observing self, body feels alien	objects seem wrong-sized, colourless, lifeless or artificial
Insight	intact – knows it is a feeling, not fact	intact – knows it is a feeling, not fact

◆ **Reality testing intact (the key line)** – this is the single feature that separates DPDR from psychosis. The patient with derealisation says the world "seems" unreal; the psychotic patient holds a delusion that it *is*. Keep this distinction ready for any short note.

● **Course & triggers** – often begins in adolescence/young adulthood; precipitated by severe stress, fatigue, panic, cannabis or hallucinogens. Transient depersonalisation is common in the general population; the *disorder* requires persistence, distress and impairment.

● TRAP

Brief depersonalisation or derealisation is normal (sleep deprivation, after a fright, fatigue) and also occurs within **panic attacks, PTSD, depression, temporal lobe epilepsy and drug intoxication**. Diagnose the standalone disorder only when symptoms are persistent/recurrent, primary, and not better explained by one of those.

5 Trance, possession & ICD-11 placement

Beyond the four core disorders sit the culturally patterned trance and possession states, the ICD-11 home for conversion, and one classic eponym.

Trance and possession

- **Trance disorder** – a marked, involuntary narrowing or loss of the usual sense of personal identity and awareness of surroundings, with restricted, repetitive movements/speech. Identity is *not* replaced by another.
- **Possession trance disorder** – the usual identity is replaced by an external "possessing" identity (spirit, deity, force), attributed by the person's culture, with later amnesia. Only a **disorder** when involuntary, unwanted, outside accepted collective practice, and causing distress or impairment.
- ◆ **ICD-11 grouping** – the dissociative disorders cluster includes dissociative amnesia, trance disorder, possession trance disorder, DID, partial DID, depersonalisation-derealisation disorder, and **dissociative neurological symptom disorder**.

Dissociative neurological symptom disorder (conversion)

- **ICD-11 home for conversion** – what was "conversion disorder" is, in **ICD-11**, classed as a *dissociative* disorder: dissociative neurological symptom disorder. Motor, sensory, gait, seizure (non-epileptic) or cognitive symptoms not explained by a recognised neurological disease. **Note: DSM-5 instead places conversion under somatic symptom and related disorders.** *Cross-ref the somatic pack.*

Ganser syndrome

- **Ganser syndrome** – a rare dissociative disorder marked by **approximate answers** (Vorbeireden: "a cow has 5 legs," 2 + 2 = 5), with clouding of consciousness, somatic conversion features and pseudohallucinations. First described in prisoners; classically termed a "hysterical pseudodementia." *Cross-ref the eponyms pack.*

● CULTURE

Possession and trance are normal, valued experiences in many religious and cultural settings; they become a **disorder** only when involuntary, distressing, impairing and outside the sanctioned practice. Culture-bound presentations (e.g. *amok*, *latah*, spirit possession) overlap this territory. *Cross-ref the culture/anthropology pack.*

6 Aetiology, differential & management

Aetiology: the trauma model

- **Trauma model** – the leading account: **overwhelming, usually early and repeated trauma** (abuse, neglect) drives dissociation as a defence. Detachment and compartmentalisation let the person survive the unbearable; with chronicity these become trait-like and structural (DID). Diathesis: high hypnotisability/dissociative capacity may predispose.

Differential diagnosis (always exclude first)

Category	What to rule out
Organic	temporal lobe epilepsy (complex partial seizures, automatisms, post-ictal amnesia), transient global amnesia, head injury, stroke, dementia, delirium, hypoglycaemia
Substance	intoxication/withdrawal: cannabis, hallucinogens, ketamine, dissociative anaesthetics, alcohol blackouts
Other psychiatric	PTSD, panic disorder, depression, psychosis, borderline PD
Factitious	feigning for the sick role (conscious, no external reward)
Malingering	feigning for external gain (conscious, clear incentive)

HOLD THIS

The cardinal organic mimic is **temporal lobe epilepsy**. Any first presentation of amnesia, fugue, or depersonalisation needs an organic screen (history, exam, EEG, neuroimaging, bloods, toxicology) **before** a dissociative diagnosis is made.

Dissociative vs organic amnesia

Feature	Dissociative (psychogenic)	Organic
Memory lost	retrograde – autobiographical, often around a stressor	anterograde – cannot form new memories
Personal identity	may be lost (esp. generalised, fugue)	usually preserved
New learning	intact	impaired
Onset / course	sudden, stress-linked, often reversible	gradual or insult-linked, less reversible
Confabulation	absent	may be present (Korsakoff)
Other signs	la belle indifference may occur; trauma history	neurological signs, abnormal EEG/imaging

Management: stabilise, then process

■ **Safety & stabilisation first** – the universal first phase: ensure physical safety, manage self-harm/suicide risk, establish a stable therapeutic alliance, build grounding and affect-regulation skills *before* any trauma processing. Premature trauma work can destabilise.

● **Phased trauma-focused therapy** – the standard frame: **(1) safety and stabilisation → (2) processing traumatic memories → (3) integration and rehabilitation**. Psychotherapy is the mainstay; no drug treats dissociation itself.

● **Treat the comorbidity** – pharmacotherapy targets *comorbid* PTSD, depression and anxiety (e.g. SSRIs), not the dissociation directly. Comorbidity is the rule, so treat it.

▲ **Caution: hypnosis & recovered memories** – hypnosis and abreaction may aid stabilisation or memory recovery in skilled hands, but carry a real risk of generating **false memories** and can be iatrogenic. The recovered-memory/false-memory debate sits here. Use cautiously, never suggestively, and never as a forensic truth test.

● RULE

For most acute dissociative amnesia and fugue, removing the person from the stressor and providing a **safe, supportive environment** allows spontaneous recovery; the prognosis for single-episode amnesia/fugue is good. DID is chronic and needs long-term phased psychotherapy. Prognosis worsens with chronicity, comorbidity and ongoing trauma.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Concept:** dissociation = disruption of the integration of consciousness, memory, identity, emotion, perception, behaviour. Trauma-linked. Continuum: absorption → peri-traumatic → DPDR → amnesia/DID. Primary (one memory) → secondary (detachment) → tertiary (separate identities).
- **Amnesia:** loss of autobiographical memory, too extensive for ordinary forgetting; retrograde, reversible, new learning intact. Types: localised (commonest), selective, generalised, continuous, systematised.
- **Fugue:** amnesia + purposeful travel + identity confusion/new identity. DSM-5 = specifier of dissociative amnesia.
- **DID:** ≥2 distinct personality states + recurrent amnesic gaps + childhood trauma. Trauma model vs sociocognitive/iatrogenic debate. Exclude malingering (external gain), factitious (sick role), iatrogenesis.
- ★ **DPDR:** depersonalisation = detached from the *self*; derealisation = detached from *surroundings*. Key line: **reality testing intact** (separates from psychosis).

- **Trance/possession:** trance = narrowed identity; possession = identity replaced by external agent + amnesia. Disorder only when involuntary, distressing, outside sanctioned cultural practice.
- **ICD-11:** conversion = **dissociative neurological symptom disorder** (DSM-5 keeps it under somatic). Ganser = approximate answers (Vorbeireden) + clouding, "hysterical pseudodementia."
- **Differential:** exclude organic first – cardinal mimic is **temporal lobe epilepsy**; also substances, PTSD, panic, depression, factitious, malingering.
- **Dissociative vs organic amnesia:** dissociative = retrograde, identity may go, new learning intact, no confabulation; organic = anterograde, identity preserved, confabulation possible.
- **Management:** safety & stabilisation first → phased trauma-focused therapy → integration. Psychotherapy is the mainstay; drugs treat comorbid PTSD/depression only. Hypnosis/recovered memories used with caution (false-memory risk).

ONE-DAY REVISION · PAPER 2

Somatic symptom disorders, made retrievable

Bodily distress with a psychological core. The chapter the examiner loves because one table settles it: who is aware of the symptom, who is aware of the motive, and whether anything external is gained. Hold conversion vs factitious vs malingering and most of the marks are already yours.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. DSM-5 and ICD-11 labels are both flagged, since either may be asked.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. The three-way contrast is the whole topic: conversion = symptom unconscious with no external gain, factitious = symptom conscious but motive unconscious (the sick role), malingering = symptom conscious and motive conscious (external incentive); the two awareness questions are independent.
2. The DSM-5 pivot is from medically unexplained symptoms to positive psychological criteria (excessive thoughts, feelings, behaviours about the symptoms); a symptom may coexist with real disease, so DSM-5 = Somatic Symptom and Related Disorders and ICD-11 = bodily distress disorder.
3. SSD vs illness anxiety disorder splits on one axis: somatic symptoms are present and distressing in SSD but minimal or absent in IAD (old hypochondriasis, now a fear of illness itself); both run ≥ 6 months.
4. Conversion is now a rule-in diagnosis from positive incompatibility signs (Hoover's sign, give-way weakness, tremor entrainment); a positive sign is NOT proof of faking, and la belle indifference is classic but unreliable (occurs in organic disease, not diagnostic).
5. Factitious disorder imposed on another (old Munchausen by proxy) is a form of child abuse and a safeguarding emergency: the diagnostic label attaches to the perpetrator, while the child is the victim, never the patient with the disorder.

1 The concept and the reclassification

These disorders share one feature: prominent bodily symptoms that cause distress or impairment, with a psychological component that is central, not incidental. The 2013 DSM-5 revision changed the defining logic of the whole group.

The shift away from “medically unexplained”

■ Old logic (DSM-IV somatoform) – a symptom counted only if it was **medically unexplained**. This needed the clinician to prove a negative, was unreliable, and felt dismissive to patients (“it is all in your head”).

● New logic (DSM-5) – diagnosis rests on **positive psychological criteria**: the patient's excessive thoughts, feelings and behaviours about their symptoms. A symptom can coexist with real medical disease; what matters is the disproportionate response to it.

- **The group renamed** – DSM-5 replaced "somatoform disorders" with **Somatic Symptom and Related Disorders**. Hypochondriasis was split; somatisation and pain disorder folded into somatic symptom disorder.

DSM-IV term	DSM-5 successor
Somatisation disorder / undifferentiated	Somatic symptom disorder (SSD)
Pain disorder	SSD, with predominant pain
Hypochondriasis	split: ~75% → SSD; ~25% → illness anxiety disorder
Conversion disorder	Conversion disorder (functional neurological symptom disorder)

The ICD-11 line: bodily distress disorder

- **ICD-11 bodily distress disorder (BDD)** – ICD-11 collapsed the old somatoform category into a single diagnosis: **bodily distress disorder**. Defined by distressing bodily symptoms plus **excessive attention** directed to them, graded mild / moderate / severe. Conceptually close to DSM-5 SSD.

● NAMING TRAP

ICD-11 **bodily distress disorder** is not the same as the older **bodily distress syndrome** (Fink, a research construct). And health anxiety with few somatic symptoms sits in ICD-11 under **hypochondriasis**, grouped with the OCD-related disorders, not with BDD. Watch the wording in the stem.

HOLD THIS

The pivot is from **medically unexplained symptoms** to **positive psychological criteria**. DSM-5 = Somatic Symptom and Related Disorders; ICD-11 = bodily distress disorder.

2 Somatic symptom disorder vs illness anxiety disorder

Two disorders that look alike at the door but split on one axis: how many somatic symptoms are actually present.

Somatic symptom disorder (SSD)

- **Core** – one or more **distressing somatic symptoms** (pain, fatigue, gut symptoms) plus **excessive thoughts, feelings or behaviours** about them: disproportionate health worry, persistent high anxiety, excessive time and energy devoted to symptoms.
- **Duration** – the symptomatic state is **persistent, typically > 6 months** (any one symptom need not persist, but the symptomatic state does). Specifiers: with predominant pain; persistent; mild / moderate / severe.

Illness anxiety disorder (IAD) · formerly hypochondriasis

- **Core** – **preoccupation with having or acquiring a serious illness**, with somatic symptoms that are **mild or absent**. High health anxiety; either excessive checking or maladaptive avoidance of doctors. Preoccupation present **≥ 6 months**, though the feared illness may change.
- **The split** – old hypochondriasis divided: those with prominent somatic symptoms became SSD; those whose problem is the **fear of illness itself**, with few symptoms, became IAD.

Feature	Somatic symptom disorder	Illness anxiety disorder
Somatic symptoms	present and distressing	minimal or absent
The focus of worry	the symptoms themselves	the *idea* of having a serious illness

Feature	Somatic symptom disorder	Illness anxiety disorder
Driving emotion	distress about the body	anxiety about disease and death
Behaviour	excessive time / energy on symptoms	care-seeking type (checking) or care-avoidant type
Duration	persistent, > 6 months	preoccupation ≥ 6 months

● RULE

The discriminator is the **presence of somatic symptoms**. Many symptoms with disproportionate response = SSD. Few or no symptoms but a fixed fear of illness = IAD. Reassurance helps neither for long.

3 Conversion disorder (functional neurological symptom disorder)

Neurological symptoms that are real to the patient but do not fit any recognised neurological disease. The diagnosis is now made on **positive evidence of incompatibility**, not merely on the absence of a lesion.

● **Core** – altered **voluntary motor or sensory function** (weakness, paralysis, tremor, gait disorder, non-epileptic seizures, blindness, aphonia, anaesthesia) with clinical findings of **incompatibility** between the symptom and recognised neurological conditions.

■ **Rule-in, not rule-out** – DSM-5 dropped the requirement that psychological stress be identifiable and the requirement to exclude feigning. The symptom is **not intentionally produced**; the patient is not faking.

Positive signs (the high-yield list)

Sign	What it shows
Hoover's sign	in functional leg weakness, hip **extension** is weak on direct testing but normal when the *opposite* hip is flexed against resistance (involuntary, so it returns). The classic positive sign.
Give-way (collapsing) weakness	strength is normal momentarily, then suddenly gives way; not the smooth fixed weakness of a pyramidal lesion
Tremor entrainment	a functional tremor changes frequency to match, or stops during, a voluntary rhythmic task with the other hand
Non-epileptic seizures	features such as long duration, closed eyes resisting opening, side-to-side movements, retained awareness; normal ictal EEG, normal prolactin

▲ **La belle indifference** – a striking lack of concern about a serious deficit. Classically described, much loved by examiners, but **unreliable**: it occurs in organic disease too and is not diagnostic. Quote it, then qualify it.

The older hysteria / dissociation link

● **Historical lineage** – conversion descends from **hysteria** (Briquet, Charcot) and Freud's idea that intolerable psychic conflict is **"converted"** into a bodily symptom, with **primary gain** (anxiety kept out of awareness) and **secondary gain** (care, role relief).

● **ICD-11 placement** – ICD-11 keeps this lineage explicit: conversion sits under **dissociative neurological symptom disorder**, within the dissociative disorders, not the somatic group. DSM-5 places it among the somatic symptom disorders. Know both homes.

● TRAP

A positive Hoover's sign or tremor entrainment is a **rule-in** for a functional disorder, not proof of malingering. The conversion patient is not consciously producing the sign. Misreading positive signs as "caught faking" is the commonest clinical error.

4 Factitious disorder

Here the symptom *is* deliberately produced. What makes it a disorder, not malingering, is the motive: there is no external reward, only the psychological pull of being a patient.

● **Core** – **intentional falsification** of physical or psychological signs or symptoms, or induction of injury or disease, associated with **identified deception**. The person presents themselves (or another) as ill, impaired or injured.

◆ **The motive (defining)** – the goal is to **assume the sick role**. There is **no obvious external incentive** (no money, no drugs, no escaped duty). This is what separates it from malingering.

Subtype	Older name	Note
Imposed on self	Munchausen syndrome	severe, chronic form; pseudologia fantastica, hospital-hopping, willingness to undergo procedures
Imposed on another	Munchausen syndrome by proxy	a caregiver fabricates or induces illness in a dependent; diagnosis is given to the **perpetrator**, not the victim

● SAFEGUARDING TRAP

Factitious disorder **imposed on another** is a recognised form of **child (or dependent) abuse** and a child-protection emergency. The diagnostic label attaches to the perpetrator; the child is the victim and needs immediate safeguarding. Never frame the victim as the patient with the disorder.

● **Methods** – fabricating history, tampering with tests (warming a thermometer, adding blood to urine), injecting faeces or insulin, taking warfarin or thyroxine covertly. Suspect when the course is implausible, signs appear only unobserved, or the patient seeks procedures eagerly.

5 Malingering and the high-yield contrast

▲ **Malingering** – the **intentional production or gross exaggeration** of symptoms motivated by an **external incentive**: money, drugs, avoiding work, military duty, school, criminal prosecution, or obtaining benefits. It is **not a mental disorder**; in DSM-5 it is a V-code (a focus of clinical attention), in ICD-11 a Z-code.

The centrepiece table: who knows what, and is anything gained

	Conversion disorder	Factitious disorder	Malingering
Symptom produced…	unconsciously (not feigned)	consciously (deliberately faked / induced)	consciously (deliberately feigned)
Motive is…	unconscious (intrapsychic conflict)	unconscious drive to be a patient	conscious and explicit
Goal / gain	no external gain (primary & secondary gain only)	the **sick role** itself; no external reward	a tangible **external incentive**
A mental disorder?	yes	yes	no (V / Z-code)

	Conversion disorder	Factitious disorder	Malingering
Behaviour when unobserved	persists	may worsen / be induced	often resolves once goal is met

QUICK RULE

Conversion = symptom unconscious, no gain. **Factitious** = symptom conscious, motive unconscious (sick role). **Malingering** = symptom conscious, motive conscious (external gain). Read the table left to right as one continuous spectrum of awareness.

MNEMONIC

"Sick role, not a paycheck"

Factitious chases the **sick role** (internal, unconscious motive); malingering chases a **paycheck** or some other external reward (conscious motive). Both feign the symptom; only the malingerer feigns it *for* something tangible.

CLASSIC TRAP

The two awareness questions are independent. Factitious sits in the middle: the **symptom is conscious** (they know they are faking it) but the **motive is unconscious** (they do not grasp why they need to be ill). Confusing factitious with malingering, or with conversion, is the single most common error on this topic.

6 Older terms, related conditions, aetiology and management

The older somatoform terms (still examinable)

- **Somatisation / Briquet syndrome** – the DSM-IV picture: many recurrent somatic complaints across multiple organ systems, beginning before age 30, running a chronic course, with extensive but unrewarding investigation. Briquet's eponym for polysymptomatic somatisation. Now subsumed under SSD.
- **Persistent (somatoform) pain disorder** – pain as the dominant complaint, judged to have a major psychological contribution. Now SSD with predominant pain; ICD-11 has **chronic primary pain** in its own pain chapter.
- **Other related labels** – **body dysmorphic disorder** moved out, to the OCD-related group. **Pseudocyesis** (false belief of pregnancy with physical signs) sits among the "other specified" somatic presentations.
- **Psychological factors affecting other medical conditions** – a genuine medical condition is present, and psychological or behavioural factors **adversely affect its course** (poor adherence, denial of risk, stress worsening asthma or arrhythmia). The medical illness is real; the psychological factor is the clinical problem.

Aetiology (lines to drop in an essay)

Predisposing & psychological

Childhood adversity, abuse and neglect; early experience of illness; **alexithymia** (difficulty naming emotion, so distress is felt bodily); insecure attachment; high trait neuroticism. Psychodynamic: distress **converted** into somatic form, with primary and secondary gain.

Cognitive, behavioural & social

Somatosensory amplification and catastrophic misinterpretation of normal sensations; selective attention to the body; the **sick role** reinforced by care and avoidance of duty. Iatrogenic reinforcement through repeated tests. More common in **women** and where psychological distress is stigmatised.

Management (the consistent answer)

■ **One consistent clinician** – a **single named doctor** coordinates care. Prevents doctor-shopping, contradictory opinions and serial investigation.

■ **Regular, brief, scheduled visits** – appointments **by the calendar, not by symptoms**, so that care is not contingent on being unwell. This de-links attention from new complaints.

■ **Avoid over-investigation** – resist unnecessary tests, referrals and procedures; each carries iatrogenic harm and reinforces illness behaviour. Examine, acknowledge the symptom as real, then limit the work-up.

■ **Treat comorbidity** – actively look for and treat **depression and anxiety**; antidepressants (SSRIs, and SNRIs / TCAs where pain dominates) help both mood and somatic symptoms.

◆ **Psychotherapy · CBT first-line** – **CBT** has the strongest evidence: it targets catastrophic illness beliefs, reduces checking and reassurance-seeking, and reframes bodily sensations. Reattribution, mindfulness-based approaches and brief psychodynamic work are alternatives. Reassurance alone does not work and may reinforce.

QUICK RULE

The management stem answers itself: **one clinician, regular brief visits, minimal investigation, treat depression/anxiety, CBT**. Validate the symptom, shift the goal from cure to function.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **The big shift:** from **medically unexplained symptoms** to **positive psychological criteria**. DSM-5 = Somatic Symptom and Related Disorders; ICD-11 = bodily distress disorder (graded mild/moderate/severe).
- **SSD vs IAD:** SSD = distressing somatic symptoms + excessive thoughts/feelings/behaviours. IAD (old hypochondriasis) = preoccupation with serious illness, symptoms **minimal or absent**. Discriminator = presence of somatic symptoms. Both ≥ 6 months.
- **Conversion (FNSD):** voluntary motor/sensory loss incompatible with disease; **rule-in** by positive signs – Hoover's sign, give-way weakness, tremor entrainment. **La belle indifference** is classic but unreliable. ICD-11: dissociative neurological symptom disorder.
- **Factitious:** intentional falsification, motive = the **sick role**, no external gain. Imposed on self = Munchausen; imposed on another (old Munchausen by proxy) = a form of **abuse**, diagnosis given to the perpetrator.
- **Malingering:** conscious feigning for an **external incentive** (money, drugs, duty). **Not a mental disorder** (V/Z-code).
- ★ **Centrepiece:** conversion = symptom unconscious, no gain · factitious = symptom conscious, motive unconscious (sick role) · malingering = symptom conscious, motive conscious (external gain).
- **Older terms:** somatisation = Briquet syndrome (polysymptomatic, onset < 30) → SSD. Persistent pain disorder → SSD with predominant pain. Body dysmorphic disorder moved to OCD-related group.
- **Psychological factors affecting other medical conditions:** the medical illness is real; psychological/behavioural factors worsen its course.
- **Management:** one consistent clinician · regular brief scheduled visits · avoid over-investigation · treat depression/anxiety · **CBT** first-line. Validate the symptom; reassurance alone fails.

ONE-DAY REVISION · PAPER 2

Personality Disorders

An enduring, pervasive, inflexible pattern of inner experience and behaviour that deviates from cultural expectation, stable from adolescence, and causes distress or impairment. The three cluster tables are the spine of the answer. Learn the one core feature of each disorder, then the depth on borderline and antisocial, then how the field moved from boxes to dimensions.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. DSM-5 keeps the 10 categories; ICD-11 has dropped them for a dimensional rating.

THE THREE CLUSTERS AT A GLANCE

Cluster A · odd / eccentric

Disorders	Paranoid, Schizoid, Schizotypal
Core thread	social oddity, constricted or suspicious relating
Keyword	the “weird” cluster; schizophrenia spectrum

Cluster B · dramatic / erratic

Disorders	Antisocial, Borderline, Histrionic, Narcissistic
Core thread	emotional dysregulation, impulsivity, disturbed relating
Keyword	the “wild” cluster; BPD and ASPD carry the marks

Cluster C · anxious / fearful

Disorders	Avoidant, Dependent, Obsessive-compulsive (anankastic)
Core thread	anxiety and fear: of rejection, of being alone, of disorder
Keyword	the “worried” cluster; most treatable

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. DSM-5 keeps 10 categorical types in 3 clusters; ICD-11 abolished the categories, rates severity first (mild/moderate/severe) then 5 trait domains, and keeps only a **borderline pattern** qualifier: the direction of travel is categorical → dimensional.
2. The 5 ICD-11 trait domains are negative affectivity, detachment, dissociality, disinhibition, anankastia, mapping onto the maladaptive poles of the Big Five.
3. BPD self-harm is recurrent and completed suicide reaches roughly 1 in 10; Linehan's biosocial model = emotional vulnerability × invalidating environment, the rationale for DBT, which carries the strongest evidence base.
4. Antisocial PD requires conduct disorder before age 15 and is not coded before age 18; psychopathy is the narrower affective-core construct measured by the Hare PCL-R (20 items, max 40, cut-off ≥30).
5. Trap: OCPD is an ego-syntonic Cluster C character style with **no true obsessions or compulsions**, whereas OCD is ego-dystonic symptoms the patient resists; and schizoid (alone, content) vs schizotypal (alone, odd cognition) vs avoidant (alone, longing) turn on desire-for-contact and odd-cognition.

1 Concept and general criteria

A personality disorder is not a single symptom but the **shape of the person**. The pattern is trait-like, not episodic, and the threshold is general before any specific type is named.

The five-part general definition

- **Enduring & pervasive** – a stable pattern of **cognition, affectivity, interpersonal function and impulse control** that shows across a broad range of personal and social situations, not just one relationship or setting.
- **Deviates from culture** – the pattern departs markedly from the expectations of the individual's culture. Culture is the yardstick, so behaviour normal in one context is not pathologised in another.
- **Inflexible** – rigid and maladaptive across situations; the person cannot flex the pattern even when it clearly costs them.
- **Early & stable onset** – traceable to **adolescence or early adulthood** and stable over time. Diagnosis under 18 is made cautiously; antisocial PD is not coded before age 18.
- **Distress or impairment** – the pattern causes clinically significant distress or impairment in social, occupational or other functioning, and is not better explained by another mental disorder, a substance, or a medical condition.
- ◆ **Ego-syntonic** – the defining experiential mark: the person sees the traits as **part of the self**, not as an alien intrusion. Contrast the **ego-dystonic** obsessions of OCD, which the patient resists. This is why insight is poor and the person rarely presents for the personality itself.

RULE

The general criteria must be met **before** a specific type is diagnosed. A pattern that is recent, situation-bound, or fully explained by a mood, psychotic or substance disorder is not a personality disorder. Enduring, pervasive, inflexible, early, distressing – all five.

2 The two classifications

The major shift in this topic: from a **categorical** model (you have it or you don't, and which of ten) to a **dimensional** one (how severe, and along which trait axes). Examiners want both, and the reason for the move.

	DSM-5 (categorical)	ICD-11 (dimensional)
Core unit	10 discrete disorders in 3 clusters (A, B, C)	a single PD diagnosis, then rated
First step	match the criteria set for a named type	rate severity: mild / moderate / severe
Then	specify which type(s); comorbid types common	specify **trait domain qualifiers**
Retained types	all ten named	categories abolished; only a **borderline pattern** qualifier kept
Criticised for	arbitrary thresholds, huge overlap, "not otherwise specified" overuse	loss of familiar clinical shorthand; clinician retraining

The five ICD-11 trait domains

After severity, the clinician flags any prominent trait domains. They map loosely onto the maladaptive poles of the Big Five.

Trait domain	Captures	Big-Five pole
Negative affectivity	broad range of negative emotions: anxiety, anger, mistrust, lability	high neuroticism
Detachment	social and emotional withdrawal, avoidance, restricted affect	low extraversion
Dissociality	disregard for others, self-centredness, callousness, deceit	low agreeableness
Disinhibition	impulsivity, recklessness, acting on immediate gratification	low conscientiousness

Trait domain	Captures	Big-Five pole
Anankastia	rigid perfectionism, control, orderliness	high conscientiousness (extreme)

■ **Borderline pattern qualifier** – the single categorical survivor in ICD-11. Added to preserve a label that drives a specific evidence base (DBT, MBT) and that clinicians found indispensable.

QUICK RULE

DSM-5 = **10 types in 3 clusters**. ICD-11 = **severity first, then 5 trait domains** (negative affectivity, detachment, dissociality, disinhibition, anankastia) **+ a borderline pattern**. The direction of travel is categorical → dimensional.

● MNEMONIC

"No Detached Dissocial Disinhibited Anankast"

The five ICD-11 domains: **N**egative affectivity, **D**etachment, **D**issociality, **D**isinhibition, **A**nankastia. Plus the borderline pattern qualifier.

3 Cluster A – odd / eccentric

The "weird" cluster. Three disorders sharing a thread of social oddity and constricted or suspicious relating. Schizotypal sits on the schizophrenia spectrum.

Disorder	Core feature	Holds in one line
Paranoid	pervasive **distrust and suspiciousness**; reads malevolence into others' motives	bears grudges, doubts loyalty, quick to counter-attack
Schizoid	**detachment** from relationships and a restricted emotional range	genuinely prefers solitude; the "loner", indifferent to praise or criticism
Schizotypal	acute social discomfort *plus* **cognitive-perceptual oddities** and eccentric behaviour	magical thinking, ideas of reference, odd speech; on the schizophrenia spectrum

● **Schizotypal on the spectrum** – shares genetic loading with schizophrenia and is classed by ICD-11 under schizophrenia-spectrum disorders, not personality disorders. The oddities are *attenuated* psychotic-like phenomena, below the threshold for psychosis.

● CLASSIC TRAP

Schizoid vs schizotypal vs avoidant, the perennial confusion. Schizoid: alone and *content* – no desire for closeness, flat affect, no oddity. Schizotypal: alone *and odd* – eccentric beliefs, perceptual distortions, magical thinking (the cognitive marker). Avoidant (Cluster C): alone but *longing* – wants closeness intensely but is paralysed by fear of rejection. The discriminator is desire-for-contact (absent in schizoid, present in avoidant) and odd-cognition (present only in schizotypal).

4 Cluster B – dramatic / emotional / erratic

The "wild" cluster, and the one that fills wards and exam papers. Emotional dysregulation, impulsivity and disturbed relating. Borderline and antisocial carry the most marks.

Disorder	Core feature	Holds in one line
Antisocial	**disregard for and violation of others' rights**; deceit, impulsivity, lack of remorse	conduct disorder before 15; not coded before age 18
Borderline	**instability** of affect, identity and relationships, with marked impulsivity	frantic efforts to avoid abandonment; recurrent self-harm

Disorder	Core feature	Holds in one line
Histrionic	pervasive **attention-seeking** and excessive emotionality	theatrical, shallow, suggestible; uses appearance to draw notice
Narcissistic	**grandiosity**, need for admiration, lack of empathy	entitled, exploitative; fragile self-esteem under the grandiosity

Borderline PD in depth

The most studied PD, defined by pervasive instability across four fronts: affect, identity, relationships and impulse control.

- **Affective instability** – rapid, reactive mood swings lasting hours, driven by interpersonal triggers; chronic emptiness; intense, poorly controlled anger.
- **Identity disturbance** – an unstable self-image and sense of self; shifting goals, values and vocational direction.
- **Splitting** – the central defence: people are experienced as all-good or all-bad, with abrupt switches (idealisation → devaluation). Drives the stormy, unstable relationships.
- **Impulsivity** – in at least two self-damaging areas: spending, sex, substance use, reckless driving, binge eating.
- **Self-harm & suicidality** – recurrent suicidal behaviour, gestures or threats, and self-mutilating behaviour. Completed suicide reaches roughly **1 in 10**.
- **Abandonment fear** – frantic efforts to avoid real or imagined abandonment; transient, stress-related paranoid ideation or dissociative symptoms under threat.

■ **Linehan's biosocial model** – BPD arises from a transaction between a constitutional **emotional vulnerability** (high sensitivity, high reactivity, slow return to baseline) and a chronically **invalidating environment**. The two amplify each other; the model is the rationale for **DBT**.

● MNEMONIC

"I DESPAIRR" → the 9 borderline criteria

Identity disturbance, **D**isturbed relationships (unstable, intense), **E**motional instability (affective lability), **S**uicidal/self-harm behaviour, **P**aranoia/dissociation under stress, **A**bandonment fear, **I**mpulsivity, **R**age (inappropriate anger), **R**elentless emptiness. Five of nine make the diagnosis.

Antisocial PD and psychopathy

- **Callousness** – the core: disregard for others' rights and feelings, deceitfulness, irritability, recklessness, irresponsibility, and a marked **lack of remorse** for harm caused.
- **Conduct disorder precursor** – diagnosis requires evidence of **conduct disorder with onset before age 15**, and the antisocial pattern is coded only from **age 18**. The developmental line runs childhood conduct disorder → adult antisocial PD.

◆ **Psychopathy & the Hare PCL-R** – psychopathy is a narrower, more severe construct than antisocial PD, adding the **affective-interpersonal** core (glibness, grandiosity, shallow affect, callousness) to the antisocial-lifestyle factor. Measured by the **Hare Psychopathy Checklist-Revised (PCL-R)**: 20 items, scored 0–2, max 40; the conventional cut-off is **≥30** (North America; ~25 in the UK). Not all antisocial PD is psychopathy.

● RULE

Antisocial PD is a behaviour-defined DSM/ICD diagnosis; **psychopathy** (Cleckley, operationalised by Hare) is a research and forensic construct with a heavier affective core. Most prisoners with antisocial PD do *not* meet the PCL-R psychopathy cut-off. The PCL-R is the strongest single actuarial predictor of violent recidivism.

5 Cluster C – anxious / fearful

The "worried" cluster. Anxiety and fear are the shared engine: of rejection, of being alone, of disorder. The most treatable cluster and the closest to common neurotic temperament.

Disorder	Core feature	Holds in one line
Avoidant	social inhibition, feelings of **inadequacy**, hypersensitivity to negative evaluation	wants closeness but avoids it for fear of rejection
Dependent	excessive **need to be cared for**; submissive, clinging behaviour	cannot make ordinary decisions alone; fears separation
Obsessive-compulsive (anankastic)	preoccupation with **order, perfectionism and control** at the cost of flexibility and openness	rigid, miserly, workaholic; rules over the point of the task

- **OCPD is ego-syntonic** – the perfectionism and rigidity feel *right* to the person; they value the traits and rarely see them as a problem. There are usually **no true obsessions or compulsions**: the disorder is a pervasive character style, not anxiety-driven rituals.

CLASSIC TRAP

OCPD vs OCD. OCPD: a Cluster C *personality* disorder – a pervasive, lifelong, **ego-syntonic** style of perfectionism, orderliness and control, with no specific obsessions or compulsions and little distress about the traits themselves. OCD: an anxiety-spectrum *disorder* – discrete **ego-dystonic** obsessions and compulsions the person resists and finds distressing, typically with insight. One is character; the other is symptom. They can co-occur, but most OCD patients do not have OCPD and vice versa.

6 Aetiology and management

Aetiology – a gene-environment transaction

- **Genetics & temperament** – personality traits are moderately heritable; early temperament (emotional reactivity, impulsivity, behavioural inhibition) is the constitutional substrate. Cluster A shares loading with schizophrenia; Cluster B (especially antisocial and borderline) shows familial impulsivity and affective instability.
- **Childhood adversity & trauma** – early abuse, neglect, inconsistent or invalidating caregiving, and disrupted attachment are strongly associated, most robustly with borderline PD. The diathesis (temperament) and the stress (adversity) interact rather than add.

Management – psychotherapy first

- **Primary modality is psychotherapy** – structured, long-term psychological therapy is the treatment of choice. Medication does not treat the personality disorder itself.

Therapy	What it targets	Best evidence in
DBT (Linehan)	emotion regulation, distress tolerance, mindfulness, interpersonal effectiveness; reduces self-harm	borderline PD (strongest evidence)
Mentalisation-based therapy (MBT)	capacity to read one's own and others' mental states; stabilises attachment	borderline PD
Schema therapy (Young)	early maladaptive schemas and modes; integrative, longer-term	borderline & other PDs
Therapeutic community	peer-led, structured milieu; social learning over months	severe PD, forensic settings

■ **Medication is adjunctive only** – targets **specific symptom domains and comorbidity**, not the personality structure. Examples: mood stabilisers or low-dose antipsychotics for affective instability, impulsivity and transient psychotic-like symptoms in BPD; SSRIs for comorbid depression or anxiety. No drug is licensed to treat a personality disorder per se; polypharmacy is to be avoided.

● TRAP

Do not call medication the treatment of personality disorder. It is **symptom-targeted and comorbidity-targeted**. The structured psychotherapies (DBT, MBT, schema therapy, therapeutic community) carry the disorder-modifying evidence. For borderline PD specifically, DBT has the strongest base; crisis prescribing should be time-limited and reviewed.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **General criteria:** enduring, pervasive, inflexible pattern, onset by adolescence/early adulthood, deviating from culture, causing distress/impairment. **Ego-syntonic** (vs ego-dystonic OCD).
- **Two systems:** DSM-5 = 10 types in 3 clusters (categorical). ICD-11 = abolished the types, rate **severity** (mild/moderate/severe) then 5 trait domains + a **borderline pattern** qualifier. Direction: categorical → dimensional.
- **ICD-11 domains:** negative affectivity, detachment, dissociality, disinhibition, anankastia (map to the maladaptive Big-Five poles).
- **Cluster A (odd):** paranoid = distrust · schizoid = detached & content · schizotypal = odd cognition, schizophrenia spectrum.
- **Cluster B (dramatic):** antisocial = rights-violation/no remorse · borderline = instability + self-harm · histrionic = attention-seeking · narcissistic = grandiosity.
- ★ **BPD:** affective instability, identity disturbance, splitting, impulsivity, self-harm/suicidality (~1 in 10 complete), abandonment fear. **Linehan biosocial** = emotional vulnerability × invalidating environment → rationale for DBT.
- **ASPD:** callousness, no remorse; needs conduct disorder before 15, coded only from 18. **Psychopathy** = narrower, affective core; **Hare PCL-R** 20 items, max 40, cut-off ≥30.
- **Cluster C (anxious):** avoidant = wants closeness, fears rejection · dependent = needs caretaking · OCPD = perfectionism/control, **ego-syntonic, no true obsessions**.
- **Three confusions:** schizoid (alone, content) vs schizotypal (alone, odd cognition) vs avoidant (alone, longing). OCPD (character, ego-syntonic) vs OCD (symptom, ego-dystonic, resisted).
- **Aetiology:** heritable temperament × childhood adversity/trauma (esp. BPD). Gene-environment transaction, not addition.
- **Management:** psychotherapy first – DBT (BPD, strongest), MBT, schema therapy, therapeutic community. Medication = symptom/comorbidity only, never the PD itself.

ONE-DAY REVISION · PAPER 2

Substance use, the exam essentials

Dependence is a syndrome, not a habit. Hold the ICD-10 six features against the DSM-5 count, fix the alcohol withdrawal timeline by hours, and keep the three antagonist-class agents (and what each blocks) straight. The intoxication and withdrawal contrasts carry most of the marks.

CONCEPT · RULE · TRAP · KEY NUMBER · MNEMONIC

The colour tells you what kind of fact it is. Doses are orientation only; prescribe to local protocol.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. Alcohol withdrawal runs in a fixed order by hours since the last drink: **tremor/autonomic 6-12 h**, alcoholic hallucinosis ~12-24 h, withdrawal seizures ~12-48 h, **delirium tremens ~48-72 h**. The sequence is the answer.
2. **Alcoholic hallucinosis occurs in a clear sensorium** with no clouding; **delirium tremens is a clouded, disoriented, autonomically unstable delirium** with visual hallucinations. The presence or absence of clouding is the discriminator.
3. The three anti-craving agents by mechanism: **disulfiram blocks aldehyde dehydrogenase (aversive)**, acamprosate modulates NMDA/glutamate (anti-craving), naltrexone is an opioid antagonist (blocks reward).
4. Opioids flip the pupil: **pinpoint (miotic) in intoxication, dilated (mydriasis) in withdrawal**; the intoxication triad is pinpoint pupils + respiratory depression + reduced consciousness, reversed by **naloxone (short-acting antagonist, re-dose)**.
5. Classification counts: **ICD-10 dependence = 3 of 6** features in the past year; **DSM-5 SUD = one disorder graded by 11 criteria** (mild 2-3, moderate 4-5, severe 6+); reward = VTA → nucleus accumbens dopamine.

1 Core concepts

Two classifications, one phenomenon. ICD-10 describes a **dependence syndrome** (a pattern, diagnosed by features); DSM-5 collapses abuse and dependence into one **substance use disorder** graded by how many criteria are met. Know both lists and how they map.

ICD-10 dependence syndrome: the classic six

Diagnose dependence when **three or more** have been present together in the past year.

Feature	Meaning
Compulsion	a strong, often overpowering desire or sense of compulsion to take the substance (craving)
Impaired control	difficulty controlling onset, termination or amount of use
Withdrawal	a physiological withdrawal state on stopping or reducing, or use to relieve/avoid it
Tolerance	increased doses needed for the same effect
Salience / primacy	neglect of alternative pleasures and interests; more time spent obtaining, using, recovering
Persistence despite harm	continued use despite clear evidence of harmful consequences

MNEMONIC

“Can't Control While Tolerating Such Persistence”

Compulsion, Control (impaired), Withdrawal, Tolerance, Salience, Persistence despite harm. Three of six in the past year = dependence.

DSM-5 substance use disorder: 11 criteria, severity by count

A single spectrum diagnosis. The 11 criteria fall into four clusters; **severity is graded by how many are met** in a 12-month period.

Cluster	Criteria (abbreviated)
Impaired control	larger amounts / longer than intended; failed attempts to cut down; much time spent; craving
Social impairment	role failure (work, home, school); use despite social problems; activities given up
Risky use	use in hazardous situations; use despite known physical or psychological harm
Pharmacological	tolerance; withdrawal

◆ **Severity grading** – **2–3** criteria = mild, **4–5** = moderate, **6 or more** = severe. DSM-5 dropped “legal problems” and added **craving**. Tolerance and withdrawal do not count if occurring under appropriate medical supervision.

Harmful use vs hazardous use

- **Hazardous use** – a pattern that *risks* harm but has not yet caused it. A risk category, not an ICD-10 diagnosis; the target of screening and brief intervention.
- **Harmful use (ICD-10)** – use that has *already* caused actual damage to physical or mental health. A diagnosis, but below the threshold of dependence. (DSM-IV called the rough equivalent “abuse”; DSM-5 merged it into SUD.)

TRAP

Hazardous = risk only, no proven harm yet (not a formal ICD-10 category). **Harmful** = demonstrable harm already done, but no dependence syndrome. Dependence sits above both. Do not use the three interchangeably.

The four pharmacological terms

- **Tolerance** – a given dose produces a diminishing effect, so more is needed. Includes pharmacodynamic (receptor adaptation), pharmacokinetic (faster metabolism) and behavioural/cross-tolerance forms.
- **Withdrawal** – a substance-specific syndrome on cessation, typically the *mirror image* of the drug's acute effect (a depressant's withdrawal is hyperexcitable).
- **Craving** – the conscious, often cue-triggered urge to use. A DSM-5 criterion and a key relapse driver.
- **Neuroadaptation** – the brain's compensatory changes to chronic exposure; the substrate underlying both tolerance and withdrawal.

The reward pathway (one line)

■ **Mesolimbic dopamine pathway** – ventral tegmental area (VTA) → nucleus accumbens (with projections to prefrontal cortex). Effectively all drugs of abuse raise dopamine here; this is the **final common reward pathway** of addiction.

HOLD THIS

ICD-10 = **syndrome, 3 of 6** features. DSM-5 = **1 disorder, count of 11** criteria (mild 2–3, moderate 4–5, severe 6+). Reward = **VTA → nucleus accumbens dopamine**.

2 Alcohol

Units and screening

◆ **Standard drink / unit** – one UK **unit = 8 g** pure alcohol; one US **standard drink ≈ 14 g**. Roughly a half-pint of beer, a small glass of wine or a single spirit measure. Grams = volume (mL) × strength (%) × 0.789 / 100.

Tool	Items	Note
CAGE	Cut down? Annoyed by criticism? Guilty? Eye-opener?	brief; ≥2 of 4 is a positive screen
AUDIT	10 items (WHO); covers consumption, dependence, harm	quantitative; ≥8 suggests hazardous use; AUDIT-C is the 3-item short form

● MNEMONIC

"CAGE"

Cut down, **A**nnoyed, **G**uilty, **E**ye-opener. Two or more positive answers warrant fuller assessment.

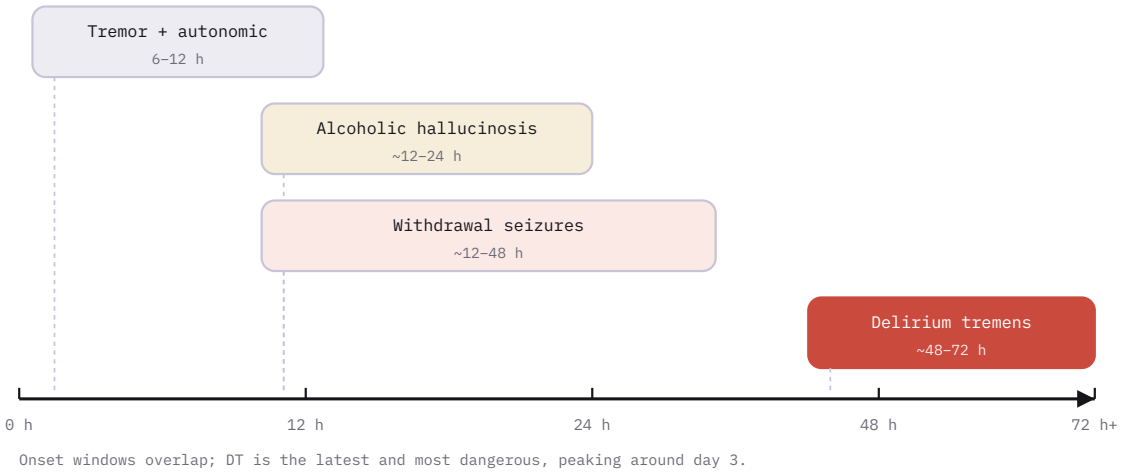
Intoxication

● **Acute intoxication** – disinhibition, slurred speech, ataxia, nystagmus, labile mood, impaired judgement, progressing to stupor and coma at high levels. CNS depressant; respiratory depression and aspiration are the lethal risks. Blackouts reflect failure to lay down memory while conscious.

The withdrawal timeline

Alcohol withdrawal is a hyperadrenergic, hyperexcitable rebound from chronic CNS depression. The hallmark is that complications appear in a **predictable order by hours since the last drink**.

ALCOHOL WITHDRAWAL TIMELINE (HOURS SINCE LAST DRINK)



Tremor and autonomic arousal at 6–12 h; alcoholic hallucinosis around 12–24 h; withdrawal (rum) fits at 12–48 h; delirium tremens at 48–72 h. The order is the answer.

Time since last drink	Picture
6–12 h	tremor, sweating, tachycardia, hypertension, nausea, anxiety, insomnia (autonomic)
~12–24 h	alcoholic hallucinosis – usually visual/tactile, **clear sensorium** (distinguishes it from DT)
~12–48 h	withdrawal ("rum") seizures – generalised tonic-clonic, usually self-limiting
~48–72 h	delirium tremens – the medical emergency

Delirium tremens

▲ **Delirium tremens (DT)** – clouding of consciousness and disorientation (a true delirium), coarse tremor, vivid **visual hallucinations** (classically small animals, Lilliputian), marked autonomic overactivity (fever, tachycardia, hypertension, profuse sweating) and agitation. Untreated mortality is appreciable (historically up to ~15%, lower with treatment). A medical emergency.

● CLASSIC TRAP

Alcoholic hallucinosis occurs in a *clear sensorium* with no clouding and no gross autonomic storm; **delirium tremens** is a clouded, disoriented, autonomically unstable delirium. Hallucinosis can also persist as a chronic auditory hallucinatory state without confusion. The presence or absence of clouding is the discriminator.

Wernicke-Korsakoff and other complications

◆ **Wernicke encephalopathy** – acute, reversible **thiamine (B1) deficiency**. Triad: **confusion, ophthalmoplegia/nystagmus, ataxia**. A medical emergency; treat with parenteral thiamine *before* glucose (a glucose load can precipitate or worsen it).

▲ **Korsakoff syndrome** – the chronic, often **irreversible** sequel: profound **anterograde amnesia** with confabulation, relatively preserved other cognition. Lesions in mammillary bodies and dorsomedial thalamus.

● MNEMONIC

"COAT" for Wernicke

Confusion, **O**phthalmoplegia, **A**taxia, give **T**hiamine. Korsakoff = the amnesic-confabulatory aftermath.

● **Other complications** – hepatic (fatty liver, hepatitis, cirrhosis), GI (gastritis, pancreatitis, varices), **cerebellar degeneration**, peripheral neuropathy, **fetal alcohol syndrome**, cardiomyopathy, pathological jealousy (Othello syndrome), and central pontine myelinolysis (from over-rapid sodium correction).

Management

■ **Detoxification** – **benzodiazepines** are first line (cross-tolerant with alcohol; they prevent and treat seizures and DT). Long-acting **chlordiazepoxide** or **diazepam**; use **lorazepam/oxazepam** in significant liver disease (no active metabolites, glucuronidated). Either fixed-dose tapering or symptom-triggered (CIWA-Ar guided) regimens.

■ **Thiamine** – give **parenteral thiamine** (e.g. Pabrinex) to prevent or treat Wernicke; always before carbohydrate load.

Once detoxified, the work is **relapse prevention** with a psychosocial backbone plus an anti-craving agent.

Agent	Mechanism	Note
Disulfiram	inhibits **aldehyde dehydrogenase** → acetaldehyde builds up → aversive flushing, nausea, palpitations if alcohol is taken	a deterrent; needs motivation and supervision; abstinence required before starting

Agent	Mechanism	Note
Acamprosate	**NMDA modulation** / restores glutamate-GABA balance; reduces craving	start after detox; renally cleared
Naltrexone	**opioid receptor antagonist**; blunts the rewarding "high" and reduces heavy drinking	avoid in opioid users (precipitates withdrawal) and significant hepatic failure

QUICK RULE

Disulfiram = aversive (ALDH block). **Acamprosate** = anti-craving (glutamate/NMDA). **Naltrexone** = blocks reward (opioid block). Nalmefene is a related option for reduction rather than abstinence.

3 Opioids

Intoxication

▲ **Opioid intoxication triad** – **pinpoint (miotic) pupils**, **respiratory depression**, and **reduced consciousness**. Add hypotension, bradycardia, hypothermia. Respiratory depression is the cause of death.

● MNEMONIC

"Pinpoint, Plummeting breath, Plummeting consciousness"

The opioid overdose triad. A comatose patient with pinpoint pupils and slow breathing is opioid toxicity until proven otherwise.

■ **Naloxone** – a competitive **opioid antagonist**; the overdose antidote. Reverses respiratory depression within minutes. **Short half-life**, so re-dosing or an infusion is often needed (the opioid may outlast the naloxone); it will precipitate acute withdrawal in dependent users.

Withdrawal

● **Opioid withdrawal** – intensely unpleasant but **not life-threatening** (unlike alcohol or benzodiazepine withdrawal). A flu-like picture: lacrimation, rhinorrhoea, yawning, sweating, **dilated pupils (mydriasis)**, **piloerection** ("cold turkey"), myalgia, abdominal cramps, diarrhoea, nausea, restlessness, dysphoria and craving.

● TRAP

Pupils flip with state: **pinpoint in intoxication**, **dilated in withdrawal**. Opioid withdrawal looks dramatic but does not kill an otherwise healthy adult; alcohol and benzodiazepine withdrawal can. Onset and duration track the drug's half-life (heroin: hours, peaks ~36–72 h; methadone: later and more prolonged).

Management

■ **Opioid substitution (OST)** – the mainstay. **Methadone**: a long-acting full μ-agonist, given as a supervised daily oral dose (caution: QT prolongation, overdose risk in induction). **Buprenorphine**: a **partial μ-agonist** with a ceiling effect (safer in overdose); precipitates withdrawal if given too early, so start when the patient is already in mild withdrawal. Buprenorphine-naloxone (Suboxone) deters injection.

● **Antagonist maintenance** – **naltrexone** for abstinent, motivated patients: it blocks the effect of any opioid taken, removing reward. Must be opioid-free first.

● **Symptomatic / detox** – **lofexidine or clonidine** (α2-agonists) dampen autonomic withdrawal; loperamide for diarrhoea, antiemetics, analgesics. Buprenorphine or methadone tapers for medically supervised withdrawal.

QUICK RULE

Overdose → **naloxone** (antagonist, short-acting). Maintenance → **methadone** (full agonist) or **buprenorphine** (partial agonist, ceiling). Relapse prevention in the abstinent → **naltrexone**.

4 Other substances

One intoxication line and one withdrawal line each. The high-yield contrasts: which withdrawals are dangerous (alcohol, benzodiazepines), which are merely unpleasant (opioids, stimulants), and the sympathomimetic stimulant picture.

Substance	Intoxication	Withdrawal
Benzodiazepines / sedatives	sedation, disinhibition, ataxia, slurred speech, anterograde amnesia, respiratory depression (worse with alcohol)	dangerous: anxiety, insomnia, tremor, perceptual disturbance, **seizures and delirium** (alcohol-like); taper slowly
Cannabis	euphoria, relaxation, conjunctival injection, dry mouth, tachycardia, increased appetite, altered time sense; anxiety/paranoia at high dose	mild: irritability, anxiety, insomnia, vivid dreams, reduced appetite
Cocaine	sympathomimetic: euphoria, grandiosity, **mydriasis**, tachycardia, hypertension, hyperthermia; risk of MI, stroke, seizures, formication ("cocaine bugs")	"crash": dysphoria, fatigue, hypersomnia, hyperphagia, vivid dreams, intense craving
Amphetamine / methamphetamine	as cocaine but longer-acting; alertness, insomnia, psychosis with paranoia; stereotypies	similar crash: hypersomnia, depression, hyperphagia, craving
Nicotine	mild stimulation, then relaxation; tachycardia	irritability, anxiety, restlessness, poor concentration, increased appetite, craving; peaks early, eases over weeks
Hallucinogens (LSD)	perceptual distortion, synaesthesia, depersonalisation, mydriasis; "bad trips"; no dependence syndrome	no physical withdrawal; **flashbacks** (HPPD – hallucinogen persisting perception disorder) can recur
Inhalants / solvents	rapid brief euphoria, disinhibition, ataxia, slurred speech; arrhythmia ("sudden sniffing death"), hypoxia	mild, inconsistent; chronic use damages brain, liver, kidney, marrow
Dissociatives (ketamine, PCP)	dissociation, analgesia, **nystagmus**, ataxia; PCP: agitation, violence, hypertension; NMDA-antagonist psychosis	mild; craving, dysphoria, anxiety on chronic use

● TRAP

Pupils sort the toxidromes: **opioids constrict (pinpoint)**; **stimulants, hallucinogens and most withdrawals dilate**. Only the depressant withdrawals (alcohol, benzodiazepines/barbiturates) are themselves life-threatening; stimulant and opioid withdrawals are unpleasant but not lethal in a healthy adult.

5 Assessment & management frameworks

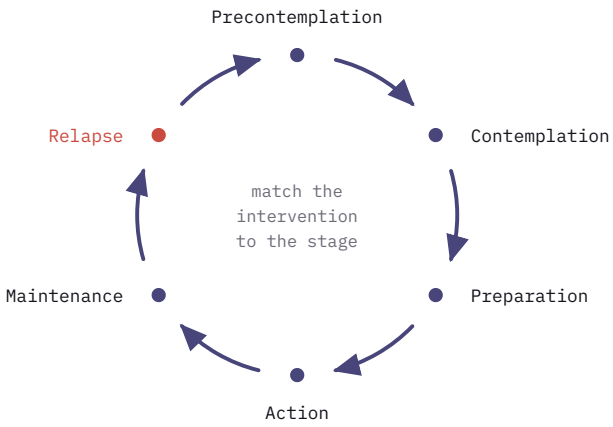
Stages of change (Prochaska & DiClemente)

The transtheoretical model: change is a cycle, not a switch. Match the intervention to the stage; relapse is part of the loop, not failure.

Stage	The person…
Precontemplation	sees no problem, not considering change
Contemplation	ambivalent; weighing pros and cons
Preparation	intends to act soon; making a plan

Stage	The person…
Action	actively changing behaviour
Maintenance	sustaining the change, guarding against relapse
Relapse	returns to old behaviour; re-enters the cycle

THE CYCLE OF CHANGE



Change runs as a spiral: precontemplation, contemplation, preparation, action, maintenance, with relapse looping back in. The clinician's job is to meet the patient where they are.

Brief intervention, MI, harm reduction

- **Brief intervention** – a short, structured, opportunistic conversation for hazardous/harmful use; the **FRAMES** elements (Feedback, Responsibility, Advice, Menu of options, Empathy, Self-efficacy) are the exam handle.
- **Motivational interviewing (Miller & Rollnick)** – a collaborative, person-centred style that resolves ambivalence and elicits the patient's own "change talk"; roll with resistance, develop discrepancy, support self-efficacy, express empathy.
- **Harm reduction** – reduce the harms of use without requiring abstinence first: needle/syringe exchange, opioid substitution, naloxone distribution, supervised consumption, safer-use advice. Pragmatic, not permissive.

Relapse prevention (Marlatt)

- **Marlatt's model** – identify **high-risk situations** and build coping; a *lapse* (a single slip) need not become a *relapse*. The **abstinence violation effect** is the danger: guilt and "I've blown it" thinking after a lapse drives a full relapse. Reframe a lapse as a learning event.

MNEMONIC

"FRAMES"

Feedback, **R**esponsibility, **A**dvice, **M**enu of options, **E**mpathy, **S**elf-efficacy. The skeleton of any brief intervention.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Classifications:** ICD-10 dependence syndrome = **3 of 6** (compulsion, impaired control, withdrawal, tolerance, salience, persistence despite harm). DSM-5 SUD = **1 disorder, 11 criteria**; mild 2–3, moderate 4–5, severe 6+. Hazardous = risk only; harmful = harm done, no dependence.
- **Reward:** all drugs of abuse raise dopamine in the mesolimbic pathway (**VTA → nucleus accumbens**), the final common reward circuit.
- **Alcohol screening:** CAGE ≥2 positive; AUDIT ≥8 hazardous (AUDIT-C = 3-item). UK unit = 8 g, US drink ≈ 14 g alcohol.
- ★ **Withdrawal timeline:** tremor/autonomic **6–12 h** · alcoholic hallucinosis (clear sensorium) **~12–24 h** · seizures **~12–48 h** · delirium tremens **~48–72 h**.
- **Hallucinosis vs DT:** hallucinosis = clear sensorium, no clouding; DT = clouded, disoriented, autonomically unstable delirium with visual hallucinations.
- **Wernicke:** confusion + ophthalmoplegia + ataxia from thiamine deficiency; parenteral thiamine before glucose. Korsakoff = irreversible anterograde amnesia + confabulation (mammillary bodies, thalamus).
- **Alcohol management:** benzodiazepine detox (chlordiazepoxide; lorazepam in liver disease), parenteral thiamine, then disulfiram (ALDH block), acamprosate (anti-craving, glutamate/NMDA), naltrexone (opioid block, blunts reward).
- **Opioid intoxication:** pinpoint pupils + respiratory depression + reduced consciousness; antidote = **naloxone** (short-acting antagonist, re-dose). Withdrawal = flu-like, dilated pupils, piloerection; unpleasant, not lethal.
- **Opioid maintenance:** methadone (full μ-agonist) or buprenorphine (partial agonist, ceiling, start in mild withdrawal); naltrexone for the abstinent; clonidine/lofexidine symptomatic.
- **Toxidrome rule:** opioids constrict pupils; stimulants/hallucinogens/withdrawals dilate. Only depressant withdrawals (alcohol, benzodiazepines) are themselves life-threatening.
- **Frameworks:** stages of change (precontemplation → contemplation → preparation → action → maintenance, with relapse) · brief intervention = FRAMES · MI elicits change talk · harm reduction · Marlatt relapse prevention (beware the abstinence violation effect).

ONE-DAY REVISION · PAPER 2

Eating Disorders

Three disorders, one fault line: anorexia restricts to a dangerously low weight, bulimia binges then compensates at a near-normal weight, binge-eating disorder binges without compensation. Hold the diagnostic triads, the medical complications, and the two safety topics that win or lose marks: the anorexia complication table and refeeding syndrome.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. Refeeding syndrome is the single highest-yield safety topic in this pack.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. Anorexia nervosa has the highest mortality of any psychiatric disorder, by two routes: medical (cardiac, electrolyte) and suicide.
2. Refeeding syndrome: an insulin surge drives phosphate, potassium and magnesium intracellularly, so the danger electrolyte is hypophosphataemia; give thiamine before carbohydrate (Wernicke) and refeed low and slow. It is a complication of treatment, not of the illness.
3. The discriminator between anorexia binge-purge type and bulimia is body weight: anorexia is at a significantly low weight, bulimia at normal or near-normal weight.
4. Russell's sign (knuckle calluses) signals self-induced vomiting, so vomiting gives a hypokalaemic metabolic alkalosis whereas laxative abuse gives a metabolic acidosis.
5. No drug is first-line for anorexia; treatment is CBT-ED in adults, FBT (Maudsley) in adolescents, while fluoxetine 60 mg/day is the one clear pharmacological win in bulimia.

1 Anorexia nervosa

The diagnosis rests on three features. Get the triad exact; examiners mark the wording.

Core feature	What it means
Energy restriction	persistent restriction of intake leading to a **significantly low body weight** for age, sex and health (DSM-5 wording; a specific BMI cut-off is no longer required, though <18.5 is the usual marker and ICD-11 sets <18.5 in adults)
Fear of weight gain	intense fear of gaining weight or becoming fat, *or* persistent behaviour that interferes with weight gain, despite being underweight
Body-image disturbance	disturbed experience of body weight/shape; undue influence of weight on self-evaluation; *or* persistent lack of recognition of the seriousness of the low weight

Subtypes

Subtype	How weight is lost	Note
Restricting type	dieting, fasting, excessive exercise; no regular binge/purge in the last 3 months	weight loss is by intake restriction and activity
Binge-eating / purging type	recurrent binge eating or purging (vomiting, laxatives, diuretics) in the last 3 months	distinguished from bulimia by the **low body weight**

● CLASSIC TRAP

The binge-purge subtype of anorexia is **not** bulimia. Both can binge and purge, but the anorexic patient is at a significantly low weight, whereas the bulimic patient is at a normal or near-normal weight. **Weight is the discriminator.**

DSM-5 severity and a dropped criterion

◆ **DSM-5 severity (by BMI)** – mild ≥17, moderate 16–16.99, severe 15–15.99, extreme <15 kg/m^2.

▲ **Amenorrhoea dropped** – the historical DSM-IV criterion of **amenorrhoea** (loss of ≥3 consecutive cycles) was **removed in DSM-5**. It excluded men, pre-menarchal girls, post-menopausal women and those on the oral contraceptive pill, and added little. Amenorrhoea is still a common clinical *finding*, just not a diagnostic requirement.

Typical cognitions

● **Overvalued ideas about shape** – self-worth judged almost entirely by weight and shape; relentless drive for thinness; control over eating felt as control over life.

● **Distorted body image** – the patient sees herself as fat despite being objectively underweight (overestimation of body size).

● **Limited insight (egosyntonic)** – symptoms are often experienced as desirable, not distressing, which drives the poor engagement and high relapse.

Epidemiology

Parameter	Anorexia nervosa
Sex	markedly female-predominant (roughly 10:1 female:male)
Age of onset	adolescence to young adulthood; bimodal peaks often quoted near 14 and 18 years
Lifetime prevalence	around 0.5–1% in women (estimates vary by criteria)
Mortality	the highest mortality of any psychiatric disorder; standardised mortality ratio markedly raised. Deaths are from medical complications and from **suicide**

HOLD THIS

Anorexia carries the **highest mortality of any psychiatric disorder**. Two routes to death: **medical** (cardiac, electrolyte) and **suicide**. This single fact frames every management decision.

2 Medical complications & refeeding

Starvation injures nearly every system. This table is a high-yield answer; the mechanism in the right column is what separates a pass from a distinction.

System	Complication	Mechanism / note
Cardiovascular	Bradycardia & hypotension	increased vagal tone; adaptive hypometabolism. A major cause of death
	prolonged QTc, arrhythmia	worsened by hypokalaemia and hypomagnesaemia
Electrolytes	Hypokalaemia, hyponatraemia, hypophosphataemia, hypomagnesaemia	purging and malnutrition; drives the cardiac risk
Skin / hair	Lanugo hair, dry skin, brittle nails, carotenoderma	fine downy body hair is a classic insulating response to starvation

System	Complication	Mechanism / note
Endocrine	amenorrhoea, low oestrogen	hypothalamic hypogonadotropic hypogonadism (low LH/FSH)
	raised cortisol, sick euthyroid	HPA activation; low T3/T4 with normal-ish TSH (low-T3 syndrome). Also growth-hormone resistance, hypoglycaemia
Bone	Osteoporosis / osteopenia	low oestrogen + low weight + high cortisol; fracture risk, often irreversible
Haematology	leucopenia, anaemia, thrombocytopenia	gelatinous marrow change (pancytopenia possible)
GI	delayed gastric emptying, constipation, raised transaminases	slowed gut motility; hepatic stress from starvation

● MNEMONIC

"Everything goes down – except cortisol, cholesterol, carotene and GH"

Starvation lowers heart rate, BP, temperature, white cells, oestrogen and T3; it *raises* cortisol, GH (with peripheral resistance), cholesterol and carotene. A quick sorting rule for a long table.

Refeeding syndrome – the safety centrepiece

The single most important topic in this pack. When a starved patient is fed too fast, the metabolic shift back to carbohydrate metabolism can be fatal. Examiners want the mechanism and the danger electrolyte.

■ **The mechanism** – refeeding (especially carbohydrate) spikes insulin. Insulin drives **phosphate, potassium and magnesium intracellularly** and stimulates cellular uptake of glucose, all of which deplete already-low serum stores. Glycogen, fat and protein synthesis consume phosphate further.

Abnormality	Why it matters
Hypophosphataemia	**the hallmark and the danger.** Causes cardiac failure, arrhythmia, respiratory failure, rhabdomyolysis, seizures, delirium. The defining feature of refeeding syndrome
Hypokalaemia	arrhythmia, weakness
Hypomagnesaemia	arrhythmia, tetany; worsens hypokalaemia and hypocalcaemia
Thiamine deficiency	refeeding raises thiamine demand; risk of **Wernicke encephalopathy**. Give thiamine *before* carbohydrate
Fluid & sodium shifts	sodium and water retention → oedema, fluid overload, cardiac decompensation

QUICK RULE

The danger electrolyte is **phosphate**. Prevent by starting low and slow, giving **thiamine first**, supplementing electrolytes pre-emptively, and monitoring phosphate, potassium and magnesium daily. Highest risk: very low BMI, little intake for >5–10 days, prior low electrolytes.

● CLASSIC TRAP

Refeeding syndrome is a complication of **treatment**, not of the illness itself. The harm comes from feeding too fast. The answer is never to withhold feeding (the patient is starving) but to refeed **cautiously** with monitoring. Thiamine is given *before* any glucose load to avoid precipitating Wernicke encephalopathy.

3 Bulimia nervosa

Recurrent binges followed by compensation, in a patient whose weight stays normal. That normal weight is exactly why it is missed.

Feature	Detail
Recurrent binges	eating an objectively large amount in a discrete period, with a **sense of loss of control**
Compensatory behaviour	self-induced **vomiting**, laxatives, diuretics, fasting, or excessive exercise
Frequency (DSM-5)	binges and compensation on average **≥1/week for 3 months**
Self-evaluation	unduly influenced by **body shape and weight**
Weight	usually **normal or near-normal** (if underweight, diagnose anorexia binge-purge type instead)

Physical signs (high-yield)

Sign	Cause
Russell's sign	calluses / abrasions on the **knuckles** from self-induced vomiting (teeth on the dorsum of the hand)
Dental erosion	gastric acid erodes enamel (perimylolysis), especially lingual surfaces; dental caries
Parotid (salivary) swelling	painless bilateral sialadenosis from repeated vomiting; raised serum amylase
Hypokalaemic metabolic alkalosis	loss of gastric H^+ and K^+ from vomiting (laxative purging tends to a metabolic acidosis instead)
Others	oesophagitis / Mallory-Weiss tears, calluses, menstrual irregularity, ipecac cardiomyopathy (rare)

● TRAP

Russell's sign points to self-induced **vomiting**, so it appears in bulimia and in the binge-purge subtype of anorexia, not in restricting anorexia or in binge-eating disorder. The acid-base picture also differs by purge route: **vomiting → hypokalaemic alkalosis**; **laxative abuse → metabolic acidosis** with hypokalaemia.

4 Anorexia vs bulimia & the other disorders

The core contrast

	Anorexia nervosa	Bulimia nervosa
Body weight	significantly low	normal or near-normal
Insight	often egosyntonic, poor insight	more egodystonic; shame, secrecy
Amenorrhoea	common (criterion now dropped)	menstrual irregularity, not amenorrhoea as a rule
Hallmark sign	lanugo, bradycardia, cachexia	Russell's sign, dental erosion, parotid swelling
Mortality	highest of any psychiatric disorder	lower, but still raised
First-line therapy	CBT-ED in adults; FBT in adolescents	CBT-ED; fluoxetine has an adjunct role

The other feeding & eating disorders

- **Binge-eating disorder (BED)** – recurrent binges with loss of control, marked distress, **but no regular compensatory behaviour**. ≥1/week for 3 months (DSM-5). The **commonest** eating disorder; associated with obesity. Distinguished from bulimia by the absence of purging/compensation.
- **ARFID** – avoidant/restrictive food intake disorder: avoidance of food on the basis of **sensory features, fear of aversive consequences (e.g. choking), or low interest in eating**, with weight loss or nutritional deficiency, but **no body-image disturbance or weight concern**. Often younger children.

- **Pica** – persistent eating of **non-nutritive, non-food substances** (chalk, soil, ice) inappropriate to developmental level, for at least 1 month. Linked to intellectual disability, pregnancy, iron deficiency.
- **Rumination disorder** – repeated **regurgitation** of food (re-chewed, re-swallowed or spat out) for ≥1 month, not due to a medical condition.

5 Aetiology

Multifactorial, biopsychosocial. No single cause; examiners want a structured spread across genetic, temperamental, sociocultural, family and neurobiological factors.

Domain	Contribution
Genetic	substantial heritability (twin estimates often quoted ~50–60% for anorexia); familial aggregation; GWAS suggests metabolic as well as psychiatric loci
Temperament	**perfectionism, harm avoidance**, obsessionality, rigidity and high constraint (anorexia); impulsivity and affective instability lean toward bulimia
Sociocultural	internalisation of the **thin ideal**, media and peer pressure, weight-related teasing, occupations valuing thinness (ballet, modelling, athletics)
Family / developmental	enmeshment, control conflicts, early feeding difficulties, childhood adversity, sexual abuse (a non-specific risk for bulimia)
Neurobiology	**serotonin (5-HT)** dysregulation (links to anxiety, perfectionism, satiety); dopamine and reward-circuit alterations; insular and frontostriatal changes in body-image and interoception; appetite peptides (leptin, ghrelin) altered, largely as a consequence of starvation

6 Management

Order of priority: stabilise the body first, then treat the mind. Psychological therapy is the mainstay; medication has a limited, adjunctive role.

Medical priority & monitoring

- **Medical comes first** – assess and correct dehydration, electrolytes and cardiac risk before anything else. Monitor weight, vitals, ECG (QTc), and bloods (K, PO_4, Mg, glucose). Treatment is usually multidisciplinary and outpatient where safe.
- **MARSIPAN principles** – the UK framework for **Management of Really Sick Patients with Anorexia Nervosa**: identify the severely ill, refeed cautiously to avoid refeeding syndrome, monitor electrolytes (esp. phosphate), use clear risk thresholds (very low BMI, bradycardia, hypotension, hypoglycaemia, deranged electrolytes) and treat in an appropriate setting with joint medical-psychiatric care.
- **Refeed cautiously** – start low and slow, thiamine before carbohydrate, pre-emptive electrolyte replacement, daily phosphate/potassium/magnesium for the first days. (See section 2.)

Psychological treatments (the mainstay)

Population	First-line	Note
Adults, anorexia	CBT-ED / CBT-E (eating-disorder-focused CBT); MANTRA and SSCM are alternatives	NICE: individual eating-disorder-focused CBT is first-line in adults
Adolescents, anorexia	Family-based treatment (FBT) / the Maudsley approach	parents take charge of refeeding, then hand control back; strongest evidence in young people
Bulimia & BED	CBT-ED (CBT-E)	guided self-help based on CBT is a common first step in bulimia/BED

Medication (limited role)

▲ **No drug is first-line for anorexia** – no medication reliably restores weight; drugs treat comorbidity, not the core disorder. Olanzapine may give modest help with weight/agitation but is not a primary treatment. Avoid QTc-prolonging agents in the cardiac-compromised.

◆ **Fluoxetine in bulimia** – the one clear pharmacological win: **fluoxetine 60 mg/day** reduces binge-purge frequency in bulimia nervosa (the only antidepressant FDA-approved for it). An adjunct to CBT, not a replacement. (Lisdexamfetamine is approved for BED.)

Indications for admission

■ **When to admit** – very low or rapidly falling weight (e.g. BMI <13–14 or severe weight loss), **bradycardia, hypotension, hypothermia**, dangerous electrolyte derangement, ECG changes, hypoglycaemia, syncope, high suicide risk, or failure of outpatient treatment. Severe cases may need treatment under mental-health-act provisions when refusing life-saving care.

HOLD THIS

Body first, then mind. **CBT-ED** first-line for adults; **FBT (Maudsley)** first-line for adolescents. **Fluoxetine 60 mg** is the one drug that helps (bulimia). Refeed cautiously; watch phosphate.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Anorexia triad:** restriction to a significantly low weight · intense fear of weight gain · body-image disturbance. Subtypes: restricting vs binge-purge. **Highest mortality of any psychiatric disorder** (medical + suicide).
- **Amenorrhoea criterion was dropped in DSM-5** (excluded men, pre-menarche, post-menopause, the pill). Still a common finding.
- **Anorexia complications:** bradycardia, hypotension, hypokalaemia, lanugo, amenorrhoea, osteoporosis, raised cortisol, sick euthyroid (low T3), leucopenia. Almost everything goes down except cortisol, GH, cholesterol, carotene.
- ★ **Refeeding syndrome:** insulin surge drives phosphate/K/Mg into cells. Danger electrolyte = **hypophosphataemia**. Give **thiamine before carbohydrate** (Wernicke), refeed low and slow, monitor daily. A complication of treatment, not the illness.
- **Bulimia:** binges + compensation (vomiting, laxatives, fasting, exercise), self-evaluation over-influenced by shape/weight, weight normal. Signs: **Russell's sign** (knuckles), dental erosion, parotid swelling, **hypokalaemic alkalosis** (vomiting) vs acidosis (laxatives).
- **Anorexia vs bulimia:** the discriminator is **body weight** (low vs normal). Russell's sign needs vomiting, so it spans bulimia and anorexia binge-purge type.
- **The others:** BED = binges without compensation (commonest, obesity-linked) · ARFID = avoidance with no body-image concern · pica = non-food substances · rumination = repeated regurgitation.
- **Aetiology:** heritable; perfectionism + harm avoidance; thin-ideal sociocultural; family/developmental; serotonin and reward-circuit neurobiology.
- **Management:** medical first (electrolytes, ECG, MARSIPAN for the severely ill). **CBT-ED/CBT-E** first-line adults · **FBT/Maudsley** adolescents. **Fluoxetine 60 mg** for bulimia; no drug first-line for anorexia. Admit for cardiac/electrolyte instability, very low weight, high suicide risk.

ONE-DAY REVISION · PAPER 2

Sexual & gender disorders, made retrievable

The clinical block: the normal response cycle, the dysfunctions sorted by phase, their causes and management, the paraphilic disorders, and the major nosological shift in gender incongruence. Hold the response-cycle sequence, the phase-to-dysfunction map, and the one distinction that separates a paraphilia from a paraphilic disorder. The three tables are the high-yield answers.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. Language here is kept clinical and respectful throughout; gender incongruence is no longer classed as a mental disorder.

II

★ HIGH YIELD TOP 5 IF NOTHING ELSE

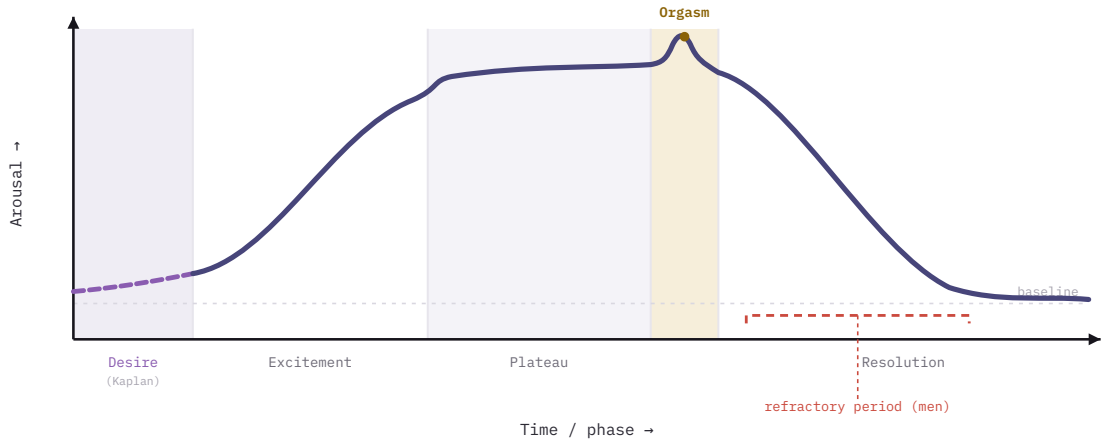
1. A paraphilia (atypical interest) becomes a paraphilic disorder only when it also causes distress/impairment to self OR entails harm or risk of harm to a non-consenting person; an unusual but consensual private interest is not a disorder.
2. ICD-11 moved gender incongruence OUT of the mental disorders chapter into conditions related to sexual health (in force 2022); DSM-5 uses gender dysphoria, focusing on the distress, not the identity.
3. Sexual response cycle: Masters & Johnson = excitement → plateau → orgasm → resolution; Kaplan prefixed the desire phase, and a refractory period follows orgasm in men only (women may have none).
4. Physiology: parasympathetic drives erection and sympathetic drives ejaculation (Point and Shoot); erection runs on nitric oxide / cyclic GMP, the target of PDE5 inhibitors, which are absolutely contraindicated with nitrates.
5. SSRIs are the classic medication cause of sexual dysfunction (low desire, delayed/absent orgasm, ED); the trap is that SSRI-induced delayed ejaculation is used therapeutically to treat premature ejaculation, and bupropion and mirtazapine carry the lowest sexual side-effect burden.

1 The normal sexual response cycle

Two names own this topic. **Masters and Johnson** described a four-phase physiological cycle from laboratory observation; **Kaplan** later prefixed a **desire** phase, giving the model its clinical use, since most dysfunctions map onto a phase.

Phase	Model	What happens
Desire (libido)	Kaplan's addition	appetite and motivation for sexual activity; precedes the bodily cycle
Excitement (arousal)	Masters & Johnson	vasocongestion: erection in men, lubrication and swelling in women; rising heart rate
Plateau	Masters & Johnson	arousal sustained at a high level just below the orgasmic threshold
Orgasm	Masters & Johnson	rhythmic muscular contractions; ejaculation in men; the peak, briefest phase
Resolution	Masters & Johnson	return to the unaroused baseline; a **refractory period** follows in men

THE SEXUAL RESPONSE CYCLE (MASTERS & JOHNSON, WITH KAPLAN'S DESIRE PHASE)



Masters and Johnson mapped excitement, plateau, orgasm and resolution. Kaplan prefixed a desire phase. A refractory period follows orgasm in men, during which further orgasm is not possible; women may not have one.

◆ **Refractory period** – the recovery interval after orgasm in **men**, during which renewed arousal to orgasm is not possible. It lengthens with age. Women typically have **no** refractory period and may be multi-orgasmic.

● **The physiology in one line** – arousal is **parasympathetic** (vasocongestion: erection, lubrication), and orgasm/ejaculation is **sympathetic**. "Point and Shoot": Parasympathetic = erection (Point), Sympathetic = emission/ejaculation (Shoot).

● MNEMONIC

"Point and Shoot"

Parasympathetic erection (Point), **S**ympathetic ejaculation (Shoot). Nitric oxide and cyclic GMP mediate erection – the target of PDE5 inhibitors.

2 Sexual dysfunctions: classify by phase

The cleanest way to hold the dysfunctions is to walk the response cycle and ask what can fail at each phase. Desire, arousal, orgasm and pain each give a small named set.

Phase affected	Dysfunction	Core feature
Desire	Male hypoactive sexual desire disorder	persistently low/absent sexual thoughts and desire (DSM-5)
	Female sexual interest/arousal disorder	DSM-5 **merges** low desire and low arousal in women into one
Arousal	Erectile disorder (ED)	difficulty attaining or maintaining erection sufficient for activity
	Female sexual interest/arousal disorder	reduced lubrication/swelling and arousal (see above)
Orgasm	Premature (early) ejaculation	ejaculation within ~1 min of penetration, before wished, with distress
	Delayed ejaculation	marked delay or absence of ejaculation
	Female orgasmic disorder (anorgasmia)	marked delay, infrequency or absence of orgasm

Phase affected	Dysfunction	Core feature
Pain	Genito-pelvic pain/penetration disorder	DSM-5 **merges** vaginismus + dyspareunia: pain, fear, or pelvic-floor tightening on penetration

DSM-5 VS ICD-11

DSM-5 made two notable mergers: (1) **female sexual interest/arousal disorder** combines low desire and low arousal in women; (2) **genito-pelvic pain/penetration disorder** combines vaginismus and dyspareunia. **ICD-11** keeps these more separate (it retains distinct vaginismus and dyspareunia, and separate hypoactive sexual desire) and groups all of them under *conditions related to sexual health*, not the mental disorders chapter. ICD-11 also adds a useful **aetiological qualifier** set (organic, psychological, mixed).

The specifiers (apply to every dysfunction)

- **Lifelong vs acquired** – **lifelong** (primary): present since the first sexual experience. **Acquired** (secondary): began after a period of normal function. Acquired onset points harder toward an organic or situational cause.
- **Generalised vs situational** – **generalised**: occurs in all situations and with all partners. **Situational**: limited to certain partners, settings or types of stimulation. A man with erections on waking or with self-stimulation but not with a partner has a **situational** problem, which suggests psychogenic rather than organic ED.
- **Duration & distress** – DSM-5 requires symptoms for roughly **6 months** and clinically significant **distress**, on roughly 75–100% of occasions, before a dysfunction is diagnosed. Occasional difficulty is normal and is not a disorder.

3 Aetiology & assessment

The first decision is always **organic vs psychogenic**, while remembering that the two usually coexist (a mixed picture). A few features sort them.

Points to psychogenic

Sudden onset, situational (one partner, not another), normal morning/nocturnal erections, intact erections with self-stimulation, clear life stressor or relationship conflict, performance anxiety, younger patient.

Points to organic

Gradual, progressive onset, generalised (all situations), absent morning erections, older patient with vascular risk (diabetes, hypertension, smoking), on relevant medication, other physical signs.

Organic causes to know

- **Vascular** – the commonest organic cause of ED: atherosclerosis, diabetes, hypertension, smoking. ED can be an **early marker of cardiovascular disease**.
- **Neurological** – spinal cord injury, multiple sclerosis, diabetic autonomic neuropathy, pelvic surgery (prostatectomy).
- **Endocrine** – hypogonadism (low testosterone), hyperprolactinaemia, thyroid disease, diabetes.
- **Medications & substances** – the most examined cause. **SSRIs** cause sexual dysfunction in a large proportion (reduced desire, delayed/absent orgasm, ED). Others: antipsychotics (via hyperprolactinaemia), beta-blockers, thiazides, antihistamines, opioids, alcohol and chronic heavy drinking.

● CLASSIC TRAP

SSRI-induced delayed ejaculation is sometimes used therapeutically to treat premature ejaculation. The same off-target effect that is a problem in depression treatment is an indication in PE. Other SSRI strategies for the prescribed dysfunction: dose reduction, a drug holiday, switching to a lower-risk agent (bupropion, mirtazapine), or adding an antidote such as sildenafil. **Bupropion** and **mirtazapine** carry the lowest sexual side-effect burden.

A focused assessment

- **Structure** – sexual history (onset, course, situational vs generalised, with self-stimulation, morning erections), full **medical and drug history**, relationship and psychosocial context, mood/anxiety screen, and a focused exam. Always interview the partner where possible.
- **Investigations** – guided, not routine: fasting glucose/HbA1c, lipids, morning testosterone, prolactin and thyroid function where indicated. **Nocturnal penile tumescence** (erections in sleep) helps separate psychogenic (preserved) from organic (absent) ED.

4 Management of dysfunctions

The principle is simple: **treat the underlying cause**, combine psychological and biological approaches, and involve the partner. A few named techniques and drug classes carry the marks.

Psychological and behavioural

- **Sensate focus** (Masters & Johnson) – graded couple exercises that **ban intercourse and orgasm** at first, with partners taking turns to give and receive non-genital then genital touch. Removes the goal, breaks **performance anxiety**, and rebuilds intimacy. The cornerstone behavioural therapy for most dysfunctions.
- **Psychoeducation & couple therapy** – correct myths, address relationship conflict and communication; CBT targets anxiety and unhelpful beliefs.
- **Premature ejaculation techniques** – the **stop-start** technique (Semans) and the **squeeze** technique (Masters & Johnson) build ejaculatory control behaviourally.

Biological, by target dysfunction

Dysfunction	First-line drug approach	Note
Erectile disorder	PDE5 inhibitors: sildenafil, tadalafil, vardenafil	contraindicated with nitrates (severe hypotension)
Premature ejaculation	**SSRIs** (dapoxetine, on-demand; or daily) or topical anaesthetics (lidocaine/prilocaine)	uses the delayed-ejaculation side-effect therapeutically
Hypoactive desire / hypogonadism	testosterone replacement *only* if deficient; treat hyperprolactinaemia	flibanserin licensed for low desire in some settings (not India routine)
SSRI-induced dysfunction	dose reduction, switch (bupropion/mirtazapine), or add sildenafil	treat the cause, not just the symptom

HOLD THIS

PDE5 inhibitors for ED (never with nitrates) · **SSRIs or topical anaesthetics for PE** · **sensate focus** as the universal behavioural base · always **treat the underlying cause** and involve the partner.

RULE

PDE5 inhibitors work by blocking phosphodiesterase-5, so cyclic GMP and the nitric-oxide vasodilation that produces erection are sustained. They need **sexual stimulation** to work (they do not initiate erection on their own). Absolute contraindication: concurrent **nitrates** or nicorandil.

5 Paraphilic disorders

The single most important distinction in this section: a **paraphilia** is an atypical sexual interest; a **paraphilic disorder** is one that, in addition, causes **distress or impairment to the person**, or entails **harm or risk of harm to others** (including any non-consenting person). An interest alone is not a disorder.

◆ **Paraphilia vs paraphilic disorder** – the DSM-5 reframing (deliberate, to reduce stigma). The atypical interest is a **paraphilia**; it becomes a **disorder** only with current distress/impairment to self, or action on non-consenting persons (or where acting would cause harm or legal consequences). This separates an unusual but consensual private interest from a clinical condition.

Disorder	Focus of the atypical interest	Consent issue
Exhibitionistic	exposing one's genitals to an unsuspecting person	non-consenting
Voyeuristic	observing an unsuspecting person who is undressing or sexual	non-consenting
Frotteuristic	touching or rubbing against a non-consenting person	non-consenting
Paedophilic	sexual interest in prepubescent children	non-consenting, illegal
Sexual sadism	arousal from inflicting suffering on another	disorder if non-consenting or distress
Sexual masochism	arousal from being made to suffer	self; disorder if distress/impairment
Fetishistic	use of non-living objects or a specific non-genital body part	self; disorder if distress/impairment
Transvestic	arousal from cross-dressing	self; disorder if distress/impairment

MNEMONIC

"Every Vain Frog Plays Sadly, Masochistically, For Two"

Exhibitionistic, **V**oyeuristic, **F**rotteuristic, **P**aedophilic, **S**adism, **M**asochism, **F**etishistic, **T**ransvestic – the eight named DSM-5 paraphilic disorders.

FORENSIC & CONSENT

The disorders that involve **non-consenting persons** (exhibitionistic, voyeuristic, frotteuristic, paedophilic, and non-consensual sadism) carry forensic and child-protection obligations: the question of consent is decisive. A consensual, distress-free atypical interest between adults is not a clinical disorder and is not a forensic matter. **Paedophilia** is the interest; **child sexual abuse** is the criminal act, and not everyone who offends has the disorder, nor does everyone with the interest offend.

Management overview

- **Psychological** – CBT (including relapse prevention and victim empathy work), management of any co-occurring disorder, and addressing risk. Engagement and motivation are central.
- **Pharmacological (for high-risk cases)** – **SSRIs** (reduce drive and any compulsive quality), **antiandrogens** (cyproterone acetate, medroxyprogesterone) and **GnRH analogues** to lower testosterone in severe or high-risk cases. Used within a clear consent and monitoring framework.

6 Gender incongruence / gender dysphoria

Terminology and classification both shifted, and the shift is the examinable point. The core principle is that **a gender identity that differs from the sex assigned at birth is not in itself a disorder**. What may need clinical attention is the **distress** that can accompany it, and the support a person may seek.

System	Term used	Where it sits
ICD-10 (old)	"transsexualism", gender identity disorder	mental and behavioural disorders
DSM-5	**Gender dysphoria**	own chapter; focus is the **distress**, not the identity
ICD-11 (current)	**Gender incongruence**	moved OUT of mental disorders into *conditions related to sexual health*

◆ **The nosological shift** – **ICD-11 removed gender incongruence from the mental disorders chapter** (2019, in force 2022) and placed it under *conditions related to sexual health*. This depathologises the identity while retaining a code so that people can access care. **DSM-5** renamed gender identity disorder to **gender dysphoria**, shifting the diagnostic focus to the *distress* arising from the incongruence, not the identity itself.

● WHY THE CHANGE MATTERS

Classifying the identity as a disorder was stigmatising and not supported by evidence. The current framing separates the **identity** (not pathological) from any **distress** (which can be helped). A retained code preserves access to affirmative healthcare and insurance coverage without labelling the person as mentally ill.

A respectful overview of care

● **Affirmative, person-centred stance** – care supports the person in understanding and, where they wish, expressing their gender. The aim is wellbeing, not to change the identity. Language follows the person's own (correct name and pronouns).

● **Multidisciplinary pathway** – mental health support, endocrinology (gender-affirming hormones where appropriate), speech therapy, and surgery for those who choose it, with informed consent throughout. Not everyone wants every step; care is individualised.

■ **The psychiatrist's role** – assessment and support, treating any co-occurring mood, anxiety or other condition, managing minority-stress effects, and contributing to a multidisciplinary plan. The role is supportive and facilitative, never to question or pathologise the identity.

● LANGUAGE & RESPECT

Use **sex assigned at birth** (not "biological sex" in a way that invalidates identity), the person's **affirmed name and pronouns**, and avoid older stigmatising terms. Gender incongruence is distinct from **differences of sex development** (intersex conditions) and from sexual orientation, which is about attraction, not identity.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Response cycle:** Masters & Johnson = excitement → plateau → orgasm → resolution. Kaplan added **desire** at the front. Refractory period in men only; women may have none.
- **Physiology:** Point and Shoot – Parasympathetic erection, Sympathetic ejaculation. Erection runs on nitric oxide / cyclic GMP (the PDE5-inhibitor target).

- **Dysfunctions by phase:** desire = hypoactive desire / female interest-arousal · arousal = ED · orgasm = premature ejaculation, delayed ejaculation, anorgasmia · pain = genito-pelvic pain/penetration.
- **DSM-5 mergers:** female sexual interest/arousal disorder (desire + arousal); genito-pelvic pain/penetration disorder (vaginismus + dyspareunia). ICD-11 keeps them more separate and groups all under conditions related to sexual health.
- **Specifiers:** lifelong vs acquired; generalised vs situational. Situational with preserved morning erections = think psychogenic; gradual + generalised = think organic.
- **Causes:** vascular (commonest organic ED, early CVD marker), neurological, endocrine, and drugs. **SSRIs** are the classic medication cause; bupropion and mirtazapine are the lowest-burden options.
- **Management:** treat the cause + involve the partner. Sensate focus (Masters & Johnson) is the behavioural base; PDE5 inhibitors for ED (never with nitrates); SSRIs / topical anaesthetics for premature ejaculation.

★ **Paraphilias:** a paraphilia is an atypical interest; a paraphilic **disorder** adds distress/impairment to self OR harm/non-consent to others. The eight named: exhibitionistic, voyeuristic, frotteuristic, paedophilic, sexual sadism, sexual masochism, fetishistic, transvestic.

- **Forensic line:** consent is decisive. Paedophilia (the interest) is not the same as child sexual abuse (the act). Pharmacology for high-risk cases: SSRIs, antiandrogens, GnRH analogues.
- **Gender:** ICD-11 moved **gender incongruence OUT of the mental disorders chapter** into conditions related to sexual health; DSM-5 uses **gender dysphoria**, focusing on distress, not identity. Care is affirmative, multidisciplinary, consent-led; the identity is never pathologised.

ONE-DAY REVISION · PAPER 2

Sleep, physiology to disorders

Learn the architecture first (stages and their EEG signature), then the cycle across the night, then the switches that run it. Draw the hypnogram and the stage-EEG table and the marks come fast. The disorder tables are direct 5-10 mark answers. Cross-link sleep to depression, narcolepsy and the parasomnias and you have answered three questions for the price of one.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. Stage percentages are approximate adult means & vary across textbooks; quote them as ranges.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. The depression sleep signature is the classic long answer: reduced REM latency (first REM comes early), increased REM density, reduced slow-wave sleep (N3) and early-morning awakening, a state marker that is sensitive but not specific.
2. N2 is defined by sleep spindles (~11-16 Hz) and K-complexes, and either one alone scores the epoch as N2; learn the full stage-EEG map (N1 theta, N2 spindles + K-complexes, N3 delta, REM sawtooth + atonia with an awake-looking EEG).
3. N3 is front-loaded into the first third of the night, REM is back-loaded and lengthens toward morning; one cycle is ~90-110 min (4-6 a night) and the first REM appears ~80-100 min after sleep onset, so a much shorter REM latency is a red flag.
4. Narcolepsy type 1 is loss of orexin/hypocretin neurons giving the tetrad CHES (Cataplexy, Hypnagogic hallucinations, EDS, Sleep paralysis), all REM intrusions into wake; cataplexy is specific to type 1, confirmed by MSLT showing short latency plus ≥2 sleep-onset REM periods (HLA-DQB1*06:02).
5. Parasomnia split: NREM disorders of arousal (sleep terrors, sleepwalking) arise from N3 in the first third, in children, with no recall; the trap is RBD, a REM disorder with loss of atonia in the latter half in older men, dream enactment with recall, heralding an alpha-synucleinopathy (Parkinson's).

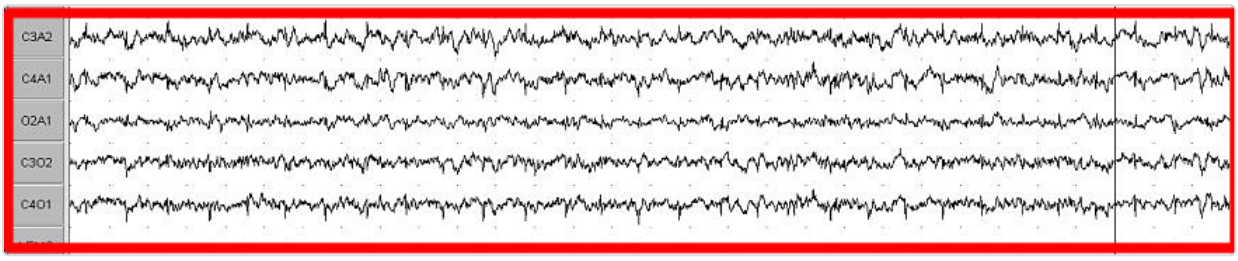
1 Sleep architecture & stages

Sleep splits into **NREM** (staged N1 → N2 → N3) and **REM**. Each stage carries a signature EEG. The exam wants the EEG correlate and the rough share of total sleep time. Modern AASM scoring merges the old stages 3 and 4 into a single **N3**.

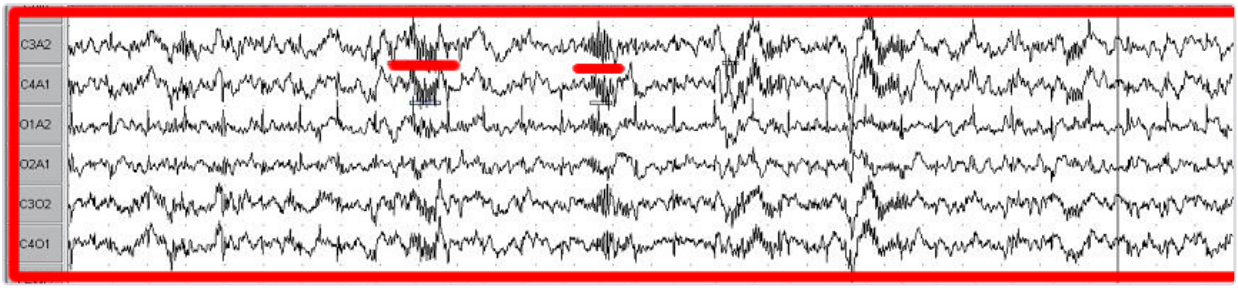
Stage	EEG correlate	Hallmark	% of sleep
Wake (eyes closed)	Alpha 8–13 Hz; beta when alert/eyes open	Alpha appears on eye closure; low-voltage fast beta when engaged	–
N1 (light, transitional)	Theta 4–7 Hz, low amplitude	Drowsy, easily roused; slow rolling eye movements; hypnic jerks; loss of alpha	~5%
N2	Theta with sleep spindles + K-complexes	True sleep onset; spindles (~11–16 Hz bursts) and K-complexes define it	~45–55%

Stage	EEG correlate	Hallmark	% of sleep
N3 (slow-wave, deep)	Delta <4 Hz, high amplitude	Hardest to rouse; growth-hormone surge; NREM parasomnias arise here	~15–20%
REM (paradoxical)	Low-voltage mixed frequency; sawtooth waves; EEG resembles wake	Muscle atonia (sparing eyes & diaphragm), vivid dreaming, rapid eye movements, autonomic variability, genital tumescence	~20–25%

REAL SLEEP EEG · N1 THEN N2, CENTRAL DERIVATIONS



N1 – low-voltage, mixed-frequency theta. The waking alpha has dropped out; no spindles or K-complexes yet.



The two short red underlines mark **sleep spindles**; the tall biphasic transients are **K-complexes** – together they define **N2**. Real polysomnography, central EEG channels. Source: MrSandman, public domain, Wikimedia Commons.

Reading the trace: the diagnostic waveforms

- **Posterior alpha rhythm** – wake with eyes closed; ~8–13 Hz over the occiput; attenuates on eye opening; its dropout marks the start of N1.
- **Vertex sharp waves** – N1; sharp negative transients maximal at the vertex (Cz); mark the descent into sleep.
- **Sleep spindles** – N2 hallmark; ~11–16 Hz (sigma), waxing-and-waning bursts lasting ~0.5–1.5 s; generated by the thalamic reticular nucleus; gate sensory input to protect sleep and support memory consolidation.
- **K-complexes** – N2 hallmark; a large biphasic transient, a sharp negative deflection then a slower positive one, lasting ≥0.5 s; occur spontaneously or are evoked by external stimuli such as a sound; thought to protect sleep and aid consolidation.
- **Delta / slow waves** – N3 (slow-wave sleep); high-amplitude (>75 microvolt) slow waves (<2 Hz); AASM scores N3 once slow-wave activity fills ≥20 percent of the epoch.
- **Sawtooth waves** – REM; notched, triangular 2–6 Hz waves that often herald bursts of rapid eye movements.

RULE

Sleep spindles and K-complexes are the two markers that define N2; the presence of either is enough to score the epoch as N2.

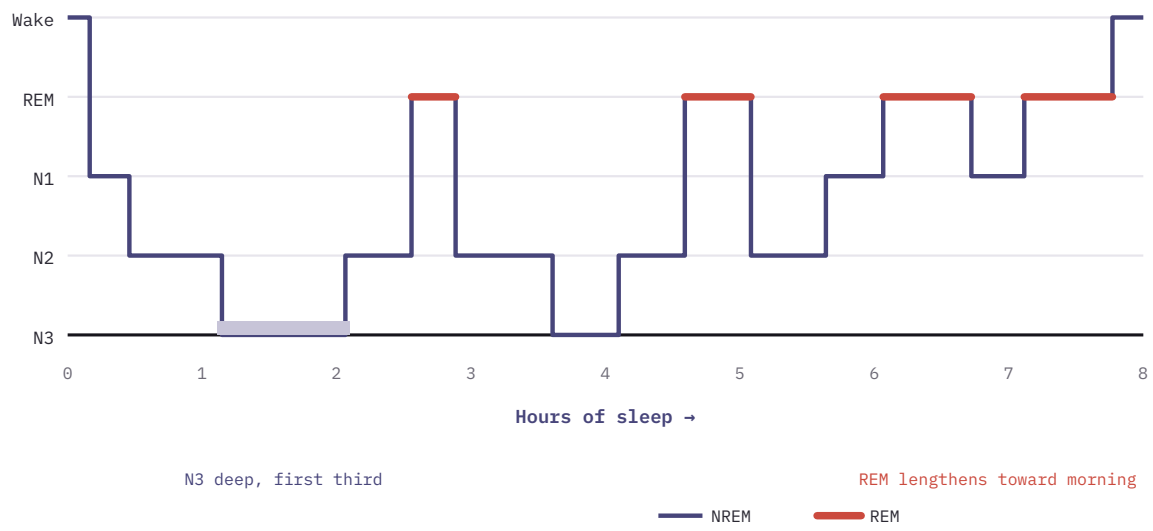
CLASSIC TRAP

REM is **paradoxical sleep**: the EEG is desynchronised and looks awake, yet the body is maximally relaxed in **atonia**. Dreaming is most vivid in REM, but mentation also occurs in NREM (sleep-terror imagery is fragmentary, not narrative). Atonia spares the extra-ocular muscles and the diaphragm, which is why the eyes dart and breathing continues.

2 Sleep across the night

One cycle runs **~90–110 minutes**; a normal night holds **4–6 cycles**. Two rules dominate. **N3 (slow-wave sleep) is front-loaded** into the first third of the night, and **REM periods lengthen toward morning**. Hence early-night waking robs you mainly of deep sleep, while a morning lie-in is mostly REM. The first REM episode appears roughly 80–100 minutes after sleep onset; a much shorter latency is a red flag (see section 5).

HYPNOGRAM · ONE NIGHT, DEEP EARLY THEN REM-RICH TOWARD DAWN



Read left to right: sleep plunges to **N3 early**, then each cycle climbs shallower as the night goes on, with **REM (coral) getting longer** each cycle. Brief wake intrusions are normal.

~5%	45–55%	15–20%	20–25%
N1	N2	N3 (SWS)	REM

HOLD THIS

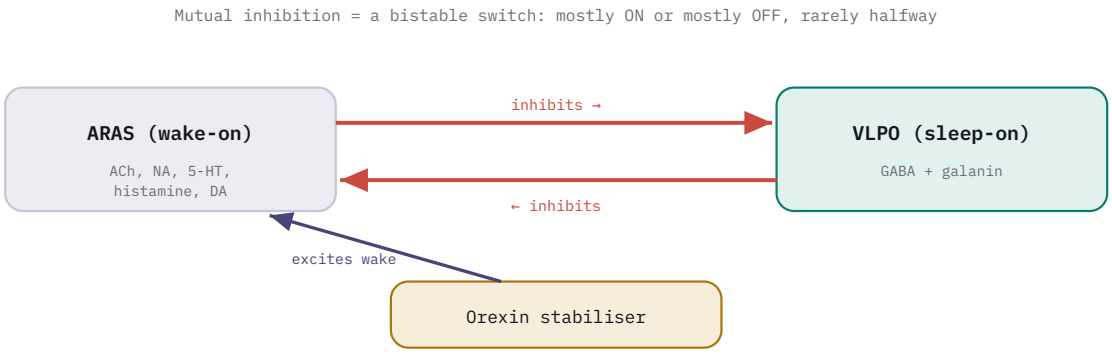
N3 front-loaded, REM back-loaded. Cycle ~90 min, 4–6 a night. First REM ~80–100 min after onset. Across the lifespan: newborns spend ~50% in REM and enter sleep through REM; SWS declines steadily with age, so the elderly have lighter, more fragmented sleep.

3 Neurobiology of sleep and wake

Wake and sleep are run by mutually inhibiting circuits, a **flip-flop switch** that snaps between states and resists the in-between. Orexin holds the switch on the wake side; lose it and the switch flickers.

System	Nucleus / source	Transmitter & role
Wake-promoting (ARAS)	Brainstem reticular formation & relays	ACh (LDT/PPT, basal forebrain), noradrenaline (locus coeruleus), serotonin (raphe), histamine (tuberomammillary nucleus), dopamine, orexin
Sleep-promoting	VLPO (ventrolateral preoptic nucleus)	GABA + galanin; the sleep "off switch", reciprocally inhibits the arousal centres
Switch stabiliser	Orexin / hypocretin (lateral hypothalamus)	Excites the arousal centres to keep wake stable; **loss → narcolepsy type 1**
Homeostatic pressure	Accumulates with waking hours	Adenosine builds → sleepiness; **caffeine is an adenosine antagonist**
Circadian timing	SCN (hypothalamus) → pineal	SCN paces the day; pineal melatonin rises in darkness, suppressed by light

THE SLEEP-WAKE FLIP-FLOP SWITCH (SAPER)



ARAS and VLPO inhibit each other, so the brain sits firmly in wake or sleep, not drifting between. Orexin (amber) biases the switch toward wake. Lose orexin and the switch destabilises: sleep and REM intrude into wake = narcolepsy.

● MNEMONIC

"VLPO is the OFF switch the brain flicks to sleep"

VLPO = GABA + galanin, the off switch. Orexin keeps it from flickering. Lose orexin → unstable switch → intrusions of sleep and REM into wake = narcolepsy with cataplexy.

REM control: the reciprocal-interaction model

Within sleep, REM is set by a second seesaw (McCarley & Hobson). **REM-ON cholinergic** pontine neurons fire against **REM-OFF aminergic** neurons (serotonin from raphe, noradrenaline from locus coeruleus).

	REM-ON (drives REM)	REM-OFF (suppresses REM)
Neurons	Cholinergic (pontine, LDT/PPT)	Aminergic: serotonergic (raphe) + noradrenergic (locus coeruleus)
During REM	Active (↑)	Silent (↓, aminergic off)
Clinical lever	Cholinergic agonists / AChE inhibitors increase REM	SSRIs, SNRIs, TCAs, MAOIs suppress REM (raise REM latency)

● RULE

REM atonia is generated by pontine REM-ON output driving medullary inhibition of spinal motor neurons. Failure of this descending inhibition is **REM sleep behaviour disorder**. The aminergic-off, cholinergic-on state of REM also underlies why most antidepressants (which raise aminergic tone) suppress REM, a clean cross-link to section 5.

Borbely's two-process model

- **Process S (homeostatic)** – sleep pressure builds with time awake (adenosine accumulation) and dissipates during sleep. The longer you are awake, the steeper the drive to sleep.
- **Process C (circadian)** – a clock-driven oscillation of sleep propensity from the SCN, independent of prior sleep. Endogenous period is **slightly longer than 24 h**, reset daily by light through the retinohypothalamic tract. Light is the strongest **zeitgeber**.
- **The interaction** – sleep is favoured when the **gap between S and C is greatest**. The model explains the mid-afternoon dip, the evening "wake-maintenance zone" just before habitual sleep, and why a night-shift worker fights both a high S and a wake-promoting C.

4 Sleep disorders as exam answers

Group them by mechanism: insomnia, breathing-related, central hypersomnia (narcolepsy), movement disorders, parasomnias (split NREM vs REM), and circadian disorders. Each row below is a ready 5-mark answer.

Disorder	Core mechanism & picture	Anchor fact
Insomnia	Difficulty initiating or maintaining sleep, or early waking, with daytime impairment, despite adequate opportunity. Most common sleep complaint	First-line CBT-I; pharmacotherapy second-line
Obstructive sleep apnoea	Repetitive upper-airway collapse → apnoeas/hypopnoeas, desaturation, arousals. Snoring, witnessed apnoeas, daytime sleepiness; obesity a key risk	Diagnosis polysomnography (AHI); treatment CPAP
Narcolepsy	Loss of orexin/hypocretin neurons (type 1, with cataplexy). Sleep architecture destabilised; REM intrudes into wakefulness	Sleep-onset REM; confirmed on MSLT
Restless legs (RLS)	Urge to move the legs, worse at rest and in the evening, relieved by movement. Dopaminergic dysfunction	Linked to iron deficiency; check ferritin
PLMD	Periodic limb movements in sleep causing repetitive arousals and unrefreshing sleep	Scored on polysomnography; often co-occurs with RLS
Circadian disorders	Internal clock misaligned with the desired schedule	Managed with timed light + melatonin

Narcolepsy: the tetrad

Type 1 narcolepsy is an **orexin/hypocretin deficiency** (low CSF hypocretin-1; strong HLA-DQB1*06:02 association; presumed autoimmune loss of lateral-hypothalamic neurons). The unstable switch lets REM phenomena bleed into wakefulness, which generates the tetrad.

Feature	What it is	REM logic
Excessive daytime sleepiness	Irresistible sleep attacks; the core, near-universal symptom	Wake cannot be held
Cataplexy	Sudden bilateral loss of muscle tone with emotion (laughter), consciousness preserved	REM atonia intruding into wake (specific to type 1)
Sleep paralysis	Transient inability to move at sleep onset or waking	REM atonia at the wake-sleep boundary

Feature	What it is	REM logic
Hypnagogic hallucinations	Vivid dream-like imagery falling asleep (hypnopompic on waking)	REM dream mentation intruding

MNEMONIC

"CHES" → Cataplexy, Hallucinations (hypnagogic), EDS, Sleep paralysis

All four are **REM intrusions** into wakefulness from a destabilised switch. Cataplexy is the one specific to orexin loss (type 1). Diagnosis: MSLT showing short sleep latency plus ≥2 sleep-onset REM periods.

Parasomnias: NREM versus REM

The single highest-yield split. Tie each cluster to the stage it arises from, the part of the night, the typical age, and whether the patient recalls anything.

NREM parasomnias (disorders of arousal)

Sleep terrors, sleepwalking, confusional arousals. Arise from **N3 deep sleep**, so in the **first third** of the night. Commoner in **children**. Partial arousal: the person is hard to wake, **amnesic** for the event, with no narrative dream recall. A sleep terror shows intense autonomic surge and a scream; sleepwalking is complex motor behaviour with open eyes but no awareness.

REM parasomnias

Nightmares arise from REM (later half of the night), with vivid recall and full alerting on waking. **REM sleep behaviour disorder (RBD)**: **loss of REM atonia** → acting out (often violent) dreams in the **latter half** of the night, *with* recall. Older men; strong link to **alpha-synucleinopathies** (Parkinson's disease, Lewy body dementia, multiple system atrophy), often years before motor signs.

CLASSIC TRAP

Sleep terror vs nightmare. **Sleep terror**: NREM (N3), first third, child, autonomic storm, no recall, inconsolable, returns to sleep. **Nightmare**: REM, last third, full recall, wakes alert and oriented. RBD is the dangerous one to flag, because the dream enactment can injure the patient or a bed partner and it heralds a synucleinopathy.

Circadian rhythm sleep-wake disorders

- **Delayed sleep phase** – the clock runs late: cannot fall asleep or wake at conventional times (the "night owl"). Common in **adolescents**.
- **Advanced sleep phase** – the clock runs early: sleepy in the early evening, wakes before dawn. Commoner in the **elderly**.
- **Shift-work & jet-lag** – an external schedule forced against the clock; sleepiness on shift, insomnia off it. Eastward travel (phase advance) is harder to adjust to than westward.
- **Non-24-hour rhythm** – the free-running clock never entrains; classic in **totally blind** individuals lacking light input. Managed with timed melatonin.

5 Sleep in psychiatric illness

High-yield table. **Reduced REM latency** and **reduced slow-wave sleep** recur across several conditions; **depression is the classic exam answer**. Reduced REM latency is sensitive but not specific.

Condition	Characteristic sleep changes
Depression	Reduced REM latency (REM comes early), increased REM density, increased total REM, **reduced SWS (N3)**, **early-morning awakening**, prolonged sleep latency, reduced sleep efficiency
Mania	**Reduced need for sleep** (feels rested on little sleep, unlike the fatigue of insomnia); reduced total sleep time; sleep loss can itself trigger or worsen an episode

Condition	Characteristic sleep changes
Anxiety / GAD	Prolonged sleep latency, difficulty maintaining sleep, increased arousals; worry-driven hyperarousal at sleep onset
PTSD	**Nightmares** (often trauma replay), fragmented sleep, increased awakenings, increased REM-related arousals; insomnia is a core symptom
Alcohol	Acutely **suppresses REM** and shortens sleep latency, then second-half fragmentation; on withdrawal **REM rebound** (vivid dreams, nightmares); chronic use reduces SWS
Schizophrenia	Reduced SWS, reduced REM latency, poor sleep efficiency and continuity; reduced sleep-spindle density reported
Dementia	Fragmentation, reduced SWS and REM, **sundowning** (evening confusion/agitation), circadian disruption, day-night reversal

THE DEPRESSION SIGNATURE

Short REM latency, high REM density, low SWS, early-morning waking. A favourite long answer. It is a state marker that often improves with treatment, and most antidepressants **suppress REM** (the opposite direction), a clean link back to section 3.

● CROSS-LINK & RISK

Insomnia and nightmares are independent risk factors for **suicide**, over and above depression severity; persistent insomnia warrants direct risk assessment. Note the therapeutic paradox in depression: a single night of **total sleep deprivation** produces a rapid but transient mood lift in some patients, consistent with REM and sleep-pressure models, though relapse on recovery sleep limits its clinical use.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Stages:** N1 theta · N2 spindles + K-complexes · N3 delta (SWS) · REM sawtooth + atonia + EEG like wake (paradoxical). Wake = alpha (eyes closed) / beta (alert).
- **Percentages:** N1 ~5%, N2 ~45–55%, N3 ~15–20%, REM ~20–25%. Cycle ~90–110 min, 4–6 a night.
- **Across the night:** N3 front-loaded (first third), REM back-loaded (lengthens toward morning). First REM ~80–100 min after onset. SWS falls with age; newborns ~50% REM.
- **Switch:** VLPO (GABA/galanin) off-switch vs ARAS wake; orexin stabilises; adenosine = pressure (caffeine antagonist). Flip-flop = bistable.
- **REM control:** REM-ON cholinergic vs REM-OFF aminergic (5-HT/NA). Most antidepressants suppress REM. Atonia from pontine-to-medullary inhibition.
- **Circadian:** SCN master clock (period >24 h), melatonin from pineal (rises in dark, suppressed by light), light = strongest zeitgeber.
- **Borbely:** Process S homeostatic (adenosine) + Process C circadian; sleep when the S-C gap is greatest.
- **Narcolepsy tetrad (CHES):** Cataplexy, Hypnagogic hallucinations, EDS, Sleep paralysis. Orexin loss, sleep-onset REM, MSLT, HLA-DQB1*06:02. Cataplexy specific to type 1.
- **Parasomnias:** NREM = N3, first third, children, no recall (terrors, sleepwalking). RBD = REM, atonia loss, latter half, older men, recall, synucleinopathy / Parkinson's.
- **Sleep disorders:** insomnia → CBT-I first-line. OSA → PSG/AHI, CPAP, obesity. RLS → iron deficiency + dopaminergic, worse evening, relieved by movement.
- ★ **Depression:** short REM latency, high REM density, low SWS, early-morning waking. Mania: reduced need for sleep. Alcohol: REM suppressed acutely → REM rebound on withdrawal.
- **Risk:** insomnia and nightmares independently raise suicide risk; dementia → sundowning + day-night reversal.

II

ONE-DAY REVISION · PAPER 2

Geriatric Psychiatry

The psychiatry of the ageing brain: what is normal cognitive ageing and what is disease, how drugs behave differently in an old body, and the one table the examiner returns to every year – delirium against dementia against depression. Hold the three Ds cold, then the four dementias, and the rest is detail.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. Full organic detail on each dementia sits in the Paper 4 organic pack; this is the geriatric-psychiatry view.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. The three Ds: delirium = acute, fluctuating, **inattention + clouded consciousness** (the core discriminators), reversible; dementia = insidious, clear consciousness, progressive; depression = "I don't know" answers that improve with cues and reverse with treatment.
2. Ageing gives reduced clearance (falling GFR is the key change) plus increased receptor sensitivity, so start low, go slow at roughly half the adult starting dose; a normal creatinine hides a low GFR, which is why lithium accumulates.
3. Severe antipsychotic sensitivity in dementia with Lewy bodies can be fatal, so avoid antipsychotics; the fluctuating cognition, visual hallucinations and parkinsonism of DLB respond instead to a cholinesterase inhibitor.
4. Always exclude reversible mimics before a neurodegenerative diagnosis: **normal-pressure hydrocephalus** is "wet, wacky, wobbly" (urinary incontinence, cognitive decline, gait apraxia) and is shunt-treatable, alongside B12, thyroid (TSH) and depression.
5. Antipsychotics in dementia carry a boxed warning of increased stroke and all-cause mortality, so use non-pharmacological measures first; risperidone holds the short-term licence for persistent aggression in Alzheimer's.

1 Ageing and the brain

Two questions open every old-age viva: what changes are normal, and how does the same drug behave differently in an old body. The whole prescribing logic of geriatric psychiatry follows from the second.

Normal cognitive ageing vs disease

Domain	Normal ageing	Points to disease
Processing speed	slows; reaction time lengthens	–
Memory	slower *retrieval*; cued recall intact; name-finding lapses	failure to **store** new memory; cueing does not help; getting lost
Crystallised knowledge	vocabulary, facts **preserved** or rise	loss of known words, semantic knowledge
Function	independent in daily activities (ADLs)	decline in instrumental then basic ADLs
Insight	aware of and bothered by lapses	often **reduced** awareness (esp. AD)

QUICK RULE

Normal ageing slows **retrieval** and is helped by cues; dementia fails to **encode** and cues do not rescue it. Lost function and lost insight push you toward disease.

Pharmacokinetics: what the old body does to the drug

The net effect of ageing is **reduced clearance** and a longer half-life, so the same dose accumulates.

Phase	Change with ageing	Consequence
Absorption	raised gastric pH, slower motility	little net change overall
Distribution	↑ body fat, ↓ total body water, ↓ albumin	lipophilic drugs (most psychotropics) gain a larger reservoir → longer half-life; more free drug if albumin low
Metabolism	↓ hepatic mass and blood flow; phase I (oxidation) falls more than phase II	slower first-pass and clearance
Excretion	↓ GFR and renal clearance (the key change)	renally cleared drugs (lithium, amisulpride, gabapentin) accumulate → toxicity

● CLASSIC TRAP

A normal serum **creatinine** in an older person can hide a low GFR, because reduced muscle mass produces less creatinine. Renal function is worse than the number suggests. This is why **lithium** needs more frequent monitoring and lower doses in the old.

Pharmacodynamics: how the old brain responds

■ **Increased sensitivity** – the ageing brain (and body) responds more strongly at the receptor for a given concentration. Greater sensitivity to **benzodiazepines** (sedation, falls, confusion), **anticholinergics** (delirium), **antipsychotics** (EPSE, postural drop) and **opioids**.

HOLD THIS

Start low, go slow. Begin at roughly half the usual adult starting dose and titrate upward gradually against response and tolerability. Reduced clearance plus increased sensitivity means standard doses overshoot.

Polypharmacy and anticholinergic burden

● **Polypharmacy** – multiple comorbidities mean many drugs; each addition raises interaction, adverse-effect and non-adherence risk. The **prescribing cascade**: a side effect is mistaken for a new illness and treated with a further drug.

◆ **Anticholinergic burden** – the cumulative load of anticholinergic drugs (some antipsychotics, TCAs, oxybutynin, antihistamines, some antiparkinsonians). High burden → **confusion, delirium, falls, constipation, urinary retention, blurred vision**, and is linked to accelerated cognitive decline. Audited with tools such as STOPP/START and the Beers criteria; minimise in anyone with cognitive impairment.

2 The dementias: an overview

Four major neurodegenerative and vascular dementias. For the geriatric viva you need the **one distinguishing feature** of each, not the full neuropathology (that sits in the organic pack). Then mild cognitive impairment, and the reversible mimics you must exclude.

Type	Distinguishing feature	Onset / course	Notes for management
Alzheimer's disease (commonest, ~60%)	**early episodic memory** loss; insidious; later language, visuospatial, apraxia	slow, steady decline	responds best to cholinesterase inhibitors
Vascular dementia (2nd commonest)	**stepwise** decline, focal signs, vascular risk factors; patchy deficits	abrupt steps after events	treat vascular risk; ChEIs not first-line
Dementia with Lewy bodies	fluctuating cognition, recurrent **visual hallucinations**, spontaneous parkinsonism, REM sleep behaviour disorder	fluctuating, progressive	severe antipsychotic sensitivity – avoid; ChEIs help
Frontotemporal dementia	**early personality / behaviour change** or progressive aphasia; memory relatively spared early; younger onset	insidious, often <65 yrs	ChEIs not indicated; behavioural management

● CLASSIC TRAP

In **dementia with Lewy bodies**, conventional antipsychotics can cause severe, sometimes fatal sensitivity reactions (marked parkinsonism, autonomic instability, neuroleptic-malignant-like states). If an antipsychotic is unavoidable for distressing psychosis, use a low-dose atypical (e.g. quetiapine) cautiously. The visual hallucinations themselves often respond to a cholinesterase inhibitor.

Mild cognitive impairment (MCI)

- MCI – objective cognitive decline *beyond* normal ageing but **without** significant loss of daily function (function is the line that separates MCI from dementia). Amnestic and non-amnestic subtypes. A risk state: amnestic MCI converts to Alzheimer's at roughly 10–15% per year, though some remain stable or revert.

Reversible causes to exclude

Before settling on a neurodegenerative diagnosis, screen for treatable mimics. These are the "do not miss" causes.

Cause	Clue	Test
B12 / folate deficiency	macrocytosis, neuropathy, subacute combined degeneration	serum B12, folate
Hypothyroidism	slowing, weight gain, cold intolerance	TSH
Normal-pressure hydrocephalus	the triad: **gait apraxia, urinary incontinence, cognitive decline** ("wet, wacky, wobbly")	CT/MRI → ventriculomegaly; LP
Depression (pseudodementia)	recent onset, low mood, "don't know" answers	mood assessment; treatment trial
Others	delirium, alcohol, medication (anticholinergics, sedatives), subdural, hypercalcaemia, neurosyphilis/HIV	history, bloods, imaging

● MNEMONIC

NPH → "Wet, Wacky, Wobbly"

Urinary incontinence (wet), cognitive decline (wacky), gait apraxia (wobbly). A potentially reversible dementia – treated by shunting. Gait usually responds best.

3 The three Ds: delirium vs dementia vs depression

This is the centrepiece of old-age psychiatry and the most reliably asked table in the paper. The three present with cognitive complaints and overlap, yet each has a separable signature. Read it down the rows: onset, course, attention, consciousness, reversibility.

Feature	Delirium	Dementia	Depression (pseudodementia)
Onset	acute (hours to days)	insidious (months to years)	subacute (weeks); often datable
Course	**fluctuating**, worse at night	slowly progressive, stable through the day	fairly stable; diurnal mood variation
Attention	grossly impaired (the core deficit)	normal early; impaired late	poor effort / inattentive, but cued-able
Consciousness	clouded / altered (the key discriminator)	clear until very late	clear
Memory	poor registration (inattention)	recent > remote loss; no benefit from cues	patchy; **improves with cueing**
Typical answers	muddled, perplexed	confabulates, near-misses, tries	**"I don't know"**; gives up
Psychomotor	hyper- or hypo-active; tremor, myoclonus	usually normal early	retardation or agitation
Perception	illusions, vivid visual hallucinations common	variable, usually later	mood-congruent if any
Reversibility	reversible if cause treated	usually irreversible / progressive	reversible with antidepressant treatment

HOLD THIS

If you remember nothing else: **delirium** = acute, fluctuating, **inattention + clouded consciousness**. **Dementia** = insidious, clear consciousness, progressive. **Depression** = "don't know" answers that improve with cues, and it **reverses with treatment**.

● CLASSIC TRAP

The three coexist far more often than they exclude. Delirium is frequently **superimposed on dementia** (dementia is the single biggest risk factor for delirium), and depression often accompanies early dementia. Any *acute* change in a person with dementia is delirium until proven otherwise – look for infection, drugs, dehydration, retention, pain. Do not anchor on the known dementia.

● MNEMONIC

Delirium causes → "I WATCH DEATH"

Infection, Withdrawal, Acute metabolic, Trauma, CNS pathology, Hypoxia, Deficiencies, Endocrine, Acute vascular, Toxins/drugs, Heavy metals. A standard screen for the reversible drivers behind an acute confusional state.

4 Late-life mood and psychosis

Late-life depression

Depression in old age is common, under-recognised, and presents differently: somatic complaints, anxiety and cognitive slowing predominate over reported sadness.

- **Presentation** – more **somatic symptoms, agitation, hypochondriasis** and cognitive impairment; less overt verbal sadness. May masquerade as physical illness or as dementia.

◆ **Depression vs dementia (pseudodementia)** – depressive pseudodementia: **recent, datable onset**, the patient *complains* of poor memory, gives **"I don't know"** answers, performance **improves with cueing**, mood is low, and it reverses with antidepressant treatment. In dementia the patient often conceals or is unaware of deficits and tries to answer. When uncertain, treat the depression and reassess cognition.

- **Vascular depression** – late-onset depression linked to cerebrovascular disease (white-matter hyperintensities on MRI). More executive dysfunction, psychomotor retardation, apathy and anhedonia; poorer and slower antidepressant response. The "vascular depression hypothesis" (Alexopoulos).

CLASSIC TRAP · SUICIDE

Suicide risk **rises with age**, and is highest in **older men**. Older people use more lethal methods, give fewer warnings, and complete more of their attempts. Physical illness, pain, isolation, bereavement and alcohol compound the risk. Always ask directly; do not be falsely reassured by an apparently low-key presentation.

Late-life psychosis

- **Very-late-onset schizophrenia-like psychosis** – new psychosis with onset **after age 60** (international consensus terminology). Features: prominent **persecutory delusions**, often **partition delusions** (belief that people/objects pass through walls/floors into the home), well-organised; relatively preserved personality and affect; female preponderance; strong association with **sensory impairment** (deafness, poor vision) and social isolation. Always exclude an organic cause and dementia first.
- **Paraphrenia** – the older term (Kraepelin; later "late paraphrenia", Roth) for a late-onset paranoid psychosis with systematised delusions and hallucinations but **preserved personality and intellect** and no progression to dementia. Largely superseded by the consensus terminology above, but examinable as a historical concept.

5 Behavioural and psychological symptoms of dementia (BPSD)

BPSD are the non-cognitive symptoms of dementia and the commonest reason for referral, carer breakdown and institutionalisation. They affect the majority of people with dementia at some point.

The symptom clusters

Agitation & aggression – restlessness, shouting, resistance to care, physical aggression.
Psychosis – delusions (often theft, infidelity, misidentification), hallucinations.
Mood – depression, anxiety, apathy.
Activity – wandering, pacing, disinhibition.

Sundowning

Worsening of confusion, agitation and behaviour in the **late afternoon and evening**. Linked to fatigue, low light, disrupted circadian rhythm and understimulation. Managed by routine, daytime light and activity, and a calm evening environment – not by sedation as first move.

HOLD THIS

Non-pharmacological first. Look for and treat a cause (pain, infection, constipation, retention, fear, boredom, an unmet need); use environmental and behavioural strategies before any drug. Most BPSD has a trigger.

- **First-line: non-drug** – identify triggers and unmet needs, person-centred care, structured activity, music, reminiscence, carer education and support, consistent routine, optimise sensory aids. Rule out delirium and pain driving the behaviour.

● CLASSIC TRAP · THE BOXED WARNING

Antipsychotics in dementia carry a regulatory warning: increased risk of **stroke / cerebrovascular events** and **all-cause mortality**. Reserve them for severe distress or risk of harm that has not responded to non-drug measures; use the lowest dose for the shortest time, with informed consent and regular review to stop. **Avoid antipsychotics in dementia with Lewy bodies** (severe sensitivity). **Risperidone** is the antipsychotic with a specific short-term licence for persistent aggression in Alzheimer's in several jurisdictions.

6 Assessment and management

Cognitive testing

Tool	What it is	Note
MMSE	11-item screen, score out of 30	quick; weak on executive/frontal function; education- and language-biased
MoCA	screen out of 30; adds executive, abstraction, attention	more sensitive to MCI and frontal-executive deficits than MMSE
ACE / ACE-III	Addenbrooke's, out of 100; five domains (attention, memory, fluency, language, visuospatial)	more detailed; helps profile the dementia subtype

Detailed scoring and cut-offs are cross-referenced in the assessment pack.

Pharmacological treatment of cognition

Drug class	Agents	Mechanism & use
Cholinesterase inhibitors	donepezil, rivastigmine, galantamine	raise synaptic acetylcholine; for **mild–moderate** AD, also DLB/PDD; symptomatic, not disease-modifying
NMDA antagonist	memantine	reduces glutamatergic excitotoxicity; for **moderate–severe** AD; can combine with a ChEI

▲ ChEI side effects – **cholinergic**: nausea, vomiting, diarrhoea, anorexia, weight loss, vivid dreams, and crucially **bradycardia / syncope** (caution with heart block, check pulse). Rivastigmine also comes as a patch (fewer GI effects).

● RULE

Cholinesterase inhibitors are **not indicated** in frontotemporal dementia (may worsen behaviour) and are not first-line in pure vascular dementia. They are the mainstay in Alzheimer's and genuinely useful for the hallucinations and fluctuations of **Lewy body** dementia.

Managing depression and psychosis cautiously

- Late-life depression – **SSRIs first-line** (sertraline, escitalopram); start low, go slow, allow a longer trial. Watch **hyponatraemia (SIADH)**, GI bleeding (esp. with NSAIDs), falls and QTc. **ECT** is highly effective and safe in the old, especially for severe, psychotic or treatment-resistant depression and where rapid response is needed. Avoid TCAs and paroxetine where possible (anticholinergic load).
- Late-life psychosis – treat the cause; low-dose atypical antipsychotic with the same stroke/mortality caution as in BPSD; correct sensory deficits and isolation in very-late-onset psychosis.

Capacity, safeguarding and elder abuse

◆ **Capacity** – decision-specific and time-specific: the person must **understand** the information, **retain** it, **weigh** it, and **communicate** a choice. Capacity is presumed unless shown otherwise; a person may have capacity for one decision and not another. Cognitive impairment does not automatically remove capacity. Act in best interests, least restrictive option, when capacity is lacking.

● **Elder abuse** – physical, psychological, financial, sexual, and **neglect** (including self-neglect); often by a known carer or relative, frequently unreported. Red flags: unexplained injuries, unexplained financial change, fearfulness around a carer, poor hygiene or unmet care needs. Carer stress is a major driver; assess and support the carer as part of management. Have a low threshold to raise a **safeguarding** concern.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Ageing PK/PD:** reduced clearance (↓ GFR is key) + increased sensitivity → **start low, go slow**, ~half the adult start dose. Normal creatinine hides low GFR; lithium accumulates.
- **Normal vs disease:** ageing slows *retrieval* and cues help; dementia fails to *encode* and cues don't. Lost function + lost insight = disease.
- **Anticholinergic burden:** confusion, delirium, falls, retention; minimise in cognitive impairment (STOPP/START, Beers).
- **Four dementias:** AD = early episodic memory · vascular = stepwise + focal signs · **DLB** = fluctuation + visual hallucinations + parkinsonism + antipsychotic sensitivity · FTD = early personality/behaviour change, younger.
- **MCI:** cognitive decline without functional loss; amnestic type converts to AD ~10–15%/yr.
- **Reversible mimics:** B12, thyroid (TSH), **NPH** (wet, wacky, wobbly), depression. Always exclude.
- ★ **Three Ds:** delirium = acute, fluctuating, **inattention + clouded consciousness**, reversible · dementia = insidious, clear consciousness, progressive · depression = "don't know" answers, improve with cues, reversible. Delirium is often superimposed on dementia.
- **Late-life depression:** somatic + agitated; pseudodementia improves with cues; vascular depression (WMH, executive, apathy); **suicide highest in older men**.
- **Late-life psychosis:** very-late-onset schizophrenia-like psychosis (>60), persecutory + **partition** delusions, sensory impairment, female; paraphrenia = the older term, personality preserved.
- **BPSD:** non-pharmacological first; find the trigger; sundowning = evening worsening. Antipsychotics carry a **stroke + mortality** warning; avoid in DLB; risperidone short-term licence in AD aggression.
- **Cognition drugs:** ChEIs (donepezil, rivastigmine, galantamine) for mild–moderate AD + DLB; memantine (NMDA) for moderate–severe. ChEIs → GI upset + **bradycardia**; not for FTD.
- **Capacity:** understand, retain, weigh, communicate; decision-specific; presumed present. Elder abuse includes neglect; support the carer; safeguard.

ONE-DAY REVISION · PAPER 2

Consultation-Liaison Psychiatry

Psychiatry at the bedside of the medically ill: the models (consultation vs liaison), the common reasons a ward calls, the psychosomatic tradition behind it, how people react to illness, the psycho-oncology and end-of-life interface, and the capacity-and-consent toolkit. The three carrying tables – reasons for referral, Alexander's holy seven, reactions to illness – are each a short-answer in waiting.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. CL = consultation-liaison; the general-hospital subspecialty also called psychosomatic medicine.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. **Consultation = the patient** (reactive, one-off, by referral); **liaison = the team** (proactive, embedded in a unit); CL blends both, and the explicit framing model is **Engel's biopsychosocial model (1977)**.
2. **The first duty in any acute change of mental state on a ward is to exclude and treat an organic cause (delirium) before reaching for a psychiatric label**: hypoactive delirium is misread as depression, agitated delirium as primary psychosis.
3. Alexander's "holy seven" (mnemonic **A HUT RUN**): Asthma, Hypertension, Ulcer (peptic), Thyrotoxicosis, Rheumatoid arthritis, Ulcerative colitis, Neurodermatitis; the list is examinable but the **specificity hypothesis (one conflict → one disease) is rejected**.
4. Capacity has four functional abilities, **understand, retain, weigh/use, communicate**: it is presumed, decision-specific and time-specific, an **unwise decision is not incapacity**, and it is never inferred from the diagnosis.
5. Demoralisation (hopelessness, helplessness, loss of meaning) is **distinct from depression**; to catch depression in cancer, lean on cognitive/affective items (hopelessness, worthlessness, guilt, anhedonia), **not the somatic ones** that overlap with the illness.

1 Definitions and models

Consultation-liaison (CL) psychiatry is the practice of psychiatry in the **general hospital**, at the interface with medicine and surgery. Its founding frame is that mind and body are one system, so the medically ill are also psychologically ill, and the two influence each other.

● **Consultation-liaison psychiatry** – the branch (US: *psychosomatic medicine*, an ABPN subspecialty since 2003, renamed *consultation-liaison psychiatry* in 2018) that assesses and manages psychiatric morbidity in patients on medical and surgical wards and in general-hospital clinics.

The two models

Model	How it works	Trigger & focus
Consultation model	case-by-case. The medical team refers a specific patient; the psychiatrist sees them, advises, and steps back	**reactive**, patient-centred; called when a problem has arisen

Model	How it works	Trigger & focus
Liaison model	the psychiatrist is **embedded** in a unit (oncology, transplant, pain), attends rounds, teaches and screens proactively	**proactive**, team- and system-centred; builds the ward's psychological skills over time

QUICK RULE

Consultation = the patient (one-off, reactive, by referral). **Liaison = the team** (continuous, proactive, embedded). Most real services blend both; "CL" names the hyphen.

The framing model: Engel's biopsychosocial

- **Biopsychosocial model (Engel, 1977)** – George Engel argued the purely **biomedical** model is reductionist; illness must be read across three interacting levels, **biological + psychological + social**. Grounded in general systems theory, it is the explicit rationale for CL work.
- **The psychiatrist in the general hospital** – sits between specialties: translates psychiatric findings into medical language, manages risk and capacity, supports staff, and treats comorbidity that otherwise lengthens stay and worsens outcome.

WHY IT MATTERS

Psychiatric comorbidity in general-hospital inpatients is common (broad estimates put significant psychological morbidity in roughly a quarter to a third of medical inpatients) and is consistently linked to **longer length of stay, poorer adherence, higher costs and worse medical outcomes**. CL is justified on outcome and economic grounds, not only humane ones.

2 Common reasons for referral

A small set of problems accounts for most ward referrals. Knowing the list, and the first move for each, is the practical core of the topic.

Reason for referral	What the CL psychiatrist does first
Delirium / acute confusion	confirm it is delirium (acute, fluctuating, inattention, altered consciousness), find and treat the **cause**; it is a medical emergency, not "just" agitation
Capacity / consent	assess decision-making capacity for the *specific* decision; capacity is presumed and decision-specific, not global
Post-self-harm assessment	psychosocial assessment after self-harm or overdose: risk, intent, mental state, supports, and a safety plan before discharge
Depression / anxiety in the medically ill	detect and treat mood and anxiety disorders that medical illness masks or mimics; weigh drug interactions and organ function
Somatic symptom & functional disorders	medically unexplained or disproportionate symptoms; avoid the organic-vs-functional split, validate distress, reduce iatrogenic harm
Substance issues	intoxication, **withdrawal** (alcohol, benzodiazepines, opioids), Wernicke prophylaxis, and motivational work
Organic vs functional diagnosis	help decide whether a presentation is medical, psychiatric or both; the most distinctively CL task

CLASSIC TRAP

Delirium is under-recognised and over-attributed to psychiatry. Hypoactive delirium is mistaken for depression; agitated delirium is mistaken for a primary psychosis or "behavioural" problem. The first duty in any acute change of mental state on a ward is to **exclude and treat an organic cause** before reaching for a psychiatric label.

MNEMONIC

“Mad, Bad, Sad & Can't Decide”

A rough taxonomy of the ward call: psychotic/confused (delirium, organic), risk/behavioural (self-harm, substance), low/anxious (depression, anxiety, somatic), and the capacity question. It maps onto the referral table above.

3 Psychosomatic medicine: concept and history

Psychosomatic medicine is the older name and intellectual root of CL: the study of how psychological factors influence the onset, course and outcome of physical disease, and the reverse.

- **The Chicago school** – the psychoanalytic-era programme led by **Franz Alexander** (with Helen Flanders Dunbar) holding that *specific* unconscious conflicts produced *specific* physical diseases (the “specificity” hypothesis).

Alexander's “holy seven”

The seven classic psychosomatic disorders Alexander linked to specific emotional conflicts. The list itself is the examinable fact; the specificity theory behind it is now rejected.

#	Classic psychosomatic disorder
1	Bronchial asthma
2	Essential hypertension
3	Peptic ulcer (duodenal)
4	Rheumatoid arthritis
5	Ulcerative colitis
6	Neurodermatitis (atopic dermatitis)
7	Thyrotoxicosis (hyperthyroidism)

MNEMONIC

“A HUT RUN” for the holy seven

Asthma, **H**ypertension, **U**lcer (peptic), **T**hyrotoxicosis, **R**heumatoid arthritis, **U**lcerative colitis, **N**eurodermatitis. Seven diseases, one discredited theory of specificity.

TRAP

The **specificity hypothesis** (one conflict → one disease) failed empirical testing and is no longer accepted. The peptic-ulcer story is the emblem: once the model psychosomatic disease, it was reframed by the discovery of **Helicobacter pylori** and NSAIDs. Stress is now seen as one contributor among biological causes, not the specific cause.

Stress and disease

- **Cannon & Selye** – **Cannon** described the “fight or flight” sympathetic-adrenal response; **Selye** described the **General Adaptation Syndrome** (alarm → resistance → exhaustion) and the HPA-axis stress response. These give the physiological bridge from psyche to soma.
- **Life events** – **Holmes & Rahe** Social Readjustment Rating Scale quantified stressful life change in “life-change units”; accumulated change correlated with subsequent illness. Established that measurable life stress precedes disease.

Type A behaviour and cardiac risk

◆ **Type A behaviour (Friedman & Rosenman)** – a pattern of **time urgency, competitiveness, hostility and impatience**, originally linked to coronary heart disease in the Western Collaborative Group Study. Later work narrowed the cardiotoxic ingredient to **hostility / anger** rather than the whole pattern; Type B is the relaxed converse.

Alexithymia

● **Alexithymia (Sifneos, 1972)** – literally "no words for feelings." A trait of **difficulty identifying and describing emotions**, an externally oriented thinking style, and impoverished fantasy life. Over-represented in somatic-symptom and functional disorders; measured by the **Toronto Alexithymia Scale (TAS-20)**.

The shift in understanding

■ **From psychosomatic to psychophysiological** – the field moved away from psychoanalytic *specificity* toward a **psychophysiological / psychoneuroimmunological** account: stress acts through the autonomic nervous system, the HPA axis and immune function (PNI) to modulate disease, within a biopsychosocial frame. "Psychosomatic" no longer means "imaginary" or "caused by the mind."

4 Psychological reactions to physical illness

Illness is a psychological event as well as a biological one. Reactions are normal first; they become disorders only when they are excessive, prolonged or disabling.

Common reactions

Reaction	What it looks like
Denial	minimising or disbelieving the diagnosis; protective early on, harmful if it blocks treatment
Anxiety	fear of the illness, procedures, dependency, death; the commonest early reaction
Depression	low mood, hopelessness, loss of role; the commonest later reaction, easily missed
Anger	"why me?"; may be displaced onto staff or family
Regression	reverting to a more dependent, childlike state; adaptive in small measure, problematic if entrenched
Sick role & illness behaviour	the social role and the way symptoms are perceived, evaluated and acted on (see below)

The sick role and illness behaviour

● **Sick role (Parsons)** – a sociological role with two **rights** (exemption from normal duties; not blamed for being ill) and two **duties** (want to get well; seek and cooperate with help).

● **Illness behaviour (Mechanic)** – how a person **perceives, evaluates and responds** to symptoms. **Abnormal illness behaviour (Pilowsky)** is a persistent, inappropriate mode of experiencing and acting on health that is out of proportion to detectable disease (the umbrella over somatisation and illness anxiety).

● TRAP

Distinguish the levels: **sick role** = the social position; **illness behaviour** = the individual's perception-and-response; **abnormal illness behaviour** = the maladaptive version. Do not collapse them into "the patient is exaggerating."

Adjustment to chronic and life-threatening illness

● **Kubler-Ross stages** – **denial → anger → bargaining → depression → acceptance**: a description of adjustment to dying (and to loss generally). Examinable, but understood as **not** a fixed sequence; people move back and forth, skip stages, or never "accept."

■ **Factors shaping coping** – the patient (personality, prior psychiatric history, coping style, intelligence, beliefs), the illness (visibility, pain, prognosis, disability, organ affected, treatment burden), and the context (social support, finances, culture, the doctor-patient relationship). These determine whether a normal reaction tips into disorder.

● MNEMONIC

Kubler-Ross: "DABDA"

Denial, **A**nger, **B**argaining, **D**epression, **A**cceptance. A map of grief and adjustment, not a timetable.

5 Psycho-oncology and specific interfaces

Cancer is the model interface: a life-threatening diagnosis, a long treatment course, and a psychological spectrum from normal distress to frank disorder.

Reactions to a cancer diagnosis

● **The adjustment-to-disorder spectrum** – most patients have normal **distress** and an **adjustment** reaction; a substantial minority develop a **depressive or anxiety disorder**. **Delirium** is common in advanced disease (metabolic, opioids, brain involvement). The aim is to detect where normal distress crosses into treatable disorder.

◆ **Demoralisation** – a syndrome of **hopelessness, helplessness, loss of meaning and existential distress**, distinct from depression (in demoralisation the capacity for pleasure can be intact, and the problem is a subjective sense of incompetence and pointlessness). Central to palliative-care psychiatry and to the assessment of the wish to hasten death.

● TRAP

Detecting depression in cancer is hard because **somatic symptoms overlap** (anorexia, weight loss, fatigue, poor sleep can be the cancer or its treatment). Lean on the **cognitive and affective** items – hopelessness, worthlessness, guilt, anhedonia, suicidal thinking – rather than the somatic ones.

Communication and breaking bad news

■ **SPIKES protocol** – a six-step structure for breaking bad news: **S**etting, **P**erception (find out what the patient knows), **I**nvitation (how much do they want to know), **K**nowledge (give it in plain language, warning shot first), **E**motions (respond empathically), **S**trategy & summary.

Palliative and end-of-life psychiatry

● **Palliative interface** – manage depression, anxiety, delirium and demoralisation; assess the **wish to hasten death** (often driven by treatable depression, uncontrolled symptoms or demoralisation, not a fixed rational choice); support communication, dignity and the family.

Other organ-system interfaces

Interface	The key examinable point
Cardiac	**depression after myocardial infarction** is common and is an **independent risk factor** for worse cardiac outcome and mortality; screen and treat (SSRIs, sertraline best evidenced post-MI)

Interface	The key examinable point
Transplant	pre-transplant psychiatric evaluation (adherence, substance use, capacity, support); post-transplant delirium, mood, and immunosuppressant (steroid, calcineurin-inhibitor) neuropsychiatric effects
HIV	high rates of depression and anxiety; HIV-associated neurocognitive disorder; delirium; adherence is central; watch antiretroviral-psychotropic interactions
Dialysis / renal	depression is the commonest psychiatric problem in end-stage renal disease; adherence and fluid restriction; capacity around withdrawal of dialysis
Chronic pain	depression, anxiety and pain are bidirectional; antidepressants (TCAs, SNRIs) and CBT help; avoid escalating opioids for distress

6 Capacity, consent and the CL toolkit

The capacity-and-consent assessment is the procedure the rest of the hospital cannot do, and the structured CL note is the product they keep.

Decision-making capacity

- **The four functional abilities** – a patient has capacity for a decision if they can **understand** the relevant information, **retain** it long enough to decide, **weigh / use** it in the balance, and **communicate** a choice. (Mental Capacity Act 2005 framing; Appelbaum's four standards are understanding, appreciation, reasoning, expressing a choice.)
- **Capacity principles** – capacity is **presumed**; it is **decision-specific** and time-specific (not all-or-none); an **unwise decision** does not equal incapacity; help the person to decide before concluding they cannot; if they lack capacity, act in their **best interests** by the least restrictive route.

TRAP

Capacity is **not** the same as the diagnosis. A person with psychosis, dementia or learning disability may still have capacity for a given decision; a person with no psychiatric diagnosis may lack it (e.g. in delirium). Assess the **function** for the **specific decision**, never infer it from the label or from disagreement with the doctor.

Consent

- **Valid consent** – requires **capacity + adequate information + voluntariness** (free of coercion). Consent is a process, not a signature; it can be withdrawn.

The structure of a good CL assessment

- **Before the bedside** – clarify the **actual referral question** (often not what is written), read the notes, drug chart and results, and speak to the referring team and nurses. The commonest CL error is answering a vaguely stated question.
- **At the bedside** – history, collateral, full mental state, and a **cognitive screen** (capacity and delirium hinge on it); review investigations and medications for organic contributors.
- **The note (the deliverable)** – state the **question asked**, the findings, a clear **formulation** (organic / psychiatric / both), and **specific, actionable recommendations** in medical language: diagnosis, risk, capacity opinion, drugs (with doses and interactions), and follow-up. Write what the team should *do*, not a psychiatric essay.

HOLD THIS

A CL referral has a **question**; the note answers it with a **formulation** and **actionable advice**. Always: rule out delirium, check capacity is decision-specific, mind drug interactions and organ function.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Models:** consultation = the patient (reactive, one-off, by referral); liaison = the team (proactive, embedded). CL = both. Framing model = Engel's biopsychosocial (1977).
- **Reasons to refer:** delirium, capacity/consent, post-self-harm, depression/anxiety in the medically ill, somatic/functional disorders, substance issues, organic-vs-functional. First duty in acute mental-state change = exclude delirium.
- **Chicago school:** Franz Alexander, specificity hypothesis (one conflict → one disease), now rejected.
- **Holy seven ("A HUT RUN"):** Asthma, Hypertension, Ulcer (peptic), Thyrotoxicosis, Rheumatoid arthritis, Ulcerative colitis, Neurodermatitis.
- **Stress:** Cannon (fight/flight) · Selye (General Adaptation Syndrome: alarm-resistance-exhaustion) · Holmes & Rahe (life-change units).
- **Type A (Friedman & Rosenman):** time-urgent, competitive, hostile; cardiotoxic ingredient narrowed to hostility. Alexithymia (Sifneos) = no words for feelings; TAS-20.
- **Shift:** psychosomatic specificity → psychophysiological / psychoneuroimmunological (ANS, HPA axis, immune) within the biopsychosocial frame.
- **Reactions to illness:** denial, anxiety, depression, anger, regression. Sick role (Parsons: 2 rights + 2 duties); illness behaviour (Mechanic); abnormal illness behaviour (Pilowsky).
- **Dying:** Kubler-Ross "DABDA" (denial, anger, bargaining, depression, acceptance) – a map, not a timetable.
- **Psycho-oncology:** distress → adjustment → depression/anxiety → delirium; demoralisation (hopelessness, loss of meaning) is distinct from depression. Use cognitive/affective items, not somatic, to catch depression.
- **Breaking bad news (SPIKES):** Setting, Perception, Invitation, Knowledge, Emotions, Strategy/summary.
- **Interfaces:** post-MI depression = independent mortality risk (sertraline) · transplant (pre-eval + immunosuppressant effects) · HIV (depression, neurocognitive) · dialysis (depression, withdrawal capacity) · chronic pain (bidirectional; TCAs/SNRIs/CBT).
- ★ **Capacity:** understand, retain, weigh/use, communicate. Presumed, decision-specific, an unwise choice is not incapacity, never inferred from diagnosis. Consent = capacity + information + voluntariness.
- **CL note:** answer the question asked → formulation (organic/psychiatric/both) → actionable advice. Always rule out delirium and check drug interactions.

ONE-DAY REVISION · PAPER 2

Psychiatric emergencies, made fast

The night-on-call core: stabilise first, recognise the drug-induced syndromes on sight, and dispose safely. The whole topic rewards a fixed sequence (safety, ABC, organic screen) and a few sharp contrasts. The NMS-versus-serotonin-syndrome table is the one to over-learn; the dystonia-versus-akathisia split is the second.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. Suicide risk detail, catatonia and delirium tremens have their own packs; cross-references are flagged.

II

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. Clonus and hyperreflexia point to serotonin syndrome, not NMS: serotonin syndrome is rapid onset (hours) with clonus, hyperreflexia and mydriasis treated with cyproheptadine, whereas NMS is slow onset (days) with lead-pipe rigidity, bradyreflexia and markedly raised CK treated with dantrolene and bromocriptine.
2. A previous suicide attempt is the single strongest predictor of completed suicide; high lethality plus ongoing intent plus absent social support pushes toward admission.
3. Acute dystonia → anticholinergic (procyclidine, benztropine), but akathisia is treated with propranolol, not an anticholinergic; giving an anticholinergic for akathisia is the classic wrong answer, and laryngeal-spasm dystonia is an airway emergency.
4. Lithium therapeutic range is 0.6-1.0 mEq/L (up to 1.2 for acute mania); toxicity above 1.5, and >2.5-3.0 mEq/L is potentially life-threatening, needing haemodialysis for severe toxicity.
5. New psychiatric symptoms with clouding of consciousness, disorientation or visual hallucinations are delirium until proven otherwise; in any alcohol-related presentation give thiamine before glucose to avoid precipitating Wernicke encephalopathy.

1 General approach to any emergency

Whatever the presentation, the opening moves are the same and in the same order. Safety, then the body, then the mind, then cause. An apparent psychiatric crisis is an organic one until you have ruled it out.

The fixed sequence

- **Safety first** – in order: **yourself, the patient, and others**. Position yourself near an exit, remove ligatures and weapons, call for staff early, never lone-assess a potentially violent patient.
- **Medical ABC** – airway, breathing, circulation, plus **D**isability (GCS, pupils, glucose) and **E**xposure. Check a capillary glucose in every altered patient: hypoglycaemia mimics almost anything.
- **Rapid risk assessment** – to self (suicide, self-harm), to others (violence), and from others/neglect (vulnerability). Frame disposition around the highest risk identified.
- **Calm, low-stimulus environment** – quiet room, reduced crowding, one person leading communication. The setting itself is a de-escalation tool.

◆ **Rule out an organic cause** – the screen: vitals, glucose, oxygen saturation, electrolytes, renal and liver function, calcium, drug and alcohol levels, infection screen, ECG, and neuroimaging where focal signs or head injury. **Sudden onset, age over 40, abnormal vitals, fluctuating consciousness, visual hallucinations or disorientation** all point organic.

● CLASSIC TRAP

New-onset "psychiatric" symptoms with **clouding of consciousness, disorientation, or visual hallucinations** are **delirium until proven otherwise**, not a primary psychosis. Visual and tactile hallucinations, waxing-waxing course and inattention are the organic flags. Treat the cause, not just the agitation.

2 The agitated or violent patient

The aim is the lowest-restrictive control that keeps everyone safe. Climb the ladder: environment and talk first, medication next, physical restraint and seclusion only when these fail or cannot be tried.

De-escalation (always first)

■ **Verbal de-escalation** – calm tone, respect personal space, one clinician speaking, acknowledge the grievance, offer realistic choices, avoid confrontation and prolonged eye contact. Remove triggers and an audience.

Rapid tranquillisation (RT)

■ **The principle** – medication to reduce dangerous agitation and distress quickly and safely, **not** to sedate into unconsciousness. Use the **oral route before parenteral**; offer oral first and only escalate to IM if refused or ineffective.

Class	Common agents	Note
Benzodiazepine	lorazepam (oral or IM), midazolam	preferred when cause unknown, in substance-related or organic states; watch respiratory depression
Antipsychotic	haloperidol; second-generation: olanzapine, risperidone, aripiprazole, IM ziprasidone	haloperidol often **with promethazine**; watch QTc and acute dystonia
Combination	haloperidol + promethazine IM; or antipsychotic + benzodiazepine	workhorse IM regimen where oral fails

■ **Monitoring after RT** – vital signs, oxygen saturation, level of consciousness and respiratory rate at regular intervals. Keep **flumazenil** (benzodiazepine reversal) and resuscitation equipment available. Avoid IM olanzapine within ~1 hour of parenteral benzodiazepine (additive cardiorespiratory depression).

QUICK RULE

Talk down → offer oral → IM if needed → restraint/seclusion last. Benzodiazepine when the cause is unclear or substance-related; antipsychotic (often + promethazine) for psychotic agitation.

Restraint and seclusion

▲ **Last resort only** – physical restraint and seclusion are used only when less restrictive measures have failed and there is imminent risk. Time-limited, continuously monitored, regularly reviewed and documented; never punitive. Avoid prone restraint (asphyxia risk). Always pursue the least restrictive option that works.

TRAP

Sedation is a **side effect to monitor**, not the goal of rapid tranquillisation. The endpoint is a calm, rousable patient. Over-sedation that compromises airway or breathing is a complication, so respiratory rate and oxygen saturation are the key observations.

3 Suicide and self-harm as an emergency

Treat every act of self-harm as an opportunity for assessment. The emergency questions are immediate: is the patient safe now, how high is the risk, and where do they go next. `(Full risk content: see the dedicated risk-assessment pack.)`

Rapid risk assessment

- **Cover** – current intent and plan, access to means, the recent act (lethality, precautions against discovery), protective factors, and reversibility. Ask directly about suicide: asking does not plant the idea.

High-risk features

Domain	Raises risk
The act	high lethality method, planning, precautions against rescue, final acts (note, giving away possessions), continued intent and regret at survival
Demographics	older age, male sex, living alone, unemployment, recent loss
Clinical	depression, psychosis with command hallucinations, substance misuse, chronic pain or illness, previous attempt (the strongest single predictor)

Immediate management and disposition

- **Make safe now** – one-to-one observation, remove means, treat the medical consequences of the act, and do not leave a high-risk patient alone.
- **Disposition** – admit (voluntary or involuntary) when intent and risk are high, supports are absent, or the act was of high lethality. A collaborative **safety plan**, means restriction and early follow-up for lower-risk patients. Treat the underlying disorder.

HOLD THIS

A **previous attempt** is the single strongest predictor of completed suicide. High lethality, ongoing intent, and absence of social support push toward admission.

4 Drug-induced emergencies (the high-yield core)

This section earns the most marks. Two malignant syndromes dominate, and the examiner's favourite is telling them apart. Learn the centrepiece table, then the four shorter entries below it.

Neuroleptic malignant syndrome (NMS)

- **Tetrad** – **lead-pipe rigidity, hyperthermia, autonomic instability, altered consciousness**, with a **raised creatine kinase** (often markedly, from muscle breakdown). An idiosyncratic reaction to dopamine blockade; onset over days, evolving slowly.
- **Management** – **stop the antipsychotic**, supportive care (cooling, IV fluids, monitor for renal failure from rhabdomyolysis). Specific agents: **dantrolene** (muscle relaxant) and **bromocriptine** (dopamine agonist); benzodiazepines also used. Consider ECT in refractory cases.

Serotonin syndrome

◆ **Triad** – **neuromuscular hyperactivity (clonus, hyperreflexia, tremor, myoclonus), autonomic instability, and altered mental state**. Diagnosed by the **Hunter criteria** in the setting of a serotonergic drug. Onset is **rapid** (hours), and clonus (especially inducible/spontaneous/ocular) is the hallmark.

■ **Management** – **stop the serotonergic drug**, supportive care and cooling, benzodiazepines for agitation and rigidity; **cyproheptadine** (a serotonin antagonist) is the specific antidote.

NMS VS SEROTONIN SYNDROME · THE CENTREPIECE CONTRAST

NMS	Serotonin syndrome
Cause: dopamine blockade	Cause: serotonin excess
Onset: days (slow)	Onset: hours (rapid)
Tone: lead-pipe rigidity	Tone: rigidity + tremor
Reflexes: normal / decreased	Reflexes: hyperreflexia
Clonus: absent	Clonus: present (hallmark)
Antidote: dantrolene, bromocriptine	Antidote: cyproheptadine

NMS is slow rigidity from dopamine blockade with normal-to-low reflexes and no clonus. Serotonin syndrome is rapid neuromuscular over-activity with hyperreflexia and clonus. The reflexes-and-clonus line is the fastest discriminator.

Feature	Neuroleptic malignant syndrome	Serotonin syndrome
Trigger	dopamine antagonist (antipsychotic); or dopaminergic withdrawal	serotonergic drug or combination (SSRI, SNRI, MAOI, tramadol, linezolid, triptans)
Onset	slow, over days	rapid, within hours (<24 h)
Neuromuscular	**lead-pipe rigidity**, bradyreflexia; tremor uncommon	**clonus** (inducible, spontaneous, ocular), **hyperreflexia**, tremor, myoclonus
Reflexes	normal or decreased	increased (hyperreflexia)
Pupils	normal	dilated (mydriasis)
Bowel sounds	normal or reduced	increased (hyperactive)
Mental state	altered consciousness, stupor	agitation, altered mental state
Autonomic	instability: fever, labile BP, tachycardia, diaphoresis	instability: fever, tachycardia, diaphoresis, labile BP
Creatine kinase	markedly raised (rhabdomyolysis)	normal or mildly raised
Diagnostic frame	clinical (Levenson criteria) + raised CK	Hunter criteria
Specific treatment	stop antipsychotic; **dantrolene**, **bromocriptine**	stop serotonergic drug; **cyproheptadine**

● MNEMONIC

“Slow & Stiff vs Fast & Flicking”

NMS = slow onset, stiff (lead-pipe), bradyreflexic. Serotonin = fast onset, flicking clonus, hyperreflexic. If you remember only one line: **clonus and hyperreflexia point to serotonin syndrome**.

Acute dystonia and akathisia

Two extrapyramidal emergencies that are easily confused and frequently paired in vivas. The first is a held abnormal posture; the second is an inner restlessness. Treatment differs.

Feature	Acute dystonia	Akathisia
What it is	sustained muscle spasm: oculogyric crisis, torticollis, trismus, tongue protrusion, laryngeal spasm (airway emergency)	subjective inner restlessness with an inability to sit still; pacing, fidgeting, rocking
Timing	early, hours to days after starting/raising an antipsychotic; younger males more prone	early to subacute, days to weeks
Treatment	anticholinergic (IM/IV procyclidine, benztropine or trihexyphenidyl); rapid relief	reduce dose; **propranolol** (first line), benzodiazepine; anticholinergics less effective
Watch for	airway compromise from laryngeal/pharyngeal spasm	misread as worsening psychosis or anxiety; linked to suicidality

● TRAP

Akathisia is treated with propranolol, not an anticholinergic; dystonia is treated with an anticholinergic. Giving an anticholinergic for akathisia or beta-blocker for dystonia is the classic wrong answer. Akathisia is easily mistaken for agitation, so the impulse to *raise* the antipsychotic worsens it.

Lithium toxicity

◆ **Levels** – therapeutic range ~**0.6–1.0 mEq/L** (up to ~1.2 for acute mania). Toxicity emerges above **1.5 mEq/L**; **>2.0 mEq/L** is moderate-to-severe and **>2.5–3.0 mEq/L** is potentially life-threatening. The therapeutic index is narrow.

● **Features** – early: **coarse tremor**, nausea, vomiting, diarrhoea, ataxia. Severe: dysarthria, gross tremor, **confusion, seizures, myoclonus, coma**, and renal impairment. Precipitants: dehydration, NSAIDs, ACE inhibitors, thiazides, sodium loss, intercurrent illness.

■ **Management** – stop lithium, correct fluids and electrolytes, IV normal saline, check levels and renal function; **haemodialysis** for severe toxicity (high levels, renal failure, or serious neurological signs).

Anticholinergic delirium (toxidrome)

● **Recognition** – **"mad, hot, dry, red, blind, full"**: delirium and agitation, hyperthermia, dry skin and mucosae, flushing, dilated pupils (blurred vision), urinary retention; tachycardia. From tricyclics, low-potency antipsychotics, antiparkinsonian and antihistaminic load.

■ **Management** – stop the offending agents, supportive care and cooling, benzodiazepines for agitation. **Physostigmine** is the specific antidote in selected severe cases (used cautiously, with cardiac monitoring).

● MNEMONIC TRAP

Anticholinergic toxidrome: **"hot as a hare, dry as a bone, red as a beet, mad as a hatter, blind as a bat, full as a flask."** Contrast with the *cholinergic* picture (wet: salivation, lacrimation, urination, defecation), where the skin is sweaty rather than dry.

5 Catatonia, delirium and withdrawal

Catatonia as an emergency

● **Recognition** – a motor-behavioural syndrome: **stupor, mutism, negativism, posturing, waxy flexibility, rigidity, echolalia/echopraxia**. Transdiagnostic (mood, psychotic, and medical/organic causes), so screen for an organic trigger.

■ **Lorazepam challenge** – a test-dose of IV/IM lorazepam (~1–2 mg); a clear improvement within minutes both supports the diagnosis and guides treatment. Benzodiazepines are first-line therapy.

▲ **Malignant catatonia** – catatonia **plus** hyperthermia, autonomic instability and rigidity. A life-threatening emergency that overlaps clinically with NMS; **avoid antipsychotics** (they can worsen it), and treat with benzodiazepines and urgent **ECT**.

◆ **ECT** – the definitive treatment for catatonia unresponsive to benzodiazepines and for malignant catatonia. (See the catatonia / ECT pack for detail.)

Delirium and alcohol withdrawal

● **Delirium** – acute, fluctuating disturbance of attention and awareness from an organic cause. A medical emergency: find and treat the cause, optimise environment, and use antipsychotics sparingly for distressing agitation. (See the geriatric pack.)

◆ **Delirium tremens** – the severe end of alcohol withdrawal, peaking around **48–72 hours**: delirium, gross tremor, autonomic storm, and visual hallucinations. High mortality if untreated. Manage with **benzodiazepines** and parenteral **thiamine before glucose** (to prevent Wernicke encephalopathy). (See the substance pack.)

● RULE

In any suspected alcohol-related presentation give **thiamine before any glucose load**. A glucose bolus into a thiamine-deficient brain can precipitate Wernicke encephalopathy (the triad: confusion, ophthalmoplegia, ataxia).

6 Disposition and the medico-legal line

The last step of every emergency is the safe destination and the legal basis for treatment. Keep the logic simple and the documentation clear.

When to admit

● **Admit when** – high and active risk to self or others, an unsafe or unsupported environment, a serious medical complication, the need for involuntary treatment, or failure of community management. Otherwise: discharge with a safety plan, means restriction and early, named follow-up.

Capacity and emergency treatment

■ **Capacity** – assess the ability to understand, retain, weigh and communicate a decision; capacity is decision-specific and can fluctuate. A patient who lacks capacity for a treatment decision may be treated in their best interests.

■ **Emergency treatment** – treatment necessary to save life or prevent serious deterioration may be given without consent in an emergency, under the relevant mental-health and capacity law, using the least restrictive option for the shortest necessary time.

MEDICO-LEGAL NOTE

Document the risk assessment, the capacity assessment, the legal basis for any treatment given without consent, and the least-restrictive justification. If it is not written, it did not happen.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Sequence:** safety (self → patient → others) → medical ABC + glucose → rapid risk assessment → calm environment → rule out organic. New psychiatric symptoms + clouded consciousness/disorientation/visual hallucinations = delirium until proven otherwise.
- **Agitation:** de-escalate → oral before parenteral → IM if needed → restraint/seclusion last. Benzodiazepine if cause unclear/substance-related; antipsychotic (often + promethazine) for psychotic agitation. Monitor respiratory rate and oxygen saturation; keep flumazenil ready.
- **Suicide:** previous attempt is the strongest predictor. High lethality + ongoing intent + no support = admit. Make safe now (1:1, remove means); ask directly.
- **NMS:** lead-pipe rigidity, hyperthermia, autonomic instability, altered consciousness, raised CK. Slow onset. Stop antipsychotic; dantrolene + bromocriptine.
- ★ **Serotonin syndrome:** clonus + hyperreflexia + autonomic + altered mental state. Rapid onset, Hunter criteria. Stop serotonergic drug; cyproheptadine. **Clonus/hyperreflexia = serotonin, not NMS.**
- **Dystonia vs akathisia:** dystonia = sustained spasm (oculogyric, laryngeal) → **anticholinergic**. Akathisia = inner restlessness → **propranolol** (not anticholinergic).
- **Lithium:** therapeutic ~0.6–1.0 mEq/L; toxic >1.5; severe >2.5–3.0. Coarse tremor → ataxia, confusion, seizures, coma. Stop, hydrate, dialyse if severe.
- **Anticholinergic delirium:** hot, dry, red, mad, blind, full. Stop agents, supportive; physostigmine in selected severe cases.
- **Catatonia:** stupor/mutism/posturing/waxy flexibility. Lorazepam challenge first; ECT if refractory. Malignant catatonia overlaps NMS → avoid antipsychotics, give ECT.
- **Withdrawal/delirium:** delirium tremens peaks 48–72 h → benzodiazepines + thiamine before glucose. Delirium = treat the cause, antipsychotics sparingly.
- **Disposition:** admit for high active risk / unsafe environment / involuntary treatment. Treat without consent only when capacity is lacking or under emergency provisions, least restrictive, documented.

ONE-DAY REVISION · PAPER 2

Psychobiology of Stress & Rhythms

Where mind meets body: the stress axes, the hormones, the immune system and the clock. Three interfaces, one logic – the brain reads the world and rewrites the body's chemistry, and the body writes back. Hold the HPA cascade and the cytokine hypothesis cold; the rest hangs off them.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. This is the mind-body interface pack: endocrine, immune, circadian. Sleep architecture lives in the companion sleep pack.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. DST non-suppression in depression is a **state marker**, **sensitive but not specific**: cortisol escapes the feedback brake, it normalises on recovery, and it cannot screen or diagnose (**false positives in Cushing's, anorexia, alcohol withdrawal**).
2. Two stress arms from one alarm: the fast **SAM** axis runs hypothalamus → adrenal medulla → adrenaline/noradrenaline in seconds, the slow **HPA** axis runs CRH → ACTH → cortisol from the adrenal cortex over minutes to hours.
3. The cytokine hypothesis: **IL-6, CRP and TNF-alpha** are raised in a depression subgroup (not all depression), high CRP predicts **poorer** SSRI response, and therapeutic interferon-alpha inducing depression is the natural experiment for causation.
4. Selye's general adaptation syndrome runs alarm → resistance → exhaustion; the key idea is **non-specificity** (same response to any stressor), split into eustress and distress.
5. Sleep deprivation (wake therapy) gives a rapid transient antidepressant response in **40-60%** of unipolar patients that relapses after recovery sleep, but is a recognised **trigger of mania** in bipolar disorder.

1 Stress physiology: the shared substrate

Every interface in this pack runs on the stress response. Learn the two pathways – one fast, one slow – and the language that frames the whole field: Selye's stages, allostasis and load.

Selye's general adaptation syndrome (GAS)

Hans Selye showed that diverse stressors (cold, toxins, restraint) produce the **same** non-specific bodily reaction. He called it the GAS and split it into three stages.

Stage	What happens	Hallmark
Alarm	acute "fight-or-flight": sympatho-adrenal-medullary surge, then HPA activation	adrenaline + cortisol rise; resistance briefly *dips* then climbs
Resistance	the body adapts and copes; cortisol stays elevated, outward arousal settles	resources mobilised, apparent coping; reserves quietly drained
Exhaustion	prolonged stress depletes reserves; adaptation fails	**stress-related disease**: ulcers, immunosuppression, atrophy

QUICK RULE

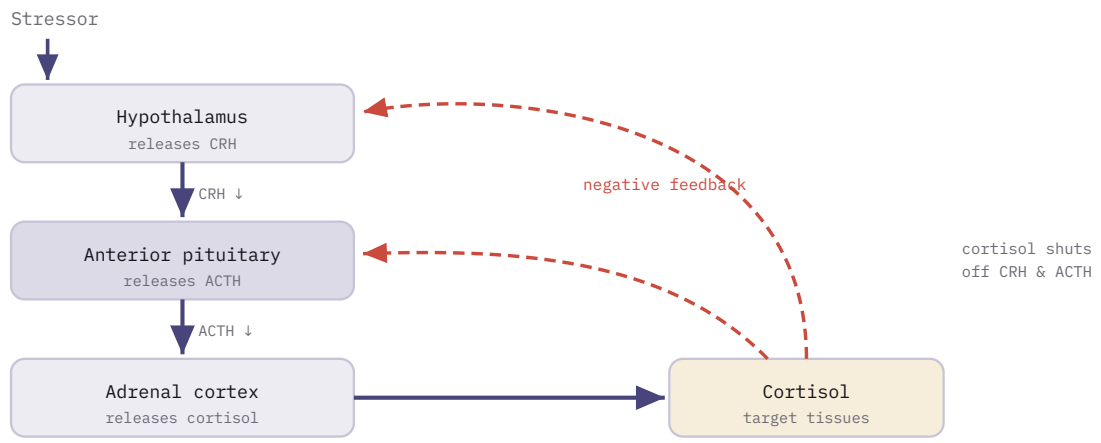
Selye's key insight is **non-specificity**: the stress response looks the same whatever the stressor. He also split stress into **eustress** (adaptive) and **distress** (harmful).

The two stress pathways

One thalamus-and-limbic alarm fires two efferent arms: a fast neural one and a slow hormonal one. Knowing which is which is the high-yield distinction.

	Fast pathway (SAM)	Slow pathway (HPA)
Full name	sympatho-adrenal-medullary axis	hypothalamic-pituitary-adrenal axis
Route	hypothalamus → sympathetic nerves → adrenal medulla	hypothalamus (CRH) → anterior pituitary (ACTH) → adrenal cortex
Messenger	adrenaline + noradrenaline (catecholamines)	cortisol (a glucocorticoid)
Timing	seconds; transient	minutes to hours; sustained
Effect	heart rate, BP, glucose, alertness up; digestion down	glucose mobilised, anti-inflammatory, catabolic; primes recovery

THE HPA AXIS AND ITS NEGATIVE FEEDBACK



CRH from the hypothalamus drives ACTH from the pituitary, which drives cortisol from the adrenal cortex. Cortisol feeds back (coral, dashed) to suppress both CRH and ACTH. In depression this feedback brake is weak, so cortisol stays high.

Allostasis and allostatic load

- **Allostasis** – "stability through change." The body maintains function by *actively varying* its set-points (cortisol, BP, glucose) to meet demand, rather than holding one fixed homeostatic value. Sterling & Eyer; developed by McEwen.

◆ **Allostatic load** – the cumulative **wear and tear** from repeated or chronic activation of the stress systems. When the load exceeds capacity (**allostatic overload**) the mediators that protect acutely become damaging: hippocampal atrophy, visceral fat, hypertension, immune dysregulation.

CLASSIC TRAP

Glucocorticoids are **anti-inflammatory** in the short term (this is why we prescribe steroids). The harm in chronic stress is not the surge itself but its *failure to switch off*: persistent cortisol plus glucocorticoid-**resistant** immune cells gives the worst of both worlds – high cortisol *and* unchecked inflammation. Allostatic load, not the acute response, is the disease driver.

2 Psychoneuroendocrinology

The study of how hormones and behaviour shape each other. The exam-relevant story is the endocrine axes in mood disorder – above all the HPA axis in depression.

The HPA axis in depression (highest yield)

◆ **Hypercortisolaemia** – a substantial subgroup of depressed patients (classically melancholic) show raised cortisol, an enlarged adrenal, blunted diurnal variation and elevated CRH in CSF. Sustained cortisol is implicated in hippocampal volume loss.

◆ **Dexamethasone suppression test (DST)** – give 1 mg dexamethasone (a synthetic glucocorticoid) at night; in health it suppresses next-morning cortisol via feedback. In depression, **non-suppression** (cortisol escapes) reflects a faulty feedback brake.

Feature	Detail
Normal result	dexamethasone suppresses cortisol the next morning
Depressed (abnormal)	non-suppression – cortisol escapes the feedback brake
Marker type	state marker, not trait: tends to normalise on recovery
Performance	sensitive but not specific; also abnormal in other conditions
Clinical note	persistent non-suppression after treatment hints at relapse risk

HOLD THIS

DST non-suppression in depression is a **state marker** that is **sensitive, not specific**. It marks an episode and tends to revert on recovery; it does not diagnose. The refined CRH-augmented **dex/CRH test** is more sensitive.

CLASSIC TRAP

The DST cannot be a screening or diagnostic test for depression: too many false positives (Cushing's, anorexia, alcohol withdrawal, weight loss, some drugs) and false negatives. “Sensitive not specific” and “state not trait” are the two phrases examiners want.

The HPT (thyroid) axis

● **Subclinical hypothyroidism** – raised TSH with normal free T4, often symptom-light, is over-represented in depression (especially treatment-resistant and rapid-cycling bipolar) and can blunt antidepressant response.

● **TRH stimulation test** – give TRH, measure the TSH rise. A **blunted TSH response** to TRH is a classic (non-specific) finding in depression; an *exaggerated* response suggests hypothyroidism.

■ **Clinical lever** – thyroid hormone (T3) is an established **augmentation** strategy in resistant depression; lithium can induce hypothyroidism, so thyroid must be monitored.

The HPG (gonadal) axis

Mood disorder clusters around points of **rapid gonadal-hormone change**. The pattern, not the molecule, is the answer here.

Window	Hormonal context	Syndrome
Perimenstrual	late luteal fall in oestrogen/progesterone	premenstrual dysphoric disorder (PMDD)
Perinatal	abrupt post-delivery hormone withdrawal	postpartum depression / psychosis
Perimenopausal	fluctuating, then falling oestrogen	raised risk of depressive episodes

QUICK RULE

Risk tracks **hormonal flux** (the rate of change), not the absolute level. The three "peri" windows – menstrual, natal, menopausal – are the high-yield list.

Growth hormone and prolactin

- **Growth hormone (GH)** – secreted in pulses, peaking in slow-wave sleep; a **blunted GH response** to clonidine (an alpha-2 agonist) is reported in depression, taken as evidence of reduced central noradrenergic sensitivity.
- **Prolactin** – under tonic **dopamine inhibition**; dopamine-blocking antipsychotics raise it (galactorrhoea, amenorrhoea, sexual dysfunction). A post-ictal prolactin rise helps distinguish a true seizure from a non-epileptic event.

3 Psychoneuroimmunology

The brain and immune system talk both ways. This bidirectional traffic links stress, infection and inflammation to mood – the cytokine hypothesis is the centre of gravity.

Bidirectional brain-immune communication

- **Brain → immune** – the HPA axis (cortisol) and the sympathetic nervous system modulate immune cells; the vagus carries a fast anti-inflammatory reflex.
- **Immune → brain** – peripheral cytokines signal the brain via leaky circumventional organs, active transport, vagal afferents, and induction of brain-side cytokines. The brain is not immune-privileged in the old absolute sense.

Cytokines and sickness behaviour

- **Sickness behaviour** – pro-inflammatory cytokines (IL-1, IL-6, TNF-alpha) acting on the brain produce a coordinated state: **fever, fatigue, anhedonia, social withdrawal, appetite and sleep loss, low mood, poor concentration**. Adaptive in acute infection (conserves energy for fighting the pathogen).

RULE

Sickness behaviour overlaps strikingly with the somatic symptoms of **depression**. This is the conceptual bridge: if cytokines can manufacture a depression-like state, chronic low-grade inflammation might drive some clinical depression. Therapeutic interferon-alpha (given for hepatitis C) induces depression in a large minority – a natural experiment supporting causation.

The inflammatory / cytokine hypothesis of depression

- **Core claim** – a subgroup of depression is an **inflammatory** state. Meta-analyses show raised IL-6, CRP and TNF-alpha in depressed patients on average.

Marker	Type	Note in depression
IL-6	pro-inflammatory cytokine	most consistently raised; predicts later depression in cohorts
CRP	acute-phase protein	raised; high CRP may predict poorer SSRI response
TNF-alpha	pro-inflammatory cytokine	raised; falls with successful antidepressant treatment

QUICK RULE

Three to memorise: **IL-6, CRP, TNF-alpha** are raised in depression. It is a **subgroup** story (inflamed vs non-inflamed depression), not all depression. Anti-inflammatory add-ons help mainly those with high baseline inflammation.

Chronic stress and immunosuppression

- **Kiecolt-Glaser & Glaser** – the foundational human work. Caregivers of dementia patients, medical students at exam time, and the bereaved showed measurable immune downshift under chronic stress.
- **Wound healing** – in the classic study, dementia caregivers healed a standardised punch-biopsy wound about **24% slower** (roughly 9 days longer) than matched controls. Exam-stressed students healed mucosal wounds more slowly too.
- **Infection susceptibility** – chronic stress impairs the antibody response to vaccination (influenza, hepatitis B) and raises susceptibility to the common cold (Cohen's deliberate-exposure studies). It also reactivates latent herpesviruses.

TRAP

Stress is not uniformly immunosuppressive. **Acute** stress can transiently *enhance* innate immunity (adaptive, mobilising defence). It is **chronic** stress that suppresses and dysregulates – shifting the response, raising basal inflammation while blunting specific immunity.

From inflammation to neurotransmitters

- **Kynurenine pathway** – one mechanistic bridge. Inflammation activates the enzyme **IDO**, which diverts **tryptophan** away from serotonin and down the kynurenine pathway. This both lowers serotonin substrate and generates neuroactive metabolites (quinolinic acid, an NMDA agonist and neurotoxin; kynurenic acid, an antagonist). A route from immune activation to monoamine and glutamate disturbance.

4 Chronobiology & chronopsychobiology

Biology keeps time. The circadian system imposes a ~24-hour rhythm on nearly every physiological variable, and its disruption is woven into psychiatric illness.

The master clock

- **Suprachiasmatic nucleus (SCN)** – the master circadian pacemaker, sitting in the **anterior hypothalamus** above the optic chiasm. It runs a self-sustaining molecular clock (transcription-translation feedback loops of clock genes) with an intrinsic period close to, but not exactly, 24 hours.
- **Melatonin** – secreted by the **pineal gland** at night under SCN control; light suppresses it. It is the body's "darkness signal," falling at dawn and rising in the evening. The pathway: retina → retinohypothalamic tract → SCN → (via sympathetic relay) pineal.

Term	Meaning
Circadian	rhythm with a ~24-hour period (Latin *circa diem*, "about a day")
Zeitgeber	"time-giver": an external cue that resets the clock; light is the strongest
Entrainment	locking the internal clock to the external 24-hour day via zeitgebers
Free-running	the rhythm with no cues; drifts to its intrinsic period (often >24 h)
Phase advance / delay	shifting the rhythm earlier (advance) or later (delay)

MNEMONIC

"Zeitgeber = time-giver; light is the loudest"

Light hitting the retina → SCN is the dominant zeitgeber. Non-photic cues (meals, exercise, social routine, exogenous melatonin) are weaker but real – the basis of social rhythm therapy.

- **Borbely's two-process model** – sleep is governed by Process S (a homeostatic sleep **pressure** that builds with wakefulness) interacting with Process C (the **circadian** rhythm). One line here; the full model lives in the companion sleep pack.

5 Rhythms & psychiatric illness

Disrupt the clock and mood follows. Three exam-favourite stories: seasonal depression, the social-zeitgeber account of bipolar disorder, and the paradoxical antidepressant effect of sleep deprivation.

Seasonal affective disorder (SAD)

- **SAD** – recurrent depression with a **seasonal pattern**, classically winter onset with atypical features (hypersomnia, hyperphagia, carbohydrate craving, weight gain). Linked to shortened winter photoperiod and a phase delay of circadian rhythms.
- **Bright light therapy** – first-line for SAD. Typically **10,000 lux for ~30 minutes in the early morning**, advancing the delayed rhythm. Effect appears within days to two weeks; can trigger (hypo)mania in bipolar patients.

Bipolar disorder and the social zeitgeber theory

- **Social zeitgeber theory (Ehlers, Frank)** – daily social routines (wake time, meals, work, social contact) act as **time cues** that stabilise the biological clock. **Life events that disrupt these routines** destabilise circadian rhythms and can precipitate episodes in vulnerable people.
- **Clinical translation** – this theory underpins **Interpersonal and Social Rhythm Therapy (IPSRT)** for bipolar disorder: regularising sleep-wake and daily routines to protect the rhythm and reduce relapse.

CLASSIC TRAP

The example examiners love: a long-haul flight or a new night shift (a social-rhythm and light disruption) precipitating a manic episode. The pathway is routine disruption → circadian destabilisation → mood episode – not the travel stress alone.

Circadian misalignment and the sleep-deprivation effect

- **Misalignment in mood disorder** – depression shows **phase-advanced** rhythms in some patients (early morning waking, early cortisol peak, blunted diurnal mood variation worst in the morning). Mania shows a markedly **reduced need for sleep** with shortened, fragmented circadian timing.

◆ **Sleep deprivation (wake therapy)** – total or partial (second-half-of-night) sleep deprivation produces a **rapid but transient** antidepressant response in roughly 40-60% of depressed patients. The catch: most **relapse after recovery sleep**. Combining it with light therapy and a phase advance helps sustain the gain.

▲ **The mania risk** – sleep loss is double-edged: therapeutic in unipolar depression but a well-recognised **trigger of mania** in bipolar disorder. A single sleepless night can tip a vulnerable bipolar patient into an episode.

HOLD THIS

Three rhythm-and-mood facts: **SAD → morning bright light** (10,000 lux); **bipolar → social rhythm disruption** precipitates episodes (IPSRT protects); **sleep deprivation** lifts unipolar depression briefly but can trigger bipolar mania.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Selye GAS:** alarm → resistance → exhaustion. Key idea = **non-specificity** (same response to any stressor). Eustress vs distress.
- **Two pathways:** fast **SAM** (adrenal medulla → adrenaline/noradrenaline, seconds) vs slow **HPA** (CRH → ACTH → cortisol from adrenal cortex, minutes-hours).
- **Allostasis** = stability through change (variable set-points); **allostatic load** = cumulative wear from chronic activation (McEwen).
- ★ **HPA in depression:** hypercortisolaemia; **DST non-suppression** = **state marker, sensitive not specific**; dex/CRH test more sensitive.
- **Other axes:** subclinical hypothyroidism + blunted TRH-stimulated TSH in depression (T3 augments); HPG → mood at peri-menstrual, peri-natal, peri-menopausal flux; prolactin under dopamine inhibition.
- **Sickness behaviour:** IL-1, IL-6, TNF-alpha → fatigue, anhedonia, withdrawal, anorexia – overlaps depression; interferon-alpha causes depression.
- **Cytokine hypothesis:** raised **IL-6, CRP, TNF-alpha** in a depression subgroup; high CRP predicts poorer SSRI response.
- **Chronic stress & immunity:** Kiecolt-Glaser caregivers heal wounds ~24% slower; impaired vaccine response; acute stress can *enhance* innate immunity (chronic suppresses).
- **Inflammation → monoamines:** IDO diverts tryptophan down the **kynurenine** pathway (quinolinic = NMDA agonist/neurotoxin).
- **Clock: SCN** (anterior hypothalamus) master pacemaker; **melatonin** from pineal at night (light suppresses); retina → retinohypothalamic tract → SCN. Zeitgeber = time-giver (light strongest); entrainment.
- **Rhythms & illness: SAD** → morning bright light 10,000 lux; **social zeitgeber theory** → routine disruption destabilises bipolar (IPSRT); depression phase-advanced.
- **Sleep deprivation:** rapid transient antidepressant (40-60%), relapses on recovery sleep; **triggers mania** in bipolar.

ONE-DAY REVISION · PAPER 2

Electrophysiology

The brain's electrical traffic, read off the scalp and the nerve. The EEG, its bands and abnormalities, the one sign that actually matters in psychiatry (diffuse slowing in delirium), the evoked and event-related potentials with the P300 as the workhorse, and the rest of the recording toolkit. Most of the marks here are pattern-recognition and one-liners.

● CONCEPT ● RULE ● TRAP ● KEY NUMBER ● MNEMONIC

The colour tells you what kind of fact it is. Underlying neurophysiology of the bands lives in the companion pack.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. Diffuse (generalised) slowing is the single most useful EEG sign in psychiatry: it points to delirium / an organic encephalopathy, not a primary psychiatric disorder, and tracks the level of consciousness. The classic trap exception is delirium tremens, where the EEG shows fast activity rather than slowing.
2. The P300 in schizophrenia is both reduced in amplitude AND delayed (prolonged) in latency (oddball paradigm, large positive wave at ~300 ms), one of the most replicated biological findings; do not state only amplitude.
3. The EEG records summed cortical postsynaptic potentials of pyramidal neurons (apical dendrites), not action potentials; amplitude is driven by synchrony, with scalp signal roughly 10-100 µV.
4. Bands by frequency: delta <4 Hz (deep sleep) · theta 4-8 Hz · alpha 8-13 Hz (relaxed, eyes closed, occipital) · beta 13-30 Hz · gamma >30 Hz; benzodiazepines & barbiturates boost beta, so a fast beta-rich record suggests sedative-hypnotics.
5. Generalised 3 Hz spike-and-wave is the signature of childhood absence (petit mal) epilepsy, provoked by hyperventilation; a spike is <70 ms and a sharp wave 70-200 ms.

1 The EEG: what it records

The EEG records the summed **postsynaptic potentials** of cortical pyramidal neurons (mainly the apical dendrites), *not* action potentials. The signal is small (tens of µV) and reflects synchronised activity in the superficial cortex directly under each electrode. Deep and subcortical generators are poorly seen.

● **Source of the signal** – graded excitatory and inhibitory postsynaptic potentials in cortical pyramidal cells, summed across large populations. Synchrony, not firing rate, drives amplitude.

◆ **Typical amplitude** – scalp EEG runs roughly **10–100 µV**; the signal is filtered and amplified before display. (For contrast, the ECG is in the millivolt range, a thousandfold larger.)

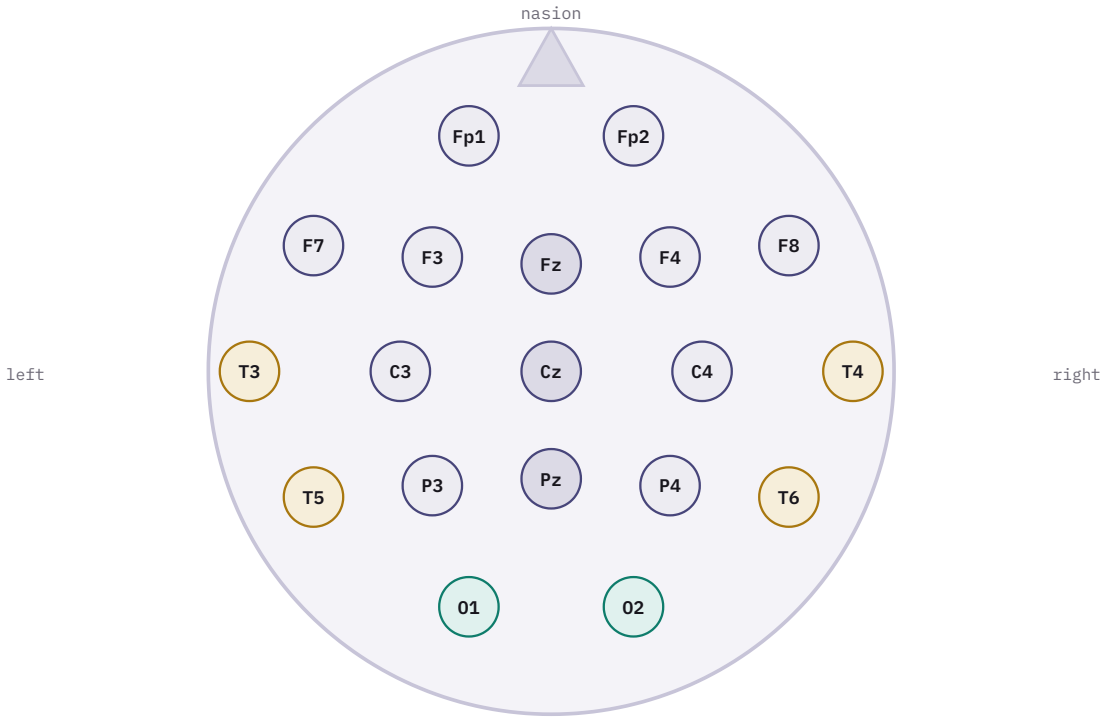
The international 10–20 system

A standardised way to place electrodes so that recordings are comparable across labs and patients. Positions are set at **10% and 20%** of the total distance between four bony landmarks: **nasion** (front), **inion** (back), and the two **preauricular points**.

● **Letter codes** – the letter names the lobe/region: **Fp** frontopolar, **F** frontal, **C** central, **P** parietal, **O** occipital, **T** temporal. **z** = midline (zero).

- **Number codes** – **even numbers on the right** hemisphere, **odd numbers on the left**; numbers rise outward from the midline.

THE INTERNATIONAL 10–20 ELECTRODE SYSTEM (SCHEMATIC, HEAD FROM ABOVE)



Head seen from above, nose at top. Letter = region (Fp/F/C/P/O/T), z = midline, odd = left, even = right. Amber ring = temporal chain, teal = occipital, darker fill = midline.

Montages: how channels are derived

- **Bipolar montage** – each channel is the voltage difference between two adjacent scalp electrodes (a chain). Localises by **phase reversal** at the maximum.
- **Referential (monopolar) montage** – each electrode is compared to a common reference (ear, mastoid, or average). Shows true amplitude; better for widespread, low-voltage activity.

Frequency bands and the states they index

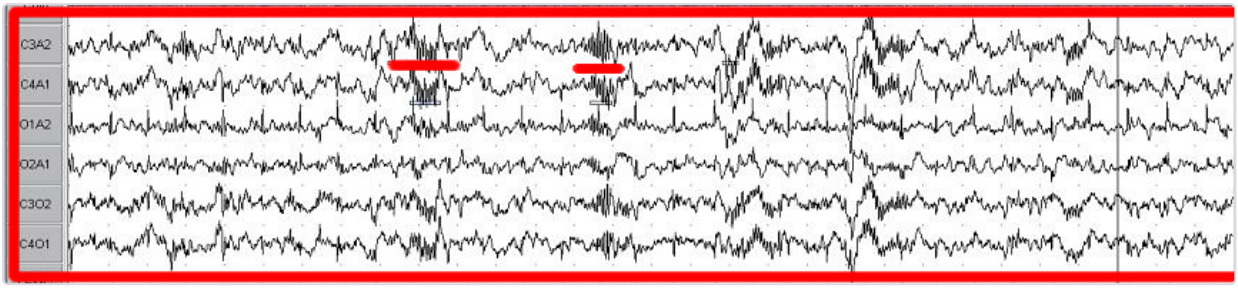
Band	Frequency	Typical state / location
Delta (δ)	< 4 Hz	deep (slow-wave, N3) sleep; pathological if awake (lesion, encephalopathy)
Theta (θ)	4–8 Hz	drowsiness, early sleep (N1), young children; meditation
Alpha (α)	8–13 Hz	relaxed wakefulness, **eyes closed**, posterior (occipital) dominant
Beta (β)	13–30 Hz	alert, eyes open, active thinking, anxiety; **boosted by benzodiazepines & barbiturates**
Gamma (γ)	> 30 Hz	active cognitive processing, feature binding, attention

● MNEMONIC

“Delta Deep, Beta Busy” → slow = asleep, fast = awake

As the brain wakes and engages, the dominant rhythm **speeds up** and its amplitude falls: delta → theta → alpha → beta. Slowing where you expect fast activity is the core abnormality.

A REAL EEG RECORDING



A real multichannel EEG (central derivations), the trace the schematics abstract. Source: MrSandman, public domain, Wikimedia Commons.

TRAP

Bands are defined by **frequency, not amplitude**. The exact cut-offs vary slightly between texts (alpha is most often quoted as 8–13 Hz, sometimes 8–12). Quote the band, the state it indexes, and the location, and you have the mark.

2 Normal variants and the abnormal EEG

The alpha rhythm and its reactivity

- **Alpha rhythm** – the posterior dominant rhythm of relaxed wakefulness, best seen **occipitally with eyes closed**. The normal adult background.
- **Alpha blocking (Berger effect)** – opening the eyes, or mental effort, **attenuates the alpha rhythm** and replaces it with low-voltage fast (beta) activity. The classic demonstration of EEG reactivity.

Epileptiform discharges

- **Spike** – a sharp transient under **70 ms** in duration, standing out from the background. The hallmark interictal epileptiform discharge.
- **Sharp wave** – the same in kind but slower, **70–200 ms**. Spike and sharp wave shade into each other; both point to cortical irritability.
- **Spike-and-wave** – a spike followed by a slow wave, in runs. **Generalised 3 Hz spike-and-wave** is the signature of childhood **absence (petit mal)** epilepsy.

Slowing: focal vs generalised

Focal slowing

Slow activity (delta/theta) over **one region**. Suggests a **localised structural lesion** beneath that area: tumour, abscess, infarct, contusion. Always investigate with imaging.

Generalised slowing

Diffuse slowing across **all regions**. Points to a **global / diffuse** process: metabolic or toxic encephalopathy, delirium, raised ICP, post-ictal state. Non-specific as to cause.

Activation procedures

Manoeuvres used during the recording to provoke latent abnormalities (chiefly epileptiform discharges).

Procedure	Method	Provokes / use
Hyperventilation	~3 min of deep breathing (hypocapnia → cerebral vasoconstriction)	3 Hz spike-and-wave of absence; some generalised epilepsies

Procedure	Method	Provokes / use
Photic stimulation	flashing strobe at a range of frequencies	photoparoxysmal response; photosensitive epilepsy
Sleep deprivation	recording after a night without sleep	raises yield of epileptiform discharges generally

QUICK RULE

Hyperventilation for absence (3 Hz spike-and-wave); **photic stimulation** for photosensitive epilepsy; **sleep deprivation** and natural/recorded sleep to raise the overall yield.

3 EEG in psychiatry and organic states

The EEG earns its keep in psychiatry mainly by separating an **organic** picture from a primary psychiatric one. In most primary psychiatric disorders it is unremarkable.

HOLD THIS

Diffuse (generalised) slowing is the single most useful EEG finding in psychiatry: it points to **delirium / an organic encephalopathy** rather than a primary psychiatric disorder. Slowing tracks the level of consciousness.

- **Delirium** – **diffuse slowing** of the background (theta then delta), roughly proportional to severity. The exception is **delirium tremens** (alcohol withdrawal), where the EEG shows *fast* activity, not slowing.
- **Dementia** – progressive **slowing** of the background with loss of the normal alpha rhythm in Alzheimer's disease; the EEG is usually *normal* in early/mild cases, so a normal EEG does not exclude dementia. Useful chiefly to flag a treatable organic cause.
- **Primary psychiatric disorders** – the routine EEG is **largely normal** in schizophrenia, mood disorders and most anxiety disorders. Its value is in *excluding* an organic mimic, not in confirming a psychiatric diagnosis.

Distinctive patterns worth knowing

Condition	Characteristic EEG
Delirium	diffuse slowing (delirium tremens = fast activity)
Hepatic encephalopathy	triphasic waves on a slow background
Sporadic CJD	periodic sharp-wave complexes (~1 Hz)
SSPE	periodic complexes (Radermecker) synchronous with myoclonus
Herpes encephalitis	PLEDs over the **temporal** lobe
Absence (petit mal)	generalised **3 Hz** spike-and-wave

Clinical and medico-legal context

- **Pre-ECT** – EEG is not a routine pre-ECT screen, but ECT induces a generalised seizure; the post-ictal EEG shows slowing, and inter-ictally repeated ECT can produce transient background slowing that resolves after the course.
- **Clozapine** – lowers the seizure threshold dose-dependently and produces EEG changes (slowing, sharp waves) in a sizeable minority. A baseline or on-treatment EEG is sometimes used when myoclonus or other warning signs appear, though it is not a universal mandate.

- **Medico-legal use** – EEG contributes to assessing organic brain disease, suspected epilepsy (including dissociative/non-epileptic seizures), and brain-death evaluation (electrocerebral silence as one supportive criterion). It is supportive evidence, not a stand-alone test of competence or intent.

DRUG EFFECTS ON THE EEG

Benzodiazepines and barbiturates increase beta activity (fast, low-voltage). **Clozapine and other antipsychotics, lithium, and TCAs** tend to *slow* the EEG and can lower the seizure threshold. So a fast (beta-rich) record suggests sedative-hypnotics; diffuse slowing suggests an organic cause or a slowing drug.

4 Evoked and event-related potentials

Average the EEG time-locked to a repeated stimulus and the random background cancels out, leaving a small, reproducible waveform. **Evoked potentials (EPs)** reflect the integrity of a sensory pathway; **event-related potentials (ERPs)** reflect cognitive processing of the stimulus.

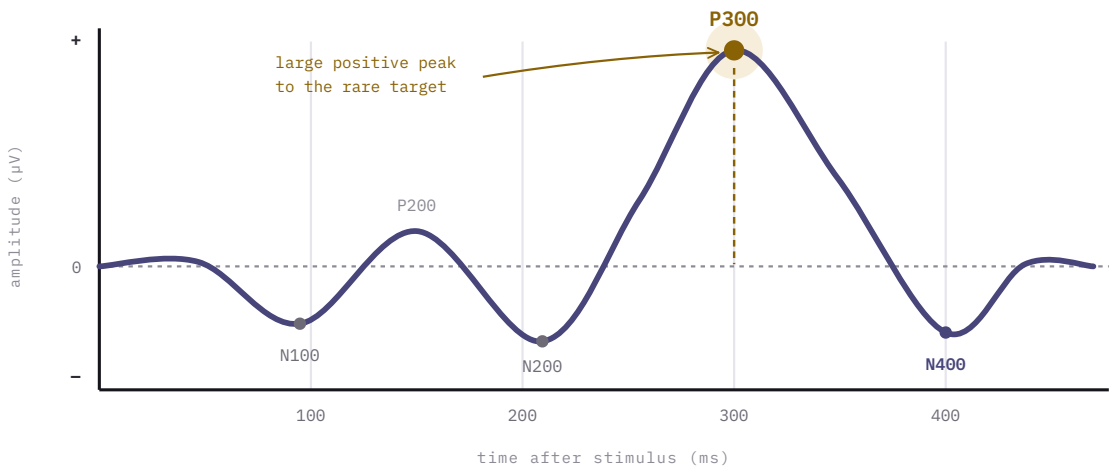
Sensory evoked potentials

- **Visual evoked potential (VEP)** – response to a reversing checkerboard; the **P100** is the key wave. Delayed P100 indicates optic nerve demyelination (a classic sign in multiple sclerosis).
- **Brainstem auditory evoked potential (BAEP / BERA)** – clicks generate waves **I–V** mapping the auditory pathway from cochlea to midbrain; used for acoustic neuroma, brainstem lesions and infant hearing screening.
- **Somatosensory evoked potential (SSEP)** – peripheral nerve stimulation tests the dorsal-column / lemniscal pathway; used in spinal cord and operative monitoring.

Event-related potentials: the high-yield ones

- **P300 (P3)** – a large **positive** wave at about **300 ms** after an infrequent, task-relevant target in an *oddball* paradigm. Indexes attention and working-memory updating. In **schizophrenia**: **reduced amplitude and delayed (prolonged) latency** – one of the most replicated biological findings.
- **Mismatch negativity (MMN)** – a **negative** wave (~150–250 ms) to a deviant in a stream of standard tones; pre-attentive (occurs without the subject attending). **Reduced in schizophrenia**; linked to NMDA-receptor function and proposed as an early/prodromal marker.
- **N400** – a **negative** wave at ~400 ms to a **semantically incongruent** word ("he spread the bread with socks"). Indexes semantic processing; abnormalities are reported in schizophrenia and the dementias.

A SCHEMATIC ERP WAVEFORM (ODDBALL PARADIGM), POSITIVE PLOTTED UP



The averaged ERP. Early waves (N100, P200) are sensory; later waves are cognitive. The **P300** is the big positive peak around 300 ms to the infrequent target; in schizophrenia it is smaller and later. N400 indexes semantic mismatch.

ERP	Polarity / latency	What it indexes	Finding in schizophrenia
P300 (P3)	positive, ~300 ms	attention, context updating	reduced amplitude, delayed latency
Mismatch negativity	negative, ~150–250 ms	pre-attentive change detection (NMDA)	reduced amplitude
N400	negative, ~400 ms	semantic processing	abnormal (context use)
P50	positive, ~50 ms	sensory gating (paired-click)	deficient gating (less suppression)

● CLASSIC TRAP

Keep the polarity straight: **P** = positive, **N** = negative; the number is the approximate latency in milliseconds. **P300 is reduced AND delayed** in schizophrenia (both amplitude down and latency up). MMN is *pre-attentive*, so its reduction is not just an attention deficit, which is why it is prized as an early marker.

5 Other electrophysiology

Polysomnography (PSG)

The overnight multi-channel sleep study. Three core channels define the sleep stage; the rest screen for sleep pathology. (Stage criteria are drilled in the sleep pack.)

Channel	Records	Scores / detects
EEG	cortical activity	sleep stage (sleep spindles, K-complexes, slow waves)
EOG	eye movements	**REM** sleep (rapid eye movements)
EMG (chin)	muscle tone	REM atonia; loss of atonia in REM behaviour disorder
Airflow + effort + SpO_2	breathing, oxygen	apnoeas/hypopnoeas (the AHI) in sleep apnoea
ECG, leg EMG	heart, limb movement	arrhythmia; periodic limb movements

● MNEMONIC

"EEG, EOG, EMG" → the three that stage sleep

Brain (stage), eyes (REM), chin muscle (atonia). REM is defined by REMs on EOG *plus* muscle atonia on chin EMG, against a low-voltage mixed-frequency EEG.

Electromyography (EMG)

● **EMG** – records electrical activity of skeletal muscle via needle or surface electrodes; with nerve conduction studies it separates **myopathic from neuropathic** weakness. In psychiatry, surface EMG is used in **biofeedback** and to index muscle tension in anxiety.

Galvanic skin response / electrodermal activity

● **GSR / EDA** – measures skin **conductance**, which rises with sweat-gland (sympathetic) activity. An index of autonomic **arousal**; the core channel in the polygraph ("lie detector") and in studies of orienting, anxiety and conditioning.

▲ **Reduced EDA reactivity** – blunted skin-conductance responses are reported in **psychopathy / antisocial personality** (weak fear conditioning) and as **electrodermal non-responding** in a subset of schizophrenia.

Magnetoencephalography (MEG)

● **MEG** – records the tiny **magnetic** fields produced by neuronal currents (via SQUID sensors). Complements EEG with **superior spatial localisation** of the source; a research and pre-surgical tool, not a routine bedside test.

QUICK-RECALL · DRILL THESE ONE-LINERS

- **EEG source:** summed cortical **postsynaptic potentials** of pyramidal neurons (not action potentials); ~10–100 µV.
- **10–20 system:** 10% / 20% of nasion–inion & preauricular distances. Letter = region (Fp/F/C/P/O/T), z = midline, **even = right, odd = left**.
- **Bands:** delta <4 (deep sleep) · theta 4–8 (drowsy) · alpha 8–13 (relaxed, eyes closed, occipital) · beta 13–30 (alert; **benzos raise beta**) · gamma >30 (active cognition).
- **Alpha reactivity:** alpha blocks (attenuates) on **eye opening** – the Berger effect.
- **Epileptiform:** spike <70 ms · sharp wave 70–200 ms · **3 Hz spike-and-wave = absence**.
- **Slowing:** focal = structural lesion; generalised = diffuse/metabolic/toxic. Activation: HV (absence), photic (photosensitive), sleep deprivation (raises yield).
- ★ **Psychiatry: diffuse slowing in delirium** is the single most useful sign (DT is the fast-activity exception). EEG largely normal in primary psychiatric disorders; used to exclude organic causes.
- **Patterns:** triphasic (hepatic) · periodic sharp waves ~1 Hz (CJD) · PLEDs temporal (HSV encephalitis).
- **EPs:** VEP P100 (optic nerve / MS) · BAEP waves I–V (acoustic neuroma, hearing screen) · SSEP (dorsal column).
- **ERPs: P300 reduced + delayed** in schizophrenia (oddball) · MMN reduced (pre-attentive, NMDA) · N400 = semantic mismatch · P50 = sensory gating.
- **Other:** PSG = EEG (stage) + EOG (REM) + chin EMG (atonia) + airflow/SpO_2 (AHI). EMG = myopathic vs neuropathic. GSR/EDA = sympathetic arousal (reduced in psychopathy). MEG = magnetic fields, superior localisation.

ONE-DAY REVISION · PAPER 2

Neuroimaging in Psychiatry

Two questions only: what the brain looks like (structure) and what it is doing (function). Hold which modality answers which, the CT-versus-MRI trade-offs, the red flags that warrant a scan in first-episode psychosis, and the handful of findings tied to each disorder. The comparison tables are the marks.

CONCEPT · RULE · TRAP · KEY NUMBER · MNEMONIC

The colour tells you what kind of fact it is. Imaging supports, it never makes, the psychiatric diagnosis.

★ HIGH YIELD TOP 5 IF NOTHING ELSE

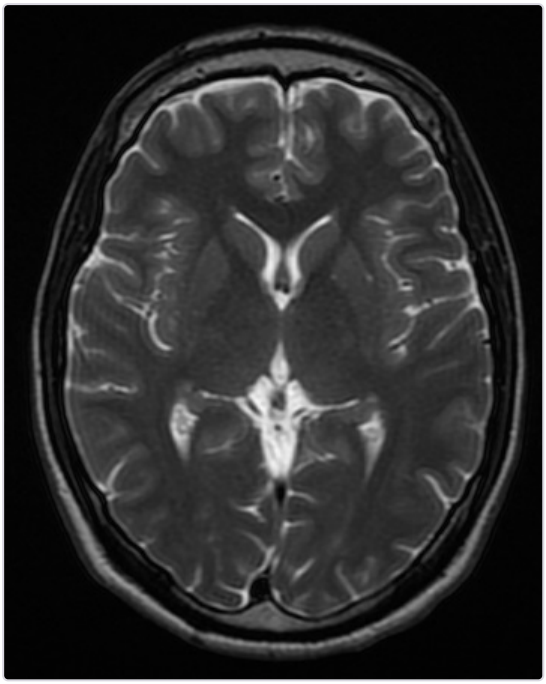
1. The whole field splits two ways: **structure** (what the brain looks like) = CT and MRI; **function** (what it does) = fMRI, PET, SPECT, MRS, DTI.
2. **CT is first-line only for acute haemorrhage** (fresh blood is bright, scan is fast); for almost everything else in psychiatry (atrophy, white matter, mesial temporal detail) MRI is superior, and **DWI shows an acute infarct within minutes, far earlier than CT**.
3. No scan makes a psychiatric diagnosis: clinically imaging only **excludes an organic mimic**, and group-level research findings have large patient-control overlap so they do not transfer to a single individual's scan.
4. Schizophrenia's most replicated structural finding is **enlarged lateral and third ventricles** with raised ventricle-to-brain ratio (present from first episode), plus functional **hypofrontality** on frontal tasks.
5. Alzheimer's signature is **medial temporal and hippocampal atrophy** on MRI plus **temporoparietal hypometabolism** on FDG-PET, but atrophy alone does not diagnose dementia (it overlaps with normal ageing).

1 The two questions, and when to image

All of neuroimaging answers one of two questions. **Structural** imaging asks what the brain looks like – its anatomy, atrophy, a bleed, a tumour. **Functional** imaging asks what the brain is *doing* – blood flow, metabolism, receptor binding. Keep that split and the modalities sort themselves.

Question	Asks	Modalities
Structure	what does the brain look like? (anatomy, lesion, atrophy, blood)	CT, MRI
Function	what is the brain doing? (flow, metabolism, receptors, tracts)	fMRI, PET, SPECT, MRS, DTI

NORMAL BRAIN MRI



Normal axial T2-weighted MRI: clear grey-white differentiation, normal ventricles and sulci. Source: Novaksean, CC BY-SA 4.0, Wikimedia Commons.

- **Clinical vs research** – in the **clinic** imaging is used to *exclude* an organic cause (rule out the lesion that mimics psychiatric illness). In **research** it maps group-level differences between patients and controls. The two uses do not transfer: a research finding does not diagnose an individual patient.

When to image a first-episode psychosis

Routine scanning of every first-episode psychosis is **not** mandated; yield is low when the history and examination are clean. Image when a **red flag** raises suspicion of an organic cause.

● RULE

Scan a first-episode psychosis when any red flag is present: **focal neurological signs**, new **seizures**, **delirium** or fluctuating consciousness, abrupt or very **atypical onset**, onset at the **extremes of age** (early childhood or new-onset late life), **headache, vomiting or other features of raised intracranial pressure**, rapid cognitive decline, or a poor response to adequate treatment. MRI is preferred over CT unless a bleed must be excluded urgently.

HOLD THIS

Structure = CT and MRI. Function = fMRI, PET, SPECT, MRS, DTI. Clinical imaging **excludes organic disease**; it does not confirm a psychiatric diagnosis.

2 Structural imaging: CT and MRI

CT (computed tomography)

- **What it is** – X-rays from many angles reconstructed into cross-sectional slices. Density-based: **bone and acute blood are bright** (hyperdense), air and fat are dark.
- **Strengths** – **fast** (seconds), widely available, the test of choice for **acute haemorrhage**, fractures and bony detail. Works when MRI is contraindicated or the patient cannot stay still.

▲ **Limits** – uses **ionising radiation**; poor **soft-tissue contrast**; the posterior fossa and brainstem are degraded by beam-hardening artefact. Iodinated contrast carries an allergy and nephrotoxicity risk.

MRI (magnetic resonance imaging)

● **What it is** – a strong magnetic field and radiofrequency pulses map the behaviour of **hydrogen protons** in tissue water. **No ionising radiation.** Superior soft-tissue contrast, the workhorse for the psychiatric brain.

Sequence	CSF / water looks…	Best for
T1	dark	anatomy; fat bright. Grey/white contrast, atrophy
T2	bright	most pathology (oedema, gliosis, demyelination) is bright
FLAIR	suppressed (dark)	T2 with CSF nulled – lesions near ventricles, white-matter hyperintensities, MS plaques
DWI	–	restricted diffusion – **acute infarct** (bright within minutes), abscess, prion disease

▲ **MRI contraindications** – ferromagnetic hazards: older **pacemakers** and implanted defibrillators, **cochlear implants**, ferromagnetic aneurysm clips, certain metallic foreign bodies (especially **intra-ocular metal**). Practical limits: severe **claustrophobia**, inability to lie still, and the loud, slow acquisition. Gadolinium contrast is avoided in severe renal impairment (nephrogenic systemic fibrosis).

CT versus MRI

	CT	MRI
Soft-tissue resolution	lower	far superior (grey/white detail)
Best shows	acute blood, bone, calcification	soft tissue, atrophy, white-matter and posterior-fossa lesions
Ionising radiation	yes	none
Speed	seconds (emergency-friendly)	minutes, motion-sensitive
Cost / access	cheaper, near-universal	costlier, less available
Contraindications	pregnancy (radiation), iodine contrast allergy	pacemaker, cochlear implant, ferromagnetic metal, intra-ocular metal, claustrophobia

● CLASSIC TRAP

Acute haemorrhage is the one place CT wins. Fresh blood is bright on CT and the scan is fast, so CT is first-line for suspected acute bleed or trauma. For almost everything else in psychiatry – atrophy, white-matter change, mesial temporal detail – **MRI is superior**. DWI on MRI shows an acute infarct within minutes, far earlier than CT.

3 Functional and molecular imaging

Functional MRI (fMRI)

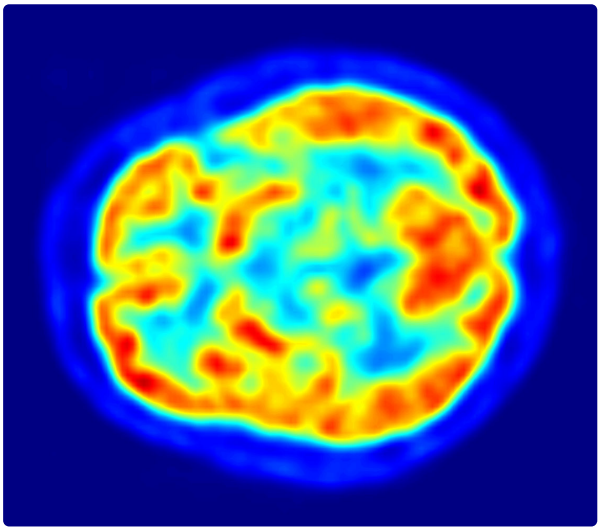
● **BOLD signal** – **blood-oxygen-level-dependent** contrast. Active neurons draw more oxygenated blood than they consume, so local **deoxyhaemoglobin falls**; because deoxyhaemoglobin is paramagnetic, the MR signal *rises*. fMRI maps activity *indirectly*, through blood flow, not by recording neurons.

● **Task vs resting-state** – **task-based** fMRI contrasts a task against a baseline to localise function. **Resting-state** fMRI maps intrinsic connectivity with no task (the **default mode network**). No ionising radiation; good spatial, modest temporal resolution.

PET and SPECT (nuclear, radiotracer-based)

- **PET** – positron emission tomography. A positron-emitting tracer is injected; **FDG (fluorodeoxyglucose)** maps **glucose metabolism** (active tissue takes up more). Other ligands map specific **receptors and transporters** – dopamine D2, serotonin, and amyloid plaque tracers. Higher resolution than SPECT but needs a cyclotron-made tracer.
- **SPECT** – single-photon emission CT. Maps **regional cerebral blood flow** with longer-lived tracers (technetium-99m HMPAO). Cheaper and more available than PET, lower resolution. DaTSCAN (a dopamine transporter ligand) separates Parkinsonian from non-degenerative tremor.
- **Amyloid & dopamine imaging** – **amyloid PET** (PiB and related tracers) images plaque load in Alzheimer's. **Dopaminergic PET/SPECT** probes the dopamine system implicated in psychosis and movement disorders.

BRAIN FDG-PET



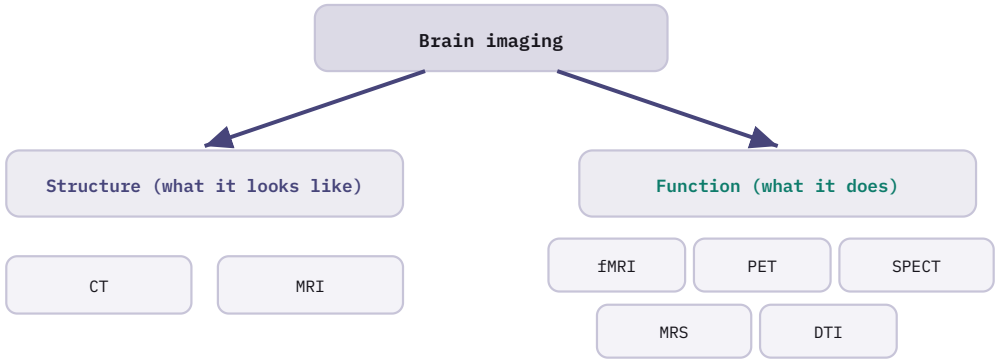
Axial FDG-PET, shown in its native colour scale: **blue = low, red/yellow = high** glucose metabolism. Functional, not structural, imaging. Source: Jens Maus, public domain, Wikimedia Commons.

MRS and DTI (MRI-based, no radiation)

- **MRS (magnetic resonance spectroscopy)** – reads brain **chemistry**, not pictures, plotting metabolite peaks. **NAA (N-acetylaspartate)** marks neuronal integrity and falls with neuronal loss; **choline** marks membrane turnover; also creatine (reference), myo-inositol, glutamate/glutamine and lactate.
- **DTI (diffusion tensor imaging)** – tracks the directional diffusion of water along **white-matter tracts** (tractography). **Fractional anisotropy (FA)** indexes tract integrity; reduced FA points to disrupted connectivity (studied in schizophrenia and ageing).

Modality	Measures	Radiation	Note
fMRI	BOLD – blood oxygenation (activity, indirect)	none	task & resting-state; connectivity
PET	glucose metabolism (FDG); receptor/transporter ligands	yes	high resolution; amyloid & dopamine
SPECT	regional cerebral blood flow	yes	cheaper, lower resolution than PET
MRS	metabolite chemistry (NAA, choline…)	none	NAA = neuronal integrity
DTI	white-matter tract integrity (FA)	none	tractography, connectivity

STRUCTURAL VS FUNCTIONAL IMAGING AT A GLANCE



Structural modalities (CT, MRI) image anatomy; functional modalities (fMRI, PET, SPECT, MRS, DTI) image activity, metabolism, chemistry or connectivity. fMRI, MRS and DTI are MRI-based and radiation-free; PET and SPECT are nuclear.

● MNEMONIC

"NAA marks the Neurons"

On MRS, **NAA** (N-acetylaspartate) tracks neuronal integrity and falls when neurons are lost; choline rises with membrane turnover. PET burns **glucose** (FDG); fMRI rides the **BOLD** wave.

4 Findings in the major disorders

These are **group-level research findings**, not diagnostic tests. They are heavily examined, so hold the headline change attached to each disorder.

Disorder	Structural	Functional / molecular
Schizophrenia	enlarged lateral & third ventricles; reduced grey matter (frontal, temporal, mesial temporal); larger ventricle-to-brain ratio	hypofrontality (reduced frontal metabolism/flow, especially on tasks); dopaminergic changes
Mood disorders	white-matter hyperintensities in late-life / vascular depression; hippocampal volume loss in chronic depression	reduced **subgenual anterior cingulate** activity; altered **amygdala** reactivity
OCD	subtle caudate and orbitofrontal changes	hyperactivity of the orbitofrontal–caudate–thalamic circuit; normalises with treatment
Alzheimer's dementia	medial temporal & hippocampal atrophy; widened sulci, ventricular enlargement	FDG-PET: **temporoparietal hypometabolism**; amyloid-PET positive

Schizophrenia

◆ **Ventricular enlargement** – the most replicated structural finding: **enlarged lateral and third ventricles** with a raised ventricle-to-brain ratio, present from first episode and not purely drug-related. Accompanied by reduced grey matter (frontal and mesial temporal) and subtle whole-brain volume loss.

● **Hypofrontality** – reduced **prefrontal** metabolism and blood flow, most evident when a frontal task is demanded (the Wisconsin Card Sorting paradigm). Maps onto negative symptoms and executive deficits.

Mood disorders

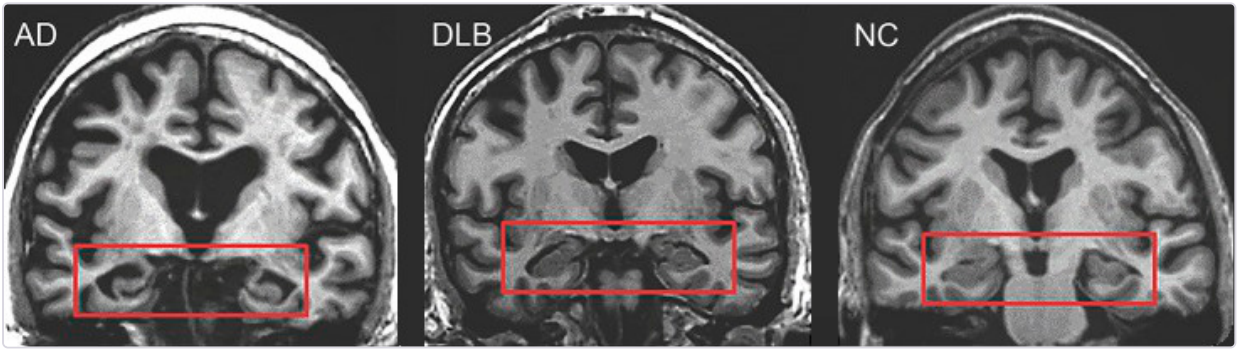
- **White-matter hyperintensities** – bright FLAIR/T2 foci in periventricular and deep white matter, characteristic of **late-life and vascular depression** (the "vascular depression" hypothesis). Reflect small-vessel disease disrupting fronto-subcortical circuits.
- **Limbic changes** – altered **amygdala** reactivity to negative stimuli and reduced **subgenual anterior cingulate** (area 25) volume/activity, a node targeted in deep brain stimulation for refractory depression.

OCD

- **The orbitofrontal–caudate circuit** – OCD is the cleanest circuit story: hyperactivity of the **orbitofrontal cortex, caudate (striatum) and thalamus** loop on functional imaging. The loop *normalises* after successful SSRI or CBT, one of the few imaging changes that tracks treatment response.

The dementias

MEDIAL TEMPORAL ATROPHY IN DEMENTIA



Coronal MRI, hippocampal region boxed: Alzheimer's (AD) shows the most medial-temporal atrophy, dementia with Lewy bodies (DLB) is relatively spared, normal control (NC) is intact. Source: McKeith et al. 2017, DLB Consortium, CC BY 4.0, Wikimedia Commons.

- **Alzheimer's atrophy** – **medial temporal and hippocampal atrophy** is the structural signature on MRI (best seen on coronal T1), with widened sulci and ex-vacuo ventricular enlargement. Hippocampal volume tracks disease stage.
- **Molecular dementia imaging** – **FDG-PET** shows **temporoparietal hypometabolism** in Alzheimer's (and a different frontotemporal pattern in FTD, useful for differentiating dementias). **Amyloid-PET** images plaque burden; a negative scan makes Alzheimer's unlikely.

TRAP

Cerebral atrophy on a scan does not diagnose dementia. Atrophy accompanies normal ageing and overlaps heavily between healthy elders and early dementia; the diagnosis stays clinical. Conversely a structurally normal scan does not exclude dementia. Imaging here *excludes mimics* (tumour, subdural, normal-pressure hydrocephalus) and supports the clinical picture – it is not the test.

5 Principles and cautions

- **Imaging supports, it does not diagnose** – there is **no scan that makes a psychiatric diagnosis**. Diagnosis stays clinical. Imaging's clinical job is to *exclude* an organic mimic; its research job is to map mechanism.

▲ **Incidental findings** – scanning healthy or psychiatric brains turns up unexpected abnormalities (cysts, small meningiomas, vascular anomalies, white-matter spots) in a meaningful minority. Most are benign, but they generate anxiety, further tests and follow-up. A reason to scan only when there is a clinical question, not reflexively.

◆ **Group vs individual** – the disorder findings above are **statistical differences between groups**, with large overlap between patients and controls. A given patient's scan usually looks normal. A research association cannot be read off a single individual's image to confirm or exclude a diagnosis.

THE ONE PRINCIPLE

Imaging in psychiatry is to **rule out organic disease** and to study mechanism. It does **not** confirm a psychiatric diagnosis, and group-level findings do not transfer to the individual scan.

QUICK-RECALL · DRILL THESE ONE-LINERS

★ **The split:** structure (what it looks like) = CT, MRI. Function (what it does) = fMRI, PET, SPECT, MRS, DTI. Clinical imaging **excludes organic disease**; it never confirms a psychiatric diagnosis.

- **CT vs MRI:** CT = fast, bone, **acute blood**, radiation, the emergency test. MRI = superior soft tissue, atrophy, white matter, **no radiation**. DWI shows acute infarct within minutes.
- **MRI sequences:** T1 anatomy (CSF dark), T2 pathology (CSF bright), FLAIR = T2 with CSF nulled (periventricular lesions, WM hyperintensities), DWI = acute stroke.
- **MRI contraindications:** pacemaker, cochlear implant, ferromagnetic clips, intra-ocular metal, claustrophobia. CT: pregnancy, iodine contrast allergy.
- **Functional:** fMRI rides the **BOLD** signal (deoxyhaemoglobin falls, signal rises); task vs resting-state (default mode network).
- **Nuclear:** PET = glucose metabolism (FDG) + receptor/transporter and amyloid ligands; SPECT = blood flow, cheaper, lower resolution. Both use radiation.
- **MRS & DTI:** MRS = chemistry, **NAA** = neuronal integrity, choline = membrane turnover. DTI = white-matter tracts, FA = integrity.
- **Schizophrenia:** enlarged lateral & third ventricles, reduced grey matter, **hypofrontality**.
- **Mood:** white-matter hyperintensities (late-life/vascular depression), amygdala & subgenual cingulate changes.
- **OCD:** hyperactive **orbitofrontal–caudate–thalamic** circuit, normalises with treatment.
- **Alzheimer's:** medial temporal & hippocampal atrophy; FDG-PET temporoparietal hypometabolism; amyloid-PET positive. Atrophy alone does not diagnose dementia.
- **Principle:** imaging supports, never makes, the diagnosis; mind incidental findings; group findings do not read off an individual scan.

ONE-DAY REVISION · PAPER 2

Landmark studies, made citable

You already know the clinical content. What banks the marks is the evidence layer: the trial name, the author, the year, and the one sentence it proved. Learn each study as a quotable line. When an examiner asks "what is the evidence for clozapine?", you answer "Kane et al, 1988" and move on.

CONCEPT · RULE · TRAP · YEAR / NUMBER · MNEMONIC

The colour tells you what kind of fact it is. Every year here has been verified against the primary source; where a year is genuinely contested, the text says so.

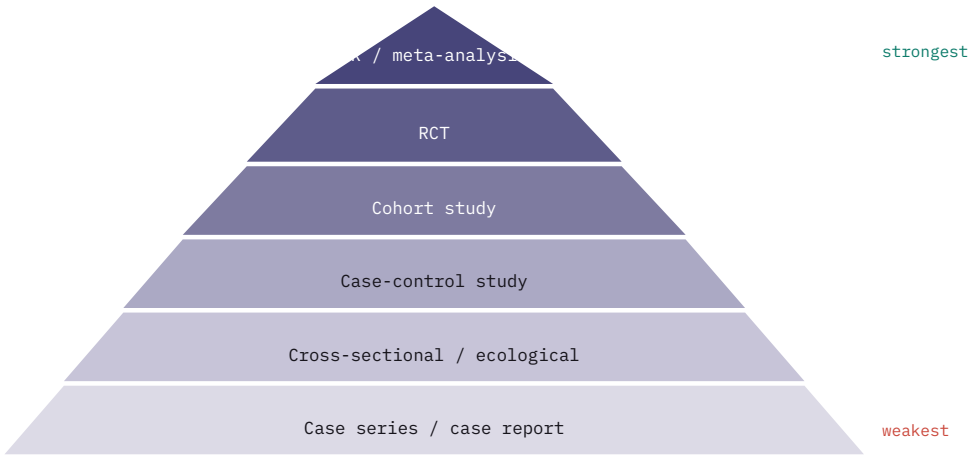
★ HIGH YIELD TOP 5 IF NOTHING ELSE

1. Kane et al, 1988 (Arch Gen Psychiatry): double-blind clozapine > chlorpromazine in treatment-resistant schizophrenia, the one landmark to quote cold; CATIE phase 2 and Leucht 2013 both echo clozapine's standalone superiority.
2. Classic trap: CATIE (Lieberman, 2005) + CUtLASS-1 (Jones, 2006) together killed the claim that SGAs are uniformly superior to FGAs; perphenazine was broadly comparable, so choose by side-effect profile and cost, not by class.
3. Oxford CEBM ranks study designs (Level 1 = SR of RCTs → Level 5 = expert opinion; grades A-D), whereas GRADE rates certainty for one outcome (RCTs start high, observational low; downgrade for RIIIP: risk of bias, inconsistency, indirectness, imprecision, publication bias).
4. Bipolar maintenance: lithium > valproate monotherapy for relapse prevention (BALANCE, Geddes, 2010) and lithium reduces suicide and all-cause death in mood disorders (Cipriani, BMJ 2013); STEP-BD (Sachs, 2007) showed adjunctive antidepressant gives no benefit over mood stabiliser alone in bipolar depression.
5. WHO IPSS / DOSMeD: schizophrenia exists transculturally with classically better outcome in developing countries, but state the caveat (ISoS long-term follow-up and case-finding criticism) to score full marks.

1 Levels of evidence & GRADE

Two systems get asked. The **Oxford CEBM hierarchy** ranks study *designs* by how far they sit from bias. **GRADE** does something different: it rates certainty in a body of evidence for one specific *outcome*. Keep them apart.

HIERARCHY OF EVIDENCE (TOP = STRONGEST, LEAST BIASED)



Higher up = less susceptible to bias and confounding. A well-conducted SR / meta-analysis of RCTs sits at the top; below the base sits expert opinion and bench reasoning, the weakest standalone evidence.

Oxford CEBM level	Example design	Strength
Level 1	SR of RCTs; single RCT with narrow CI	strongest
Level 2	cohort study; SR of cohorts; low-quality RCT	strong
Level 3	case-control study; SR of case-control	moderate
Level 4	case series; poor cohort / case-control	weak
Level 5	expert opinion; bench / mechanistic reasoning	weakest

- **Grades of recommendation** – CEBM levels translate into grades **A** (consistent level-1 evidence), **B** (level-2/3 or extrapolation from level-1), **C** (level-4), **D** (level-5 or inconsistent/inconclusive). Grade A is the firmest recommendation.

GRADE: certainty in the evidence

GRADE rates an **outcome**, not a single study. Each body of evidence starts at a default and is then moved up or down.

Starting point

RCTs start HIGH. Observational studies start **LOW.** Five downgrade factors and three upgrade factors then move the rating.

Four certainty levels

High → Moderate → Low → Very low. Each reflects confidence that the true effect lies close to the estimate.

5 reasons to downgrade

1. Risk of bias (poor methods)
2. Inconsistency (heterogeneity)
3. Indirectness (wrong population / comparison / outcome)
4. Imprecision (wide CI, few events)
5. Publication bias

3 reasons to upgrade (observational only)

1. Large effect size
2. Dose–response gradient
3. Plausible confounding would have *reduced* a real effect (bias works against the finding)

MNEMONIC

Downgrade = "RIIIP"

Risk of bias, **I**nconsistency, **I**ndirectness, **I**mprecision, **P**ublication bias. Upgrades apply to observational data only: large effect, dose-response, opposing confounding.

RULE

Do not confuse the two. CEBM ranks a **design** in the abstract; GRADE rates the **certainty for one outcome** after looking at the actual trials. A meta-analysis of RCTs can still earn only "low" certainty in GRADE if the trials are biased, heterogeneous and imprecise.

2 Schizophrenia trials

Trial	Author, year, journal	What it established (the exam line)
Clozapine landmark	Kane et al, **1988**, *Arch Gen Psychiatry*	clozapine > chlorpromazine in treatment-resistant schizophrenia
CATIE	Lieberman et al, **2005**, *NEJM*	very high all-cause discontinuation; perphenazine (FGA) broadly comparable
CUtLASS-1	Jones et al, **2006**, *Arch Gen Psychiatry*	no clear quality-of-life advantage of SGAs over FGAs
EUFEST	Kahn et al, **2008**, *Lancet*	first-episode; SGAs better retained, core efficacy broadly similar
Northwick Park	Crow, Johnstone et al, **1986**, *BJP*	first-episode; maintenance neuroleptic cuts relapse; longer untreated illness → worse outcome
15-drug MTM	Leucht et al, **2013**, *Lancet*	antipsychotics differ on a continuum; the FGA/SGA split is not a clean efficacy divide

◆ **Kane et al, 1988** – the one to quote cold. Double-blind clozapine vs chlorpromazine in patients who had failed multiple neuroleptics; clozapine clearly superior. This single trial established clozapine as **the** drug for treatment resistance, a status no later trial has overturned.

● **CATIE** – large NIMH pragmatic effectiveness trial. Primary outcome was **all-cause discontinuation**, which was high across every arm (around three-quarters stopped). Olanzapine was modestly best on time-to-discontinuation; the old, cheap **perphenazine was broadly comparable** to the newer drugs. In phase 2, **clozapine outperformed other SGAs**, echoing Kane.

● **CUtLASS-1** – pragmatic UK NHS trial: clinician chooses an FGA or a (non-clozapine) SGA, then randomises within class. Found **no quality-of-life or symptom advantage** for SGAs, and if anything a slight edge to FGAs. With CATIE, it dismantled the blanket claim that "SGAs are better."

CLASSIC TRAP

CATIE + CUtLASS together killed the idea that second-generation antipsychotics are *uniformly* superior on efficacy. **Clozapine is the one exception** whose superiority survives scrutiny (Kane 1988). Leucht 2013 then showed drugs sit on a continuum, so "FGA vs SGA" is a poor way to choose. Choose by side-effect profile, cost and the individual patient, not by class.

3 Mood disorder trials

Trial	Author, year, journal	What it established (the exam line)
STAR*D	Rush et al, **2006**, *Am J Psychiatry*	depression often needs sequential treatment; ~1/3 remit at step 1

Trial	Author, year, journal	What it established (the exam line)
STEP-BD	Sachs et al, **2007**, *NEJM*	adjunctive antidepressant no better than mood stabiliser alone in bipolar depression
BALANCE	Geddes et al, **2010**, *Lancet*	lithium (alone or with valproate) > valproate monotherapy for relapse prevention
Lithium & suicide	Cipriani et al, **2013**, *BMJ*	lithium reduces suicide and all-cause death in mood disorders
AD continuation	Geddes et al, **2003**, *Lancet*	continuing an antidepressant after remission roughly halves to thirds the relapse odds
21 antidepressants	Cipriani et al, **2018**, *Lancet*	all 21 beat placebo; they differ in efficacy and acceptability (network meta-analysis)

- STAR*D – the largest real-world **sequenced** trial in major depression. Step 1 was citalopram: about **one-third remitted**. Remission accumulated across switch and augment steps, but the chance of response **fell with each further step**. The exam point: depression frequently needs more than one treatment, and earlier steps work better.
- STEP-BD – in bipolar depression, adding an **antidepressant to a mood stabiliser gave no benefit** over the mood stabiliser alone, with no significant excess switch to mania in the main analysis. The basis for caution with antidepressants in bipolar depression.
- BALANCE – open-label bipolar I maintenance: lithium monotherapy and lithium-plus-valproate were both **better than valproate monotherapy** at preventing relapse; combination numerically best. Supports lithium as a first-line maintenance agent.

● LITHIUM & SUICIDE

A consistent body of evidence (Cipriani et al, *BMJ* 2013, updating the earlier meta-analyses) links **lithium with reduced suicide, self-harm and all-cause death** in mood disorders, an anti-suicidal effect not clearly shown for other mood stabilisers and only partly explained by relapse prevention. A recurring exam favourite, and part of why lithium stays first-line in maintenance.

4 ECT, course & epidemiology

The ECT evidence

◆ UK ECT Review Group, 2003 – systematic review and meta-analysis in the *Lancet*. Established that **real ECT > sham ECT**, that **ECT > pharmacotherapy** for short-term efficacy in depression, that **bilateral > unilateral**, and higher dose > lower dose. The single citable source for "ECT works in depression."

The WHO cross-cultural schizophrenia studies

Study	What it examined	The exam sentence
WHO IPSS (Intl Pilot Study of Schizophrenia, from 1966; first report 1973)	cross-national symptom profile & course, 9 countries	schizophrenia **exists across cultures** with a recognisable core syndrome; outcome appeared **better in developing centres** (India, Nigeria, Colombia)
WHO DOSMeD (Determinants of Outcome of Severe Mental Disorders, 1980s)	incidence & 2-year outcome across 10 countries	broadly **similar incidence** of narrowly defined schizophrenia worldwide; again **better outcome in developing settings**

● EXAM PEARL

The "better outcome in developing countries" finding is **heavily debated**. The long-term ISoS follow-up showed only about half the developing-country "best outcome" cases held up, and critics question case-finding, follow-up and the developed/developing dichotomy. Score the marks by stating the classic finding *and* the caveat.

Psychosocial predictors of relapse

- **Expressed emotion (Brown; Vaughn & Leff)** – high family **EE** (criticism, hostility, emotional over-involvement) predicts schizophrenia **relapse**. Brown originated the construct and the Camberwell Family Interview; **Vaughn & Leff (1976)** refined its measurement and confirmed the relapse link. The basis for family intervention in high-EE households.
- **Life events (Brown & Birley, 1968)** – an excess of independent **life events** clusters in the weeks just before the onset or relapse of schizophrenia. EE and life events are the two psychosocial relapse predictors to name together.

5 Guidelines: what to cite, when

Examiners want the **headline recommendation** and the right source name, not dosing detail. Know what each guideline *is* so you cite the appropriate one.

Guideline	What it is	Cite it when…
NICE	UK national, evidence-graded guidance (NHS); condition-by-condition	you want the authoritative, systematically-reviewed recommendation
APA	American Psychiatric Association practice guidelines; GRADE-based	a US-style, consensus-plus-evidence recommendation is wanted
IPS CPG	Indian Psychiatric Society Clinical Practice Guidelines	the answer should reflect **Indian practice and context**
Maudsley	Maudsley Prescribing Guidelines in Psychiatry; a *prescribing* reference, not a care pathway	the question is about **drug choice, dosing or switching**

Headline recommendations (drill these)

- **Schizophrenia** – antipsychotic + offer **CBT** + **family intervention** (especially high-EE families). **Clozapine** when two adequate antipsychotic trials (one a SGA) have failed = treatment resistance.
- **Depression** – **stepped care** by severity; combine pharmacotherapy with psychotherapy in moderate-to-severe illness. Continue the antidepressant **at least 6 months after remission** (longer if recurrent) – the Geddes 2003 logic.
- **Bipolar** – **lithium first-line for maintenance** / relapse prevention; caution with antidepressant monotherapy; treat acute mania with an antipsychotic and/or a mood stabiliser.

QUICK RULE

NICE = UK national guidance · APA = American · **IPS CPG** = Indian Psychiatric Society (cite for Indian context) · Maudsley = the prescribing reference (cite for drug/dose questions).

QUICK-RECALL · DRILL THESE ONE-LINERS

- **Evidence:** Oxford CEBM ranks *designs* (Level 1 = SR of RCTs → Level 5 = expert opinion; grades A–D). GRADE rates *one outcome*: RCTs start high, observational low; downgrade for **RIIIP**, upgrade observational for large effect / dose–response / opposing confounding.
- ★ **Kane et al, 1988 (Arch Gen Psychiatry):** clozapine > chlorpromazine in **treatment-resistant schizophrenia** – the landmark.

- **CATIE (Lieberman, NEJM 2005):** high all-cause discontinuation; perphenazine comparable; clozapine best in phase 2 – questioned routine SGA superiority.
- **CUtLASS-1 (Jones, 2006):** no clear QoL advantage of SGAs over FGAs. **EUFEST (Kahn, Lancet 2008):** first-episode, SGAs better retained, efficacy similar.
- **Northwick Park (Crow/Johnstone, BJP 1986):** first-episode; maintenance neuroleptic cuts relapse (46% vs 62%); longer untreated illness predicts worse outcome. **Leucht (Lancet 2013):** 15 antipsychotics on a continuum, FGA/SGA split unhelpful.
- **STAR*D (Rush, 2006):** ~1/3 remit at step 1; sequential steps add remission but response falls each step.
- **STEP-BD (Sachs, NEJM 2007):** adjunctive antidepressant **no better** than mood stabiliser alone in bipolar depression.
- **BALANCE (Geddes, Lancet 2010): lithium > valproate** monotherapy for bipolar maintenance.
- **Lithium & suicide (Cipriani, BMJ 2013):** lithium reduces suicide and death in mood disorders. **AD continuation (Geddes, Lancet 2003):** continuing antidepressants prevents relapse. **21 antidepressants (Cipriani, Lancet 2018):** all > placebo, differ in efficacy/acceptability.
- **UK ECT Review Group (Lancet 2003):** ECT > sham and > drugs short-term; bilateral > unilateral.
- **WHO IPSS / DOSMeD:** schizophrenia transcultural; classically **better outcome in developing countries** (now debated, ISoS).
- **Vaughn & Leff (1976):** high **expressed emotion** → relapse · **Brown & Birley (1968): life events** precede onset/relapse.
- **Guidelines:** NICE (UK) · APA (US) · IPS CPG (India) · Maudsley (prescribing). Schizophrenia: clozapine after two failed trials. Depression: continue AD ≥6 months after remission. Bipolar: lithium first-line maintenance.