WEAVE SPRINT
PAPER I · VOLUME THREE

MD and DNB psychiatry, theory revision

Foundations of Psychiatry

Behaviour, Measurement and Evidence

How people are moved, how behaviour is learned, and how personality has been explained. Then the instruments that measure the mind, and how to read evidence.

Dr. Wilfred D'souza · Dr. Niharika Reddy

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WEAVE SPRINT · PAPER I

Foundations of Psychiatry

THE WEAVE SPRINT SERIES

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WRITTEN BY

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First compiled 2026. Free to read, share and print for study.

The clinical content is written for doctors and doctors in training. It is a study and revision aid, not a substitute for current guidelines or clinical judgement. Verify doses and thresholds against the current references and your local protocols before any clinical use.

For every resident who has ever sat up the night before,
afraid it will not be enough.

ACKNOWLEDGEMENT

A word of thanks

I want to begin by thanking something I cannot quite name. Call it whatever you like. I have never been certain what to call it, and I have stopped needing to be. There is a part of this work that does not feel like mine. The line that arrives before I have thought of it, the connection I did not plan, the steadiness on a morning I had no reason to be steady. I only hold the pen. Something moves through the holding, and I have learned to thank it without asking it for a name.

Someone once asked me why I give all of this away. Why hand out the notes I sat up making, to the very people sitting the same exam beside me. It is a fair question. If I had kept them, I would probably have scored a few marks more than the next person, and I might have been thought of as the better student. I have turned that over honestly, and I have decided it is a small thing to want. What I get instead, by putting the work in your hands, is the chance to take a little of the fear out of a great many nights. That trade is not close. Easing that fear is not a detour on the way to becoming a good psychiatrist. It is the thing itself. I did not come here to be the best student in the room. I came here to be a good psychiatrist.

And to Niharika, who reads everything first and knows when to be honest and when to wait; and to the residents who study from these pages and write back with the corrections and the questions: thank you. The book is better for you, and so am I.

Dr. Wilfred D'souza

Foreword

This third volume closes the opening paper with its tools. Social and learning psychology explain how a person is shaped by other people and by consequences; psychometry and descriptive psychopathology supply the words and the tests that the clinical papers will use without explaining; and research methods decide which claims are allowed in at all.

The notes were written to be understood, not just revised. Each chapter is a full account of its subject, from the mechanism up to the point the exam tests, so that you can read to learn the first time and read to recall every time after. Where a fact is worth holding exactly, it is marked. Where a mistake is easy to make, it is named before you make it. The colour code on the next pages tells your eye what kind of fact it is looking at before your mind has to decide, and after a few pages it stops being a key you consult and becomes a sense you carry.

Read it in order if you are meeting the material, or open it anywhere if you are returning to it. The contents at the front and the index at the back both jump to the page you want. However you use it, the hope held in these pages is plain: that one ordered, trustworthy place to study from makes the work a little lighter, and that you walk into the hall steadier than you would have otherwise.

HOW TO USE THIS BOOK

The colour memory code

Every note in this volume speaks one visual language, so once you learn it in the first chapter you read the rest without thinking about it.

Concept and rule	The core idea and the principle that follows from it. When a line is petrol, it is the thing to understand, and the thing worth being able to state.
Key number	A figure to hold exactly: a dose, a threshold, a prevalence, a cut-off. Amber means memorise this number, not just the shape of it.
Trap	The mistake the exam is waiting for. When a line is coral, it is warning you off an error that looks right until it costs you the mark.

Hold this is the single line from a topic you must carry out of it. **Good to remember** gathers, for each named thing, what it is, when it matters, and the one number or formula worth keeping. Both help a tired brain find the centre of a dense page fast.

The contents at the front is clickable, and so is the index at the back: every entry jumps to its page. Read to learn once, and to recall as often as you need.

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PART V

V

Social, Learning & Personality Psychology

How people are moved by other people, how behaviour is learned, and how personality has been explained.

01 Orientation

02 Social psychology

03 Learning and behaviour

04 Personality and psychodynamic theory

05 Special topics

06 Discriminate

07 Apply and consolidate

Social, Learning & Personality Psychology

Seven sections · 20 topics · one complete notes document

These notes complete the psychology of the paper by covering the three domains the General and Developmental Psychology notes deliberately held back: the social, the learned and the theorised person. Where the General and Developmental Psychology notes taught the general functions and the developmental theorists, these notes cover **how people act on each other, how behaviour is shaped, and how personality is modelled.** Social psychology comes first (attitudes and their change, attribution and the biases of social thinking, conformity and obedience, prejudice, aggression, the crowd), then learning and behaviour (classical and operant conditioning, Bandura's social learning, and the therapies built directly on them), then the rest of psychodynamic and personality theory (the defences and the Vaillant hierarchy, the object-relations school, and the trait, type, humanistic and positive-psychology models). Freud's models, Adler and Jung are taught in full on the General and Developmental Psychology notes and only cross-referenced here. The examinable skill is discrimination: telling classical from operant from social learning, one personality model from another, and one social bias from the next, then placing a case in the right paradigm.

HOW TO USE THESE NOTES

The theory-heavy topics (attitudes, attribution, the learning paradigms, the personality models) follow the same flow as the General and Developmental Psychology notes: the idea in brief, then the **theories in the order they appeared** (what each fixed, what stayed debated), then the **currently accepted** position, then a **behavioural or neural correlate**, then a worked example. Figures declare one axis and code it in colour and shape: **petrol** marks structure and the neutral case, **coral** marks conflict, pathology or the point of debate, and **marigold** highlights the accepted answer. The boxes marked **Good to remember** gather every named theorist, mechanism and classification in one place, so the reference facts sit apart from the reasoning. One patient-facing note carries through the whole ecosystem: no diagnosis is named to a patient, but this is doctor-facing revision, so the full clinical vocabulary is used.

Orientation

1 · Orientation: the three domains as a map

The General and Developmental Psychology notes gave you the general functions of the mind and the developmental theorists, taught through five competing lenses. This material takes the territory that those lenses opened but did not pay off, and it turns out to be three large domains, each defined by a different **unit of analysis**. The **social** person is moved by the presence, judgement and pressure of *other people*; the **learned** person is behaviour *shaped by consequence and by observation*; the **theorised** person is personality itself, *modelled* by a succession of schools that each claimed to say what a person fundamentally is. Fix these three as a map before any of the detail, because the paper rarely asks you to defend one domain against another; it asks you to

recognise which domain a given experiment, effect or theorist belongs to, and to attach the right name (Kaplan & Sadock 2024).

The exam skill across this whole chapter is placement. An item about a subject who conforms to a unanimous but wrong majority is **social psychology** (Asch); an item about a response that grows or shrinks according to its consequence is **learning** (Skinner); an item asking which trait dimension captures anxiety-proneness is **personality** (neuroticism, in the Big Five). This orientation lays the three domains side by side, binds each to its unit of analysis and its key figures, and marks the two boundaries you must not cross: the psychodynamic *founders* (Freud's topographic and structural models, Adler, Jung) are taught in full on the General and Developmental Psychology notes and are only cross-referenced here, and the psychological *tests* (reliability, validity, the specific instruments) are the Psychometry, Assessment and Descriptive Psychopathology notes. The rest of these notes pays the map off.

1.1 Why three domains, and what each one counts as data

Each domain answers a different question about behaviour. Social psychology asks how the individual is changed by the real, imagined or implied presence of others, so its data are situations and the people in them. Learning and behaviour ask how experience lawfully changes what an organism does, so its data are stimuli, responses and their contingencies. Personality asks what stable internal structure makes one person reliably different from another across situations, so its data are consistencies over time. The same clinical fact sits differently in each: a patient's avoidance of a crowded market is, to social psychology, a matter of anticipated evaluation and the behaviour of the crowd; to learning, a conditioned fear response maintained by negative reinforcement (escape lowers anxiety, so avoidance is strengthened); to personality, an expression of high trait neuroticism and low extraversion. None is wrong; the discipline for the viva is to name the domain the question is pitched at (Kaplan & Sadock 2024; Tasman 2015).

1.2 The three domains as a map

Bind each domain to its unit of analysis and its key figures. This is the schema for the whole chapter: read the domain, hold what it counts as data, and attach the names examiners expect. Read the definitions once now as the index; every later topic is a special case of one of these three.

Social psychology

Unit of analysis: *the individual in the presence of others*. How attitudes form and change, how we explain behaviour (attribution), how the group bends the individual (conformity and obedience), and how intergroup processes produce prejudice, aggression and crowd behaviour. Key figures: Leon Festinger (cognitive dissonance, attitude change), Solomon Asch (conformity to a majority), Stanley Milgram (obedience to authority), Gordon Allport (attitudes, the nature of prejudice). This domain owns the attitudes, attribution, conformity, prejudice, aggression and crowd topics of these notes.

Learning and behaviour

Unit of analysis: *the lawful change in behaviour produced by experience*. Three paradigms: classical (Pavlovian) conditioning of a stimulus to a response, operant (instrumental) conditioning of a behaviour by its consequence, and observational learning by watching a model. Key figures: Ivan

Pavlov (classical conditioning), B. F. Skinner (operant conditioning, building on Thorndike's law of effect), Albert Bandura (observational or social learning). This domain grounds behaviour therapy and underwrites the conditioning topics.

Personality

Unit of analysis: *the stable internal structure that makes a person consistently themselves*. A succession of traditions: the psychodynamic (drives, defences, object relations), the trait (dimensions on which everyone can be scored), the type (discrete categories), the humanistic (the self striving toward growth), and the social-cognitive (person and situation in reciprocal interaction). Named anchors examiners want: the ego defence mechanisms, object relations, and above all the Big Five or Five Factor Model, the modern trait consensus (McCrae & Costa 1990).

> **PEARL**
>
> The three domains in one line: social psychology studies the person *between other people*, learning studies the person *shaped by consequences*, and personality studies the person *as a stable structure*. If you can say whether a claim is about the *situation*, the *contingency*, or the *trait*, you have placed the domain and can go looking for the right name.

1.3 The unit of analysis is the fastest discriminator

When an item could belong to two domains, decide by what it treats as the cause. The three domains overlap at the edges, and the paper exploits this. The reliable test is to ask what the explanation makes the cause of the behaviour. If the cause is *someone else* (a majority, an authority, an outgroup, an audience), it is social psychology. If the cause is a *past pairing or consequence* (a bell that once predicted food, a reward that followed an action, a model who was seen to be reinforced), it is learning. If the cause is a *durable internal disposition* that would show up across many situations, it is personality. The domains also hand off historically: behaviour therapy grew out of the learning domain in the 1950s and 1960s, social-cognitive personality theory borrowed observational learning from Bandura, and social psychology and personality meet in the person-situation debate. Knowing the seams is itself examinable (Kaplan & Sadock 2024).

DOMAIN	UNIT OF ANALYSIS (WHAT CAUSES THE BEHAVIOUR)	SIGNATURE PHENOMENA	KEY FIGURES
Social psychology	**Other people (real, imagined or implied)**	Attitudes and dissonance, attribution, conformity and obedience, prejudice, aggression, the crowd	Festinger, Asch, Milgram, Allport
Learning and behaviour	**Past pairings and consequences**	Classical conditioning, operant conditioning, observational learning	Pavlov, Skinner, Bandura
Personality	**A stable internal disposition**	Defences and object relations, traits, types, the striving self, reciprocal person-situation interaction	Freud (the General and Developmental Psychology notes), the Big Five (McCrae & Costa)

The three domains of these notes on one axis: the fastest discriminator is what each treats as the cause of behaviour. Signature phenomena and figures are paid off in the domain topics; here, hold the parallelism.

RULE **Place the item by its unit of analysis, not its content.** If the cause is other people it is social; if it is a past pairing or consequence it is learning; if it is a durable internal disposition it is personality.

HOLD THIS

Social = the person between others; learning = the person shaped by consequences; personality = the person as a stable structure. Name which of the three a stem is pitched at before reaching for any figure.

1.4 The boundary with the General and Developmental Psychology notes and with Psychometry

Know where each thread starts and stops. This chapter deliberately picks up threads the General and Developmental Psychology notes opened, so keep the cross-references clean and do not re-teach. The *founders* of the psychodynamic tradition are the General and Developmental Psychology notes material: Freud's topographic and structural models, Alfred Adler's individual psychology, and Carl Jung's analytical psychology are taught there in full, and the personality topic here uses them only as the psychodynamic tradition's starting point rather than re-deriving them. In the other direction, the personality domain names the Big Five and the defence mechanisms, but the *measurement* of personality (the NEO inventory, the MMPI, projective tests, and the reliability and validity of any scale) belongs to the Psychometry, Assessment and Descriptive Psychopathology notes. Likewise, learning here is the theory of conditioning; the clinical behaviour therapies that apply it (systematic desensitisation, exposure, token economy) are developed with the treatments. Treat this topic as the index page: when a later claim needs placing, flip back here, decide the domain by its unit of analysis, and then follow the cross-reference to where it is finished (Kaplan & Sadock 2024).

COMMON TRAP

Do not let the personality topic pull you back into re-teaching Freud, Adler and Jung; those are the General and Developmental Psychology notes and are only cross-referenced here. Equally, do not answer a personality-*testing* question (a scale's reliability, an inventory's validity) inside these notes; that is Psychometry. The personality topic here is about the *theories* of what a person is, not the *instruments* that measure it.

1.5 How these notes is wired, and the theory-evolution spine

The theory-heavy topics each run as an evolution of theories. Within each domain, the examinable topics that carry competing theories (attitudes and attitude change, attribution, the three learning paradigms, and the personality-theory topic) are best learned the same way the general functions were on the General and Developmental Psychology notes: the theories in the order they appeared, each stating what it fixed from the last and what it left debated, then the currently accepted position, its behavioural or neural correlate where one exists, and a worked example. So attitude change runs from balance and dissonance theories to the modern dual-process (elaboration-likelihood) account; attribution runs from the naive-scientist models to the recognition of the fundamental attribution error and modern social cognition; the learning paradigms run from Pavlov and Thorndike through Skinner to Bandura and the cognitive turn; and personality runs from the psychodynamic school through trait and type approaches to the humanistic and social-cognitive traditions and the Big Five consensus. Hold that spine now; the domain topics pay it off.

MNEMONIC

SLP: See, Learn, Portray

The three domains of these notes, in teaching order: **S**ocial psychology (how others *See* and move me), **L**earning and behaviour (how I *Learn* from consequence and model), **P**ersonality (how theory *Portrays* the stable self). SLP is the master mnemonic for this chapter: every topic here is a See, a Learn, or a Portray. Under each letter hang the lead names, Festinger-Asch-Milgram-Allport under S, Pavlov-Skinner-Bandura under L, and the psychodynamic-trait-type-humanistic-social-cognitive traditions ending in the Big Five under P.

GOOD TO REMEMBER

- **Social psychology (domain):** unit of analysis = the individual in the presence of others; owns attitudes, attribution, conformity and obedience, prejudice, aggression, the crowd.
- **Leon Festinger (1957):** cognitive dissonance and attitude change; the discomfort of holding an action and a belief that conflict, resolved by changing the attitude.
- **Solomon Asch:** conformity to a unanimous majority even when it is plainly wrong.
- **Stanley Milgram:** obedience to authority; ordinary people administer harm when instructed by a legitimate authority.
- **Gordon Allport:** attitudes and the nature of prejudice.
- **Learning and behaviour (domain):** unit of analysis = the lawful change in behaviour produced by experience; three paradigms below.
- **Ivan Pavlov:** classical (Pavlovian) conditioning; a neutral stimulus paired with a significant one comes to evoke the response.
- **B. F. Skinner:** operant (instrumental) conditioning; behaviour is controlled by its consequences, building on Thorndike's law of effect.
- **Albert Bandura:** observational (social) learning; behaviour acquired by watching a model.
- **Personality (domain):** unit of analysis = the stable internal structure that makes a person consistently themselves; five traditions, psychodynamic, trait, type, humanistic, social-cognitive.
- **Big Five / Five Factor Model (McCrae & Costa 1990):** the trait consensus; extraversion, agreeableness, conscientiousness, neuroticism, openness (mnemonic OCEAN).
- **Cross-references (do not re-teach):** psychodynamic founders (Freud topographic and structural, Adler, Jung) are the General and Developmental Psychology notes; the psychological tests and their reliability and validity are the Psychometry, Assessment and Descriptive Psychopathology notes.

Social psychology

2 · Attitudes, attitude change and cognitive dissonance

How the mind takes a stance, and how that stance is moved. An **attitude** is a learned evaluative disposition toward a person, object or idea, and the psychology of **attitude change** is the study of how such evaluations are formed, resist, and shift. The exam wants this as a chain of theories in the order they appeared: a first structural account of what an attitude is (the tripartite ABC model), the discovery that attitudes and behaviour often diverge (LaPiere), then successive theories of change, the **yale** communication approach, Leon **festinger**'s cognitive-dissonance theory, Bem's rival self-perception account, and finally Petty and Cacioppo's dual-route **elaboration likelihood** model of **persuasion**. Each fixed a limitation of the one before it, and the field settled on a dual-process synthesis that is now the **currently accepted** position.

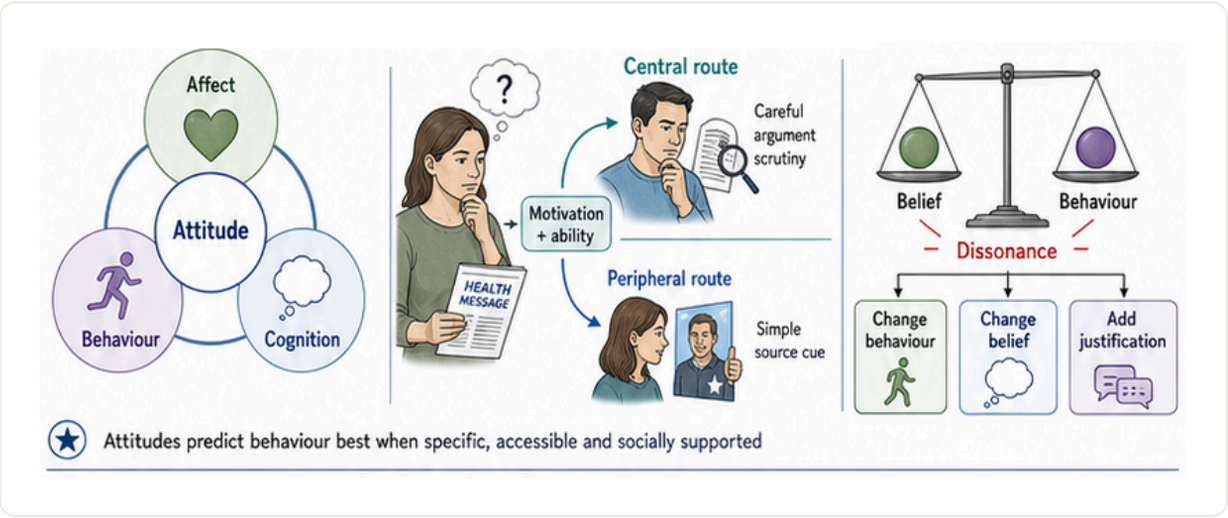


Fig 5.1 Attitudes, persuasion and cognitive dissonance.

This matters for psychiatry beyond social-science trivia. Attitude change is the engine of every psychoeducation programme, adherence intervention and public-health campaign; cognitive dissonance is the explicit mechanism of motivational interviewing and of the dissonance-based eating-disorder prevention programmes now in the guidelines (New Oxford Textbook of Psychiatry). Treat the persuasion structure and the dissonance paradigm as the two highest-yield items; treat the numeric details of the classic experiments as standard textbook figures to reproduce, not to over-claim.

2.1 What an attitude is: the tripartite ABC model

Affect, behaviour, cognition. The classic structural definition holds that an attitude has three components, the **ABC model**: an *affective* component (the feelings an object evokes, "I dislike smoking"), a *behavioural* component (the disposition to act, "I avoid smokers"), and a *cognitive* component (the beliefs held about the object, "smoking causes cancer"). An attitude is thus a relatively enduring, learned evaluation that predisposes a person to respond consistently, favourably or unfavourably, toward a class of objects. It is distinguished from a transient *mood* (no object), a *belief* (the cognitive element alone, without the evaluation), and a *value* (a broader, more abstract guiding principle under which many attitudes sit).

2.2 Attitude formation and function

How evaluations are acquired. Attitudes are learned, chiefly through three routes: *classical conditioning* (an object paired repeatedly with positive or negative affect acquires that valence, the basis of advertising and of the "mere exposure" effect, in which familiarity alone breeds liking); *operant conditioning* (attitudes that are rewarded or socially approved are retained); and *social/observational learning* (attitudes modelled by parents, peers and media are copied, Bandura). A complementary *functional* view (Katz) asks what an attitude does for the person: a *knowledge* function (organising a complex world), an *instrumental/utilitarian* function (maximising reward), an *ego-defensive* function (protecting self-esteem, the route by which prejudice can serve a defensive need), and a *value-expressive* function (signalling identity). The function an attitude serves predicts which persuasive appeal will move it.

2.3 The attitude-behaviour gap: LaPiere

Attitudes predict behaviour only weakly. The foundational problem that every later theory had to reckon with is that expressed attitudes and actual behaviour often diverge. Richard LaPiere's classic field study (LaPiere 1934) is the exam anchor: travelling the United States with a young Chinese couple, he found they were refused service at only one of over 250 hotels and restaurants, yet when he later wrote to the same establishments asking whether they would serve Chinese guests, the large majority said they would not. Attitude (expressed prejudice) and behaviour (actual courteous service) were sharply discordant. Later work (Ajzen and Fishbein; the theory of reasoned action and its successor the theory of planned behaviour) reconciled this: attitudes predict behaviour well only when they are *specific* to the behaviour, *strong*, *accessible*, and when perceived social norms and perceived control are added to the model. The gap is not a refutation of attitudes but a specification of when they bite.

> **COMMON TRAP**
>
> Do not read LaPiere as proving attitudes are irrelevant. The correct lesson is that general attitudes poorly predict specific acts, and that behaviour is co-determined by norms and situational constraint. This is exactly why a health campaign that shifts a general attitude ("smoking is bad") may fail to change a specific behaviour unless it also targets intention, norms and perceived control.

2.4 Theory one: the Yale/Hovland persuasion approach

Who says what to whom, through what channel. The first systematic theory of attitude change came from Carl Hovland and colleagues at Yale after the Second World War (the **yale** communication and attitude-change programme). It treated **persuasion** as a message-learning process and dissected it into four classes of variable, the durable exam checklist:

Source

The communicator. Persuasion rises with source *credibility* (expertise and trustworthiness), *attractiveness* and *similarity*. The *sleeper effect* is the Yale finding that a message from a low-credibility source can gain influence over time as the message dissociates from its discredited source.

Message

Its content and structure: one-sided versus two-sided arguments, the strength and order of arguments, and the calibrated use of *fear appeals* (moderate fear paired with a clear coping action persuades; overwhelming fear provokes denial).

Channel

The medium of delivery (face-to-face, print, broadcast), and its match to message complexity.

Audience

Recipient characteristics: prior attitude, intelligence, self-esteem, involvement, and forewarning (which mobilises resistance).

What it fixed and what it left open. The Yale model fixed the absence of any framework by giving a systematic, empirically testable taxonomy of persuasion variables. What it left unexplained was the *internal* process: it described which inputs move attitudes but not the

psychological mechanism by which a person changes their own mind, and it could not explain why the same variable (say, an attractive source) sometimes mattered and sometimes did not.

2.5 Theory two: Festinger's cognitive dissonance

The discomfort of holding inconsistent cognitions drives change. Leon Festinger (Festinger 1957, *A Theory of Cognitive Dissonance*, Stanford University Press) proposed that when a person holds two psychologically inconsistent cognitions, or acts in a way discrepant from a held belief or from the view of the self, they experience an aversive state of tension, **cognitive dissonance**, that they are motivated to reduce (Tasman Psychiatry). Because behaviour that has already occurred is usually the fixed element, the cheapest route to consonance is often to change the *attitude* to fit the act. Festinger's originating observation was a doomsday cult whose members, after the predicted apocalypse failed to arrive, did not abandon their belief but *intensified* their proselytising, reducing the dissonance between "I gave up everything for this prophecy" and "the prophecy was false" by recruiting others (Festinger 1957; Tasman Psychiatry). Dissonance is now often reframed as a *self* theory: the discomfort is sharpest when the discrepant act threatens the view of oneself as competent and moral (Aronson et al. 2007, cited in Tasman Psychiatry).

2.6 Festinger: the three classic dissonance paradigms

Induced compliance, effort justification, post-decision. Three experimental paradigms recur in exams and each demonstrates a distinct route by which dissonance shifts an attitude.

Induced compliance (the "one dollar / twenty dollar" study)

Festinger and Carlsmith (1959) had subjects perform a dull task and then paid them either a small or a large sum to tell a waiting person the task was enjoyable. Those paid the *small* sum later rated the task as more genuinely enjoyable than those paid the large sum. The small payment was insufficient external justification for the lie, so dissonance ("I said it was fun, but it was boring") was resolved by internally revising the attitude; the large payment supplied ample external justification, so no dissonance and no attitude change followed. Counter-attitudinal behaviour changes the attitude most when external incentive is minimal.

Effort justification

Aronson and Mills (1959): people who underwent a severe, embarrassing initiation to join a dull group later valued the group more highly than those admitted easily. High effort or suffering for a poor outcome creates dissonance that is resolved by inflating the outcome's worth ("I would not have gone through that for nothing"). This underlies the loyalty produced by harsh hazing and demanding training.

Post-decision dissonance

After a difficult choice between two similar options, the rejected option's attractive features and the chosen option's flaws generate dissonance, which is reduced by *spreading of alternatives*: the chosen option is subsequently rated more highly and the rejected one more poorly. This is why people rationalise a purchase or a career choice after committing to it.

■ RULE **In induced compliance, the smaller incentive produces the larger attitude change.** Too little external justification for the act forces the person to justify it internally by revising the attitude.

CLINICAL ANCHOR

Cognitive dissonance is the mechanism named inside motivational interviewing: the clinician selectively elicits and reflects the patient's own *change talk* so that the discrepancy between current behaviour (drinking, non-adherence) and a valued goal (health, family, self-respect) becomes salient, and the resulting dissonance is resolved in the direction of change rather than of defending the status quo. It also explains "effort justification" in therapy itself: a patient who has invested heavily in treatment is motivated to value its gains.

2.7 Theory three: Bem's self-perception, the rival account

Attitude inferred from behaviour, no inner tension required. Daryl Bem (1967) offered a behaviourist rival to dissonance. His **self-perception theory** proposes that people often do not consult a pre-existing internal attitude at all; instead, like an outside observer, they *infer* their attitude from watching their own behaviour and the circumstances in which it occurred ("I ate the whole salad, so I must like salad"). Applied to the Festinger and Carlsmith result, the small-payment subject observes "I said the task was fun for only a dollar, so I must actually have found it somewhat fun," with no aversive arousal needed. **What it fixed and what it left open.** Self-perception fixed dissonance theory's untestable reliance on an unobservable inner "tension." The debate was settled empirically rather than by one theory winning: dissonance operates when behaviour is clearly *counter-attitudinal* and produces measurable physiological arousal, whereas self-perception better describes attitude formation in *novel or weakly held* domains where no prior attitude exists to be contradicted. Both mechanisms are real; they occupy different regions of the same map.

2.8 Theory four: the Elaboration Likelihood Model (central vs peripheral routes)

Two roads to a changed attitude. Richard Petty and John Cacioppo's **elaboration likelihood** model (1986) is the integrative dual-process theory that reconciled the earlier work and remains the framework of choice. It proposes that persuasion travels one of two routes depending on how much a person is *motivated* and *able* to think about (elaborate) the message.

Central route

Engaged when motivation and ability are high (the issue is personally relevant, the person is undistracted and capable). The recipient effortfully scrutinises the *quality of the arguments*. Attitude change by the **central route** is strong, enduring, resistant to counter-persuasion, and predictive of behaviour.

Peripheral route

Engaged when motivation or ability is low (low involvement, distraction, time pressure). The recipient relies on simple *cues* outside the argument's merit, source attractiveness, expert credentials, message length, sheer number of arguments, or the mood of the moment. Attitude change by the **peripheral route** is weaker, more transient, and easily reversed.

FEATURE	CENTRAL ROUTE	PERIPHERAL ROUTE
Trigger	High motivation *and* ability to think	**Low motivation *or* low ability (distraction, low involvement)**
What is processed	Argument quality, effortful scrutiny (elaboration)	**Peripheral cues: source attractiveness/credibility, message length, number of arguments, mood**
Resulting attitude	Strong, enduring, resistant to counter-persuasion, predicts behaviour	**Weak, transient, easily reversed, poor behaviour prediction**
Parallel model	Systematic processing (Chaiken)	Heuristic processing (Chaiken)

The discriminator is the middle two rows: what the recipient actually processes (arguments vs cues) and how durable the resulting attitude is; the trigger is set by motivation and ability.

What it fixed. The ELM explained the previously puzzling inconsistency of the Yale variables: a peripheral cue such as an attractive source persuades only when elaboration is low, whereas under high elaboration argument quality dominates and the cue fades. It absorbed the Yale taxonomy, dissonance and self-perception into a single account of *when* each process operates. Chaiken's parallel heuristic-systematic model makes the same central distinction between systematic (effortful) and heuristic (cue-based) processing.

2.9 Currently accepted position

Dual-process persuasion and constructed, context-sensitive attitudes. The **currently accepted** synthesis is dual-process: attitude change proceeds through a thoughtful, elaboration-heavy central/systematic route or an automatic, cue-driven peripheral/heuristic route, with the balance set by motivation and cognitive capacity (Petty and Cacioppo 1986). Alongside this, contemporary social cognition treats attitudes not as fixed objects retrieved intact from memory but as *constructed* at the moment of judgement and sensitive to context, framing and momentary accessibility. The field also distinguishes *explicit* attitudes (deliberate, self-reported) from *implicit* attitudes (automatic, activated without intention and measured by reaction-time tasks such as the Implicit Association Test), which can diverge from explicit ones and independently predict spontaneous behaviour.

HOLD THIS

The persuasion arc is Yale to Festinger to Bem to the ELM. External input-output (Yale), then internal-motivation dissonance (Festinger), then a behaviourist rival (Bem's self-perception), resolved into the dual-process ELM (central = argument quality, peripheral = cues).

2.10 Behavioural and neural correlate

Where attitudes predict action, and the neurobiology of dissonance. Behaviourally, the attitude-behaviour link is strongest for accessible, specific, strongly held attitudes, and **implicit attitudes** add predictive power for fast, unmonitored behaviour where explicit attitudes fail. The *neural correlate* of dissonance is beginning to be mapped: functional imaging associates the aversive state of holding inconsistent cognitions, and its resolution, with the *dorsal anterior*

cingulate cortex and *anterior insula* (a conflict- and discomfort-detecting network), while the down-regulation of that signal as the attitude is revised engages *dorsolateral prefrontal cortex*; evaluative attitude signals themselves recruit *ventromedial prefrontal cortex* and the amygdala. These localisations are an emerging literature rather than a settled map, so treat them as direction-of-travel, not as fixed exam facts.

2.11 Worked example: a smoking-cessation campaign

The theories applied to one health-behaviour campaign. Take a district anti-smoking campaign and read it through the whole chain. *Attitude structure (ABC):* the target attitude has a cognitive component (beliefs about harm), an affective component (fear, disgust) and a behavioural component (buying, lighting up). *Attitude-behaviour gap:* a poster that shifts the general belief "smoking is harmful" will not, on LaPiere's lesson, change the specific act unless it also targets intention, injunctive norms ("people I respect disapprove") and perceived control (a quit-line, nicotine replacement). *Yale variables:* a credible source (a respected physician), a two-sided message pre-empting the "but my grandfather smoked to ninety" counter-argument, a moderate fear appeal *coupled with* a concrete coping action, delivered through a channel the audience uses. *Dissonance:* rather than lecturing, have smokers publicly argue the case for quitting to younger peers, the dissonance-based, induced-compliance move, so the discrepancy between their advocacy and their own smoking presses toward change; this is exactly the logic of the dissonance-based Body Project used for eating-disorder prevention (New Oxford Textbook of Psychiatry). *ELM routing:* for a highly involved smoker planning to quit, use the central route with strong argument quality for durable change; for a low-involvement teenager, peripheral cues (an admired influencer, vivid imagery) may be the only lever that lands, accepting that such change is more fragile and needs reinforcement.

INDIA

Persuasion variables are culturally weighted. In much of Indian practice, *source* credibility rests heavily on perceived authority and seniority (the treating consultant, the elder, the community leader), and *norms* are family- and community-level rather than individual, so an adherence or public-health message routed through the family and a trusted authority figure often out-performs an individually addressed appeal. Fear appeals on tobacco and alcohol must be paired with a locally reachable coping action, or they default to denial.

MNEMONIC

"ABC of attitudes; Yale asks Who-What-Which-Whom; dissonance = $1 > $20"

ABC: Affective, Behavioural, Cognitive components. **Yale:** Source (who), Message (what), Channel (which medium), Audience (to whom). **Dissonance direction:** the *smaller* incentive ($1) produces the *larger* attitude change, because low external justification forces internal justification. And for the ELM: *Central = think, Peripheral = cues.*

PEARL

The one-line arc for an examiner: the field moved from an external, input-output account of persuasion (Yale) to an internal-motivation account (Festinger's dissonance), met a behaviourist rival (Bem's self-perception), and resolved both into a dual-process model (Petty and Cacioppo's ELM, central versus peripheral) that specifies *when* each earlier mechanism operates. Name that sequence and you have framed the entire topic.

GOOD TO REMEMBER

- **Attitude (ABC model):** a learned evaluative disposition with Affective, Behavioural and Cognitive components.
- **Attitude formation:** classical conditioning, operant conditioning, social/observational learning (Bandura); mere-exposure effect; Katz's functions (knowledge, instrumental, ego-defensive, value-expressive).
- **LaPiere (1934):** Chinese-couple field study; expressed attitude (prejudice) discordant with actual behaviour (service given). The attitude-behaviour gap.
- **Ajzen and Fishbein:** theory of reasoned action / planned behaviour; attitudes predict behaviour via intention, norms and perceived control.
- **Yale / Hovland approach:** persuasion as message-learning; Source, Message, Channel, Audience; sleeper effect; fear-appeal calibration.
- **Festinger (1957):** cognitive dissonance; aversive tension from inconsistent cognitions drives attitude change; doomsday-cult origin; now a self theory (Aronson et al. 2007).
- **Festinger and Carlsmith (1959):** induced compliance, the $1 > $20 study; smaller incentive, larger attitude change.
- **Aronson and Mills (1959):** effort justification; severe initiation raises valuation of the group.
- **Post-decision dissonance:** spreading of alternatives after a hard choice.
- **Bem (1967):** self-perception theory; attitude inferred from own behaviour, no inner tension; rival to dissonance, better for novel/weak attitudes.
- **Petty and Cacioppo (1986):** elaboration likelihood model; central route (argument quality, high elaboration, durable) vs peripheral route (cues, low elaboration, transient); Chaiken's heuristic-systematic parallel.
- **Currently accepted:** dual-process persuasion; attitudes as constructed and context-sensitive; explicit vs implicit attitudes (Implicit Association Test).
- **Neural correlate (emerging):** dissonance signalled by dorsal anterior cingulate cortex and anterior insula; resolution via dorsolateral prefrontal cortex; evaluative attitude signals in ventromedial prefrontal cortex and amygdala.
- **Clinical use:** motivational interviewing exploits dissonance via change talk; dissonance-based prevention (Body Project) for eating disorders (New Oxford).

3 · Attribution theory and social cognition

Two questions, one machinery. **Social cognition** is the umbrella: how we perceive, encode, store, retrieve and use information about people and social situations, running largely on **schemas** (organised knowledge structures for persons, roles and events) and cognitive short-cuts called **heuristics**. **Attribution** is the best-studied slice of it: the process by which people explain the causes of behaviour and events, deciding whether an act sprang from the person (disposition) or from the situation. For the exam this topic is a clean historical spine (Heider to Weiner) that ends in a set of named **attribution biases**, and it is examined both as pure psychology and as the

cognitive substrate of depression (attributional style), paranoia (personalising bias) and prejudice (Kaplan & Sadock 2024; Tasman 2015).

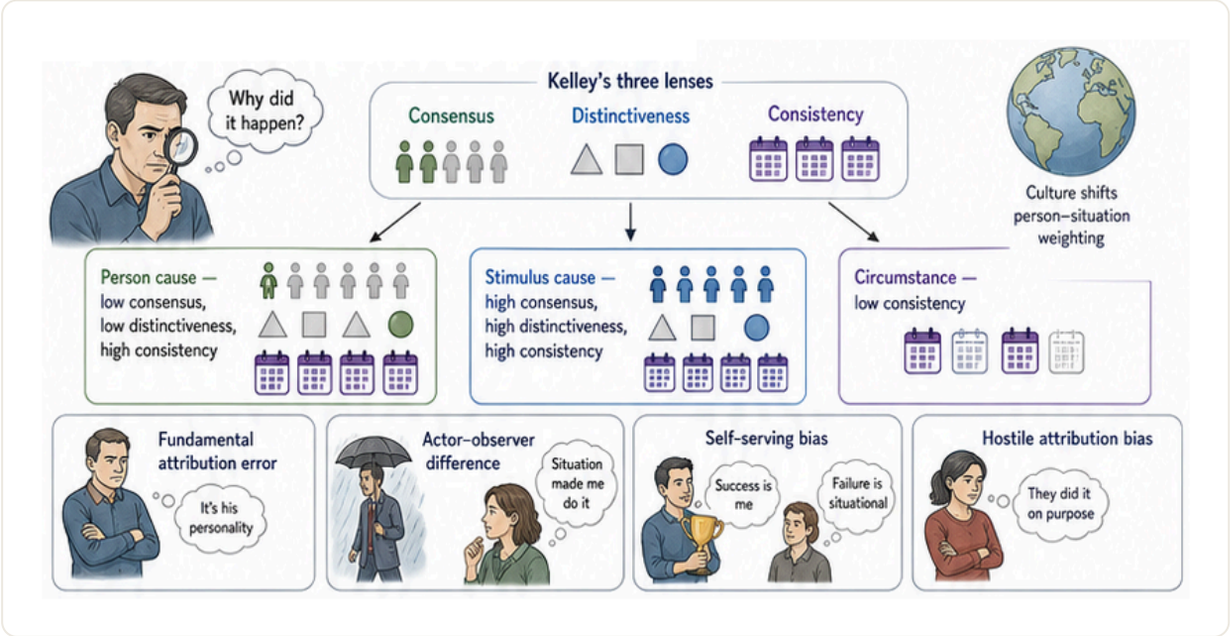


Fig 5.2 Attribution: how we infer causes.

The spine runs in strict date order, each theory fixing a gap left by the last: **Heider (1958)** named the person-versus-situation split; **Jones and Davis (1965)** asked when we may read a disposition off a single act; **Kelley (1967)** asked how we combine information across situations; **Weiner (1974)** re-cut attributions along abstract dimensions for achievement and emotion. The **currently accepted** position is not a further grand theory but the recognition that these normative models are systematically distorted by predictable errors, the attribution biases, whose neural substrate sits in the social-cognition (mentalizing) network. Because this topic names dated theorists, dimensions and hard classifications of bias, it closes with a good-to-remember box enumerating every one (Kaplan & Sadock 2024; Tasman 2015).

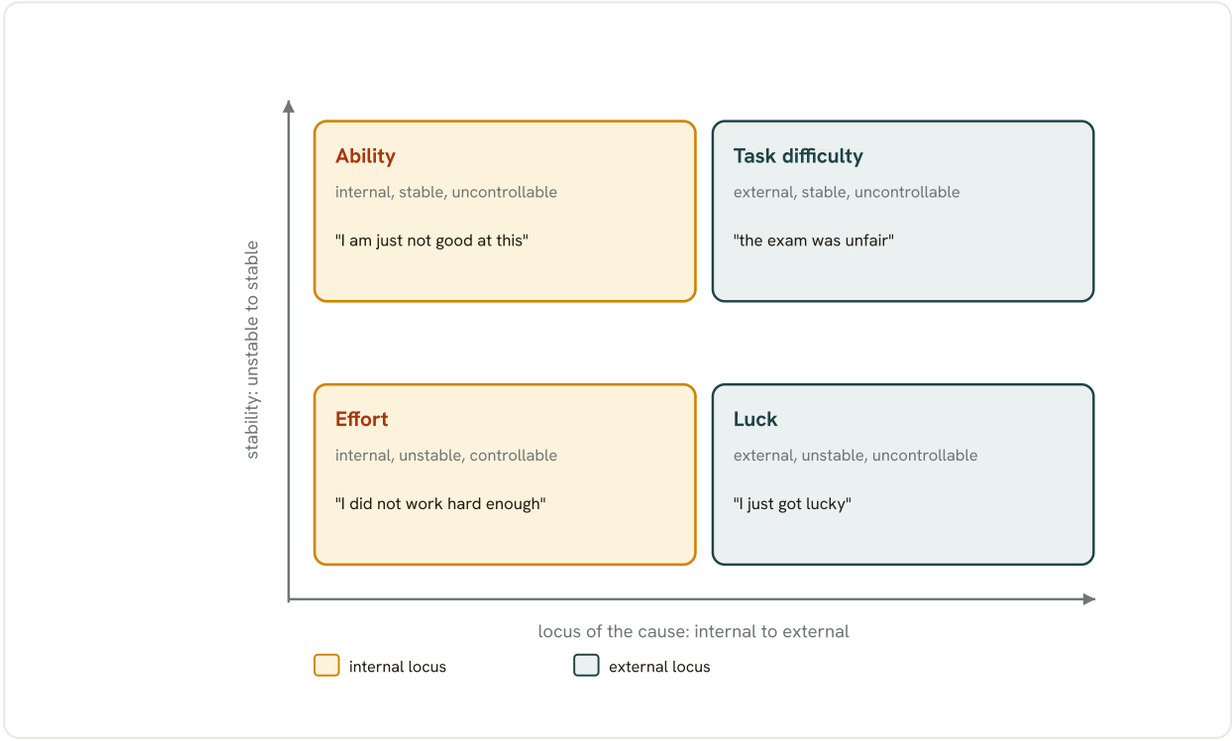


Fig 5.3 Weiner's dimensional model of attribution, shown on two of its axes: whether the cause is inside the person (internal) or outside (external), and whether it is stable or unstable across time. The four classic achievement attributions fall into the cells: ability (internal, stable), effort (internal, unstable), task difficulty (external, stable) and luck (external, unstable). A third axis, controllability, cuts across these: effort is controllable while ability and luck are not, which is why effort attributions carry the most motivational weight. Colour codes the locus: marigold for internal causes, petrol for external.

3.1 Social cognition: the frame the theories live inside

Attribution is one operation within social cognition, so define the frame first. Social cognition is the study of how people make sense of themselves and others: perceiving faces and emotions, inferring intentions and mental states (theory of mind), forming and applying impressions, and choosing behaviour on that basis. It is not a blank-slate computation. Incoming social data is filtered through **schemas**, pre-existing mental templates (a person schema, a role schema, a self-schema, an event schema or *script*) that let us fill gaps and predict, at the cost of stereotyping and confirmation bias. Because full rational analysis of every social act is impossible, the mind leans on **heuristics**, fast rules of thumb (representativeness, availability, anchoring), and on **dual-process** operation: fast, automatic, effortless System 1 judgements versus slow, controlled, effortful System 2 reasoning (Kahneman's framing, the modern default account). Attribution is precisely the schema-and-heuristic-driven machinery we use to answer one recurring social question: *why did that happen?* (Kaplan & Sadock 2024).

3.2 Heider (1958): naive psychology and the person-situation split

The founding move: treat the ordinary person as an intuitive scientist. Fritz Heider's *The Psychology of Interpersonal Relations* (1958) launched attribution theory with the idea of **naive** (or common-sense) **psychology**: everyday people continuously seek the causes of behaviour and do so with an implicit, lay causal theory. Heider's core distinction, which every later model inherits, is between **internal (personal or dispositional)** causes located in the actor (ability, effort, personality, mood) and **external (situational or environmental)** causes located outside the

actor (task difficulty, luck, social pressure, circumstance). Heider observed that perceivers tend to see behaviour as flowing from the person and to under-weight the situation, planting the seed of what would later be called **correspondent inference** and the fundamental attribution error. What he left debated: *when* and *how* the perceiver actually decides between the two, which the next theories set out to specify (Heider 1958; Kaplan & Sadock 2024).

3.3 Jones and Davis (1965): correspondent inference theory

What it fixed: it specified when a single act licenses a dispositional inference. Edward Jones and Keith Davis proposed **correspondent inference** theory: an observer makes a *correspondent* inference when they conclude that a behaviour corresponds to a stable underlying disposition (a hostile act means a hostile person). Their contribution was to list the conditions under which such an inference is warranted. It is stronger when the behaviour is *freely chosen* (not coerced), when it produces **non-common effects** (outcomes unique to that choice and not shared by the alternatives, which narrow down the actor's real motive), and when it is *low in social desirability* (unexpected, norm-violating behaviour is more informative about the person than conventional behaviour, since anyone would do the conventional thing). Hedonic relevance (the act affects the perceiver) and personalism (it seems aimed at the perceiver) further strengthen the dispositional reading. What it left debated: it handled inference from a *single* episode but said little about how we integrate information across *many* occasions, which Kelley then addressed (Jones & Davis 1965; Kaplan & Sadock 2024).

3.4 Kelley (1967): the covariation model and causal schemata

What it fixed: it gave a rule for combining information across situations and time. Harold Kelley's **covariation** model treats the lay perceiver as an intuitive statistician who attributes an effect to the cause with which it *covaries*. Given multiple observations, the perceiver weighs three kinds of information:

Consensus

Do *other people* behave the same way toward this stimulus? High consensus (everyone reacts this way) points *away* from the actor, toward the stimulus or situation.

Distinctiveness

Does this actor behave this way *only* toward this stimulus, or toward many stimuli? High distinctiveness (only this stimulus) points toward the stimulus; low distinctiveness (the actor does it to everything) points toward the person.

Consistency

Does the actor behave this way toward this stimulus *across time and settings*? High consistency is a precondition for a confident attribution of either kind; low consistency drives attribution to transient circumstance.

The classic pattern: high consensus + high distinctiveness + high consistency yields an *external (stimulus)* attribution ("the film really is funny, everyone laughs, he laughs only at it, and every time"); low consensus + low distinctiveness + high consistency yields an *internal (person)* attribution ("only he laughs, he laughs at everything, and always, so it is him"). When information is incomplete (a single observation), Kelley proposed that perceivers fall back on stored **causal**

schemata: pre-formed beliefs about how causes combine, such as the discounting principle (the role of a given cause is discounted if other plausible causes are present) and the augmentation principle (a cause is upgraded when it produces an effect despite an opposing force). What it left debated: covariation describes the ideal normative computation but not the systematic ways real perceivers deviate from it, nor the emotional consequences of attributions, which Weiner took up (Kelley 1967; Kaplan & Sadock 2024).

RULE **An external (stimulus) attribution needs all three Kelley cues high: consensus, distinctiveness and consistency.** Low consensus plus low distinctiveness plus high consistency points instead to the person.

3.5 Weiner (1974 onward): the dimensional model of achievement attributions

What it fixed: it abstracted attributions into dimensions and linked them to emotion, expectancy and motivation. Bernard Weiner reframed the field for *achievement* situations (success and failure at a task). Rather than cataloguing specific causes (ability, effort, task difficulty, luck), he classified any cause along orthogonal dimensions:

Locus (of causality)

Locus internal versus external: is the cause inside the person (ability, effort) or outside (task difficulty, luck)? Governs self-esteem and pride: internal success feels good, internal failure wounds.

Stability

Stability stable versus unstable: is the cause enduring (ability, task difficulty) or variable (effort, mood, luck)? Governs *expectancy* of future outcomes: a failure attributed to a stable cause predicts continued failure and breeds hopelessness; an unstable cause leaves hope.

Controllability

Controllable versus uncontrollable: could the person have altered the cause (effort is controllable; innate ability and luck are not)? Governs social emotions such as guilt, shame, anger, and pity, and others' willingness to help.

Crossing locus and stability gives Weiner's canonical two-by-two: *ability* (internal, stable), *effort* (internal, unstable), *task difficulty* (external, stable), and *luck* (external, unstable). This model is the bridge from attribution to clinical psychiatry: the *reformulated learned-helplessness* theory of depression (Abramson, Seligman and Teasdale 1978) holds that a depressogenic **attributional style** explains bad events by internal, stable, global causes ("it is me, it always will be, it ruins everything"), directly borrowing Weiner's locus and stability axes (Weiner 1985; Tasman 2015; Kaplan & Sadock 2024). What it left debated: like its predecessors it is a *normative* account of how attributions should be made and what they cause, which set up the corrective era of documented biases.

3.6 The currently accepted position: attribution is systematically biased

The modern consensus is not a new grand theory but a catalogue of predictable errors. The **currently accepted** view is that the elegant normative models above (correspondent inference, covariation, dimensional analysis) describe an ideal the mind rarely meets: real attribution is fast,

motivated and schema-driven, and it deviates in reliable directions. These are the **attribution biases**, and they are the highest-yield part of the topic.

GOOD TO REMEMBER

- **Fundamental attribution error (FAE):** the pervasive tendency, when explaining *others'* behaviour, to over-attribute it to disposition and under-weight the situation (also called correspondence bias; Ross 1977; Gilbert & Malone 1995).
- **Actor-observer asymmetry:** we explain *our own* behaviour situationally ("I snapped because the day was awful") but *others'* behaviour dispositionally ("he snapped because he is aggressive"); driven partly by differing perceptual focus and by richer self-knowledge (Jones & Nisbett 1972).
- **Self-serving bias:** we attribute our *successes* to internal, dispositional causes (ability, effort) and our *failures* to external causes (bad luck, unfair task), protecting self-esteem; its reversal (internal for failure, external for success) marks the depressive attributional style.
- **Just-world bias / hypothesis:** the belief that the world is fair, so people get what they deserve; leads to victim-blaming (Lerner).
- **False-consensus bias:** we over-estimate how far others share our own attitudes, choices and behaviour, treating our own view as the norm (Ross).

BIAS	WHOSE BEHAVIOUR	DIRECTION OF THE ERROR	FUNCTION OR DRIVER
Fundamental attribution error	Other person	**Over-weights disposition, under-weights situation**	Actor is the perceptual figure; automatic dispositional inference
Actor-observer asymmetry	Self vs other	**Self = situational, other = dispositional**	Different visual focus; more knowledge of one's own constraints
Self-serving bias	Self	**Success = internal, failure = external**	Self-esteem protection (motivational)
Just-world bias	Victim / other	**Blames the victim for their misfortune**	Need to believe outcomes are deserved and the world controllable
False-consensus bias	Self projected onto others	**Over-estimates how many share one's view**	Own position is cognitively available as the anchor

The five exam-standard attribution biases: what target they distort, the direction of the distortion, and why it happens (Kaplan & Sadock 2024; Tasman 2015).

HOLD THIS

The spine is Heider to Jones-Davis to Kelley to Weiner, and the modern answer is not a fifth theory but the biases. The reversed self-serving bias (failure read as internal, stable and global) is the depressive signature.

COMMON TRAP

Do not merge the **actor-observer** asymmetry with the **self-serving** bias. Actor-observer is about the *self-versus-other* contrast regardless of outcome (I am situational, you are dispositional). Self-serving is about the *success-versus-failure* contrast within the self (my wins are me, my losses are the world). They can be dissociated: the depressive shows a *reversed* self-serving bias (failure attributed internally, stably and globally) while the actor-observer pattern may persist. The **fundamental attribution error** is specifically about explaining *others*; it is not a self-versus-other contrast on its own.

3.7 Neural correlate: the social-cognition (mentalizing) network

Attribution has a definable neurobiology within the wider social brain. The **neural correlate** of attributing mental states and causes to others is the *mentalizing* or theory-of-mind network, whose reliably implicated nodes are the *medial prefrontal cortex*, the bilateral *temporo-parietal junction* (especially the right TPJ, engaged when representing another's beliefs and intentions), the *posterior superior temporal sulcus* (reading biological motion and gaze), the *precuneus and posterior cingulate*, and the *temporal poles* (social-semantic knowledge). The *amygdala* and *fusiform face area* feed the rapid, affective appraisal of faces and threat that biases attribution before deliberate reasoning engages. In psychiatry these systems are the substrate of the **social cognition** deficits catalogued in schizophrenia (impaired theory of mind, a *personalising* attributional bias that over-attributes negative events to other people rather than to circumstances, and jumping-to-conclusions), and they are the explicit target of Social Cognition and Interaction Training (SCIT) (Roberts, Penn & Combs 2016; Kaplan & Sadock 2024).

3.8 Culture: attribution is not universal

The fundamental attribution error is partly a Western phenomenon. A major qualification to the whole tradition is cross-cultural: people from *individualist* (typically Western) cultures, which foreground the autonomous person, show a stronger dispositional bias and a more robust fundamental attribution error, whereas people from *collectivist* (many East Asian and South Asian) cultures, which foreground the group, relationships and context, attribute behaviour more readily to *situational* and relational causes and show a weaker FAE. The self-serving bias is likewise attenuated, and sometimes a *self-effacing* pattern appears, in collectivist settings where modesty is normative. The lesson for the exam and for Indian practice is that the "fundamental" error is not a fixed feature of human cognition but is shaped by culturally transmitted models of the person (Nisbett and colleagues; Kaplan & Sadock 2024).

INDIA

India is a broadly collectivist, family-embedded context, and this reshapes attribution in the clinic. Distress is often explained through situational, relational and supernatural or karmic frames (family conflict, evil eye, planetary influence, fate) rather than through internal, dispositional or biomedical ones. A clinician who reads a patient's situational explanation as denial, or who imposes a Western dispositional-illness model, will mis-attune. It also predicts a softer fundamental attribution error in how families explain a relative's behaviour: an act is more likely read as caused by circumstances and relationships than by fixed character, which can protect the patient from blame but can also delay recognition of illness.

3.9 Worked example: reading one late arrival five ways

Run one everyday event through the whole apparatus. A colleague, Ravi, arrives thirty minutes late to a meeting you are chairing.

Heider

You immediately sort the cause into internal (Ravi is careless, disorganised) or external (the traffic, a prior emergency). The pull toward the internal reading is Heider's seeded observation.

Jones and Davis

If Ravi *freely* chose to leave home late and lateness is socially undesirable, you make a correspondent inference: he is an unreliable person. If he was ordered to finish another task first (coerced, low choice), the inference weakens.

Kelley (covariation)

You consult three streams. Consensus: do others also arrive late to *your* meetings (high consensus points to your meeting or its timing, an external cause)? Distinctiveness: is Ravi late only to this meeting or to everything (low distinctiveness points to Ravi)? Consistency: is he late to this meeting every week (high consistency licenses a firm attribution)? Low consensus + low distinctiveness + high consistency yields "it is Ravi."

Weiner

If you frame lateness as a performance failure, you locate its cause by **locus** and **stability**: "lazy" (internal, stable) predicts he will always be late and invites anger; "stuck in unusual traffic" (external, unstable) predicts nothing and invites sympathy. Controllability decides whether you feel anger (he could have left earlier) or pity (he could not).

The biases (currently accepted)

The **fundamental attribution error** makes you leap to "Ravi is unreliable" and ignore the flooded road. The **actor-observer** asymmetry means that when *you* were late last week you blamed the traffic, but when Ravi is late you blame Ravi. Had it been *your* lateness excused by traffic and *your* punctuality credited to your discipline, that is the **self-serving** bias. If you assume most people would judge Ravi as harshly as you do, that is false-consensus; if you conclude he "deserves" the reputational cost, that edges toward just-world thinking. In a collectivist frame, a colleague may instead default to "something must have happened at home," a situational reading and a softer FAE.

PEARL

If four things survive into the exam: (1) the *date-ordered spine* Heider (person vs situation) to Jones and Davis (correspondent inference from one act) to Kelley (covariation: consensus, distinctiveness, consistency) to Weiner (locus, stability, controllability); (2) Kelley's rule that *external* attribution needs high consensus + high distinctiveness + high consistency; (3) the five biases, with the *fundamental attribution error* as the flagship and the reversed self-serving bias as the depressive signature; (4) that the FAE is culturally softened in collectivist settings.

MNEMONIC

Kelley = CDC (Consensus, Distinctiveness, Consistency)

The three covariation cues, read like the disease-control acronym so it sticks. Pair it with the external-attribution rule "**all three high** (consensus + distinctiveness + consistency) points **out** to the stimulus." For Weiner, remember the two-by-two by its four cells: **A**bility, **E**ffort, **T**ask difficulty, **L**uck ("AETL"), read as internal-stable, internal-unstable, external-stable, external-unstable.

GOOD TO REMEMBER

- **Social cognition:** how we perceive, store and use social information; runs on schemas, heuristics and dual-process (System 1 automatic / System 2 controlled) cognition.
- **Attribution:** the sub-process of explaining the causes of behaviour, internal (dispositional) versus external (situational).
- **Heider (1958):** naive (common-sense) psychology; person-versus-situation split; seeded correspondent inference and the FAE.
- **Jones and Davis (1965):** correspondent inference theory; a dispositional inference is warranted when behaviour is freely chosen, has non-common effects, and is low in social desirability.
- **Kelley (1967):** covariation model; three cues = consensus, distinctiveness, consistency; external attribution needs all three high; causal schemata with discounting and augmentation principles for incomplete data.
- **Weiner (1974 onward):** dimensional model of achievement attribution; locus (internal/external), stability (stable/unstable), controllability; four cells = ability, effort, task difficulty, luck; locus drives self-esteem, stability drives expectancy, controllability drives social emotion.
- **Attributional style (Abramson, Seligman, Teasdale 1978):** depressogenic style attributes bad events to internal, stable, global causes (reformulated learned helplessness).
- **Fundamental attribution error (Ross 1977):** over-attributing others' behaviour to disposition; also called correspondence bias (Gilbert & Malone 1995).
- **Actor-observer asymmetry (Jones & Nisbett 1972):** self explained situationally, others dispositionally.
- **Self-serving bias:** success internal, failure external (self-esteem protection); reversed in depression.
- **Just-world bias (Lerner):** belief the world is fair, so victims must deserve their fate; drives victim-blaming.
- **False-consensus bias (Ross):** over-estimating how far others share one's own views.
- **Currently accepted:** the normative models are systematically distorted by these biases; attribution is fast, motivated and schema-driven.
- **Neural correlate:** mentalizing (theory-of-mind) network = medial prefrontal cortex, temporo-parietal junction (esp. right TPJ), posterior superior temporal sulcus, precuneus/posterior cingulate, temporal poles; amygdala and fusiform face area for rapid affective appraisal.
- **Clinical link:** personalising attributional bias and theory-of-mind deficits in schizophrenia; target of SCIT.
- **Culture:** individualist cultures show a stronger FAE and self-serving bias; collectivist cultures attribute more situationally and show a weaker FAE (Nisbett).

4 · Group dynamics: conformity, obedience and group processes

Why this matters. Social psychology studies the **typical** forces that govern how the social world shapes behaviour, where clinical psychiatry studies the atypical (Tasman 2015). Its central lesson, running from Sherif through Milgram, is that much of what looks like character is actually **situational**: ordinary people conform, obey and diffuse responsibility when the situation pulls for it. This is the counterweight to the **fundamental attribution error** (over-crediting internal traits), and it is why a psychiatrist reads a patient's behaviour against its social field, not only against personality.

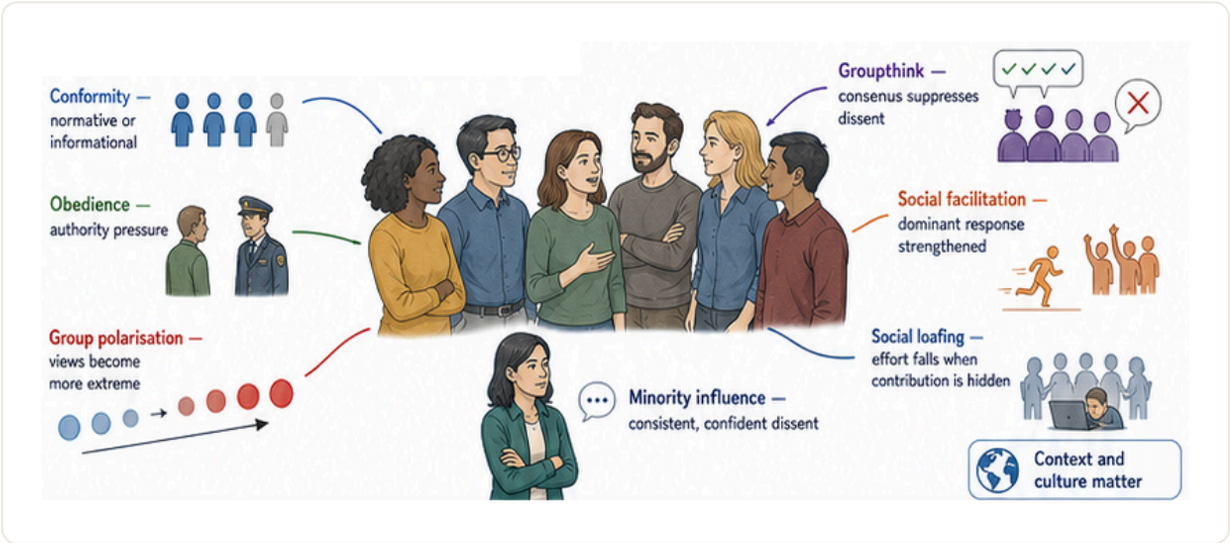


Fig 5.4 Social influence in groups.

The topic is a reliable exam staple: examiners want the **named experiment** matched to the **named mechanism** (Asch to normative influence, Milgram to the agentic state, Janis to groupthink, Zajonc to social facilitation). Build the answer as a spine of classic studies in the order they appeared, then close with the currently accepted situationist synthesis and its neural correlate.

4.1 What a group is: norms, roles, cohesion

A **group** is two or more people who interact, are interdependent, share goals and perceive themselves as a unit (a distinction from a mere aggregate, such as a bus queue). Three structural properties organise it and generate all the phenomena below.

Norms

Shared, often implicit rules about how members should behave and think; the reference point people default to when a situation is ambiguous.

Roles

Expected patterns of behaviour attached to a position in the group (leader, scapegoat, guard, prisoner); powerful enough to override individual disposition.

Cohesion

The sum of forces binding members to the group and to one another. Moderate cohesion aids function; excessive cohesion breeds loss of individuality, seen as fusion, contagion and groupthink (Tasman 2015).

Status and communication structure

The hierarchy of prestige and the channels through which information flows; both shape who conforms to whom.

4.2 Conformity, era 1: Sherif and the birth of a norm

Sherif, autokinetic effect (1935). A stationary pinpoint of light in a dark room appears to move (an **autokinetic** illusion). Asked individually how far it moved, people gave idiosyncratic estimates; placed in groups, their estimates **converged on a shared group norm** that then persisted when they were tested alone again. Sherif showed that in a genuinely ambiguous

situation a norm is **internalised**, not merely complied with. What it **fixed**: it demonstrated norm *formation* experimentally. What it **left debated**: because the stimulus was ambiguous, it could not tell us whether people would conform against the plain evidence of their own eyes. That question is Asch's.

4.3 Conformity, era 2: Asch and the line-judgement studies

Asch, line-judgement paradigm (1950s). A naive participant judged which of three comparison lines matched a standard, a task with an **unambiguous** correct answer, while a group of confederates unanimously gave the wrong answer on set trials. Roughly a third of naive responses went along with the majority, and about three-quarters of participants conformed at least once, while a control group erred almost never. Asch proved that people will publicly deny plain perceptual evidence under group pressure. Two mechanisms were later separated (Deutsch and Gerard 1955).

Normative influence

Conforming to be liked or accepted and to avoid rejection; produces public compliance without private belief change. Dominant in Asch, where the answer was obvious.

Informational influence

Conforming because others are treated as a source of valid information; produces private acceptance. Dominant in Sherif, where the answer was ambiguous.

Factors that raise conformity: unanimity of the majority; larger majority size up to about three or four, after which it plateaus; higher group cohesion and attractiveness; task difficulty or ambiguity; public rather than private responding; lower individual status or expertise. **Factors that lower it:** a single dissenting ally who breaks unanimity (the strongest single reducer); private or anonymous responding; higher personal competence on the task; and cultural individualism relative to collectivism.

~1/3	~75%	3-4
OF RESPONSES CONFORMED (ASCH)	CONFORMED AT LEAST ONCE	MAJORITY SIZE WHERE EFFECT PLATEAUS

RULE **A single dissenting ally who breaks unanimity is the strongest single reducer of conformity.** It is unanimity, not sheer numbers, that does most of the pressing.

COMMON TRAP

Sherif is *not* the same as Asch. Sherif used an **ambiguous** stimulus and demonstrated **informational** influence with genuine norm internalisation; Asch used an **unambiguous** stimulus and demonstrated **normative** influence with mere public compliance. Examiners deliberately swap the two.

4.4 Obedience: Milgram and the shock experiments

Milgram, obedience to authority (1963 onward; Milgram 1974). Prompted by the trial of Adolf Eichmann and Arendt's thesis that the Holocaust's perpetrators were ordinary bureaucrats rather than sadistic monsters (Tasman 2015), Milgram tested how far ordinary people would go

under instruction. A participant, cast as "teacher," was ordered by an experimenter to deliver escalating "electric shocks" to a "learner" (a confederate) for wrong answers. A striking majority continued to the maximum voltage when told to do so by the authority figure, and none of the minority who refused insisted the study be stopped or checked on the victim (Tasman 2015). Obedience is **compliance with a direct order from a perceived authority**, and is distinct from conformity to peers.

Milgram's two explanations (Tasman 2015): first, *conformism* from a lack of a reference point about how to behave in an unfamiliar situation; second, and central, **agentic state** theory. In the agentic state the obedient person redefines himself as an **instrument carrying out another's wishes** and feels minimal personal responsibility for the outcome. A contemporary parallel is **moral disengagement**, documented in prison guards assigned to execution teams (Osofsky, Bandura and Zimbardo 2005).

Situational factors that changed obedience rates: *proximity of the victim* (obedience fell as the learner became more physically present, and fell sharply when the teacher had to press the learner's hand onto a plate); *proximity and presence of the authority* (obedience dropped when the experimenter gave orders by telephone rather than in the room); *legitimacy of the authority* (a prestigious institutional setting raised obedience; an ordinary setting or an ordinary-man "experimenter" lowered it); and the presence of *disobedient peers*, whose defiance freed the participant to refuse. The lineage runs back to the **authoritarian personality** construct (Adorno et al. 1950), which had tried, less successfully, to locate obedience in a trait rather than the situation.

4.5 Roles, deindividuation and the Stanford prison experiment

Zimbardo, Stanford prison experiment (1971). Screened, ordinary young men were randomly assigned to be "guards" or "prisoners" in a simulated prison. Guards rapidly became abusive and prisoners passive and distressed, and the study, planned for two weeks, was halted early. Zimbardo read it as evidence of the **power of situations and assigned roles** to transform behaviour, mediated by **deindividuation**: a loss of individual self-awareness and self-restraint under anonymity, uniforms and group immersion, which lowers accountability and releases behaviour the person would not otherwise show. The same construct underlies the aggression of anonymous crowds.

COMMON TRAP

Modern critique, stated soberly: the Stanford study is now read cautiously. It had **no control condition**, a tiny non-representative sample, and, on later evidence, the experimenter and guards were **coached and led** toward the cruel behaviour rather than it emerging spontaneously; some guard conduct appears to have been demand characteristics and performance. Cite it as a *demonstration and a cautionary tale about roles and situational power*, not as clean experimental proof. Milgram, with its manipulated variables and control comparisons, is the sturdier evidence of situational power.

4.6 Group decision processes: polarisation and groupthink

Groups do not simply average their members' views; they systematically shift them.

Group polarisation

After discussion, a group's decision becomes **more extreme in the direction the members already leaned** (the older "risky shift" is one instance, but caution can be amplified too). Driven by informational influence (hearing more arguments on the favoured side) and normative influence (aligning with the group's valued position).

Groupthink (Janis 1972)

A mode of thinking in **highly cohesive, insulated groups under directive leadership and stress**, where the drive for unanimity overrides realistic appraisal of alternatives, degrading decision quality. Janis drew it from foreign-policy fiascos (the Bay of Pigs is the classic case).

Janis's symptoms of groupthink cluster into three groups: *overestimation of the group* (an illusion of invulnerability; belief in the group's inherent morality); *closed-mindedness* (collective rationalisation; stereotyping of outgroups and opponents); and *pressure toward uniformity* (self-censorship of doubts; a shared illusion of unanimity; direct pressure on dissenters; and self-appointed **mindguards** who shield the group from discordant information). Antidotes: a designated devil's advocate, leaders withholding their own view early, and inviting outside critique.

4.7 Presence of others: social facilitation and social loafing

Social facilitation (Zajonc 1965). The mere presence of others (as audience or co-actors) raises **arousal**, which strengthens the **dominant response**. The consequence depends on the task: on *easy or well-learned* tasks the dominant response is correct, so performance **improves**; on *difficult or novel* tasks the dominant response is often wrong, so performance **worsens**. This drive-arousal account resolved a century of contradictory audience findings into one rule.

Social loafing. When individual contributions are **pooled and cannot be identified**, people exert less effort than when working alone (the effect first noted in group rope-pulling). It is the near-opposite of facilitation, driven by the same diffusion logic as the bystander effect: identifiability, evaluation and personal accountability all reduce it.

4.8 Minority influence: how the few change the many

Moscovici (1969 onward). Against the majority-centred tradition above, Moscovici showed that a **consistent, confident minority** can convert the majority. The critical variable is **behavioural consistency** over time and across members; a wavering minority has no effect. Minority influence tends to produce **conversion** (genuine, often private and delayed attitude change through informational processing) rather than the surface **compliance** that majorities extract. This is the mechanism behind social innovation and the slow success of dissenting movements.

4.9 Currently accepted position and its neural correlate

The **currently accepted** synthesis is **situationism** tempered by interactionism: behaviour is a joint product of person and situation, but classic social psychology corrected a durable bias by showing how strongly the situation pulls, and how routinely observers underweight it (the

fundamental attribution error). Modern work reframes conformity within **social-identity** and self-categorisation theory (people conform to norms of groups they identify with, not to strangers indiscriminately) and within dual-process models separating fast normative alignment from slower informational persuasion.

HOLD THIS

Match each named study to its named mechanism: Asch to normative influence, Milgram to the agentic state, Zimbardo to deindividuation, Janis to groupthink, Zajonc to social facilitation, Moscovici to minority conversion. Much that looks like character is situation.

Neural correlate. Conformity has a measurable **neurobiology**. When a person's judgement conflicts with the group, activity in the **posterior medial frontal cortex / dorsal anterior cingulate** and **anterior insula** tracks the "social prediction error," the felt cost of deviating; the size of this signal predicts subsequent conforming. Reward circuitry, the **ventral striatum and ventromedial prefrontal cortex**, updates value toward the group's position when one aligns, giving conformity the signature of reinforcement learning. Obedience and moral disengagement engage reduced **medial prefrontal** self-agency signalling, consistent with the agentic state (the sense of acting as another's instrument). These are localisation claims to verify against a current source before quoting in a viva.

4.10 Worked example and the clinical read

Everyday. A junior doctor on a ward round privately doubts a senior's plan but stays silent (Asch normative pressure and Milgram's legitimate authority), the team's shared confidence hardens into an illusion of unanimity (groupthink), and no one calls the near-miss because "someone senior will have checked" (diffusion of responsibility). The fix is structural: name a devil's advocate, invite the most junior view first, and make each check individually accountable.

CLINICAL ANCHOR

Group phenomena are the working material of **group psychotherapy**: cohesion is a curative factor (Kaplan and Sadock 2024), but the same cohesion can tip into pretend-mode groupthink where patients answer for one another and stop mentalising (Bateman and Fonagy). Ward culture, family systems and treatment teams all show conformity, role capture and diffusion, and the clinician's job is often to restore individual accountability and dissent.

INDIA

In hierarchical, collectivist and family-centred settings, normative conformity and deference to authority (senior clinician, elders, the joint family) run high, which raises both helpful cohesion and the risk of unspoken dissent in consent and treatment decisions. Explicitly soliciting the quietest voice and separating family consensus from the patient's own view is a practical counter to culturally amplified groupthink.

MNEMONIC

SAME ZOO: Groups Just Zap Loafers, Minorities Convert

Sherif (norm formation), **A**sch (conformity), **M**ilgram (obedience), **E**ffect-bystander (Latané and Darley), **Z**imbardo (roles), **ZOO** reminds you these are the caged classics. Then the processes: **G**roup polarisation, **J**anis (groupthink), **Z**ajonc (social facilitation), **L**oafing, **M**oscovici (minority conversion).

GOOD TO REMEMBER

- **Group:** interacting, interdependent, shared-goal unit; structured by norms, roles, cohesion, status.
- **Sherif (1935), autokinetic effect:** ambiguous stimulus, group norm forms and is internalised; informational influence.
- **Asch (1950s), line judgement:** unambiguous stimulus, about a third of responses conform, roughly three-quarters conform at least once; normative influence.
- **Normative vs informational influence (Deutsch and Gerard 1955):** to be accepted (public compliance) vs to be correct (private acceptance).
- **Conformity raisers:** unanimity, majority size to about three, cohesion, ambiguity, public response; single ally is the strongest reducer.
- **Milgram (1974), shock experiments:** majority obey an authority to maximum shock; obedience is compliance with an order, not peer conformity.
- **Agentic state:** obedient person feels an instrument of another's will, minimal own responsibility.
- **Milgram situational factors:** victim proximity, authority proximity and legitimacy, and disobedient peers all move obedience.
- **Authoritarian personality (Adorno et al. 1950):** earlier trait account of blind acceptance of authority, superseded by the situationist reading.
- **Zimbardo (1971), Stanford prison:** roles and situational power; deindividuation; modern critique = no control, coached guards, demand characteristics.
- **Deindividuation:** loss of self-awareness and restraint under anonymity, releasing disinhibited behaviour.
- **Group polarisation:** discussion pushes the group further in its pre-existing direction (risky shift is one case).
- **Groupthink (Janis 1972):** cohesive, insulated, stressed group sacrifices appraisal for unanimity; symptoms = invulnerability, rationalisation, out-group stereotyping, self-censorship, illusion of unanimity, pressure on dissenters, mindguards.
- **Social facilitation (Zajonc 1965):** mere presence raises arousal, boosts easy tasks, impairs hard ones (dominant-response rule).
- **Social loafing:** reduced effort when contributions are pooled and unidentifiable; countered by accountability.
- **Minority influence (Moscovici 1969):** a consistent minority produces conversion (deep, private change), not mere compliance.
- **Bystander effect and diffusion of responsibility (Latané and Darley 1970):** less helping when others are present; belongs with prosocial and aggression behaviour (see the aggression and prosocial behaviour topic), cross-referenced here.
- **Situationism (currently accepted):** behaviour = person plus situation; fundamental attribution error over-weights traits; neural correlate in dorsal anterior cingulate, anterior insula and ventral striatum for conformity.

5 · Prejudice, stereotypes and intergroup relations

Why this sits on the exam spine. Intergroup psychology is the social-psychology chapter that maps directly onto the clinician's daily reality: the **stigma** of mental illness is, mechanically, prejudice applied to a psychiatric out-group. Examiners want the clean triad (thought, feeling, action), the named theories in the order they emerged, and the one intervention with the strongest evidence base behind it (the contact hypothesis). Answer it as an evolution of theories, each fixing the previous one's blind spot, and land it on the stigma application.

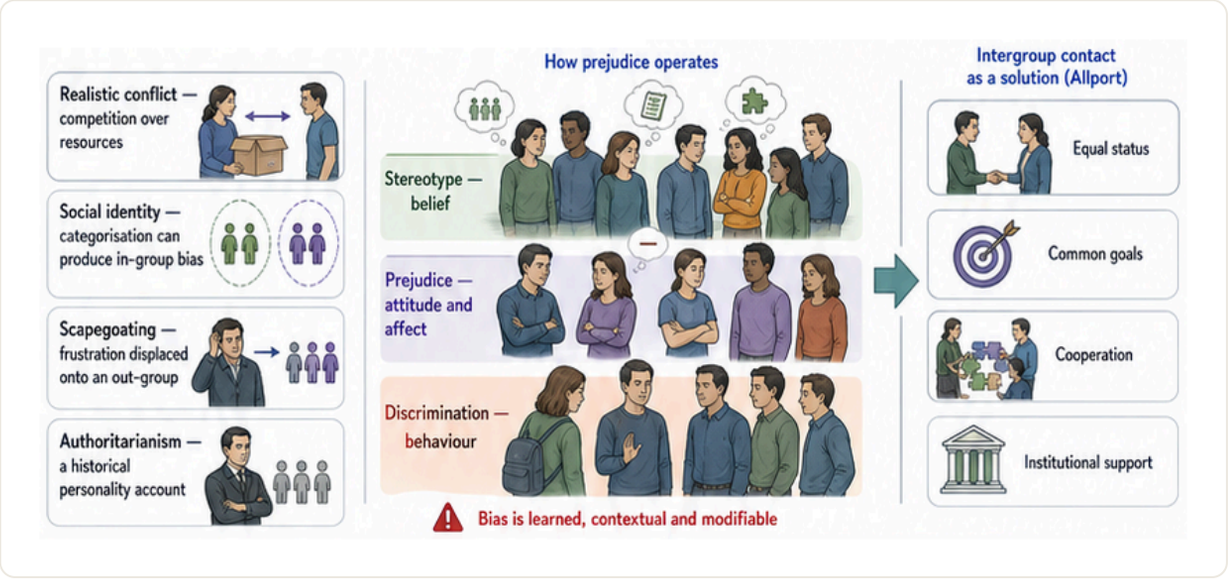


Fig 5.5 Prejudice and intergroup relations.

The organising move is to separate three things that lay usage blurs. A **stereotype** is a belief, a **prejudice** is a feeling or attitude, and **discrimination** is a behaviour. They correlate but dissociate: a clinician can hold an accurate-seeming stereotype without hostile affect, or feel prejudice yet not discriminate when institutional rules forbid it. Keeping the three levels distinct is what turns a vague answer into a scorable one (Kaplan & Sadock 2024).

5.1 The triad: stereotype, prejudice, discrimination

Three levels, one target group. The cognitive, affective and behavioural components of an intergroup attitude. Allport (1954) framed prejudice as "an antipathy based on a faulty and inflexible generalisation," which already fuses the belief (generalisation) with the feeling (antipathy).

Stereotype (cognitive)

An over-generalised, relatively fixed belief about the attributes of a social group, applied to its members regardless of individual variation. A cognitive schema, and therefore cognitively economical: it lets the perceiver "go beyond the information given." Can be positive or negative in content but is error-prone by construction.

Prejudice (affective attitude)

A pre-judgement, a negative (occasionally positive) attitude and emotional orientation toward a group and its members, held in advance of, and resistant to, disconfirming evidence. The evaluative, feeling-toned layer sitting on the stereotype.

Discrimination (behaviour)

The overt differential treatment (usually unfavourable) of individuals on the basis of their group membership. The behavioural output; the level at which law and institutions intervene.

COMPONENT	PSYCHOLOGICAL LEVEL	EVERYDAY MARKER
Stereotype	**Cognition (belief / schema)**	"People with schizophrenia are unpredictable"
Prejudice	**Affect (attitude / feeling)**	Discomfort, fear or dislike toward that group
Discrimination	**Behaviour (action)**	Refusing to rent a room to a psychiatric patient

The cognition to affect to behaviour spine: stereotype feeds prejudice, prejudice licenses discrimination, but the links are probabilistic, not automatic.

HOLD THIS

Stereotype is a belief (cognition), prejudice is a feeling (affect), discrimination is an act (behaviour). Keeping the three levels apart is what turns a vague answer into a scorable one; the stigma of mental illness is this triad applied to a psychiatric out-group.

5.2 Allport's classic account (1954): the foundational text

Where the field starts. Gordon Allport's *The Nature of Prejudice* (1954) is the origin point examiners expect you to name. He defined prejudice as antipathy from faulty, inflexible generalisation, located its cognitive engine in normal **categorisation** (the mind sorts the social world into groups to save effort, and prejudice is the pathological overreach of this normal process), and proposed a scale of acting-out from mild to lethal: antilocution (hostile talk), avoidance, discrimination, physical attack, and extermination. His lasting bequest was two-fold: prejudice grows out of ordinary cognition rather than a special pathology, and it can be reduced by structured intergroup contact (the **contact hypothesis**, 5.7). *What it left debated:* Allport described the individual and cognitive roots richly but did not fully explain why prejudice tracks group competition and group identity, which the next theories addressed.

5.3 The authoritarian personality (Adorno, 1950): prejudice as personality

Locating prejudice inside the person. Adorno and colleagues (1950), working in the shadow of fascism and from a psychodynamic frame, argued that prejudice is an expression of a personality type forged by harsh, status-anxious parenting. The **authoritarian** personality shows submission to authority, aggression displaced onto weaker out-groups, rigid conventional thinking and intolerance of ambiguity, indexed by the F-scale (F for fascism). *What it fixed:* it explained the individual differences Allport's cognitive account under-weighted, why some people are far more prejudiced than others in the same culture. *What it left debated:* a personality-only account cannot explain how whole populations shift into or out of prejudice rapidly, or why prejudice appears even among people with no authoritarian traits, which pushed the field toward situational and group-level theories.

5.4 Realistic conflict theory (Sherif): prejudice from competition

Prejudice as rational group interest. Muzafer Sherif's **realistic conflict** theory holds that intergroup hostility arises when groups compete for scarce, mutually exclusive resources; cooperation for shared goals dissolves it. The evidence is the **Robbers Cave** field experiment (1954): two groups of boys at a summer camp, once placed in competition, rapidly developed **in-group** solidarity and **out-group** hostility (name-calling, raids, flag-burning). Crucially, simple contact did not repair it; hostility only fell when the experimenters imposed **superordinate goals**, problems (a broken water supply, a stuck truck) that neither group could solve alone. *What it fixed:* it grounded prejudice in real group dynamics rather than individual pathology, and it foreshadowed the contact conditions. *What it left debated:* Robbers Cave showed competition is *sufficient* for hostility, but later work showed it is not *necessary*, group identity alone can produce bias without any conflict, which social identity theory demonstrated.

5.5 Social identity theory (Tajfel): prejudice from categorisation alone

The minimal condition for bias. Henri Tajfel's **social identity** theory (Tajfel & Turner) argues that people derive part of their self-esteem from the groups they belong to, and therefore favour the in-group to enhance a positive social identity. The decisive evidence is the **minimal-group paradigm**: when participants are sorted into groups on a trivial, arbitrary basis (a coin toss, a preference for one abstract painter), with no history, no contact and no competition, they still allocate more reward to anonymous in-group members. **In-group** favouritism emerges from mere categorisation. The mechanism runs categorisation, then identification, then social comparison, with a drive to see one's own group as positively distinct. *What it fixed:* it proved prejudice does not require competition (fixing the gap RCT left) or an authoritarian personality; a bare "us and them" split suffices. *What it left debated:* it explains the readiness to divide but is often paired with realistic conflict and cognitive-schema accounts to explain the *content* and *intensity* of specific prejudices.

5.6 Scapegoating: prejudice as displaced frustration

The out-group as a target of convenience. The **scapegoating** account (frustration-aggression tradition, Dollard and colleagues; taken up by Allport) holds that frustration or economic hardship generates aggression that cannot safely be aimed at its true or powerful cause, so it is displaced onto a weaker, visible minority. Historically this fits the correlation between economic downturn and rising hostility toward minorities. It complements rather than competes with the other theories: it supplies a motive (displaced frustration) that categorisation (5.5) and the authoritarian dynamic (5.3) then channel toward a particular out-group.

GOOD TO REMEMBER

- **Allport (1954), *The Nature of Prejudice*:** prejudice = antipathy from faulty, inflexible generalisation; roots in normal categorisation; scale of acting-out (antilocution to extermination); proposed the contact hypothesis.
- **Adorno et al. (1950), authoritarian personality:** prejudice as a personality type (submission, displaced aggression, rigidity), indexed by the F-scale; harsh parenting origin.
- **Sherif, realistic conflict theory / Robbers Cave (1954):** competition for scarce resources drives in-group vs out-group hostility; superordinate goals reduce it.
- **Tajfel & Turner, social identity theory:** in-group favouritism from mere categorisation; minimal-group paradigm; categorise to identify to compare.
- **Scapegoating (frustration-aggression, Dollard et al.):** displaced aggression onto a weaker visible out-group.
- **Pettigrew & Tropp (2006):** meta-analysis of 500+ studies confirming contact reduces prejudice; optimal conditions helpful but not essential.

5.7 Reducing prejudice: the contact hypothesis and Allport's conditions

The currently accepted intervention. The **contact hypothesis**, first set out by Allport (1954), is the position with the strongest evidence and is the **currently accepted** account of prejudice reduction: bringing groups into contact reduces prejudice, but only reliably when the contact meets certain conditions. Allport specified four: **equal status** between the groups within the contact situation, **common (superordinate) goals**, intergroup **cooperation** rather than competition, and **institutional / authority support** (norms and sanction that endorse the contact). Robbers Cave (5.4) is the field demonstration of the goal and cooperation conditions. Without these conditions, mere proximity can entrench hostility rather than dissolve it.

CLASSIC TRAP Post Pettigrew and Tropp (2006), Allport's four conditions are **facilitating and optimising, not strict prerequisites**; contact still reduces prejudice, more weakly, without them, so do not state them as absolute requirements.

MNEMONIC

E C C I

Allport's four contact conditions: **E**qual status, **C**ommon goals, **C**ooperation, **I**nstitutional support.

5.8 The modern evidence: Pettigrew and Tropp (2006)

What the meta-analysis actually showed. Pettigrew and Tropp's (2006) meta-analysis pooled several hundred studies and confirmed that intergroup contact reliably reduces prejudice, with effects generalising beyond the specific individuals met. Their refinement of Allport matters for exams: the four optimal conditions *enhance* the effect but are **not strictly necessary**, contact reduces prejudice even without the full set, and a large part of the effect is mediated by reduced anxiety and increased empathy / perspective-taking rather than by belief change alone. This is the modern, currently accepted picture: contact works, the conditions optimise it, and affect (anxiety, empathy) is a key mediating pathway.

COMMON TRAP

Do not state Allport's four conditions as strict prerequisites for any effect at all. Post Pettigrew and Tropp (2006), the examiner-correct line is that the conditions are *facilitating and optimising*, not absolutely necessary; contact still reduces prejudice, more weakly, without them. Conversely, do not claim "mere contact always helps": Robbers Cave shows unstructured contact under competition can worsen hostility.

5.9 Neural correlate of intergroup bias

The neurobiology of us-and-them. The **neural correlate** of prejudice centres on threat and social-cognition circuitry. The **amygdala** shows differential activation to out-group faces (a rapid, often implicit threat response), modulated by top-down control; the **medial prefrontal cortex** is engaged less when perceiving extreme, dehumanised out-groups (reduced mentalising, the neural signature of failing to attribute a full mind to the other); and the **anterior cingulate cortex** and **dorsolateral prefrontal cortex** support the effortful regulation and override of automatic bias. The reading is dual-process: a fast, amygdala-linked implicit reaction and a slower, prefrontally mediated controlled evaluation, the same fast-versus-controlled architecture that runs through modern social cognition.

5.10 Worked example: the stigma of mental illness as applied prejudice

The clinical application. The **stigma** of mental illness is the textbook clinical instance of the triad (cross-reference the community psychiatry / stigma material). Map it directly: the **stereotype** is the belief that people with mental illness are dangerous, incompetent or to blame; the **prejudice** is the fear and desire for social distance those beliefs generate; the **discrimination** is the concrete harm, denial of housing, employment, insurance and even parity in medical care. Link (2001) and Corrigan's work distinguish *public stigma* (the community's endorsement of the stereotype and resulting discrimination) from *self-stigma* (the patient internalising it, with loss of self-esteem and the "why try" effect that erodes help-seeking and adherence). The anti-stigma toolkit is exactly the intergroup-theory toolkit: education corrects the stereotype, protest challenges discrimination, and structured *contact* with people who have lived experience, framed with equal status and cooperation per Allport, is the most effective single lever, consistent with Pettigrew and Tropp (2006).

CLINICAL ANCHOR

When a discharged patient cannot get a job or a rented room, name the mechanism precisely: that is *discrimination* (behaviour) driven by *prejudice* (affect) built on a *stereotype* (belief). It anchors the anti-stigma plan, and it enforces the system rule that a diagnostic label is never handed to a patient as a bare word, because labelling amplifies self-stigma and the "why try" effect.

INDIA

The Mental Healthcare Act 2017 explicitly prohibits discrimination against persons with mental illness and asserts a right to equality and non-discrimination in treatment and services, translating the discrimination limb of the triad into statutory protection. In practice, marriage-related and family stigma remain dominant drivers of concealment and delayed care in Indian settings.

PEARL

One-line thread for the whole topic: prejudice is a normal cognitive process (categorisation) gone wrong (Allport), sharpened by personality (Adorno), competition (Sherif) and mere group identity (Tajfel), and the one evidence-based cure is optimal-condition contact (Allport's ECCI, validated by Pettigrew and Tropp), which is also the front-line tool against the stigma of mental illness.

6 · Aggression and prosocial behaviour

Two mirror-image questions. Social psychology asks why humans harm each other and why they help each other, and the exam pairs them as two halves of one topic. Both halves are examined the same way: as a **succession of theories**, each fixing a flaw in the one before, converging on a modern integrative account with a named neural substrate. For **aggression** the arc runs from Freud's death instinct and Lorenz's ethology, through the **frustration-aggression** hypothesis and Berkowitz's revision, through Bandura's social learning, to the general aggression model, with serotonin, the amygdala and prefrontal cortex as the biology. For **prosocial** behaviour and **altruism** the arc runs from the puzzle of the unhelpful crowd (the **bystander** effect) to the competing motivational accounts (**empathy-altruism** versus **social exchange**) and the evolutionary accounts (**reciprocity** and kin selection).

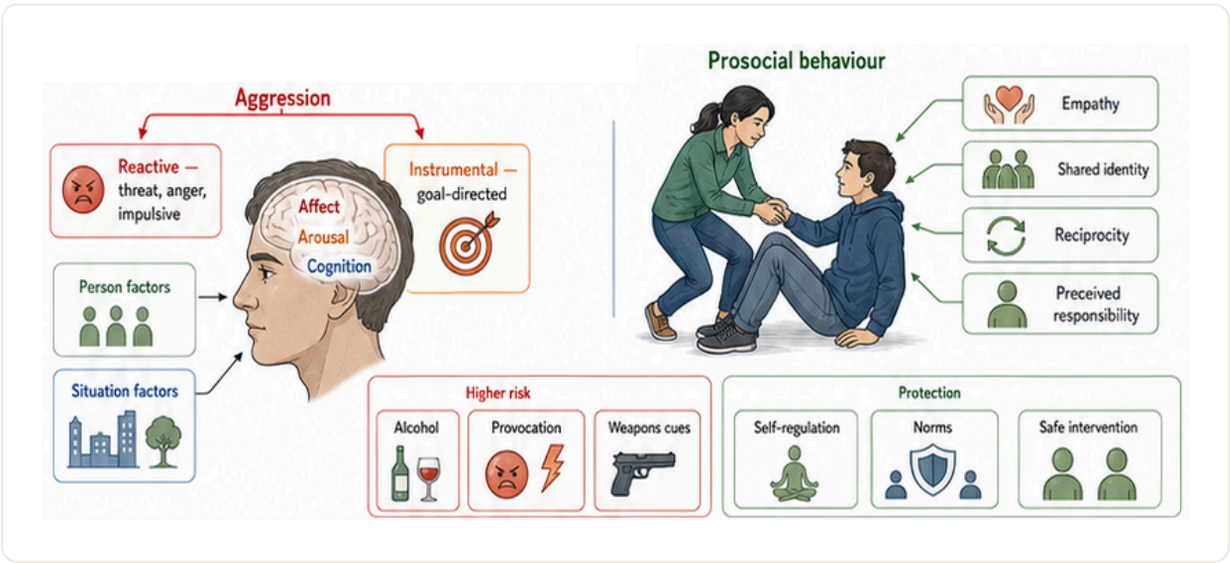


Fig 5.6 Aggression and prosocial behaviour.

The topic matters clinically because these are the mechanisms behind violence risk, impulsive aggression in personality disorder and intermittent explosive disorder, and the diffusion of responsibility that lets a ward or a bystander fail to act in an emergency; the empathy and reciprocity accounts also underpin how the therapeutic and helping relationship works (Kaplan &

Sadock 2024; Tasman 2015). Treat the theory sequence below as a system to enumerate: state what each theory added and what it left debated.

6.1 Defining aggression: reactive versus instrumental

Behaviour intended to harm. **Aggression** is behaviour carried out with the intent to harm another who is motivated to avoid it; intent is what separates it from accidental injury (Kaplan & Sadock 2024). The single most useful clinical division is by function. **Reactive (affective, impulsive) aggression** is hot: it is unplanned, arises from frustration, threat or anger, is accompanied by autonomic arousal, and serves no goal beyond discharge; **instrumental (proactive, predatory) aggression** is cold: it is deliberate, calculated and goal-directed, with low arousal, and is used as a means to an end (Bateman & Fonagy 2016). The distinction maps onto psychopathology: reactive aggression is sensitised by trauma and dominates in impulsive and borderline presentations, whereas planned instrumental aggression is the form that best distinguishes psychopathy from ordinary antisocial behaviour (Bateman & Fonagy 2016). Keep this pair in mind, because the early drive and frustration theories explain reactive aggression well but instrumental aggression poorly.

RULE **The reactive-versus-instrumental split drives management.** Hot, serotonin-linked reactive aggression (borderline, intermittent explosive disorder) responds to SSRIs and skills work; cold instrumental aggression in psychopathy does not, and is a risk-management problem.

6.2 Instinct and ethological theories: Freud and Lorenz

Aggression as an innate drive that must discharge. The earliest accounts treated aggression as a built-in biological force. Sigmund Freud, after the First World War, proposed a death instinct, **Thanatos**, a drive toward destruction that is either turned outward as aggression or inward as self-harm and depression; on this view aggression is an inevitable internal pressure seeking release (Kaplan & Sadock 2024). Konrad Lorenz, from ethology, argued in parallel that aggression is an evolved instinct with survival value (spacing out competitors, establishing dominance hierarchies, defending the young), driven by an accumulating internal energy that is released by an external **releasing stimulus**. Both are **hydraulic** models: pressure builds and must be let out, which gave rise to the (now discredited) idea of **catharsis**, that venting aggression harmlessly lowers it. *What they fixed:* nothing before them; they established aggression as biologically rooted, not merely learned. *What they left debated:* the drive was untestable, the hydraulic energy was never measured, and the catharsis prediction failed empirically (venting tends to increase, not reduce, later aggression). This pushed the field toward a specifiable trigger.

6.3 The frustration-aggression hypothesis: Dollard and Miller

Every frustration breeds aggression. The Yale group, John Dollard, Neal Miller and colleagues, proposed the **frustration-aggression** hypothesis (Dollard, Doob, Miller, Mowrer & Sears 1939), translating the psychoanalytic drive into testable learning-theory terms (Kaplan & Sadock 2024). Its original strong claim was reciprocal: frustration (the blocking of goal-directed behaviour) always produces a readiness for aggression, and all aggression presupposes frustration. It introduced **displacement** (aggression is redirected onto a safer, substitute target when the true

source is too powerful or unavailable, the classic account of scapegoating) and **catharsis** as the reduction of the aggressive drive after an aggressive act. *What it fixed:* it gave aggression an identifiable, manipulable cause (frustration) in place of an unmeasurable instinct. *What it left debated:* the strong form was quickly falsified, because frustration often yields not aggression but withdrawal, sadness or renewed effort, and aggression frequently occurs with no frustration at all. Neal Miller himself softened it in 1941 to say frustration produces instigations to a number of responses, of which aggression is only one.

6.4 Berkowitz's cue-arousal reformulation

Frustration produces readiness, cues pull the trigger. Leonard Berkowitz revised the hypothesis into the **cue-arousal** (later cognitive-neoassociation) model (Berkowitz 1989). Frustration, he argued, does not directly produce aggression; it produces **negative affect** and a readiness to aggress, but whether aggression actually occurs depends on the presence of **aggressive cues** in the situation, learned stimuli associated with aggression. His **weapons effect** demonstration showed that the mere presence of a gun in the room increased aggressive responding in provoked participants (Berkowitz & LePage 1967). The mature cognitive-neoassociation account holds that any aversive event (frustration, pain, heat, foul odour), not just goal-blocking, generates negative affect, which automatically primes an associative network of aggression-related feelings, thoughts and motor responses; higher-order appraisal can then amplify or inhibit the impulse. *What it fixed:* it explained why frustration only sometimes leads to aggression (cues and appraisal moderate it) and widened the cause from frustration to all aversive stimulation. *What it left debated:* it still centred on internal affect and said little about how aggressive behaviour is acquired in the first place.

6.5 Social learning of aggression: Bandura and the Bobo doll

Aggression is learned by watching. Albert Bandura's **social learning** theory answered the acquisition question: aggression, like other behaviour, is learned through **observation** and **imitation** of models, and does not require the observer to be reinforced directly (Bandura 1973; cross-referenced in the social-learning topic). In the famous **Bobo doll** experiments, young children who watched an adult model attack an inflatable Bobo doll later reproduced the specific aggressive acts when given access to the doll, while children who had not seen the model did not (Bandura, Ross & Ross 1961); a follow-up showed the effect transferred from a filmed model and even to a live clown (Bandura, Ross & Ross 1963). This was evidence for learning by observation in the absence of direct reinforcement (Companion to Psychiatric Studies). Whether the child then performs the learned aggression depends on **vicarious reinforcement** (seeing the model rewarded raises imitation; seeing the model punished lowers it) and on the child's **self-efficacy** for the act. Optimal conditions for observational learning include a live model, perceived similarity to the model, seeing the model reinforced, and familiarity with the model (Companion to Psychiatric Studies). *What it fixed:* it explained the transmission of aggression across people and generations (family violence, media violence) that instinct and frustration theories could not. *What it left debated:* it needed integration with the affect and arousal mechanisms, which the next model supplied.

6.6 The currently accepted position: the general aggression model

An integrative input-route-outcome framework. The **currently accepted** synthesis is the **general aggression model** (GAM) of Anderson and Bushman (Anderson & Bushman 2002), which folds the earlier theories into one cyclical process rather than replacing them. *Inputs* are of two kinds: **person factors** (trait aggressiveness, sex, beliefs, attitudes, genetic loading) and **situation factors** (provocation, frustration, aggressive cues such as weapons, pain, heat, alcohol). These act through three interacting *internal routes*, the present **cognition** (hostile thoughts and scripts), **affect** (anger, negative affect, from Berkowitz), and **arousal**. Appraisal and decision processes, immediate and automatic or reappraised and controlled, then yield the *outcome*: a thoughtful or an impulsive action, whose social consequences feed back to update the person's knowledge structures across encounters. GAM thereby contains Berkowitz's affect, Bandura's learned scripts and knowledge structures, and the older arousal notions inside a single loop. *What it fixed:* it unifies previously rival theories and accommodates both reactive and instrumental aggression. *What it leaves debated:* its breadth makes it hard to falsify, and the relative weight of each input remains contested.

6.7 Neurobiology of aggression

The neural correlate: a serotonin-limbic-prefrontal circuit. The **neural correlate** of impulsive aggression is a triad. First, low central **serotonin** (5-HT) function is the most replicated neurochemical correlate: reduced cerebrospinal-fluid 5-HIAA (the main serotonin metabolite) is associated with impulsive aggression, violent suicide attempts and low behavioural inhibition, supporting the model of serotonin as a brake on impulsive aggressive responding (Kaplan & Sadock 2024). Second, the **amygdala** generates the threat-driven reactive aggressive response, and its regulation by the **ventromedial and orbitofrontal prefrontal cortex** is what normally restrains it; reduced prefrontal control over an over-reactive amygdala is the circuit-level signature of reactive aggression, and reduced grey-matter volume has been reported in violent individuals with comorbid psychopathy and antisocial personality disorder (Bateman & Fonagy 2016). Third, **testosterone** correlates modestly with dominance-seeking and aggression, most consistently in interaction with low serotonin and low cortisol rather than as a simple main effect. A well-known gene-environment example is the low-activity **MAOA** variant, which predicts later antisocial and aggressive behaviour chiefly in men who were also maltreated in childhood (Caspi et al. 2002), illustrating that biology sets a threshold that experience then moves.

- **Low serotonin** - the most replicated neurochemical correlate: reduced CSF 5-HIAA marks impulsive aggression, so serotonin acts as a brake.
- **Amygdala under prefrontal control** - the amygdala drives the reactive aggressive response; reduced ventromedial/orbitofrontal restraint is the circuit signature of reactive aggression.
- **MAOA x maltreatment (Caspi 2002)** - the low-activity MAOA variant predicts later aggression chiefly in men maltreated in childhood: biology sets a threshold, experience moves it.

CLINICAL ANCHOR

The reactive versus instrumental split drives management. Reactive, serotonin-linked impulsive aggression (borderline personality disorder, intermittent explosive disorder) responds to SSRIs, mood stabilisers and skills-based work on arousal and appraisal; cold instrumental aggression in psychopathy does not respond to these and is a risk-management, not a pharmacological, problem (Kaplan & Sadock 2024; Bateman & Fonagy 2016). Note also that the catharsis idea, that letting a patient vent anger discharges it, is empirically wrong and is not a therapeutic technique.

6.8 Prosocial behaviour and the bystander effect

Why a crowd fails to help. **Prosocial** behaviour is voluntary action intended to benefit another; **altruism** is the special case of prosocial behaviour motivated by concern for the other rather than for self-gain. The founding finding is the **bystander** effect: an individual is significantly less likely to help a person in need when other potential helpers are present, because the responsibility to act is distributed across everyone present, termed **diffusion of responsibility** (Latané & Darley 1970; Tasman 2015). Prompted by the 1964 Kitty Genovese case, Latané and Darley showed experimentally that help becomes slower and less likely as group size rises. The effect operates through three social-cognitive processes: *diffusion of responsibility* (each onlooker feels less personally obliged), **pluralistic ignorance** (each reads the inaction of others as a signal that no emergency exists), and *evaluation apprehension* (fear of intervening wrongly and being judged). The lesson, carried into resuscitation training, is to defeat diffusion by assigning responsibility to one named person.

6.9 The bystander decision tree

Five sequential yes-or-no steps to helping. Latané and Darley modelled helping as a **decision tree**: help occurs only if the bystander clears every step, and a no at any step ends the process (Latané & Darley 1970).

STEP	THE QUESTION	WHAT BLOCKS A "YES" (FAILURE MODE)
1. Notice	Do I notice something is happening?	**Distraction, hurry, self-absorption**
2. Interpret	Is this an emergency?	**Pluralistic ignorance: others look calm, so I under-read it**
3. Responsibility	Is it my responsibility to act?	**Diffusion of responsibility across the crowd**
4. Decide how	Do I know what to do?	**Lack of competence or knowledge of the right action**
5. Act	Do I implement help?	**Evaluation apprehension, fear of danger or cost**

Each stage is a filter; the more people present, the more likely the process stalls at steps 2 and 3, which is the bystander effect.

6.10 Why we help: empathy-altruism versus egoistic accounts

Is helping ever truly selfless? Once someone decides to help, the debate is about motive. Two families of theory compete. The **empathy-altruism** hypothesis (Batson) holds that feeling genuine *empathic concern* for a sufferer produces truly altruistic motivation, a desire to relieve the other's distress as an end in itself, not to relieve one's own; Batson's experiments dissociated the two by making escape easy, showing that high-empathy participants still helped even when they could walk away, which a purely selfish account cannot explain (Batson 1991). Against this stand the **egoistic** accounts, which argue all helping ultimately serves the self. The **negative-state relief** model (Cialdini) proposes that witnessing distress makes the observer feel bad and that helping is a way to repair one's own mood, so the real goal is self-soothing. The **social exchange** account frames helping as a cost-benefit transaction: we help when the rewards (reciprocation, social approval, relief of distress, self-esteem) outweigh the costs, so apparent altruism is disguised self-interest. The debate is not fully resolved, but the empathy-altruism evidence is generally taken to show that genuinely other-directed motivation exists alongside egoistic motives.

6.11 The evolutionary accounts: reciprocity and kin selection

How can selfish genes build a helper? Evolutionary biology explains the paradox that costly helping persists. Two mechanisms do most of the work (Kaplan & Sadock 2024). **Kin selection** (William Hamilton) shows that a gene causing an individual to sacrifice for relatives can spread because relatives carry copies of that gene: Hamilton's rule states an altruistic act is favoured when $C < B \times r$, the cost to the actor is less than the benefit to the recipient times the coefficient of relatedness (r = **0.5** for a parent, child or full sibling), a concept captured by **inclusive fitness**. It explains parental sacrifice and why one would risk more for two siblings or eight cousins than for one. **Reciprocity**, or reciprocal altruism (Robert Trivers), explains helping between *non-kin*: I help you now because you are likely to help me later, an exchange that pays off when repeated interactions allow cheaters to be detected and excluded, modelled by the **prisoner's dilemma** (Trivers 1971; Kaplan & Sadock 2024). On this view social emotions are the enforcement machinery of reciprocity: gratitude and friendship reward cooperators, while anger, guilt and the urge to punish defectors police cheating (Kaplan & Sadock 2024). Together, kin selection and reciprocity supply the ultimate (evolutionary) explanation, while empathy and social exchange supply the proximate (psychological) one.

HOLD THIS

Catharsis is empirically false: venting tends to raise, not lower, later aggression. The examinable current model of aggression is Berkowitz's (frustration is one aversive input creating a readiness that cues and appraisal then shape), folded into the general aggression model.

TRAP Batson's empathy-altruism argues **for** genuinely other-directed helping; negative-state relief and social exchange are the egoistic rebuttals, not versions of it.

6.12 Worked example

One scene, every theory. A junior doctor is jostled and insulted by an intoxicated relative in a crowded casualty. His flash of anger and impulse to shove back is *reactive aggression*, primed by the aversive provocation (Berkowitz's negative affect) and the aggressive cues around him, gated by his prefrontal control over the amygdalar threat response; that he learned to keep his hands down comes partly from having modelled senior colleagues who de-escalate (Bandura). Moments later the relative collapses. The dozen onlookers freeze: each reads the others' stillness as evidence it is not serious (pluralistic ignorance) and assumes someone else will act (diffusion of responsibility), the *bystander* effect. The doctor overrides it by pointing at one nurse and saying "you, call the code," collapsing diffusion onto a named person. Whether he then feels genuine *empathic concern* for the patient (empathy-altruism) or is partly relieving his own distress (negative-state relief) and expecting professional credit (social exchange) is the motive debate, but the deep reason a species evolved to help a stranger at all traces to *reciprocity* and, for his own child at home, to *kin selection*.

COMMON TRAP

Do not confuse the two aggression subtypes with the two theory families: reactive aggression is not the same as the frustration-aggression theory, and instrumental aggression is not social learning. Also, the frustration-aggression hypothesis in its original strong form (frustration always causes aggression, all aggression follows frustration) is *wrong*; the examinable current form is Berkowitz's, where frustration is one aversive input producing a readiness that cues and appraisal then shape. And empathy-altruism is Batson's claim *for* genuine altruism; negative-state relief and social exchange are the egoistic rebuttals, not versions of it.

MNEMONIC

"NIRA-DA" for the bystander decision tree

Notice, **I**nterpret as emergency, take **R**esponsibility, decide how to **A**ct, then **DA**re to do it. Five sequential gates; a single "no" ends the sequence and no help is given.

INDIA

Diffusion of responsibility is a live problem in Indian public spaces: crowded roads and railway platforms where accident victims are watched but not helped are the bystander effect in the field. The Supreme Court's Good Samaritan guidelines (2016) were designed precisely to remove the cost side of the helping decision, protecting a helper from legal and procedural harassment so that steps 4 and 5 of the decision tree become easier to clear.

GOOD TO REMEMBER

- **Freud (Thanatos):** death instinct, aggression as an innate drive turned outward or inward.
- **Lorenz (ethology):** aggression an evolved instinct with an accumulating energy released by a releasing stimulus; hydraulic model.
- **Catharsis idea:** venting lowers aggression; empirically false (venting tends to increase it).
- **Dollard & Miller et al. 1939 (frustration-aggression):** frustration always breeds aggression; introduced displacement and scapegoating. Strong form falsified.
- **Miller 1941:** softened it, frustration instigates several responses, aggression being one.
- **Berkowitz (cue-arousal / cognitive-neoassociation):** frustration and any aversive event create negative affect and readiness; aggressive cues (the weapons effect, Berkowitz & LePage 1967) release it.
- **Bandura (social learning):** Bobo doll (1961; filmed and live-clown versions 1963), learning by observation and imitation without direct reinforcement; vicarious reinforcement governs performance.
- **Anderson & Bushman 2002 (general aggression model):** currently accepted; person and situation inputs act via cognition, affect and arousal, through appraisal, to a thoughtful or impulsive outcome, then feed back.
- **Reactive vs instrumental aggression:** hot, impulsive, frustration-driven (borderline, IED) versus cold, planned, goal-directed (psychopathy).
- **Neurobiology:** low serotonin (reduced CSF 5-HIAA) disinhibits impulsive aggression; amygdala drives reactive aggression under ventromedial/orbitofrontal control; testosterone modestly linked; low-activity MAOA x childhood maltreatment (Caspi 2002).
- **Latané & Darley 1970 (bystander effect):** help less likely as onlookers increase; diffusion of responsibility, pluralistic ignorance, evaluation apprehension; five-step decision tree.
- **Batson (empathy-altruism):** empathic concern produces genuinely altruistic motivation (helping persists even when escape is easy).
- **Cialdini (negative-state relief):** egoistic; helping repairs the helper's own bad mood.
- **Social exchange / reciprocity:** helping is a cost-benefit transaction; apparent altruism serves the self.
- **Trivers 1971 (reciprocal altruism):** helping non-kin because of expected later return; modelled by the prisoner's dilemma; social emotions enforce it.
- **Hamilton (kin selection):** altruism to relatives favoured when $C < B \times r$ ($r = 0.5$ for parent/child/sibling); inclusive fitness.

7 · Crowd and mob psychology

What happens to the individual mind when it is dissolved into a crowd. A **crowd** is a large, temporary, co-present gathering of people; a **mob** is a crowd that has turned to intense, often aggressive, collective action. The psychology of the crowd asks a single durable question: does a person in a crowd lose their individual judgement and become swept along by the mass, or do crowds behave in a patterned, meaningful way that reflects who the people in them are? The exam wants this as a chain of theories in the order they appeared: Gustave **le bon**'s original account of the crowd as a primitive collective mind, the mid-century theory of **deindividuation**, Turner and Killian's **emergent-norm** theory, and finally Reicher's **social identity** model, which is the modern corrective. Each theory fixed a limitation of the one before it.

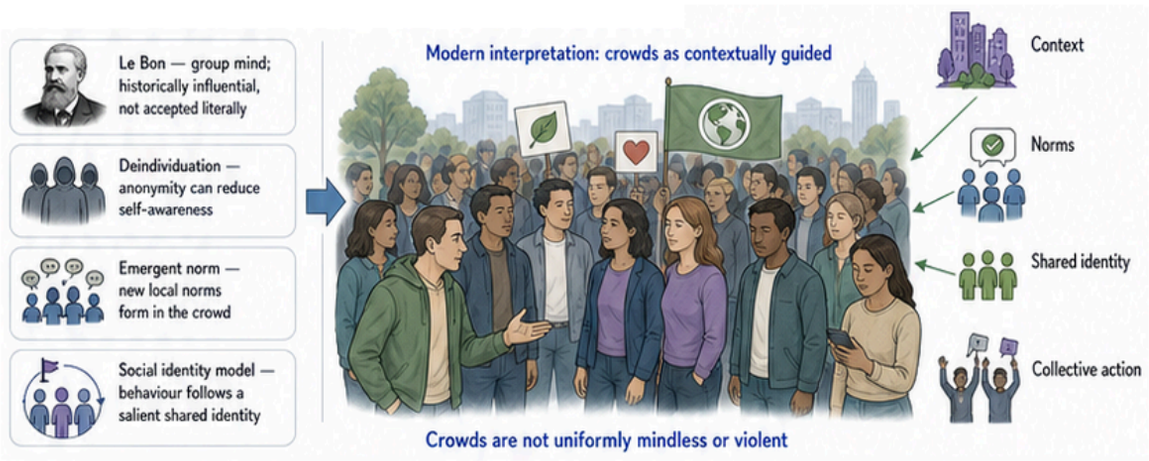


Fig 5.7 Crowd psychology: from group mind to shared identity.

This matters clinically and forensically, not as social-science trivia. Crowd dynamics underlie **mass psychogenic illness** (epidemic hysteria), the panic and crush injuries of stampedes, and the behaviour of riots, all of which a psychiatrist may be asked to explain in a medico-legal report or a public-health inquiry (Companion to Psychiatric Studies). Treat the four-theory sequence and the Le Bon-to-Reicher shift as the highest-yield items; treat mass hysteria as the clinical anchor.

7.1 Defining the crowd, the mob and collective behaviour

A vocabulary before the theories. *Collective behaviour* is the umbrella term for the relatively spontaneous, unstructured action of people responding together to a common situation. Within it, a *crowd* is defined by physical co-presence and a shared focus of attention; a *mob* is an emotionally aroused crowd acting toward a target (a lynch mob, a riot); a *panic* is a crowd in flight from a perceived threat. Two mechanisms recur across every theory and are worth fixing first: *suggestibility*, the readiness to accept and act on ideas or emotions coming from others without critical appraisal, and *contagion*, the rapid, seemingly automatic spread of an emotion or behaviour through a gathering. Le Bon put both at the centre; the later theories progressively reinterpreted them.

7.2 Theory one: Le Bon and the group mind (1895)

The crowd as a single primitive organism. Gustave Le Bon's *The Crowd: A Study of the Popular Mind* (Le Bon 1895) is the founding text and the classic exam anchor. Writing in the wake of the French revolutionary crowds, he argued that when individuals gather, a *collective* or **group mind** forms and the person is transformed. Three mechanisms drive this:

Anonymity

Submerged in the crowd and feeling unidentifiable, the individual loses the sense of personal responsibility that normally restrains conduct, and yields to instincts they would otherwise control.

Contagion

Emotions, ideas and acts spread through the crowd like an infection, each person imitating the next, so that feeling and behaviour sweep through the mass with little resistance.

Suggestibility

The crowd enters a hypnotic, quasi-trance state in which critical faculties are suspended; the person becomes highly open to suggestion and is driven by the images and slogans put before them.

The result, in Le Bon's picture, is a crowd that is impulsive, irritable, intellectually inferior to its members, prone to extremes of emotion, and morally regressed to a "primitive" level: the individual "descends several rungs on the ladder of civilisation." **What it fixed and what it left debated.** Le Bon fixed the absence of any theory by naming the phenomenon and giving it mechanisms, and his account was enormously influential on later thinking about propaganda and on Freud's group psychology. What it left debated, and what every later theory attacked, was its assumption that the crowd is uniformly irrational, mindless and homogeneous, that it destroys the individual's identity rather than expressing it, and its politically loaded contempt for the "masses."

7.3 Theory two: deindividuation (Festinger, Zimbardo, Diener)

Anonymity and lost self-awareness lower restraint. The mid-twentieth-century response kept Le Bon's core insight that the crowd loosens restraint but recast it in the testable language of experimental social psychology as **deindividuation**. Festinger, Pepitone and Newcomb (1952) coined the term for a state in which people in a group are not seen or attended to as individuals, which reduces inner restraints and increases behaviour that would ordinarily be inhibited. Philip Zimbardo (1969) systematised the antecedents: anonymity, diffusion of responsibility, large group size, arousal and altered states all reduce self-observation and evaluation-apprehension, releasing impulsive, often antisocial action; in his study, hooded, unidentifiable participants delivered more apparent shocks than clearly identified ones. Ed Diener (1976, 1980) sharpened the mechanism to *reduced self-awareness*: it is the loss of *private* self-focus, of attention to one's own standards, that de-regulates behaviour, not mere anonymity to others. A widely cited naturalistic illustration is that anonymity-promoting conditions such as darkness and disguise are associated with more disinhibited behaviour, for example the finding that trick-or-treating children were more likely to steal extra sweets when anonymous and in a group than when alone and identified (Diener et al. 1976).

What it fixed and what it left debated. Deindividuation fixed Le Bon's untestable "group mind" by replacing it with measurable variables (anonymity, group size, self-awareness) that could be manipulated in the laboratory. What it left debated is that anonymity does not reliably produce *antisocial* behaviour at all: anonymous, deindividuated crowds sometimes become more generous, more helpful or more conformist to a peaceful norm, which a theory of pure restraint-loss cannot explain. The direction of behaviour, not just its intensity, needed a theory.

7.4 Theory three: emergent-norm theory (Turner and Killian)

Crowds are not norm-less; they build a new norm on the spot. Ralph Turner and Lewis Killian's **emergent-norm** theory (Turner and Killian, *Collective Behavior*, 1957) rejected the idea that crowds are uniform and driven by contagion. In the ambiguous, unstructured situation a crowd faces, no existing rule tells people how to act; a few distinctive individuals then behave in a salient way (for example, throwing the first stone or beginning to flee), their conduct is taken as defining "what people here are doing," and it crystallises into a *new, situation-specific norm* that the

rest feel pressure to follow. The crowd only *appears* unanimous and contagious; in reality members differ in motive, and it is normative social pressure and the desire not to deviate visibly, not hypnotic contagion, that produces the observed uniformity. **What it fixed and what it left debated.** Emergent-norm theory fixed the assumption of mindlessness: crowd action is now rational and norm-governed, just governed by a norm that emerged in the moment rather than one brought in from outside. What it left debated is *where the new norm comes from* and why particular crowds converge on particular norms, hostile in one case, festive in another; the theory describes the process of norm formation but not its content, and it still treats the crowd as a set of strangers negotiating from scratch.

7.5 Theory four: the social identity model (Reicher), the modern corrective

Crowds act on a shared social identity, not on lost identity. Stephen Reicher's **social identity** model of crowd behaviour (Reicher 1984, building on Tajfel and Turner's social identity and self-categorisation theory) is the decisive correction to Le Bon and the position the field now holds. Its central claim inverts Le Bon: in a crowd the person does not *lose* identity but *shifts* from personal identity to a shared *social* identity, categorising the self as a member of the relevant group ("we, the protesters"; "we, the fans"). Behaviour then becomes not random or de-regulated but *patterned by the norms and values of that group identity*. This explains what deindividuation could not: crowds are selective, not indiscriminate. In Reicher's analysis of the 1980 St Pauls riot in Bristol, the crowd's violence was bounded and targeted, directed at police and symbols of the state and confined to the neighbourhood, not a blind orgy of destruction, precisely because it expressed a specific community identity and its grievances. The later *elaborated social identity model* (ESIM, Reicher, Stott, Drury) adds that crowd conflict is often *generated by the interaction* with an out-group such as the police: indiscriminate, heavy-handed control that treats a whole peaceful crowd as hostile can create a new, oppositional shared identity and escalate disorder that did not initially exist. **What it fixed.** The social identity model fixed the deepest error in the whole tradition, the equation of "crowd" with "irrational" and "identity-less." It shows crowd behaviour to be socially meaningful, group-normed and, crucially, shaped by how the crowd is policed, which is why it now underpins evidence-based public-order policing.

7.6 Currently accepted position

The social identity account, with deindividuation demoted to a special case. The **currently accepted** position is that crowd behaviour is not the loss of the rational individual to a primitive group mind but the shift to a shared social identity whose norms then guide conduct (Reicher; Reicher, Stott and Drury). Le Bon's contagion is reinterpreted: what looks like automatic emotional spread is the rapid, mutual recognition and enactment of a common in-group norm among people who now categorise themselves together. Deindividuation is retained only in a limited, reinterpreted form, the SIDE model (Social Identity model of Deindividuation Effects, Reicher, Spears and Postmes), which holds that anonymity in a group does not abolish norms but *strengthens adherence to the salient group norm* by making personal individuality less salient than group membership. The through-line is decisive: crowds are norm-governed and identity-driven, not mindless.

HOLD THIS

The crowd sequence rescues the crowd's rationality: Le Bon (group mind) to deindividuation to emergent-norm to Reicher's social identity model. In a crowd the person does not lose identity but shifts from personal to shared social identity, and behaviour follows that group's norms.

7.7 Neural and behavioural correlate

Where restraint and self-monitoring live. There is no single "crowd centre," but the mechanisms the theories describe map onto known systems. The reduced self-monitoring and behavioural disinhibition of deindividuation are consistent with attenuated engagement of the *medial and lateral prefrontal cortex*, the substrate of self-referential processing and of top-down inhibitory control over prosocial norms; reduced private self-awareness is, in effect, reduced recruitment of these self-monitoring circuits. Emotional contagion, the rapid spread of affect through a crowd, is linked to fast, automatic *affect-sharing and mirroring processes* (limbic structures including the amygdala and the anterior insula, and the human mirror-neuron system), which transmit fear and arousal person-to-person below the level of deliberation. Social categorisation, the engine of the social identity model, engages the same *medial prefrontal and temporoparietal* "social brain" regions that distinguish in-group from out-group. The behavioural correlate at the level of the crowd is the crush dynamics of a panicking mass: as density rises, individual locomotion is overridden by physical coupling between bodies, producing turbulent, wave-like "crowd quakes" in which people are injured by compressive asphyxiation rather than by trampling.

TRAP Do not label every stampede death "mass panic": most are **physical crush and compressive asphyxia at critical density**, and survivors usually behaved cooperatively until physically overwhelmed.

CLINICAL ANCHOR

Mass psychogenic illness (epidemic or mass hysteria). This is contagious dissociative or conversion phenomena spreading rapidly through a group under conditions of shared anxiety, with no organic cause found (Companion to Psychiatric Studies). It classically strikes closed, stressed institutions, most often schools, where fainting, dizziness, breathlessness or non-epileptic collapse spread from person to person, typically along lines of sight and friendship. A famous, still-debated example is the 1955 outbreak among nurses at the Royal Free Hospital during a polio epidemic, read by some as atypical infection and by others as mass hysteria (McEvedy and Beard 1970, cited in Companion to Psychiatric Studies). The clinical task is a careful differential from a genuine toxic or infectious outbreak: mass psychogenic illness shows rapid onset and remission, spread by sight and sound rather than by a plausible physical vector, a preponderance of subjective symptoms with normal investigations, and often an index case with a visible acute symptom. Management is calm separation of affected individuals, avoidance of dramatic medical spectacle that amplifies suggestion, credible reassurance, and correcting the anxiety-laden rumour that feeds it.

INDIA

Crowd crush at mass religious gatherings and festivals is a recurring public-health problem in India, where enormous congregations converge on constrained spaces (narrow temple approaches, riverbank ghats, festival grounds). Fatal stampedes at such events illustrate the crowd-dynamics point exactly: deaths are overwhelmingly from progressive crowd compression and asphyxia at critical density, often triggered by a rumour, a sudden surge toward a darshan or a blocked exit, not by generalised "panic" in the Le Bon sense. The psychiatric and forensic contribution is to resist the lazy "mass panic" narrative and point instead to identifiable failures of crowd management, density control, flow direction, communication, and to the behavioural finding that people in crushes typically continue cooperative, help-seeking behaviour until physically overwhelmed. Mass psychogenic illness outbreaks in Indian residential schools and factories are also periodically reported and follow the same anxiety-and-suggestion pattern.

COMMON TRAP

Do not present Le Bon as the correct modern account. In an exam, name Le Bon (1895) as the historically foundational but now-superseded theory, and be explicit that the contemporary consensus is the social identity model (Reicher): crowds are not mindless, contagion-driven and identity-less, they act on a shared social identity and its norms. A second trap: do not equate every crowd death with "panic." Most stampede fatalities are physical crush and compressive asphyxia at high density, and survivors usually behaved cooperatively; the label "mass panic" is often a post-hoc misattribution.

MNEMONIC

Le Bon's Dogs Emerge Social

The four theories in the order they appeared: **Le Bon** (group mind, 1895), **D**eindividuation (Festinger, Zimbardo, Diener), **E**mergent-norm (Turner and Killian), **S**ocial identity (Reicher, the modern corrective). Read the sequence as a steady rescue of the crowd's rationality: from a primitive group mind, to lost restraint, to a norm built on the spot, to a shared identity that was there all along.

GOOD TO REMEMBER

- **Le Bon (1895), *The Crowd*:** first theory; anonymity + contagion + suggestibility create a primitive, irrational "group mind" and loss of individual responsibility. Now superseded.
- **Deindividuation (Festinger, Pepitone and Newcomb 1952; coined the term):** group immersion reduces inner restraints and inhibited behaviour is released.
- **Zimbardo (1969):** anonymity, diffusion of responsibility, group size and arousal drive deindividuated, disinhibited action.
- **Diener (1976, 1980):** the core mechanism is reduced *private self-awareness*, not mere anonymity to others.
- **Emergent-norm theory (Turner and Killian 1957):** in ambiguous situations crowds create a new, situation-specific norm; uniformity comes from normative pressure, not contagion.
- **Social identity model (Reicher 1984):** in a crowd the person shifts from personal to shared *social* identity; behaviour is patterned by group norms, not lost. The modern corrective to Le Bon.
- **ESIM (Reicher, Stott, Drury):** crowd conflict is often generated by out-group (police) action; indiscriminate control creates oppositional identity and escalates disorder.
- **SIDE model (Reicher, Spears, Postmes):** anonymity in a group strengthens adherence to the salient group norm rather than abolishing norms.
- **Contagion:** rapid automatic spread of emotion/behaviour through a gathering; reinterpreted by modern theory as mutual enactment of a shared in-group norm.
- **Mass psychogenic illness / epidemic (mass) hysteria:** contagious dissociative-conversion symptoms in an anxious group with no organic cause; classic in schools; Royal Free Hospital 1955 (McEvedy and Beard 1970).
- **Currently accepted position:** social identity account (Reicher, Stott, Drury); deindividuation retained only as the reinterpreted SIDE effect.
- **Stampede mechanism:** deaths are from crowd compression and asphyxia at critical density ("crowd quakes"), not from Le Bon-style mass panic.

Learning and behaviour

8 · Classical conditioning

The oldest theory in learning, still the model of fear. **Classical conditioning** (also called Pavlovian or respondent conditioning) is the associative process by which a neutral stimulus, through repeated pairing with a biologically significant event, comes itself to elicit an anticipatory response. It is the first of the two great learning paradigms (the other being operant/instrumental conditioning) and the one the exam ties most tightly to psychopathology: conditioned fear is the working model of phobia, conditioned nausea is the model of anticipatory vomiting in chemotherapy, and conditioned cue reactivity is the model of relapse in addiction (cross-refer SUD). For a CORE answer you must trace the theory in date order, from Pavlov's reflex physiology, through Watson and Rayner's demonstration that human emotion obeys the same law, to the modern cognitive turn (Rescorla and Wagner) that redefined conditioning as prediction rather than mere pairing (Kaplan & Sadock 2024; Tasman 2015).

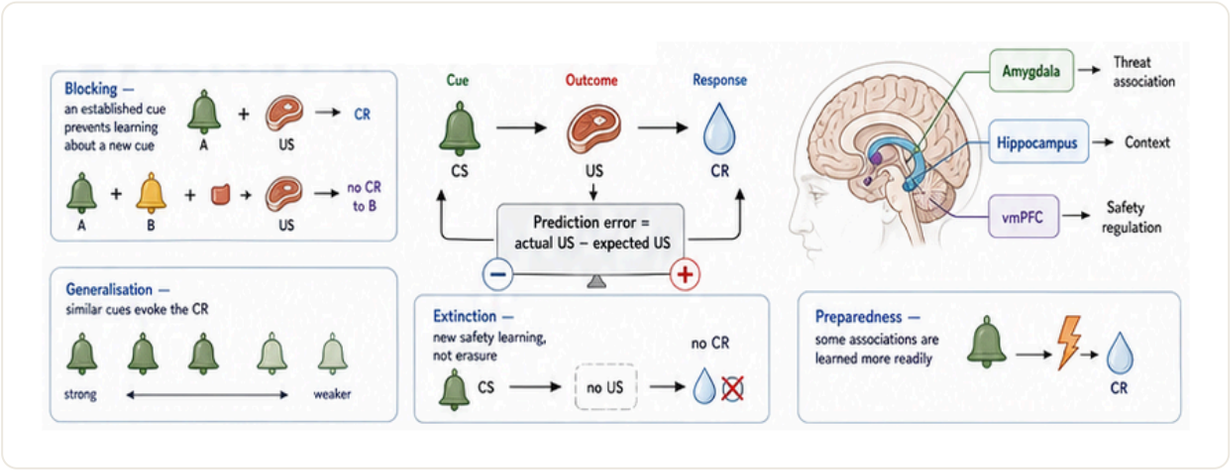


Fig 5.8 Classical conditioning is prediction.

The spine runs in strict historical order, each stage fixing what the last left open: **Pavlov (1927)** gave the reflex vocabulary (US, UR, CS, CR) and the four core phenomena (acquisition, extinction, spontaneous recovery, generalisation); **Watson and Rayner (1920)** proved the law applied to human fear (Little Albert) and launched the behaviourist manifesto; the mid-century **timing analysis** specified how the CS and US must be arranged in time; and the **Rescorla and Wagner (1972)** model overturned the naive idea that contiguity alone is enough, recasting learning as driven by surprise and contingency, with **preparedness** (Seligman; the Garcia effect) adding an evolutionary constraint. The **currently accepted** view is that classical conditioning is prediction-error learning under biological constraint, and its clearest **neural correlate** is amygdala-based fear conditioning with prefrontal control of extinction. Because this topic names dated theorists, defined phenomena and a hard timing classification, it closes with a good-to-remember box enumerating every one (Kaplan & Sadock 2024).

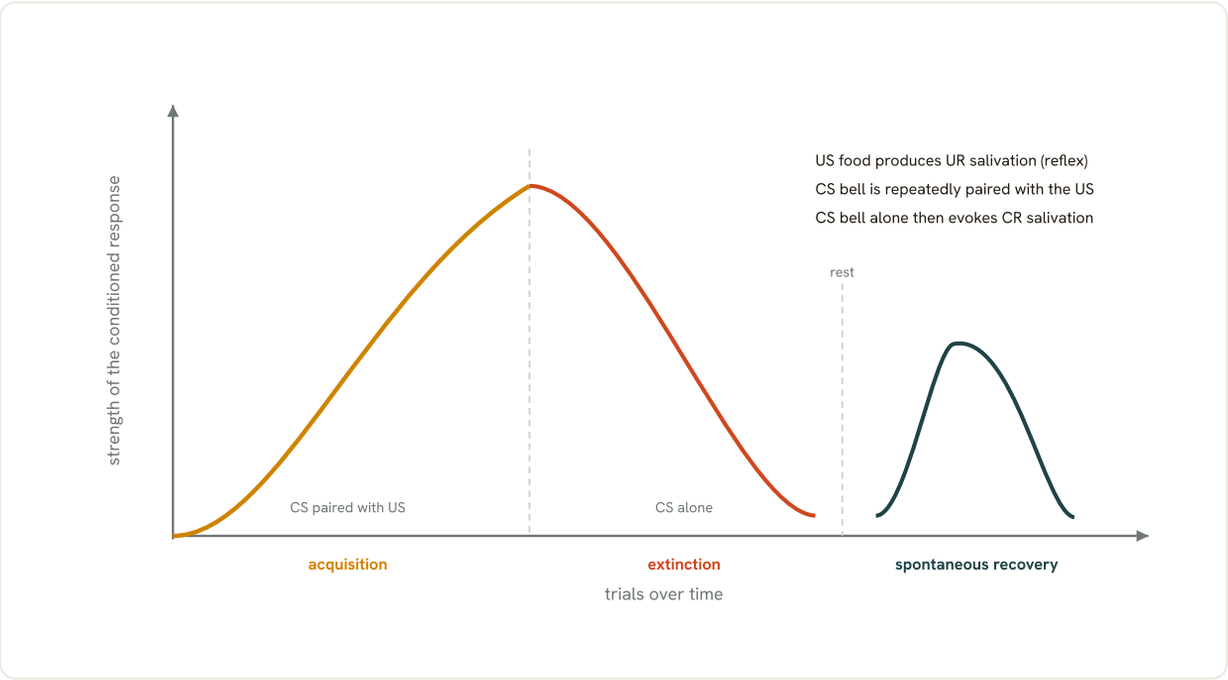


Fig 5.9 The course of classical conditioning. During acquisition the conditioned stimulus (the bell) is repeatedly paired with the unconditioned stimulus (food), and the conditioned response grows to an asymptote. Presenting the conditioned stimulus alone produces extinction, a fall in the response. After a rest the response returns, weaker, as spontaneous recovery, showing that extinction suppresses rather than erases the learning. The unconditioned stimulus and response are the inborn reflex; the conditioned stimulus and response are what the animal learns.

8.1 Pavlov (1927): the reflex vocabulary

The founding move: an automatic reflex can be transferred to an arbitrary signal. The Russian physiologist Ivan Pavlov, working in the reflex tradition, rang a bell and then gave a dog food; after a few pairings the dog salivated to the bell alone, anticipating the food (Kaplan & Sadock 2024). The events are named so the paradigm applies anywhere:

Unconditioned stimulus (US, UCS)

The stimulus that *unconditionally* elicits a response before any learning: the food.

Unconditioned response (UR, UCR)

The innate, reflexive response to the US: salivation to food.

Neutral stimulus (NS)

A stimulus that initially elicits no relevant response: the bell before pairing.

Conditioned stimulus (CS)

The former neutral stimulus *after* pairing, which now elicits the response conditional on the CS to US pairing: the bell.

Conditioned response (CR)

The learned, anticipatory response to the CS: salivation to the bell (usually weaker than the UR).

Read the arithmetic once and it never confuses: US produces UR by nature; NS produces nothing; pair NS with US repeatedly; the NS becomes a CS and now produces a CR. What Pavlov left open was *why* pairing sometimes works and sometimes does not, which the twentieth-century revisions answered (Tasman 2015).

8.2 The four core phenomena of Pavlovian learning

Every classical-conditioning answer must name these four. They are examined as pure psychology and as the mechanics of exposure therapy.

Acquisition

Acquisition is the initial learning phase in which the CR is established and strengthened over successive CS to US pairings; it is faster when the US is intense or salient and when the CS is novel.

Extinction

Extinction is the weakening and eventual disappearance of the CR when the CS is presented repeatedly *without* the US. It does not erase the original learning; it lays down new "the CS is safe" learning on top of it. Extinction is the theoretical engine of exposure therapy (Kaplan & Sadock 2024).

Spontaneous recovery

After extinction and a rest interval, presenting the CS again elicits the CR in diminished strength, proving the original association was not destroyed. Clinically it is the model for the return of a treated phobia after a symptom-free gap.

Stimulus generalisation and discrimination

Generalisation is the spread of the CR to stimuli resembling the CS (a child bitten by a spaniel fears all dogs). *Discrimination* is the complementary narrowing: the organism learns to respond only to the exact CS and not to similar stimuli, achieved by pairing the true CS with the US while presenting similar stimuli without it.

A fifth, higher-order (second-order) conditioning, extends the reach of a CS: once a CS reliably elicits a CR, pairing a *new* neutral stimulus with that established CS (with no US present) makes the new stimulus a CR-eliciting signal in its own right, which is how conditioned fear can spread to cues never once paired with the original trauma (Kaplan & Sadock 2024).

8.3 Watson and Rayner (1920): Little Albert and the behaviourist manifesto

What it fixed: it proved human emotion obeys Pavlov's law, not just canine salivation. John B. Watson, who had declared in his 1913 behaviourist manifesto that psychology should study only observable behaviour and treat consciousness as outside its scope, set out with Rosalie Rayner to show that a human emotion could be conditioned. In the **Little Albert** experiment (1920) an eleven-month-old infant was shown a white rat (initially neutral, even pleasant) while a steel bar was struck loudly behind his head (the US, producing the UR of startle and fear). After a few pairings the rat alone (now the CS) elicited crying and withdrawal (the CR of conditioned fear). Crucially the fear *generalised* to a rabbit, a dog, a fur coat and a Santa Claus mask, demonstrating stimulus generalisation of a conditioned emotion (Kaplan & Sadock 2024). Watson thereby claimed emotions are built by conditioning, not inherited fully formed, the empirical anchor of his environmentalist manifesto ("give me a dozen healthy infants..."). The complementary case, *Peter* (deconditioned of an animal fear by pairing a caged rabbit, moved gradually closer, with a favourite food), is the historical seed of systematic desensitisation (Kaplan & Sadock 2024). What it left debated: *which* arrangements of CS and US in time actually produce learning, and why some pairings fail, taken up next.

CLINICAL ANCHOR

Little Albert is the textbook model of specific phobia: a neutral object becomes a fear cue by pairing with an aversive event, then generalises. The mirror image, Peter, is the model of exposure-based cure. Both together make the two-line clinical story the exam wants: fear is conditioned in and extinguished out.

8.4 The timing of CS and US: delay, trace, simultaneous, backward

What it fixed: it specified the temporal arrangement that makes conditioning strong or absent. Because the CS works by *predicting* the US, its onset must precede the US for good learning. The four classic arrangements, in descending order of effectiveness, are a high-yield MCQ:

PARADIGM	TEMPORAL ARRANGEMENT	STRENGTH OF CONDITIONING
Delay conditioning	CS begins, then continues (overlaps) until the US is delivered	**Strongest; the standard optimal arrangement**
Trace conditioning	CS begins and *ends*, then a gap (trace interval), then the US	Weaker; must hold a memory trace of the CS across the gap
Simultaneous conditioning	CS and US presented at the same instant	Poor; the CS predicts nothing new
Backward conditioning	US precedes the CS (CS comes after)	**Weakest, often inhibitory; the CS signals the US is over**

The four Pavlovian timing paradigms ordered by efficacy: delay is best, backward is worst, and the rule is that the CS must come before and predict the US (Kaplan & Sadock 2024).

RULE **The CS must come before and predict the US.** Efficacy runs delay > trace > simultaneous > backward, with backward (US first) often inhibitory because the CS then signals the US is over.

8.5 Rescorla and Wagner (1972): conditioning is prediction, not contiguity

What it fixed: it replaced "pairing causes learning" with "surprise causes learning". Research from the late 1960s showed that a CS to US pairing does *not* guarantee conditioning. Two findings forced the change. In **blocking** (Kamin), if one CS already reliably predicts the US, a second CS added to it acquires little conditioning, because it carries no new information. In the *contingency* experiments (Rescorla), the organism treats the CS as a signal only when the US is *more probable* in the presence of the CS than in its absence; if the US occurs equally often with and without the CS, no conditioning develops despite many pairings. The lesson is that **contingency** (predictive relationship), not mere **contiguity** (co-occurrence), drives learning (Kaplan & Sadock 2024). The *Rescorla-Wagner model* made this formal: the change in a CS's associative strength on any trial is proportional to the discrepancy between the US that *actually* occurs and the US already *predicted* by all cues present, that is, the **prediction error** (or surprise). A fully predicted US drives no further learning (error is zero); an unexpected US drives strong learning; a US smaller than predicted drives inhibitory learning. This "learning is driven by prediction error" idea is the direct ancestor of the dopaminergic reward-prediction-error account

in modern computational psychiatry. What it left debated: *which* CS to US associations form easily and which resist, answered by preparedness (Kaplan & Sadock 2024).

HOLD THIS

Conditioning is driven by contingency (prediction), not mere contiguity (co-occurrence). A fully predicted US drives no further learning; surprise does, which is why blocking occurs and why classical conditioning is now read as prediction-error learning under biological constraint.

8.6 Preparedness: Seligman, taste aversion and the Garcia effect

What it fixed: not all pairings are equal; evolution biases which associations form. The Rescorla-Wagner era treated the CS and US as interchangeable, but Seligman's concept of **preparedness** (1971) holds that organisms are biologically *prepared* to associate certain CS to US combinations far more readily than others, because those associations were adaptive over evolutionary time. The decisive evidence is *conditioned taste aversion*, the *Garcia effect* (Garcia and Koelling): a rat that becomes ill hours after tasting a novel flavour will avoid that flavour thereafter, and this learning violates the ordinary rules, because it forms after a *single* trial and across a delay of hours between CS (taste) and US (nausea) that would abolish any normal Pavlovian pairing. Critically, tastes are readily associated with *illness* but not with shock, whereas audio-visual cues are readily associated with *shock* but not illness (belongingness). Preparedness explains why human phobias cluster on evolutionarily threatening objects (snakes, spiders, heights, blood) rather than on modern dangers (electric sockets, cars) that are as often paired with pain (Kaplan & Sadock 2024).

CLINICAL ANCHOR

The Garcia effect is the direct model of *anticipatory nausea and vomiting* in chemotherapy: the clinic, the smell of the infusion suite or a particular food eaten before a cycle becomes a CS that elicits nausea (CR) before the cytotoxic drug (US) is even given. Single-trial, delay-bridging taste-aversion learning is exactly why patients develop food aversions after one bad cycle.

8.7 The currently accepted position and its neural correlate

Conditioning is prediction-error learning under biological constraint, and fear conditioning has a mapped circuit. The **currently accepted** synthesis keeps Pavlov's phenomena but reinterprets them: acquisition is the accrual of associative strength driven by prediction error; extinction is *new inhibitory learning* (not erasure), which is why the response returns as spontaneous recovery, renewal (context change) and reinstatement; and preparedness sets which associations form. The **neural correlate** is best defined for conditioned fear: the *amygdala*, specifically the basolateral complex, is where CS and US converge and the fear memory is stored, with the central nucleus driving the autonomic and behavioural fear output; the *hippocampus* encodes the context (essential for context-dependent renewal); and the *ventromedial prefrontal cortex* drives extinction learning and top-down inhibition of the amygdala (the LeDoux and Milad-Quirk circuitry). Extinction being new learning rather than unlearning is precisely why exposure therapy relapses when the patient leaves the treatment context, and why extinction is

now augmented pharmacologically (for example D-cycloserine facilitating NMDA-dependent extinction consolidation) (Kaplan & Sadock 2024; Stahl 2021).

PEARL

Extinction is not forgetting. The CR that "disappeared" in the clinic can come back three ways: *spontaneous recovery* (time passes), *renewal* (the CS is met in a new context) and *reinstatement* (the US is encountered again). This single fact explains post-exposure phobia relapse, and it is the highest-value clinical pearl of the whole topic.

8.8 Clinical yield: phobia, chemotherapy nausea, addiction cue reactivity

Three disorders, one mechanism. Classical conditioning is examinable chiefly as the substrate of three clinical phenomena (Kaplan & Sadock 2024; New Oxford Textbook 2020):

Conditioned fear to phobia

A neutral object paired with an aversive event becomes a fear CS, generalises, and is maintained by avoidance (which prevents extinction). Treatment is extinction by graded exposure.

Conditioned nausea in chemotherapy

Anticipatory nausea and vomiting is a prepared, taste-aversion-like CR to clinic cues; treatable by behavioural desensitisation and by minimising cue salience.

Cue reactivity in addiction

Drug-paired cues (paraphernalia, places, people, the sight of a needle) become CSs that, via Pavlovian conditioning, elicit conditioned craving, autonomic arousal and conditioned compensatory (tolerance-like) responses, driving relapse. Cue-exposure therapy attempts extinction; the renewal effect explains why extinction in a clinic often fails to transfer to the street (cross-refer SUD).

8.9 Worked example: the dental phobia, read through the whole apparatus

Run one everyday fear through every term. A man tenses and feels his knees weaken merely on entering the waiting room of a dentist he is visiting for a routine cleaning (Tasman 2015).

Acquisition

A previous painful procedure (the US) elicited a fear-and-pain response (the UR). The dentist's office, paired with that pain, became a CS.

Conditioned response

Now the office alone (CS) elicits fear (the CR): sweating, weak knees, dread, before any drilling.

Generalisation

The fear spreads: the smell of antiseptic, the sound of a drill next door, even a white coat can now trigger it.

Higher-order conditioning

The appointment-reminder text, paired only with the already-feared office and never with pain, itself starts to trigger dread.

Preparedness

Pain-linked cues condition readily and fast; a single bad extraction can establish the phobia, echoing prepared, few-trial fear learning.

Extinction and its limits

Repeated painless visits (CS without US) extinguish the fear. But after a year's gap the dread can return (*spontaneous recovery*), or reappear at a *new* clinic (*renewal*), because extinction added a "safe" meaning without deleting the "dangerous" one.

Neural correlate

The office-fear association is held in the basolateral amygdala; the return of fear in a new clinic reflects hippocampal context coding; successful exposure depends on ventromedial prefrontal cortex driving extinction over the amygdala.

COMMON TRAP

Do not confuse classical with operant conditioning. In *classical* conditioning a *stimulus* comes to elicit an involuntary, reflexive response (the response is *elicited* by an antecedent CS: salivation, fear, nausea). In *operant* conditioning a *voluntary behaviour* is strengthened or weakened by its *consequences* (reinforcement or punishment). A second frequent slip: "extinction" means different things in the two paradigms only superficially; in both it is the decline of the response when the maintaining event (the US, or the reinforcer) is withheld, but in neither does it erase the original learning.

MNEMONIC

Timing efficacy: "Delay Dominates, Backward is Broken" (D > T > S > B)

Delay (overlapping CS) is strongest, then Trace (gap), then Simultaneous, then Backward (weakest, often inhibitory). The single rule underneath: the CS must come **before** and **predict** the US. For the reflex terms, remember the US to UR pair is *unlearned* (both begin with the natural reflex) and the CS to CR pair is *conditional* on training.

INDIA

Cue-reactivity conditioning is directly usable in Indian addiction practice, where alcohol and tobacco (including gutka and khaini) dependence is highly cue-bound: the tea-shop, the after-meal ritual, the paan corner and specific companions act as powerful CSs for craving. Relapse-prevention counselling that identifies and plans around these conditioned cues (a behavioural, low-cost intervention) fits resource-limited settings well, and the renewal effect warns that skills learned in an inpatient de-addiction ward may not transfer home unless practised in the patient's own high-risk contexts.

GOOD TO REMEMBER

- **Classical (Pavlovian, respondent) conditioning:** a neutral stimulus paired with a US comes to elicit an anticipatory CR; the model of conditioned fear, nausea and craving.
- **Pavlov (1927):** reflex vocabulary (US, UR, CS, CR) and the core phenomena; the dog-bell-salivation paradigm.
- **US / UR:** unconditioned (unlearned) stimulus and its innate reflex response (food to salivation).
- **CS / CR:** conditioned (learned) stimulus and its trained response (bell to salivation); the CR is usually weaker than the UR.
- **Acquisition:** the initial phase of learning and strengthening the CR over CS to US pairings.
- **Extinction:** decline of the CR when the CS is repeatedly presented without the US; new inhibitory learning, not erasure; basis of exposure therapy.
- **Spontaneous recovery:** reappearance of an extinguished CR (diminished) after a rest interval; proves the original learning survives.
- **Stimulus generalisation / discrimination:** spread of the CR to similar stimuli / learned narrowing to the exact CS.
- **Higher-order (second-order) conditioning:** a new neutral stimulus paired with an established CS becomes CR-eliciting without the US.
- **Watson and Rayner (1920), Little Albert:** conditioned fear of a white rat (US = loud noise) that generalised; empirical anchor of the behaviourist manifesto (Watson 1913).
- **Timing paradigms:** delay (strongest) > trace > simultaneous > backward (weakest); CS must precede and predict the US.
- **Rescorla-Wagner (1972):** learning is driven by prediction error (surprise); explains blocking; contingency, not mere contiguity, produces conditioning.
- **Blocking (Kamin):** a CS that predicts nothing new (a second cue already predicts the US) acquires little conditioning.
- **Preparedness (Seligman 1971):** organisms are biologically biased toward some CS to US associations; explains the object-selectivity of phobias.
- **Garcia effect / conditioned taste aversion:** single-trial, long-delay taste-to-illness learning; belongingness (taste with illness, not shock); model of chemotherapy anticipatory nausea.
- **Neural correlate:** basolateral amygdala stores the fear CS to US association; hippocampus codes context (renewal); ventromedial prefrontal cortex drives extinction.

9 · Operant conditioning and reinforcement schedules

Learning by consequence, and the timetables that shape it. Where classical conditioning is about associations between stimuli, **operant conditioning** (also called instrumental conditioning) is about how the consequences of a voluntary action change the future probability of that action. A behaviour that operates on the environment is an **operant**; if its consequence strengthens it, the consequence is a **reinforcement**, and if the consequence weakens it, the consequence is a **punishment**. The exam wants this as an evolutionary sequence of ideas: Thorndike's law of effect from the puzzle box, Skinner's formalisation of the operant and the four-cell reinforcement-versus-punishment matrix, the derived techniques (shaping, chaining, extinction, stimulus control), the four schedules of reinforcement with their signature response patterns, and the modern reward-learning synthesis anchored to dopamine.

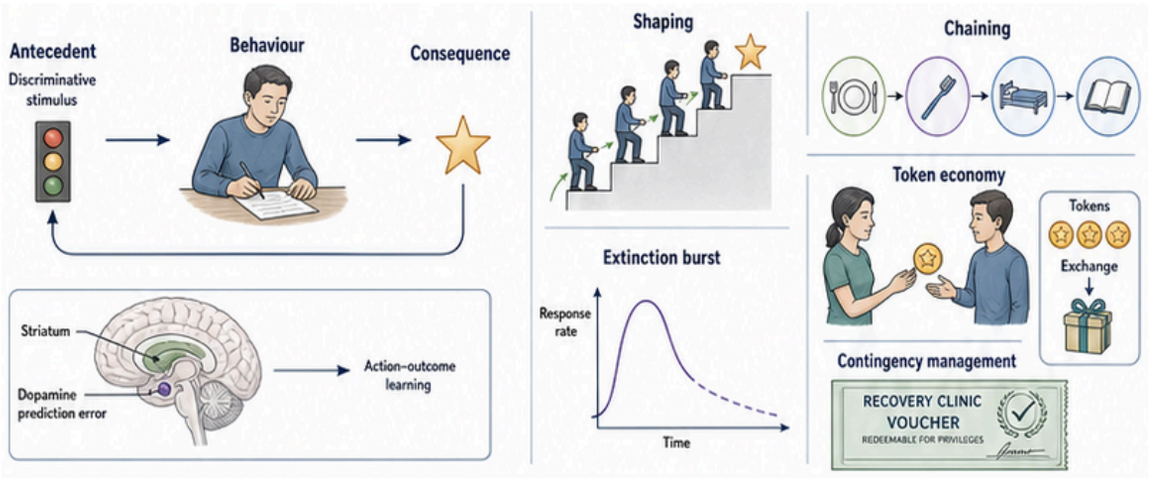


Fig 5.10 Operant learning: consequences select behaviour.

This is one of the highest-yield theory blocks in the behavioural-science paper because the whole of behaviour therapy, applied behaviour analysis, token economies and contingency management is built on it (Kaplan & Sadock 2024). Treat the four-cell matrix and the four schedules as the two must-reproduce items; the clinical arm (token economy, contingency management) is developed in the behaviour-therapy topic and is only cross-referenced here.

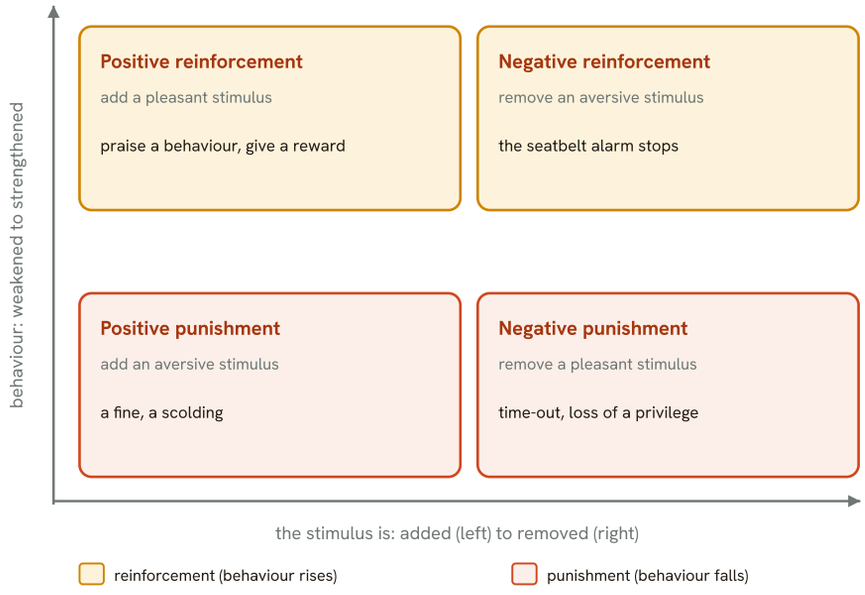


Fig 5.11 The operant matrix. "Positive" and "negative" mean the stimulus is added or removed, not good or bad; "reinforcement" and "punishment" mean the behaviour is strengthened or weakened. Reading the two axes gives the four operations: positive reinforcement (add a pleasant stimulus), negative reinforcement (remove an aversive one), positive punishment (add an aversive one) and negative punishment (remove a pleasant one). The commonest exam trap is confusing negative reinforcement, which strengthens behaviour, with punishment, which weakens it.

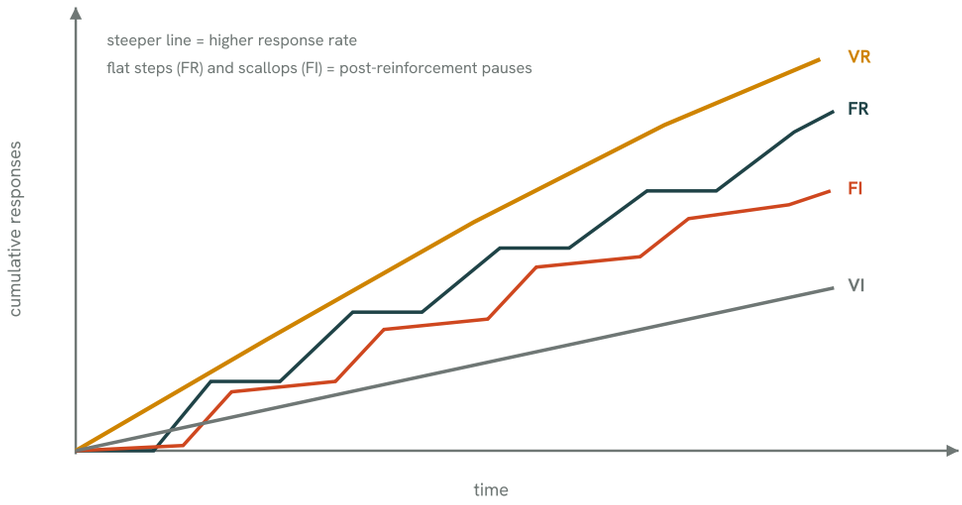


Fig 5.12 The four schedules of reinforcement as cumulative-response curves. Ratio schedules (reinforce after a number of responses) drive higher rates than interval schedules (reinforce the first response after a time). Variable schedules produce steady responding, while fixed schedules produce a pause after each reinforcement: the fixed-ratio step and the fixed-interval scallop. The variable-ratio schedule (VR) gives the highest, steadiest rate and the greatest resistance to extinction, which is why it is the schedule of gambling.

9.1 Definition: the operant and the law of consequences

A behaviour controlled by its consequences, not by an antecedent trigger. The defining contrast with Pavlovian learning is that a respondent is *elicited* and controlled by an antecedent stimulus, whereas an **operant** is *emitted* and controlled by its consequences (Kaplan & Sadock 2024). A rat is not compelled to press a lever the way it is compelled to salivate to food; it presses because pressing has been followed by a reinforcer, and if the reinforcer is withdrawn the pressing stops. Clinically this distinction is the first fork in any behavioural analysis: if a child acts out in class, the clinician asks whether the behaviour is a respondent (change its antecedents) or an operant (change its consequences) before intervening (Kaplan & Sadock 2024).

9.2 Theory one: Thorndike's law of effect (the puzzle box)

The first systematic account, from a hungry cat in a box. Edward Thorndike (1874 to 1949) placed a hungry cat in a "puzzle box" from which it could escape to food by operating a latch (Thorndike 1913, cited in Tasman Psychiatry). At first the cat unlatched the door only by accident; on being replaced it escaped progressively faster, until it operated the latch immediately. From this Thorndike derived the **law of effect**: a response followed by a satisfying consequence is strengthened (stamped in) and one followed by an annoying consequence is weakened (Kaplan & Sadock 2024). **What it fixed and what it left debated.** The law of effect fixed the absence of any mechanism for goal-directed learning, giving a lawful relation between action and outcome. What it left debated was the internal process: Thorndike's "stamping in" was a blunt strengthening metaphor with no account of what the animal actually learned, a gap the later action-outcome view had to fill.

9.3 Theory two: Skinner formalises the operant

The free operant and the experimental analysis of behaviour. In the late 1930s B. F. Skinner (Skinner 1938) enclosed a rat in a chamber (the operant chamber, popularly the "Skinner box") with a lever it was free to press repeatedly whenever it "wanted," earning a food pellet each time (Kaplan & Sadock 2024). This "free operant" arrangement, unlike Thorndike's discrete escape trials, let response *rate* be measured continuously and made behaviour a lawful function of its scheduled consequences. Skinner held that operant principles, reinforcement, punishment, extinction and stimulus control, could in principle explain virtually all human behaviour (Skinner 1938, cited in Tasman Psychiatry), and it was the specific application of these principles that became *applied behavioural analysis* and the engine of the 1950s to 1960s behaviour-therapy movement (Kaplan & Sadock 2024).

9.4 The core four-cell matrix: reinforcement versus punishment

Two questions: is a stimulus added or removed, and does the behaviour strengthen or weaken. The single most examined structure in this topic is the two-by-two matrix built from crossing *add versus remove* a stimulus with *strengthen versus weaken* the behaviour. "Positive" and "negative" here mean added and removed (like arithmetic signs), *not* pleasant and unpleasant; "reinforcement" always strengthens and "punishment" always weakens.

Positive reinforcement

An appetitive stimulus is *added* after the behaviour, strengthening it. A child praised for tidying their room tidies more (Tasman Psychiatry).

Negative reinforcement

An aversive stimulus is *removed* or avoided by the behaviour, strengthening it. A student studies to avoid failing; taking a painkiller removes pain and so is reinforced. Note it *increases* behaviour and is not punishment.

Positive punishment

An aversive stimulus is *added* after the behaviour, weakening it. A reprimand added after talking back.

Negative punishment

An appetitive stimulus is *removed* after the behaviour, weakening it. The classic form is *time-out from reinforcement* and the loss of privileges or a fine (Kaplan & Sadock 2024).

	STIMULUS ADDED (POSITIVE)	STIMULUS REMOVED (NEGATIVE)
Behaviour strengthened	Positive reinforcement (add appetitive: praise, food)	**Negative reinforcement (remove aversive: escape, avoidance)**
Behaviour weakened	Positive punishment (add aversive: reprimand, shock)	**Negative punishment (remove appetitive: time-out, fine)**

The columns are the discriminator most candidates get wrong: the sign is about adding versus removing a stimulus, never about the stimulus being nice or nasty. Negative reinforcement increases behaviour; punishment decreases it.

HOLD THIS

Positive/negative = a stimulus added or removed; reinforcement/punishment = behaviour up or down. Decode the two words independently, and never call negative reinforcement (which increases behaviour) a punishment.

COMMON TRAP

Negative reinforcement is *not* punishment. Both "negatives" involve removal, but negative reinforcement removes an *aversive* stimulus and so *increases* behaviour, whereas negative punishment removes an *appetitive* stimulus and so *decreases* it. The second trap: punishment suppresses a behaviour only temporarily and teaches no alternative, so a reprimanded child may suppress the urge to talk back yet learn no better way to communicate, or may even escalate if the scolding is the only attention (a positive reinforcer) on offer (Tasman Psychiatry).

9.5 Primary versus secondary (conditioned) reinforcers

Innately rewarding versus reward by association. A *primary reinforcer* is biologically based and rewarding without prior learning: food, water, warmth, nurturance (Tasman Psychiatry). A *secondary* or *conditioned reinforcer* has no intrinsic value and becomes reinforcing only by being paired with a primary reinforcer: money, praise, a grade, and the tokens of a token economy (Kaplan & Sadock 2024). A lollipop reinforces a child by primary means; a word of praise reinforces by conditioned means (Tasman Psychiatry). A *generalised* conditioned reinforcer such as money is one paired with many different primaries, which is why it reinforces so broadly. This is the conceptual basis of the token economy, developed in the behaviour-therapy topic.

9.6 The Premack principle

A high-probability behaviour reinforces a low-probability one. Rather than testing reinforcers by trial and error, the **Premack principle** (David Premack) predicts them from preference: if you rank a person's freely chosen activities by time spent, access to a *more-preferred* activity will reinforce a *less-preferred* one (Kaplan & Sadock 2024). For a child who prefers pinball to candy, pinball reinforces candy-eating; for one with the reverse preference, candy reinforces pinball. In everyday form this is "grandma's rule," first you eat your vegetables, then you get dessert. The refinement is that even a low-preference activity can reinforce a higher one if the person has been deprived of it below baseline (the response-deprivation account) (Kaplan & Sadock 2024).

9.7 Shaping by successive approximation, and chaining

Building a behaviour that never occurs spontaneously. **Shaping** is the operant technique of reaching a behavioural goal in stepwise fashion by selectively reinforcing closer and closer approximations to the desired behaviour (Kaplan & Sadock 2024). To teach a rat to press a high lever, you first reinforce facing it, then approaching, then touching, then pressing, raising the criterion at each step. Clinically a physician's praise for each successive step of a health-promoting change shapes a durable lifestyle change (Tasman Psychiatry). *Chaining* is the linked cousin: a complex sequence is built by joining individual responses, each response producing the discriminative stimulus for the next and the terminal reinforcer maintaining the whole chain (as in teaching a multi-step self-care routine, often taught in reverse as backward chaining).

9.8 Extinction and the extinction burst

Withhold the reinforcer and the behaviour declines, but not smoothly. *Extinction* is the decline of an operant when its reinforcer is no longer delivered (Kaplan & Sadock 2024): a tantrum maintained by parental attention fades when attention is consistently withheld. The clinically vital caveat is the *extinction burst*, a transient *increase* in the frequency, intensity and variability of the behaviour immediately after reinforcement stops, before it declines. Parents who relent during the burst inadvertently reinforce a louder tantrum on a leaner schedule. Extinction does not erase the original learning: *spontaneous recovery*, *renewal*, *reinstatement*, *rapid reacquisition* and *resurgence* (return of an old behaviour when a replacement is itself extinguished) all show the learning is suppressed and context-bound, not destroyed (Kaplan & Sadock 2024). This is exactly why exposure gains relapse and why relapse-prevention work matters.

9.9 Discriminative stimuli and stimulus control

When behaviour comes under the control of a signal. A *discriminative stimulus* (S-D) is a cue in whose presence a response is reinforced; behaviour that reliably occurs in its presence and not its absence is said to be under *stimulus control* (Kaplan & Sadock 2024). Stimulus control is learned by *discrimination*, distinguishing two similar stimuli because one signals reinforcement and the other does not: a child learns to leave class when the school bell rings but not when a telephone rings (Tasman Psychiatry). Its complement is *generalisation*, responding to stimuli resembling the trained S-D. Clinically, stimulus-control procedures are the backbone of insomnia treatment (the bed becomes a discriminative stimulus for sleep, not for lying awake) and of trigger management in addiction.

9.10 Continuous versus partial reinforcement

Every response, or only some. Under a *continuous* reinforcement schedule every target response is reinforced; this produces the fastest acquisition of a new behaviour. Under a *partial* (intermittent) schedule only some responses are reinforced, which produces slower acquisition but far greater persistence. In the natural world few actions are reinforced every time, so partial schedules are the rule (Kaplan & Sadock 2024). The best practice is therefore to acquire a new behaviour on continuous reinforcement, then thin to a partial schedule to maintain it.

9.11 The four schedules of reinforcement and their response patterns

Ratio versus interval, crossed with fixed versus variable. Partial schedules are classified on two axes: whether reinforcement depends on the *number* of responses (ratio) or on *time* elapsed (interval), and whether the requirement is *fixed* (constant) or *variable* (averages a value but varies trial to trial) (Kaplan & Sadock 2024). Ratio schedules tie reinforcement directly to work done and so drive higher response rates than interval schedules.

Fixed ratio (FR)

Every nth response is reinforced (FR-10 reinforces every 10th). High steady rate with a brief *post-reinforcement pause* after each delivery. Everyday form: piece-rate pay, one reward per fixed quota.

Variable ratio (VR)

Reinforcement after an *average* number of responses that varies unpredictably. Produces the *highest, steadiest response rate and the greatest resistance to extinction* of all four; this is the gambling / slot-

machine schedule, because the next pull might always be the payoff (Kaplan & Sadock 2024).

Fixed interval (FI)

The first response after a fixed time (say 60 seconds) is reinforced. Produces the classic *fixed-interval scallop*: responding nearly stops just after reinforcement, then accelerates as the interval's end approaches. Everyday form: cramming that intensifies as a scheduled exam nears.

Variable interval (VI)

The first response after an *average, varying* interval is reinforced. Produces a low but very steady, extinction-resistant rate. Everyday form: repeatedly checking email, since a new message becomes available at unpredictable times (Kaplan & Sadock 2024).

SCHEDULE	REINFORCED WHEN	RESPONSE PATTERN	EVERYDAY EXAMPLE
Fixed ratio (FR)	After a set number of responses	High rate, post-reinforcement pause	Piece-rate pay
Variable ratio (VR)	After an average, varying number	**Highest rate, most resistant to extinction (the gambling schedule)**	Slot machine
Fixed interval (FI)	First response after a set time	**Scalloped: pause then acceleration toward interval's end**	Cramming before a dated exam
Variable interval (VI)	First response after an average, varying time	Low but steady, extinction-resistant	Checking email for new messages

Discriminators: ratio schedules out-rate interval schedules (reinforcement tracks work); variable schedules out-persist fixed ones (unpredictability blocks the "just been reinforced" pause). Variable ratio is the peak on both counts.

RULE **Ratio out-rates, variable out-lasts, so the variable-ratio schedule wins on both: the highest, most extinction-resistant rate.** It is the gambling schedule; FI gives the scallop.

9.12 The partial-reinforcement extinction effect

Intermittently reinforced behaviour resists extinction longest. The *partial-reinforcement extinction effect* (PREE) is the paradoxical finding that a behaviour reinforced only intermittently persists *longer* when all reinforcement is withdrawn than one that had been continuously reinforced (Kaplan & Sadock 2024). It is paradoxical because the more sparsely rewarded behaviour is the more durable one. One account is that partial reinforcement trains responding *through* non-reward and frustration, so a run of unrewarded responses does not signal "extinction has begun." This is the mechanistic reason variable-ratio behaviours like gambling are so hard to extinguish, and why a tantrum a parent reinforces only occasionally (giving in "just this once") becomes especially entrenched.

TRAP The partial-reinforcement extinction effect is counter-intuitive: **intermittently reinforced behaviour resists extinction longer than continuously reinforced behaviour**, so the more sparsely rewarded habit is the more durable one.

9.13 Escape and avoidance learning; two-factor theory

Both are negative reinforcement, but avoidance is subtler. In *escape* learning the response terminates an aversive event already present (leaving a noisy room stops the noise). In *avoidance* learning the response prevents an aversive event that has not yet occurred, which raises a puzzle: how can the non-occurrence of something reinforce? Mowrer's *two-factor (two-process) theory* answers it by combining both learning types: first, Pavlovian conditioning makes a warning cue elicit fear; second, the avoidance response is negatively reinforced by the *reduction of that conditioned fear* (Kaplan & Sadock 2024). This is the standard learning model of the anxiety disorders: the obsessive-compulsive patient washes to reduce contamination anxiety, the agoraphobic stays home to escape fear of panic-associated places, and the person with bulimia vomits to reduce learned post-meal anxiety, each avoidance response reinforced by fear reduction and thereby maintained (Kaplan & Sadock 2024). Because the feared outcome is prevented, the belief that it would have happened is never disconfirmed, which is precisely why exposure with response prevention is the treatment.

> **CLINICAL ANCHOR**
>
> The negative-reinforcement trap runs through psychiatry. Compulsions, safety behaviours, avoidance in PTSD, and much substance use are maintained because the behaviour removes an aversive internal state (anxiety, craving, withdrawal), which reinforces it and blocks new learning. The therapeutic move, exposure with response prevention, deliberately breaks the negative-reinforcement contingency so the feared consequence can be disconfirmed and the fear extinguished.

9.14 Currently accepted position: reward learning as action-outcome association

From "stamping in" to a computational reward-learning account. The **currently accepted** view has moved beyond Thorndike's mechanical strengthening. The modern synthesis holds that the organism learns an *action to outcome* association (much as Pavlovian learning is stimulus to outcome), and that reinforcers act less as automatic "stampers" than as guides and motivators of goal-directed behaviour (Kaplan & Sadock 2024; Bouton 2016). A second consensus is the goal-directed versus habitual distinction: early instrumental behaviour is goal-directed and sensitive to the value of its outcome, whereas heavily overtrained behaviour becomes an outcome-insensitive *habit*, a shift central to modern models of compulsion and addiction. The quantitative law of effect (the matching law) further specifies that a behaviour's rate depends not only on its own reinforcement but on the reinforcement available for all competing behaviours, which is why enriching a young person's environment with alternative reinforcers lowers the rate of a maladaptive one (Kaplan & Sadock 2024).

9.15 Neural correlate: the dopamine reward-prediction-error system

Where reinforcement is computed in the brain. The *neural correlate* of operant reinforcement is the midbrain dopamine system. Phasic firing of dopamine neurons in the *ventral tegmental area* and *substantia nigra pars compacta*, projecting to the *ventral striatum (nucleus accumbens)* and prefrontal cortex, encodes a *reward prediction error*: dopamine rises when an outcome is better than predicted, is unchanged when it is exactly as predicted, and dips when it is worse than

predicted (Schultz 2016). This teaching signal is the biological implementation of the law of effect and explains why unpredictable schedules are so potent: a variable-ratio payoff is by design a persistent source of positive prediction error, which is the neurobiology behind gambling's grip and the dopaminergic reward pathway's central role in every substance use disorder. The goal-directed-to-habitual transition maps onto a shift from ventral (accumbens) to dorsolateral striatal control.

9.16 Worked example: an outpatient with alcohol dependence

The whole apparatus applied to one patient. A man drinks each evening. *Operant analysis:* drinking is an operant maintained by *negative reinforcement*, it removes the aversive states of anxiety and mounting withdrawal, and by *positive reinforcement*, the euphoriant and social rewards. *Discriminative stimuli:* the pub, payday and certain company are S-D cues that occasion drinking; stimulus-control work removes or re-signals them. *Schedule:* the intoxication reward arrives on a rich, near-continuous schedule, whereas the harms are delayed and intermittent, so present reinforcement dominates. *Extinction and its burst:* early abstinence brings a surge in craving and drink-seeking (an extinction burst) before decline, which must be anticipated so a lapse is not read as failure. *Shaping and Premack:* abstinence is built by reinforcing successive approximations (a dry evening, then a dry week), and higher-probability healthy activities are arranged to reinforce lower-probability recovery tasks. *Neural:* the alcohol cue drives a dopaminergic prediction-error spike in the ventral striatum, and over years control shifts toward dorsal-striatal habit, which is why willpower alone fails. The formal clinical delivery of these contingencies, contingency management and token systems, is developed in the behaviour-therapy topic.

MNEMONIC

"Positive = ADD, Negative = REMOVE; Reinforce = UP, Punish = DOWN. Schedules: VR is king."

For the matrix, decode the two words independently: the first word is whether a stimulus is added (positive) or removed (negative); the second is whether behaviour goes up (reinforcement) or down (punishment). For the schedules, remember **"Ratio out-rates, Variable out-lasts"**: ratio schedules give higher rates, variable schedules give more persistence, so *variable ratio* wins on both, the gambling schedule. For the patterns: **FI = scallop, VR = gambling.**

INDIA

Operant contingencies are culturally and economically loaded. In Indian family systems the most powerful reinforcers are frequently social and relational (parental approval, community standing) rather than material, so a behavioural plan routed through the family reinforcement network usually out-performs an individually addressed token scheme. In gambling and alcohol work, the variable-ratio structure of local betting and the ready, near-continuous availability of alcohol in many settings are the contingency realities any plan must plan around.

PEARL

The one-line arc for an examiner: Thorndike's law of effect (behaviour is selected by its consequences) was formalised by Skinner into the operant and the reinforcement-punishment matrix, elaborated into the techniques (shaping, extinction, stimulus control) and the four schedules (with variable ratio the most powerful), and is now understood as action-outcome reward learning implemented by the dopamine reward-prediction-error signal. Negative reinforcement, not punishment, is the thread that ties compulsions, avoidance and addiction together in the clinic.

GOOD TO REMEMBER

- **Operant conditioning:** behaviour changed by its consequences; the operant is emitted and controlled by consequences, unlike the elicited respondent.
- **Thorndike (1874 to 1949), law of effect (1913):** puzzle-box cats; satisfying consequences stamp a response in, annoying ones stamp it out.
- **Skinner (1938):** the free operant, operant chamber ("Skinner box"), and the experimental analysis of behaviour; founder of the reinforcement-schedule framework.
- **Positive reinforcement:** add appetitive stimulus, behaviour increases (praise, food).
- **Negative reinforcement:** remove/avoid aversive stimulus, behaviour increases (escape, avoidance); NOT punishment.
- **Positive punishment:** add aversive stimulus, behaviour decreases (reprimand, shock).
- **Negative punishment:** remove appetitive stimulus, behaviour decreases (time-out, fine).
- **Primary vs secondary reinforcer:** primary = innately rewarding (food, water); secondary/conditioned = rewarding by pairing (money, praise, tokens).
- **Premack principle (David Premack):** access to a more-preferred behaviour reinforces a less-preferred one ("grandma's rule").
- **Shaping:** reinforcing successive approximations to a target behaviour; chaining links responses into a sequence.
- **Extinction:** withholding reinforcement declines the behaviour; extinction burst = transient surge first; learning suppressed, not erased (spontaneous recovery, renewal, resurgence).
- **Discriminative stimulus / stimulus control:** a cue signalling reinforcement gains control of the behaviour; learned by discrimination, opposed by generalisation.
- **Fixed ratio (FR):** every nth response; high rate with post-reinforcement pause.
- **Variable ratio (VR):** average varying number; highest rate, most resistant to extinction; the gambling schedule.
- **Fixed interval (FI):** first response after a set time; the "scallop" (pause then acceleration).
- **Variable interval (VI):** first response after an average varying time; low but steady, extinction-resistant.
- **Continuous vs partial; PREE:** partial (intermittent) reinforcement resists extinction longer than continuous, the partial-reinforcement extinction effect.
- **Escape vs avoidance; two-factor theory (Mowrer):** Pavlovian fear plus operant fear-reduction; the learning model of anxiety disorders and compulsions.
- **Neural correlate:** dopamine reward-prediction-error signal (VTA/SNc to ventral striatum), Schultz 2016; goal-directed to habitual shift = ventral to dorsal striatum.

10 · Social learning theory (Bandura)

How behaviour is learned by watching, not only by doing. **Social learning** theory, developed by Albert Bandura at Stanford, is the bridge between the older behaviourism of Watson and

Skinner and the cognitive psychology that followed. Its central claim is that people acquire behaviour, emotions and attitudes by **observational learning**, watching a model and its consequences, without ever needing direct reinforcement of their own. Bandura integrated the principles of classical and operant conditioning with observational learning to formulate the tenets of the theory (Kaplan & Sadock 2024), and in doing so placed *cognition*, thoughts, images, beliefs and expectations, at the centre of learning. For the exam, treat this as the pivot on which twentieth-century learning theory turns: the move from **radical behaviourism**, which refused to study the unobservable mind, to the *social-cognitive* account in which expectancies mediate every learned response.

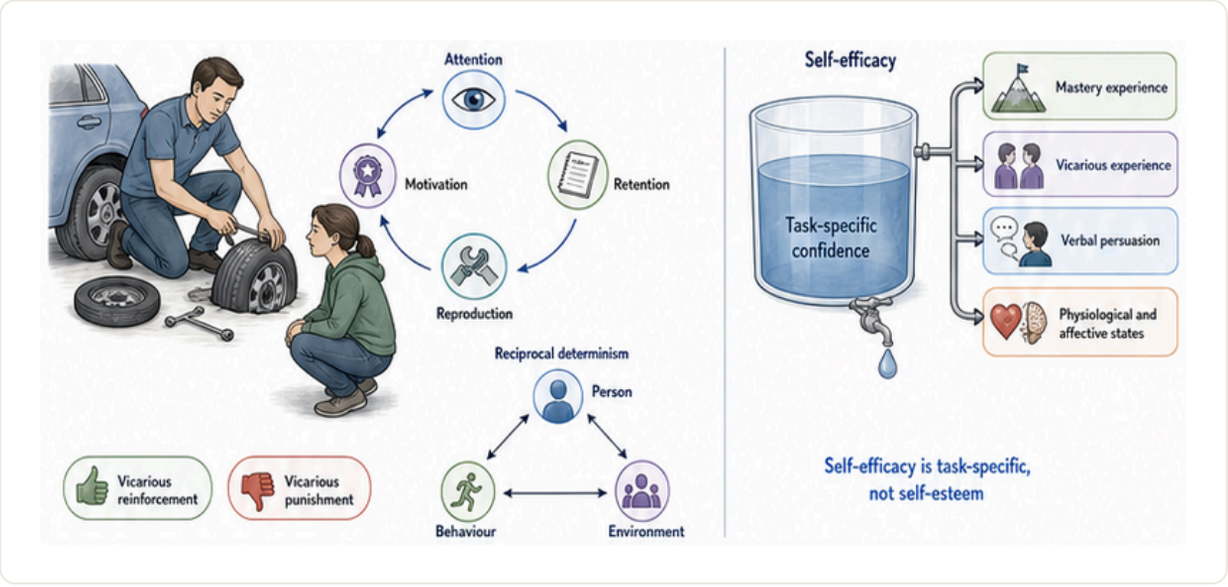


Fig 5.13 Observational learning and self-efficacy.

The clinical reach is large and directly examinable. Bandura's work is one of the three pillars of the 1960s cognitive revolution (with Beck and Ellis), it supplies the theoretical basis for modelling and participant-modelling treatments, and its later concept of **self-efficacy** underpins cognitive behaviour therapy, graded exposure and essentially every behaviour-change intervention now in use (Kaplan & Sadock 2024). Follow the standard arc: define the theory, walk the era in the order it appeared (radical behaviourism, then Rotter and Bandura's social learning, then Bandura's mature social-cognitive theory with reciprocal determinism and self-efficacy), state the currently accepted position, name its behavioural and neural correlate, and close with a worked clinical example.

10.1 What social learning theory claims

Learning through observation, mediated by cognition. Social learning theory (later renamed by Bandura himself as *social-cognitive theory*) holds that most human behaviour is learned by observing others and the consequences their behaviour brings, then reproducing or inhibiting it accordingly. Through observational learning, the process of observing the actions and reactions of others can effect a change in an individual's behaviour without that individual needing direct experience of the situation (Kaplan & Sadock 2024). The theory keeps the conditioning laws of the behaviourists but adds an indispensable cognitive layer: what is stored is not a reflex but a

symbolic *representation* of the modelled act and an *expectancy* about its likely outcome. This is why it is classed as a *social-cognitive* rather than a purely behavioural theory.

10.2 The starting point: radical behaviourism and its limit

The mind ruled out of court. The theory Bandura reacted against was **radical behaviourism**. Steeped in the tradition of Watson, radical behaviourists eschewed the study of cognition, holding that thought processes were mentalistic constructs not observable or verifiable by others and therefore outside science; directly changing behaviour was held to change the associated thoughts and feelings, so direct access to thoughts was deemed unnecessary (Kaplan & Sadock 2024). **What it left debated.** Its fatal gap was that it could not explain learning that occurs with no direct reinforcement of the learner at all, a child acquiring a whole complex behaviour merely by watching an adult, or a fear caught from a frightened parent. It also had no account of the obvious fact that the same reinforcement produces different learning in different people, because it refused to admit the mediating cognitions (expectancies, beliefs) that make the difference. Bandura's theory was built precisely to close that gap.

10.3 Rotter's social learning theory: expectancy and locus of control

The first cognitive amendment to reinforcement. Julian Rotter (1916 to 2014) offered an early version of social learning theory that first inserted cognition into the reinforcement account. Rotter proposed that human behaviour is guided not only by the actual history of reinforcement but also by plans, goals and *expectations* of success: a person performs a behaviour if they believe it is likely to lead to a valued goal (Kaplan & Sadock 2024). From this he derived **locus of control**: individuals with a history of success develop a generalised expectancy that they can control their lives (an *internal* locus of control), whereas those whose efforts have generally failed come to believe outcomes are a matter of luck or the decisions of powerful others (an *external* locus of control). **What it fixed and left open.** Rotter fixed behaviourism's silence on expectancy, showing that subjective probability of reward, not just reward history, drives behaviour. What he left for Bandura was the *mechanism of acquisition*: Rotter explained why we choose an act, but not how a novel behaviour is learned in the first place without ever performing it.

10.4 Bandura's observational learning and modelling

Acquisition without performance. Bandura's decisive addition was **modelling**: a novel behaviour can be acquired in full simply by observing a model perform it, before the observer has ever enacted or been reinforced for it. This is *observational* or *vicarious* learning, and it explains the transmission of language, skills, social conventions, fears and aggression far more economically than trial-and-error conditioning could. Bandura distinguished *acquisition* (learning the behaviour, which happens on observation) from *performance* (whether it is later enacted, which depends on expected consequences), a distinction the behaviourists could not draw. The Companion to Psychiatric Studies lists the empirically established optimum conditions for observational learning: *live modelling*, *perceived similarity* between observer and model, *seeing the model receive reinforcement*, and *familiarity* with the model (Companion to Psychiatric Studies).

10.5 The four subprocesses of observational learning

Attention, retention, reproduction, motivation. Bandura specified that observational learning proceeds through four component subprocesses, each of which must succeed for a modelled behaviour to be learned and shown. This is a classic exam list.

Attention

The observer must attend to the model and perceive the relevant features of the behaviour. Attention is raised by model salience, prestige, perceived similarity to the self, and the observer's own arousal and interest.

Retention

The observed behaviour must be encoded and stored as a symbolic (imaginal or verbal) representation in memory, so it can be recalled later in the model's absence. Rehearsal strengthens retention.

Reproduction (motor reproduction)

The observer must be physically and cognitively capable of converting the stored representation into action, comparing their own performance against the remembered model and refining it.

Motivation (motivational processes)

Even a fully acquired behaviour is performed only if the observer is motivated to do so. Motivation is supplied by anticipated consequences: direct reinforcement, *vicarious* reinforcement (seeing the model rewarded), and self-reinforcement.

10.6 Vicarious reinforcement and vicarious punishment

Consequences that fall on the model steer the observer. A cornerstone of the theory is that reinforcement need not fall on the learner. In **vicarious** reinforcement, seeing a model rewarded for a behaviour increases the observer's likelihood of performing it; in vicarious punishment, seeing a model punished decreases it. The individual thus learns not only on the basis of their own experience but also through vicarious reinforcement from the observation of others (Kaplan & Sadock 2024). This mechanism preserves the acquisition-performance distinction: watching a model be punished does not erase the observer's *learning* of the behaviour, it merely suppresses its *performance*, which reappears if the incentive changes. Vicarious reinforcement is the cognitive engine that lets the four subprocesses translate observation into action.

10.7 The Bobo doll experiments

Aggression transmitted by modelling. The empirical anchor of the theory is the Bobo doll series (Bandura, Ross and Ross 1961). Bandura produced a film of one of his students attacking an inflatable toy, a Bobo doll, and showed it to young children; when the children were subsequently given access to the doll, they copied the student's behaviour and attacked the doll (Companion to Psychiatric Studies). In a more striking extension, the experiment was repeated using a *live* clown instead of the doll (Bandura et al. 1963), and again the children copied the aggressive actions and struck the live clown after watching the film (Companion to Psychiatric Studies). The findings gave scientific legitimacy to the social learning perspective (Kaplan & Sadock 2024) by demonstrating learning of a complex behaviour, physical aggression, purely by observation, in the complete absence of any direct reinforcement of the child. Bandura's demonstration of modelling effects in the 1960s is the reason concern over depicted violence in

television, film, social media and video games is framed in observational-learning terms (Kaplan & Sadock 2024).

COMMON TRAP

Do not say the Bobo doll study proves aggression is simply "imitated." Its precise and examinable point is that a complex behaviour was *acquired by observation with no direct reinforcement of the child*, refuting the radical-behaviourist requirement that the learner be reinforced. Note also that later variants manipulated *vicarious* consequences (models rewarded, punished, or neither): children who saw the model punished showed less spontaneous aggression, yet reproduced it when later offered an incentive, proving the behaviour had been *learned* though not *performed*.

10.8 Reciprocal determinism

Person, behaviour and environment interact both ways. Bandura's mature social-cognitive theory replaced the behaviourists' one-way causal arrow (environment shapes behaviour) with **reciprocal determinism**: the person (cognitions, expectancies, self-efficacy beliefs), their behaviour, and the environment operate as three factors that continuously and mutually influence one another. The environment shapes behaviour, but behaviour also selects and alters the environment, and both are filtered through and act upon the person's cognitions. K&S illustrate the interaction directly: the same observed event, a peer being teased by a class bully, is learned differently depending on the child (an inhibited child thinks "that could happen to me" and withdraws in anxiety; a rambunctious child focuses on the bully and imitates the aggression), and the environment's response to each child's reaction then feeds back to reinforce or extinguish it (Kaplan & Sadock 2024). Reciprocal determinism is what makes the theory *interactionist* rather than environmentally deterministic.

10.9 Self-efficacy: definition and the four sources

The belief in one's own capability to execute a behaviour. Bandura's most influential single concept is **self-efficacy**: a person's belief in their capability to organise and execute the actions needed to manage a given situation or attain a goal. It is not global self-esteem but a task-specific and situation-specific judgement of "can I do this?" In the social-cognitive account of personality, the generalised sense of self-efficacy is treated as the *central individual-difference variable*: people are characterised primarily by their beliefs in their ability to control their lives, because those beliefs powerfully determine the effort they invest in adapting to their surroundings (Kaplan & Sadock 2024). Bandura held that efficacy beliefs are built from four sources, in descending order of potency.

Mastery experience (performance accomplishment)

The most powerful source: succeeding at the task oneself. Repeated personal success raises efficacy; repeated failure, especially early, lowers it. This is why graded, success-yielding exposure is the strongest efficacy-builder in therapy.

Vicarious experience (modelling)

Seeing similar others succeed ("if they can do it, so can I") raises efficacy, most strongly when the model is perceived as similar to the self. The direct clinical link to observational learning.

Verbal (social) persuasion

Being credibly encouraged or persuaded that one is capable. Weaker and more fragile than the first two, but it can raise the effort a person is willing to try.

Physiological and affective state

The person reads their own bodily and emotional arousal as information: a pounding heart and dread are interpreted as evidence of incapacity, so reducing arousal (or reappraising it) raises efficacy. Explains why calming physiology aids performance.

RULE **Mastery experience is the most powerful source of self-efficacy.** This is why graded, success-yielding exposure, not reassurance, is the strongest efficacy-builder in therapy.

10.10 Self-efficacy and self-perpetuating maladaptive behaviour

Why low efficacy is self-confirming. Low self-efficacy is not merely a symptom; in Bandura's account it is a cause that maintains disorder. Individuals who lack a strong sense of self-efficacy in social situations avoid them, and as a consequence fail to learn the social skills that would have enhanced their confidence, so self-defeating behaviours that look irrational from outside are understandable within the dynamics of learning (Kaplan & Sadock 2024). The avoidance protects the low efficacy belief from disconfirmation, a vicious cycle that maps precisely onto the maintenance model of the anxiety disorders and social phobia. Raising self-efficacy, chiefly through mastery experiences delivered as graded exposure, breaks the loop.

10.11 Currently accepted position

Social-cognitive theory as the settled synthesis. The **currently accepted** position is Bandura's mature *social-cognitive theory*: behaviour is learned through observation and direct experience, is mediated throughout by cognitive processes (symbolic representation, outcome expectancies and self-efficacy beliefs), and is co-determined by the reciprocal interaction of person, behaviour and environment. Social-cognitive approaches are among the most influential frameworks in current personality and behaviour-change research (Kaplan & Sadock 2024). All contemporary cognitive behaviour therapy protocols rest on the principles of social learning theory (Kaplan & Sadock 2024): the radical-behaviourist prohibition on studying cognition has been fully abandoned, and the "cognitive turn" Bandura, Beck and Ellis initiated in the 1960s is now orthodox. The concept has also generalised, with later theorists (for example Hazel Markus's "possible selves") extending social learning from specific behaviours to whole identities (Kaplan & Sadock 2024).

HOLD THIS

Bandura's move is acquisition without direct reinforcement: behaviour is learned by watching a model (Bobo doll), through four subprocesses and vicarious reinforcement, and organised around self-efficacy. Learning (acquisition) is distinct from doing (performance).

10.12 Behavioural and neural correlate

Where modelling lives in the brain. Behaviourally, observational learning is expressed as imitation, vicarious conditioning (acquiring a conditioned emotional response by watching another's), and the graded rise in performance and persistence that tracks rising self-efficacy. The candidate *neural correlate* of imitation and action-observation is the *mirror neuron system*, first

described in macaque premotor cortex and, in humans, mapped to a network centred on the *inferior frontal gyrus / ventral premotor cortex* and the *inferior parietal lobule*, which discharges both when an action is performed and when the same action is observed, providing a plausible substrate for learning by watching. Vicarious emotional learning (learning fear by observing another's fear) recruits the same fear circuitry as direct conditioning, notably the *amygdala*. These localisations are an active and still-debated literature (the behavioural specificity of "mirror" responses in humans remains contested), so treat them as the direction of travel rather than as fixed exam facts.

10.13 Worked clinical example: modelling and self-efficacy in a specific phobia

The theory applied to a dog phobia. Take a child with a specific phobia of dogs. *Origin (observational learning):* the fear may never have involved a bite; it can be caught by observing a frightened parent recoil from dogs (vicarious emotional learning), which is why phobias run in families. *Reciprocal determinism:* the child's fear cognition drives avoidance (behaviour), which shrinks their exposure to friendly dogs (environment), which prevents disconfirmation and entrenches the fear, each factor feeding the others. *Self-efficacy:* the child holds a low efficacy belief, "I cannot cope near a dog." *Treatment built from the theory:* Bandura's own participant modelling is the prototype, the therapist first *models* calm approach to the dog (vicarious experience), often using a peer model similar to the child to maximise perceived similarity, then guides the child through *graded mastery experiences* of approaching and touching the dog, the most potent efficacy source, while keeping arousal low so the child does not read their own racing heart as proof of danger (physiological state) and offering credible encouragement (verbal persuasion). Rising self-efficacy and repeated non-catastrophic outcomes extinguish the avoidance. This is exactly the logic of graded exposure in modern CBT, and it is drawn straight from social learning theory (Kaplan & Sadock 2024).

CLINICAL ANCHOR

Self-efficacy is the concept that ties Bandura to everyday psychiatric practice. Graded exposure works chiefly by delivering *mastery experiences*; participant modelling adds *vicarious experience*; the therapeutic relationship supplies *verbal persuasion*; and arousal-reduction skills address the *physiological state*. When an intervention "builds confidence," the mechanism you should name in an exam is a rise in task-specific self-efficacy across Bandura's four sources.

INDIA

Observational learning is culturally weighted toward high-status and familiar models. In much Indian practice, adherence and health behaviours shift most when the model is a trusted elder, senior clinician or community figure the patient perceives as similar and authoritative, satisfying Bandura's "perceived similarity," "prestige" and "familiarity" conditions at once. Peer-modelling and family-inclusive demonstrations therefore often outperform individually delivered instruction.

MNEMONIC

Subprocesses "A RRM"; efficacy sources "My Very Persuasive Physiology"

A-R-R-M = the four observational-learning subprocesses in order: *A*ttention, *R*etention, *R*eproduction, *M*otivation. **My Very Persuasive Physiology** = the four self-efficacy sources in descending potency: *M*astery experience, *V*icarious experience, verbal *P*ersuasion, *P*hysiological/affective state.

PEARL

The one-line arc for an examiner: learning theory moved from radical behaviourism (mind excluded) to Rotter's expectancy-and-locus-of-control amendment, then to Bandura's social learning, in which behaviour is acquired by observation (Bobo doll) through four subprocesses and vicarious reinforcement, governed by reciprocal determinism, and organised around self-efficacy (four sources), the whole now the "social-cognitive" theory that underpins CBT and exposure. Name that sequence and the "cognitive turn" and you have framed the topic.

GOOD TO REMEMBER

- **Albert Bandura:** founder of social learning / social-cognitive theory; integrated classical and operant conditioning with observational learning at Stanford.
- **Observational (vicarious) learning:** behaviour acquired by watching a model and its consequences, with no direct reinforcement of the learner.
- **Four subprocesses (A-R-R-M):** Attention, Retention, Reproduction (motor), Motivation.
- **Optimum conditions for observational learning:** live modelling, perceived similarity, seeing the model reinforced, familiarity with the model (Companion to Psychiatric Studies).
- **Vicarious reinforcement / punishment:** consequences falling on the model raise or suppress the observer's performance; learning persists even when performance is suppressed (acquisition vs performance).
- **Bobo doll (Bandura, Ross & Ross 1961):** children copied filmed aggression toward the inflatable doll; live-clown replication (Bandura et al. 1963). Aggression transmitted by modelling without direct reinforcement.
- **Reciprocal determinism:** person (cognition), behaviour and environment continuously and mutually interact; interactionist, not environmentally deterministic.
- **Self-efficacy:** task-specific belief in one's capability to execute a behaviour; the central individual-difference variable in social-cognitive personality theory (Kaplan & Sadock 2024).
- **Four sources of self-efficacy (descending potency):** mastery experience, vicarious experience (modelling), verbal persuasion, physiological/affective state.
- **Julian Rotter (1916 to 2014):** early social learning theory; behaviour guided by expectancy of a valued goal; internal vs external locus of control.
- **Radical behaviourism (Watson tradition):** excluded cognition as unobservable; Bandura's "cognitive turn" (with Beck and Ellis, 1960s) overturned it.
- **Currently accepted:** social-cognitive theory; all contemporary CBT protocols rest on social learning theory principles (Kaplan & Sadock 2024).
- **Neural correlate (debated):** mirror neuron system, inferior frontal gyrus / ventral premotor cortex and inferior parietal lobule for action observation; amygdala for vicarious fear learning.
- **Clinical reach:** participant modelling and graded exposure build self-efficacy chiefly via mastery experiences; underpins behaviour change generally.

11 · Learning applied to behaviour therapy, ABA and learned helplessness

The learning paradigms are not just theory; they are the therapies. Everything built in the classical and operant topics converges here: the three learning paradigms (respondent, operant, social/observational) each generated a family of clinical techniques, and one paradigm applied to a natural experiment generated a whole model of depression. This is the topic that ties the behavioural-science paper to the clinical papers. The examiner wants you to be able to walk from a learning principle to the named therapy it produced, and back again: from **reciprocal inhibition** to **systematic desensitisation**, from operant reinforcement to the **token economy** and **applied behaviour analysis**, from Pavlovian-plus-operant fear to **exposure** and **flooding**, and from uncontrollable aversive events to **learned helplessness** as a model of depression.

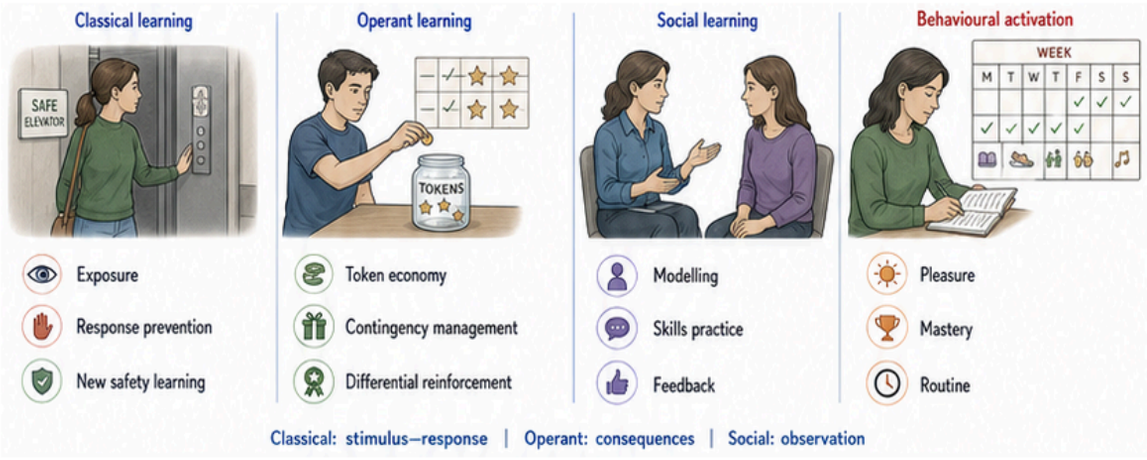


Fig 5.14 Learning mechanisms in behaviour therapy.

Behaviour therapy is defined as the systematic application of principles derived from learning theory to the modification of maladaptive behaviour (Kaplan & Sadock 2024). The field arose in the 1950s, in three centres almost at once, out of dissatisfaction with the length and unfalsifiability of psychoanalysis: the United States (Skinner and the operant tradition), South Africa (Wolpe), and the United Kingdom (Eysenck and Rachman) (Kaplan & Sadock 2024; Tasman Psychiatry). The standard way to organise the many techniques is by their parent paradigm, which is exactly how this topic is built, followed by the learned-helplessness thread and its attributional reformulation.

11.1 Definition and the three-wave frame

Behaviour therapy as applied learning theory, developing in three waves. Behaviour therapy treats maladaptive behaviour as learned, and therefore as unlearnable by the same principles, without any requirement to uncover unconscious conflict (Kaplan & Sadock 2024). The historical scaffold examiners like is the three-wave model (Kaplan & Sadock 2024). The *first wave* (1950s to 1960s) was pure classical-and-operant behaviour therapy: systematic desensitisation, exposure, aversion, token economies. The *second wave* (1970s onward) added cognition, growing out of Bandura's social learning theory into cognitive behaviour therapy (Beck, Ellis); it is the dominant system worldwide today. The *third wave* (from the 1990s) added acceptance, mindfulness and

values (DBT, ACT), and emerged in part from Jacobson's finding that behavioural activation alone matched full CBT for depression. This topic covers the behavioural (first-wave) engine and its direct descendants.

11.2 From classical conditioning: reciprocal inhibition and systematic desensitisation (Wolpe 1958)

The first named behaviour therapy, built on counterconditioning. Joseph Wolpe, a South African psychiatrist frustrated by psychoanalysis, published *Psychotherapy by Reciprocal Inhibition* (Wolpe 1958). **Reciprocal inhibition** is the principle that two physiologically antagonistic states cannot be held at once: if a response incompatible with anxiety (deep relaxation) is evoked in the presence of an anxiety-provoking stimulus, the bond between that stimulus and anxiety is weakened (Companion to Psychiatric Studies; Kaplan & Sadock 2024). **Systematic desensitisation** operationalises this in three components (Kaplan & Sadock 2024): (1) training in a competing response, usually progressive deep muscle relaxation; (2) construction of an *anxiety hierarchy*, a graded list of feared situations rated by subjective units of distress (SUDS) from least to most frightening; and (3) graded pairing, in which the relaxed patient imagines each hierarchy item in ascending order, moving up only when the current item no longer evokes anxiety. **What it fixed and what it left debated.** It fixed the slowness and indirectness of insight therapy for phobias, giving a rapid, replicable, testable method. What it left debated was its *mechanism*: Wolpe attributed the effect to counterconditioning (reciprocal inhibition), but later work suggested the relaxation and the hierarchy may be inessential and that plain habituation and extinction through exposure do most of the work, a debate the exposure therapies settled.

11.3 From classical conditioning: exposure, flooding and implosion

Confront the fear directly and let the anxiety extinguish. **Exposure** therapy rests on the premise that fear, acquired by associative learning, is eliminated by systematic contact with the feared object, event or situation until the conditioned anxiety habituates and extinguishes (Kaplan & Sadock 2024). It can be graded (as in in-vivo desensitisation) or intensive. In **flooding**, the patient is exposed immediately to the feared stimulus at full strength, without relaxation, and stays until anxiety peaks and falls; the imaginal, catastrophically-vivid variant is *implosion (implosive therapy)* (Kaplan & Sadock 2024). Exposure is almost always paired with *response prevention*, blocking the escape and avoidance and other anxiety-reducing rituals so that the feared consequence can be disconfirmed; exposure and response prevention (ERP) is the treatment of choice for obsessive-compulsive disorder (Kaplan & Sadock 2024). Distinguish *within-session* extinction (anxiety falling during one exposure) from *between-session* extinction (peak anxiety and time-to-settle both falling across sessions); the second predicts durable gain. **What it fixed.** Exposure fixed the mechanistic ambiguity of desensitisation by showing the graded hierarchy and relaxation are helpful but not necessary: extinction through contact is the active ingredient, and this is the currently accepted account of how anxiety-focused behaviour therapy works.

11.4 From classical conditioning: aversion therapy and cue exposure

Two opposite uses of the conditioned stimulus. **Aversion** therapy pairs an unwanted appetitive behaviour with an unpleasant stimulus so that the behaviour itself comes to elicit discomfort. Historically it used a subthreshold electric shock or an emetic such as apomorphine paired with alcohol, and the covert form, *covert sensitisation*, pairs the imagined behaviour with an imagined aversive scene. Its use has narrowed sharply on both efficacy and ethical grounds, but it remains an examinable technique (in alcohol dependence, some paraphilias historically). *Cue exposure* is the mirror-image application in addiction: the patient is repeatedly exposed to drug- or alcohol-associated cues while consumption is prevented, so the conditioned craving response to those cues extinguishes (Stahl, Substance Use). Both are classical-conditioning techniques; aversion builds a new conditioned aversion, cue exposure extinguishes an existing conditioned craving.

GOOD TO REMEMBER

- **Reciprocal inhibition (Wolpe 1958):** relaxation, an anxiety-antagonistic state, weakens the stimulus-anxiety bond; the basis of systematic desensitisation.
- **Systematic desensitisation:** relaxation training + graded anxiety hierarchy (SUDS) + graded imaginal/in-vivo pairing; classical-conditioning (counterconditioning) technique.
- **Exposure:** systematic contact with the feared stimulus until anxiety habituates and extinguishes; graded or intensive.
- **Flooding / implosion:** immediate full-strength exposure without relaxation; implosion is its imaginal variant; usually with response prevention (ERP for OCD).
- **Aversion therapy:** pairs an unwanted behaviour with an aversive stimulus (shock, apomorphine); covert sensitisation is the imaginal form.
- **Cue exposure:** extinguishes conditioned craving by exposing to drug cues while blocking use (addiction).

TECHNIQUE	PARENT PARADIGM	MECHANISM	SIGNATURE USE
Systematic desensitisation	Classical	**Reciprocal inhibition / counterconditioning + graded hierarchy**	Specific phobia
Exposure / flooding / implosion	Classical	**Habituation and extinction of conditioned fear (with response prevention)**	OCD, phobias, PTSD
Aversion / covert sensitisation	Classical	Conditioning a new aversion to the behaviour	Alcohol dependence, some paraphilias
Cue exposure	Classical	Extinction of conditioned craving	Substance use disorders
Token economy	Operant	Secondary (conditioned) reinforcement	Chronic inpatient wards
Contingency management	Operant	**Positive reinforcement of a verified target (abstinence)**	Substance use disorders

TECHNIQUE	PARENT PARADIGM	MECHANISM	SIGNATURE USE
Shaping / differential reinforcement	Operant	Reinforcing successive approximations / other behaviour (DRO)	Autism, intellectual disability
Time-out / response cost	Operant	Negative punishment (removing an appetitive stimulus)	Child behaviour programmes
Modelling / skills training	Social learning	**Observational learning (Bandura)**	Social skills, assertiveness
Behavioural activation	Operant / social	Restoring response-contingent positive reinforcement	Depression

The whole of behaviour therapy sorted by its parent learning paradigm. The discriminator columns are the mechanism: classical techniques act on stimulus-response bonds (counterconditioning, extinction), operant techniques act on consequences (reinforcement, punishment), social-learning techniques act by observation.

RULE **Name a behaviour therapy by its parent paradigm and its mechanism.** Classical acts on stimulus-response bonds (desensitisation, exposure); operant acts on consequences (token economy, contingency management, ABA); social learning acts by observation (modelling, skills training).

11.5 From operant conditioning: the token economy

A whole-ward contingency system built on secondary reinforcers. The **token economy** is a structured programme in which patients earn tokens (a generalised secondary reinforcer) for performing specified target behaviours, later exchanging them for backup reinforcers such as privileges or goods (Kaplan & Sadock 2024; Companion to Psychiatric Studies). Its logic is pure operant: the token has no intrinsic value but, by pairing with many backup rewards, becomes a generalised conditioned reinforcer that can be delivered immediately and precisely. Ted Ayllon and Nathan Azrin established the first comprehensive token economy at Anna State Hospital, Illinois, producing marked improvements in the targeted behaviour of chronic inpatients with severe mental illness (Kaplan & Sadock 2024). The historical antecedent examiners cite is Alexander Maconochie's early-19th-century points system for prisoners at Norfolk Island (Kaplan & Sadock 2024). Token economies flourished on chronic wards and in residential and school settings, though their use has declined with deinstitutionalisation.

11.6 From operant conditioning: contingency management, shaping and differential reinforcement

Reinforcing verified target behaviour, one approximation at a time. *Contingency management* is the systematic delivery of a positive reinforcer (a voucher, prize or privilege) contingent on an objectively verified target behaviour, most classically a clean urine drug screen in substance use disorder treatment (Kaplan & Sadock 2024; Stahl, Substance Use); it is among the most robustly evidenced psychosocial treatments in addiction. *Shaping* reaches a behaviour that never occurs spontaneously by reinforcing successive approximations to it, the operant backbone of teaching self-care and language in intellectual disability. *Differential reinforcement* reduces an unwanted behaviour not by punishing it but by reinforcing an alternative: *differential reinforcement of other*

behaviour (DRO) reinforces the absence of the problem behaviour, and *differential reinforcement of an incompatible behaviour* (DRI) reinforces a response that cannot co-occur with it (Tasman Psychiatry; Kaplan & Sadock 2024). These are the constructive alternatives to punishment across child and developmental work.

11.7 From operant conditioning: time-out and response cost (the punishment-side techniques)

Reducing behaviour by removing an appetitive stimulus. Both are forms of *negative punishment*. *Time-out* (time-out from positive reinforcement) removes the person from the reinforcing environment for a brief period contingent on the problem behaviour, so the behaviour no longer accesses reinforcement (Kaplan & Sadock 2024). *Response cost* removes a specified quantity of an already-held reinforcer (tokens, points, privileges) contingent on the behaviour, and is often built into a token economy. Because punishment suppresses without teaching an alternative, best practice pairs these with a differential-reinforcement procedure that builds the desired behaviour at the same time.

11.8 Applied behaviour analysis (ABA): the systematic clinical application of operant principles

Functional analysis, the ABC chain, and its use in autism and intellectual disability. **Applied behaviour analysis** (**ABA**) is the systematic application of operant learning principles to socially significant behaviour, and it is the umbrella under which shaping, differential reinforcement, chaining and the rest are clinically deployed (New Oxford Textbook of Psychiatry). Its engine is the *functional analysis*: rather than treat a behaviour as a fixed trait, ABA analyses the *ABC chain*, the **antecedent**, the **behaviour**, and the **consequence** that maintains it (Kaplan & Sadock 2024, "the ABCs of therapy"). A functional analysis typically resolves a challenging behaviour into one of a small set of maintaining functions: escape or avoidance of a demand, attention-seeking, tangible access to a wanted item or activity, and automatic or sensory reinforcement (Oxford Handbook of DBT). Once the function is known, the treatment follows from it: an escape-maintained behaviour is treated differently from an attention-maintained one. ABA is the best-evidenced psychosocial intervention for the core deficits and challenging behaviours of autism spectrum disorder and intellectual disability, where it is used to build communication, self-care and social skills and to reduce self-injury and aggression (New Oxford Textbook of Psychiatry). The child-psychiatry detail of autism and intellectual-disability management is developed in Paper III (child).

11.9 From social learning: modelling, skills training and behavioural activation

Learning by observation, and re-engaging with reinforcing activity. Albert Bandura's social learning theory holds that behaviour is acquired not only by direct conditioning but by *observational learning*, watching a model perform a behaviour and experience its consequences (Kaplan & Sadock 2024). Its clinical arm is *modelling* and *social skills training*: the therapist demonstrates a target behaviour (assertiveness, a social response), the patient rehearses it with feedback and reinforcement, and it is generalised to real settings; participant modelling, in which the patient performs the feared action alongside the model, is a potent phobia treatment. *Behavioural activation* (BA) is the depression-specific technique that treats low mood as, in part, a state of reduced response-contingent positive reinforcement (drawing on Lewinsohn's

reinforcement model of depression); it systematically schedules and grades pleasant and mastery activities to restore that reinforcement (Kaplan & Sadock 2024). BA is examinable because Jacobson's component analysis and Dimidjian and colleagues' 2006 trial showed BA alone matched full CBT and antidepressant medication for severe depression, which helped launch the third wave.

11.10 Learned helplessness: Seligman's shuttle-box experiments

Uncontrollable aversive events produce passivity. **Learned helplessness** is the paradigm through which learning theory generated a model of depression. Working with dogs, Martin **Seligman** and colleagues exposed animals to inescapable electric shock; when later placed in a shuttle-box where a simple response (jumping a barrier) would let them escape, these animals failed to learn to escape and passively endured the shock, whereas animals given escapable shock, or none, learned readily (Seligman 1975; Kaplan & Sadock 2024; Tasman Psychiatry). Seligman argued the crucial variable was not the shock but its *uncontrollability*: the animals had learned that outcomes were independent of their responses, and this learned expectation of uncontrollability generalised into motivational, cognitive and emotional deficits (reduced initiation of action, failure to learn that responding now works, and blunted affect). He proposed **learned helplessness**, defined as the belief that it is futile to initiate personal action to reverse aversive circumstances, as an experiential analogue of human depression (Kaplan & Sadock 2024).

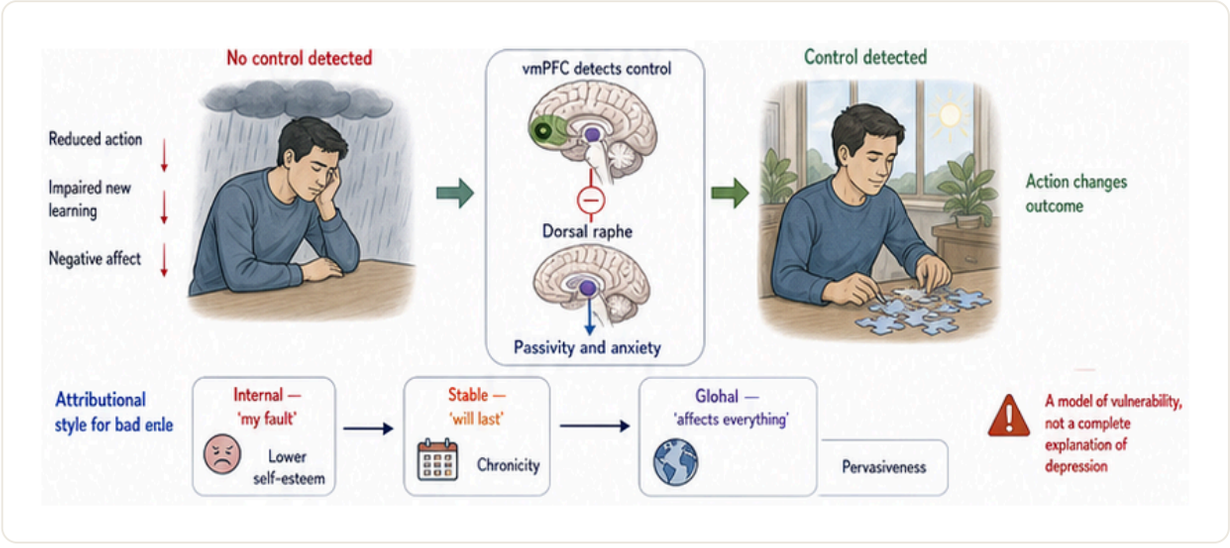


Fig 5.15 Learned helplessness, control and explanatory style.

11.11 The attributional reformulation (Abramson, Seligman and Teasdale 1978)

What fixed the original model: it is not helplessness itself but how you explain it. The original animal model could not explain why, faced with the same uncontrollable event, some people become depressed and others do not, nor why human depression involves self-blame. The *attributional (cognitive) reformulation* (Abramson, Seligman & Teasdale 1978) fixed this by inserting a cognitive step: what matters is the *causal attribution* a person makes for the uncontrollable event, their characteristic *attributional (explanatory) style* (Tasman Psychiatry). The depressogenic style attributes bad events along three dimensions: *internal* ("it is my fault") rather

than external, *stable* ("it will last forever") rather than unstable, and *global* ("it will affect everything") rather than specific (Tasman Psychiatry; Kaplan & Sadock 2024). Internal attributions explain the loss of self-esteem, stable ones the chronicity, and global ones the pervasiveness of depressive symptoms. This was later extended into the *hopelessness theory* of depression (Abramson, Metalsky & Alloy 1989), in which a stable and global attributional style, plus a negative event, generates hopelessness, a proximal sufficient cause of a hopelessness subtype of depression. This reformulation is what made learned helplessness a genuinely cognitive model and the bridge to Beck's cognitive theory.

HOLD THIS

The depressogenic attributional style for bad events is internal, stable and global (I See Grief). Internal drives low self-esteem, stable drives chronicity, global drives pervasiveness; for positive events the depressive style is the reverse.

11.12 Currently accepted position and the neural correlate

From learned helplessness to learned passivity, with a mapped circuit. The **currently accepted** position, argued by Maier and Seligman on the paradigm's fiftieth anniversary, actually inverts the original interpretation: passivity in the face of prolonged aversive events is the animal's *default, unlearned* response, and what is genuinely *learned* is the *detection of control*, which then inhibits that default passivity. In other words, the organism does not learn to be helpless; it fails to learn that it has control. The *neural correlate* is now well specified in this reformulation: prolonged uncontrollable stress activates the *dorsal raphe nucleus* serotonergic system, driving passivity and anxiety, while the *ventromedial prefrontal cortex* detects the presence of behavioural control and, when control is present, inhibits the dorsal raphe response (Maier & Seligman 2016). The broader neurobiology of learned helplessness implicates the same stress systems central to depression, the hypothalamic-pituitary-adrenal axis and forebrain serotonergic and dopaminergic circuits sensitised by early-life and chronic stress (Tasman Psychiatry). This dovetails with the wider mood-disorder correlates developed in the mood disorders notes.

11.13 Learned helplessness as a model of depression

Why the paradigm earns its place in psychiatry. Learned helplessness sits alongside Beck's cognitive model and Lewinsohn's reinforcement model as one of the three great learning-and-cognition models of depression (Kaplan & Sadock 2024). Its explanatory yield is real: it accounts for the passivity, motivational deficit and low hostility seen in some depressed patients; the reformulated version accounts for the self-blame, chronicity and pervasiveness through the internal-stable-global attributional triad; and it makes a testable therapeutic prediction, that changing expectations of uncontrollability and modifying attributional style should relieve depression, which is precisely what cognitive therapy sets out to do (Kaplan & Sadock 2024). Its role reaches beyond depression to a general disposition relevant to post-traumatic stress disorder, chronic pain and states of social powerlessness (Kaplan & Sadock 2024). The standard caveat is that the leap from animal analogue and self-report inventory to clinical depression must be made

cautiously, and that recent work implicates genetic vulnerability in how readily helpless behaviour is learned (Kaplan & Sadock 2024).

11.14 Worked example: a patient with panic disorder and agoraphobia

The learning-to-therapy chain applied to one presentation. A woman develops panic attacks and begins avoiding buses and crowded markets. *Classical arm:* the market has become a conditioned stimulus for panic; the behavioural analysis identifies the antecedent cue (crowds), the behaviour (fleeing), and the consequence (immediate anxiety relief) that negatively reinforces the avoidance. *The therapy that follows:* a graded anxiety hierarchy is built (from imagining a quiet shop to standing in a crowded market at peak hour, rated by SUDS), and either systematic desensitisation (relaxation paired with each step) or, more efficiently, graded in-vivo exposure with response prevention is used, so she stays in each situation until anxiety habituates and the catastrophic prediction is disconfirmed. *Operant arm:* her partner's reassurance and permission to leave were positively reinforcing the avoidance, so a contingency is arranged to reinforce staying, not fleeing. *Social-learning arm:* the therapist models entering the feared setting first (participant modelling). *Learned-helplessness arm:* if she has come to believe her panic is uncontrollable and to attribute it internally, stably and globally ("I am broken, this will never change, it ruins everything"), the cognitive work targets exactly that attributional style, restoring a sense of control, which is the learned-helplessness reformulation put to clinical use.

CLINICAL ANCHOR

When you name a behaviour therapy in an exam or on a ward round, say which paradigm it comes from and what it does mechanistically. "Systematic desensitisation, a classical-conditioning technique working by reciprocal inhibition and graded exposure." "Contingency management, an operant technique reinforcing verified abstinence." "Behavioural activation, restoring response-contingent positive reinforcement in depression." The examiner is testing whether you can move between the principle and the technique, not whether you can list therapies.

COMMON TRAP

Do not confuse the two intensive exposures. *Flooding* is real-life or imaginal full-strength exposure; *implosion* is specifically the imaginal, deliberately-exaggerated form. And systematic desensitisation is *graded* and paired with relaxation (counterconditioning), whereas flooding is *ungraded* and deliberately without relaxation (extinction). A second trap: in the attributional reformulation the depressogenic style is internal-stable-global for *negative* events; for positive events the depressive style is the reverse (external-unstable-specific), a symmetry examiners like to probe.

MNEMONIC

"I See Grief" for the depressogenic attributional style: Internal, Stable, Global.

The three attributional dimensions that make learned helplessness depressogenic for bad events are **I**nternal (my fault), **S**table (permanent), **G**lobal (affects everything): I-S-G, "I See Grief". Internal drives low self-esteem, Stable drives chronicity, Global drives pervasiveness. For the techniques, sort by paradigm: classical = desensitisation/exposure/aversion/cue; operant = token economy/contingency management/ABA/time-out; social = modelling/skills/behavioural activation.

INDIA

ABA and structured behavioural programmes for autism and intellectual disability are resource-intensive and unevenly available across Indian settings, so parent-mediated behavioural training, which teaches caregivers to run the antecedent-behaviour-consequence analysis and deliver reinforcement at home, is often the pragmatic backbone rather than clinic-delivered one-to-one ABA. In addiction work, contingency management fits Indian de-addiction settings well because its reinforcers can be modest and locally meaningful, and cue exposure maps onto the culturally specific drinking and substance cues a plan must address.

PEARL

The one-line arc for an examiner: the three learning paradigms each became a therapy family, classical conditioning gave systematic desensitisation (Wolpe, reciprocal inhibition), exposure and flooding, aversion and cue exposure; operant conditioning gave the token economy (Ayllon and Azrin), contingency management, shaping and differential reinforcement, and applied behaviour analysis with its functional analysis of the antecedent-behaviour-consequence chain for autism and intellectual disability; and social learning gave modelling, skills training and behavioural activation. Learned helplessness (Seligman) turned uncontrollable aversive events into a model of depression, and its attributional reformulation (Abramson, Seligman and Teasdale 1978, internal-stable-global) made it cognitive and the bridge to CBT.

GOOD TO REMEMBER

- **Behaviour therapy:** systematic application of learning theory to modify maladaptive behaviour; arose 1950s (Skinner, Wolpe, Eysenck/Rachman); three-wave model (behavioural, cognitive, acceptance-based).
- **Wolpe (1958), *Psychotherapy by Reciprocal Inhibition*:** reciprocal inhibition (relaxation inhibits anxiety) underlies systematic desensitisation.
- **Systematic desensitisation:** relaxation + graded anxiety hierarchy (SUDS) + graded pairing; classical/counterconditioning; for specific phobia.
- **Exposure:** contact with the feared stimulus until anxiety habituates/extinguishes; within-session vs between-session extinction.
- **Flooding / implosion:** immediate full-strength exposure without relaxation; implosion = imaginal exaggerated form; with response prevention (ERP for OCD).
- **Aversion therapy:** pair unwanted behaviour with aversive stimulus (apomorphine, shock); covert sensitisation = imaginal.
- **Cue exposure:** extinguishes conditioned craving in addiction.
- **Token economy (Ayllon & Azrin, Anna State Hospital):** operant; tokens as generalised secondary reinforcers exchanged for backups; antecedent = Maconochie.
- **Contingency management:** operant; positive reinforcement of verified target (clean urine screen) in substance use disorder.
- **Shaping / differential reinforcement:** reinforce successive approximations / reinforce other (DRO) or incompatible (DRI) behaviour.
- **Time-out / response cost:** negative punishment (removing an appetitive stimulus).
- **Applied behaviour analysis (ABA):** systematic operant application; functional analysis of the ABC (antecedent-behaviour-consequence) chain; four functions (escape/avoidance, attention, tangible, sensory); best-evidenced for autism and intellectual disability (Paper III child).
- **Modelling / skills training (Bandura):** social-learning technique via observational learning; participant modelling for phobias.
- **Behavioural activation (Jacobson; Dimidjian 2006):** restores response-contingent positive reinforcement in depression (Lewinsohn roots).
- **Learned helplessness (Seligman 1975):** inescapable shock in dogs, shuttle-box; uncontrollability produces passivity; belief that action is futile; a model of depression.
- **Attributional reformulation (Abramson, Seligman & Teasdale 1978):** depressogenic explanatory style for bad events = internal, stable, global; extended to hopelessness theory (Abramson, Metalsky & Alloy 1989).
- **Currently accepted (Maier & Seligman 2016):** passivity is the default; control-detection is what is learned; neural correlate = ventromedial prefrontal cortex detecting control inhibits the dorsal raphe serotonergic passivity response.

Personality and psychodynamic theory

12 · Meyer, Rank and the wider psychodynamic map

The depth-psychology map, and the two figures these notes owns. Classical **depth psychology** radiates from three primary schools: Sigmund Freud's **psychoanalysis**, Alfred Adler's individual psychology, and Carl Jung's analytical psychology. All three, together with their apparatus (the **topographic model** of conscious, preconscious and unconscious; the **structural model** of id, ego and **superego**; and the great defections of Adler and Jung) are taught in full on the General and Developmental Psychology notes and are only named and cross-referenced here.

What these notes adds is the pair of Paper-1 scientists who sit at the edges of that map and are examinable in their own right: Adolf Meyer, whose **psychobiology** is the American holistic-formulation tradition that anticipated the modern biopsychosocial model, and Otto Rank, the analyst whose **birth trauma** and **will therapy** broke from Freud and seeded humanistic and existential practice.

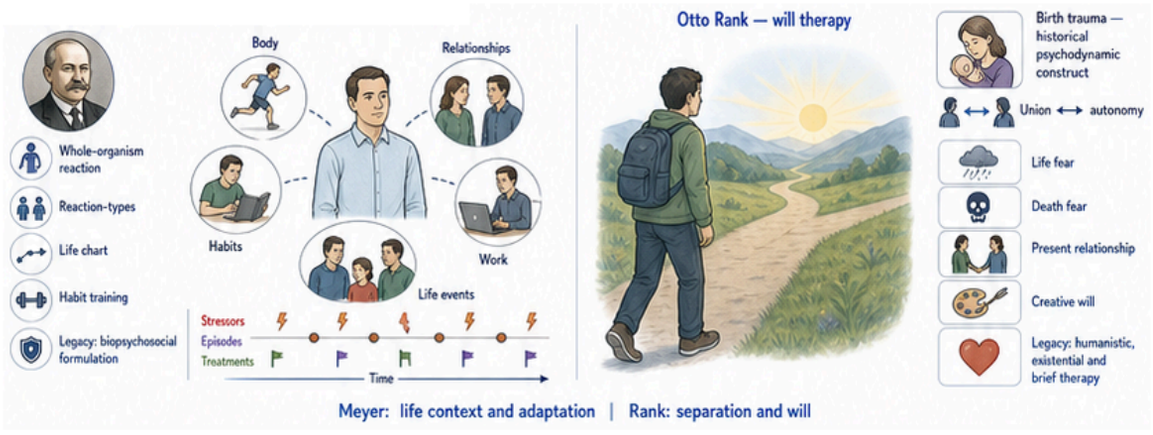


Fig 5.16 Meyer and Rank: two exits from Freud.

The exam framing is precise. Freud, Adler and Jung are General and Developmental Psychology items; do not re-teach them here. Meyer and Rank are the two names these notes must deliver, each as a short evolution-of-ideas: what each figure took from the psychoanalytic mainstream, what he replaced, and what survives in contemporary practice. Meyer feeds directly into formulation and nosology; Rank feeds into the humanistic and existential therapies. The neo-Freudian relationalists (Horney, Sullivan, Fromm) are named at the end only as a bridge into the object-relations and interpersonal topic developed next.

12.1 The three primary schools: named, and referred to the General and Developmental Psychology notes

Freud, Adler, Jung, taught in full elsewhere. The organising spine of depth psychology is three schools, each with its own founder, its own account of the unconscious, and its own therapeutic aim.

Psychoanalysis (Freud)

The parent school. Its models of mind are the **topographic model** (conscious, preconscious, unconscious) and the later **structural model** (id, ego, **superego**); its engine is intrapsychic conflict, drive and defence, with the Oedipus complex central. Taught in full on **the General and Developmental Psychology notes**.

Individual psychology (Adler)

The first major defection. Replaces libido as prime mover with striving for superiority to compensate for inferiority; emphasises social interest, the family constellation and the unified goal-directed self. Cross-referenced to **the General and Developmental Psychology notes**.

Analytical psychology (Jung)

The second defection. Adds the collective unconscious, archetypes, the persona and shadow, individuation, and the introversion-extraversion typology. Cross-referenced to **the General and Developmental Psychology notes**.

Everything below is the delta these notes add: two Paper-1 scientists whom the General and Developmental Psychology notes does not cover.

TRAP Do not re-teach Freud, Adler and Jung here: they are cross-referenced only. The two examinable figures in this topic are **Meyer (psychobiology) and Rank (birth trauma)**.

12.2 Adolf Meyer: the man and the idea of psychobiology

The whole organism reacting to its life. Adolf Meyer (1866 to 1950), Swiss-born and Baltimore-based, was the dominant figure in early twentieth-century American psychiatry, directing the Henry Phipps Psychiatric Clinic at Johns Hopkins (New Oxford Textbook of Psychiatry). His system, **psychobiology**, treated the person as a single biological unit, maintaining internal and external equilibrium while coping with new situations, and understood mental disorder not as a discrete brain lesion nor as a purely intrapsychic drama but as a maladaptive *reaction of the whole organism* to its life circumstances (The Psychotherapy Guidebook). Meyer was influenced by Hughlings Jackson's levels of nervous integration and by American pragmatism (James, Dewey), giving the system a concrete, optimistic, anti-dogmatic character that deliberately avoided the metapsychological speculation of the European schools.

12.3 Meyer's ergasiology: reaction-types instead of one-word diagnoses

Ergasia: behaviour as work of the organism. Dissatisfied with the reductive one-word diagnoses of Kraepelinian nosology, Meyer classified disorders as *reaction-types*, patterned maladaptive responses of the person to circumstance rather than fixed disease entities. In the 1920s he coined *ergasia* (from Greek *ergon*, work) for behaviour and mental activity as a whole, and its plural *ergasias* for specific behavioural units, the vocabulary now labelled **ergasiology**. Organic brain reactions were "anergasia", toxic psychoses "dysergasia", and so on (The Psychotherapy Guidebook). The idiosyncratic ergasia terms were never adopted into formal nomenclature, but the underlying *reaction* concept was: it entered American classification directly, so that DSM-I (1952), strongly shaped by Meyer's views, described conditions as "reactions" (for example schizophrenic reaction, manic-depressive reaction) rather than as diseases (Shorter Oxford Textbook of Psychiatry).

12.4 Meyer's method: the life chart, distributive analysis and habit training

Plot the life, then re-train the habits. Meyer's clinical signature is the **life chart**: the psychiatrist plots the patient's life on a single time-line from birth to the present, penciling in major life events alongside the onset and worsening of illness, so that recurrent associations between circumstance and symptom become visible (Kaplan & Sadock 2024). The biographical data gathered this way were then worked through by *distributive analysis* (the patient examines the components of the problem and their relative weight) and *distributive synthesis* (reconstructing healthier patterns), with therapy proceeding as *habit training*, the modification of unhealthy adaptations through guidance, re-education and direction, engaging the healthier part

of the patient's own ego. Meyer held this approach especially valuable for psychotic patients, and he was an early architect of the mental-hygiene and community-care movements alongside Clifford Beers (The Psychotherapy Guidebook).

CLINICAL ANCHOR

Meyer's life chart is the direct ancestor of the timeline every trainee draws in a formulation: the longitudinal plotting of stressors, episodes and treatments to expose temporal patterns. His reaction-type view is the seed of the modern *biopsychosocial* formulation, illness read as the whole person's maladaptive reaction to biological, psychological and social forces rather than as an isolated diagnosis. When you write "onset followed the loss of employment and coincided with sleep disruption", you are doing Meyerian psychobiology.

12.5 Otto Rank: the birth trauma

Separation from the mother as the root of anxiety. Otto Rank (1884 to 1939), one of Freud's closest early collaborators, made his decisive move in *The Trauma of Birth* (1924). He took Freud's own passing idea that the violent physical separation of birth is the prototype of anxiety and made it the *central* source, arguing that all later anxiety re-enacts the primal **birth trauma** (Kaplan & Sadock 2024; Wolberg). Two opposed strivings follow from it: an impulse to return to the womb and restore prenatal security (expressed as dependent, clinging attachment), and an impulse for rebirth and independence (expressed as the will to separate and individuate). Rank named the two poles of this conflict the *life fear* (the dread that asserting one's separateness and creativity will isolate one from others) and the *death fear* (the dread of losing one's individuality by being absorbed into another). This relocation of the origin of anxiety from the Oedipal, father-centred conflict to the *pre-Oedipal*, mother-centred separation is Rank's core contribution and the point on which he parted from Freud.

12.6 Rank's will therapy and his influence downstream

Mobilise the will in the present relationship. Rank's therapeutic system, set out in *Will Therapy* (1936), abandoned the archaeological search for buried content and made the therapeutic *relationship in the present* the curative agent (Wolberg). Therapy is patient-centred and active: the analyst mobilises the patient's constructive "creative will impulse" toward self-direction and self-realisation, and studies the patient's reactions to the inevitable *separation* at the end of treatment as the working-through of the birth-derived conflict between union and autonomy. Insight and unconscious content are secondary to the corrective relational experience. With Ferenczi and Stekel, Rank thereby pioneered short-term, flexible, active psychotherapy. His influence runs forward on two lines: Carl Rogers absorbed Rank's philosophy of self-realisation and the patient-centred stance into humanistic *client-centred therapy*, and Rank's emphasis on will, separation, isolation and the confrontation with individuality prefigured the *existential* therapies (Wolberg). His functional-casework legacy also shaped American social work through Jessie Taft and Virginia Robinson.

COMMON TRAP

Do not attribute the concept of the "birth trauma" to Freud, and do not confuse Rank's *will therapy* with Adler's will-to-power or with the later existentialists' terms. Rank is Paper-1 examinable as a distinct figure: birth trauma (1924), will therapy (1936), pre-Oedipal focus, break from Freud, influence on humanistic and existential therapy. Also keep Rank's *birth trauma* (a psychodynamic construct about separation anxiety) separate from Stanislav Grof's later "birth matrices" and from any obstetric use of the phrase.

12.7 Meyer versus Rank: two ways off the Freudian main line

Same era, opposite exits. Both men worked at the edge of psychoanalysis in the same decades, but they left it in opposite directions: Meyer outward toward biology, society and pragmatic formulation, Rank inward toward the pre-Oedipal relationship and the will. The table fixes the discriminators.

FEATURE	ADOLF MEYER (PSYCHOBIOLOGY)	OTTO RANK (WILL THERAPY)
Core construct	**Mental illness as a maladaptive *reaction* of the whole organism**	**Anxiety rooted in the *birth trauma*; life fear versus death fear**
Break point from Freud	Rejected metapsychology for holistic, pragmatic biography; not a pupil-defector but a parallel American tradition	Moved the origin of anxiety from Oedipal to *pre-Oedipal* separation; formal break from Freud
Signature method	The *life chart*, distributive analysis and synthesis, habit training	Active, patient-centred *will therapy*; the present relationship and its ending are curative
Unit of classification	Reaction-types; the *ergasia* vocabulary (ergasiology)	Personality as normal, neurotic, or creative-artist types
Enduring influence	American nosology (DSM-I "reactions"); the biopsychosocial formulation; community psychiatry	Rogerian client-centred therapy; existential therapy; short-term dynamic therapy; functional social work

The two discriminator rows are the core construct (organism-level reaction versus birth-trauma separation anxiety) and the enduring influence (formulation and nosology versus humanistic and existential therapy).

12.8 Currently accepted position and its correlate

Meyer survives as method, Rank as lineage. Neither man's system is practised intact, but each is metabolised into current thinking. The **currently accepted** descendant of Meyer's psychobiology is the *biopsychosocial* model (Engel, 1977) and the longitudinal case formulation, both routine in contemporary training; Meyer's specific ergasia nomenclature is obsolete, but the "reaction/adjustment" logic persists in categories such as adjustment disorder and in the whole-person emphasis of formulation. Rank's system likewise survives not as a standalone therapy but as an acknowledged root of the humanistic and existential traditions and of brief dynamic therapy. The *neural correlate* is meaningful chiefly for the underlying constructs rather than the schools: separation-related distress, Rank's territory, maps onto a well-characterised separation-

distress circuitry involving the *amygdala*, *anterior cingulate cortex* and periaqueductal grey and the oxytocin and endogenous-opioid attachment systems (a neurobiology developed with the attachment material in the General and Developmental Psychology notes), while Meyer's stressor-to-illness reaction is the same stress-response neurobiology (HPA axis, amygdala, hippocampus, prefrontal cortex) that his life chart set out to make visible clinically.

HOLD THIS

Meyer survives as method (the biopsychosocial formulation and the DSM-I "reactions"); Rank survives as lineage (root of humanistic, existential and brief dynamic therapy). Neither system is practised intact today.

12.9 Worked example: one patient, read through both lenses

A first manic episode after migration and job loss. Take a young adult presenting with a first manic episode some months after moving city for work and then losing the job. *Through Meyer:* draw the life chart, the migration, the isolation, the sleep disruption and the job loss plotted against symptom onset, so the episode is formulated as the whole organism's maladaptive reaction to a cluster of stressors on a biological diathesis, and the plan targets habit re-training (sleep, routine, re-employment) alongside medication, the biopsychosocial formulation in action. *Through Rank:* the same migration is a concrete *separation*, reviving the birth-derived tension between the pull toward dependent security (returning home, clinging) and the push toward independent individuation (the reason for leaving), and the "life fear" of standing alone is live material; a Rankian-descended, relationship-focused brief therapy would work with the patient's will and with the meaning of separation rather than excavate childhood content. The two readings are complementary, not rival: Meyer organises the formulation, Rank supplies one lens on its psychological content.

PEARL

The fastest way to hold these two apart in the exam: *Meyer = the chart* (biography plotted, whole-organism reactions, feeds nosology and formulation), *Rank = the birth* (birth trauma, pre-Oedipal separation, will therapy, feeds humanistic and existential therapy). Freud, Adler and Jung are on **the General and Developmental Psychology notes**; if a Paper-1 stem names Meyer or Rank, it is testing exactly the deltas above.

GOOD TO REMEMBER

- **Three primary schools (the General and Developmental Psychology notes):** psychoanalysis (Freud), individual psychology (Adler), analytical psychology (Jung), named here, taught in full on the General and Developmental Psychology notes.
- **Topographic model:** conscious, preconscious, unconscious (Freud, the General and Developmental Psychology notes).
- **Structural model:** id, ego, superego (Freud, the General and Developmental Psychology notes).
- **Adolf Meyer (1866 to 1950):** founder of psychobiology; person as a biological unit; illness as a maladaptive reaction of the whole organism.
- **Ergasiology / ergasia:** Meyer's reaction-type vocabulary (anergasia, dysergasia); never entered formal nomenclature.
- **Life chart:** Meyer's biographical time-line of life events against illness onset; ancestor of the modern formulation timeline.
- **Distributive analysis and synthesis; habit training:** Meyer's therapeutic method.
- **Meyer's nosological legacy:** shaped DSM-I (1952) "reaction" terminology; precursor of the biopsychosocial model and community psychiatry.
- **Otto Rank (1884 to 1939):** Freud collaborator who broke away; pre-Oedipal, mother-centred focus.
- **Birth trauma (The Trauma of Birth, 1924):** Rank's relocation of the root of anxiety to the primal separation at birth; life fear versus death fear.
- **Will therapy (Will Therapy, 1936):** Rank's active, patient-centred, present-relationship therapy; mobilises the creative will; separation at termination is curative.
- **Rank's influence:** Rogerian client-centred (humanistic) therapy, existential therapy, short-term dynamic therapy, functional social work.
- **Neo-Freudian bridge:** Horney, Sullivan, Fromm, the relational/interpersonal turn developed in the object-relations topic next.

13 · Defence mechanisms and the Vaillant hierarchy

A **defence mechanism** is an automatic, unconscious mental process the ego uses to protect against the anxiety generated by intrapsychic conflict, unacceptable impulses, and external stressors (Kaplan & Sadock 2024). The concept is Freudian in origin but was first catalogued and systematised by **Anna Freud** in *The Ego and the Mechanisms of Defence* (1936), which moved the defences from scattered clinical observations into a coherent list. The examiner's favoured organising frame, and the one that carries the most marks, is **George Vaillant**'s arrangement of the defences into a maturity **hierarchy**: from the most pathological (psychotic) up through immature and neurotic to the most adaptive (mature). The same American spelling, defense mechanism, appears throughout the US texts and the DSM.

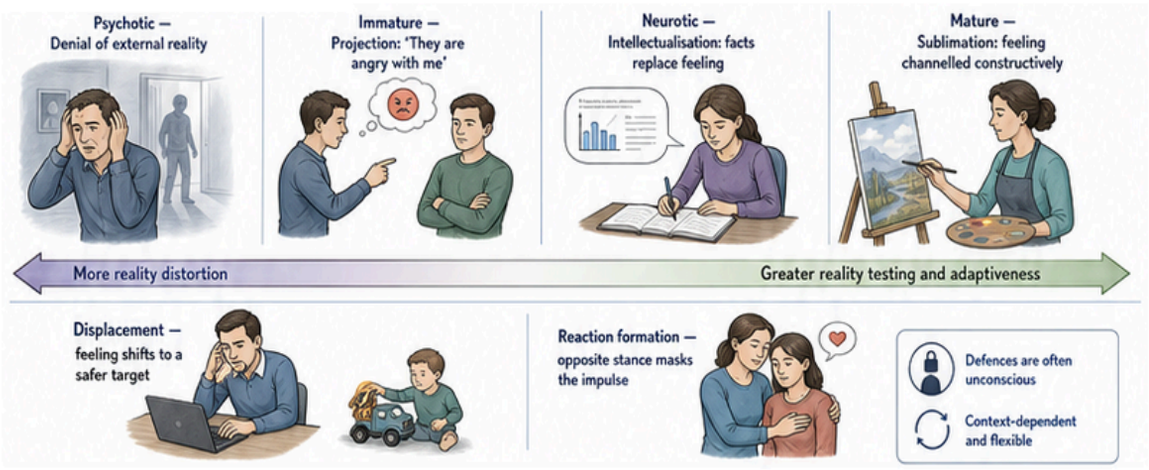


Fig 5.17 Defence mechanisms in action.

Why it matters. Defences are not inherently pathological. They are part of normal development and everyone uses the whole range, but the *habitual predominant level* a person deploys under stress is diagnostically and prognostically informative. The immature cluster (splitting, projective identification) anchors the borderline and other severe personality disorders, while the mature cluster predicts good long-term outcome. Learn the four tiers, one crisp definition and one clinical example each, and the disorder anchors, and this topic is essentially closed.

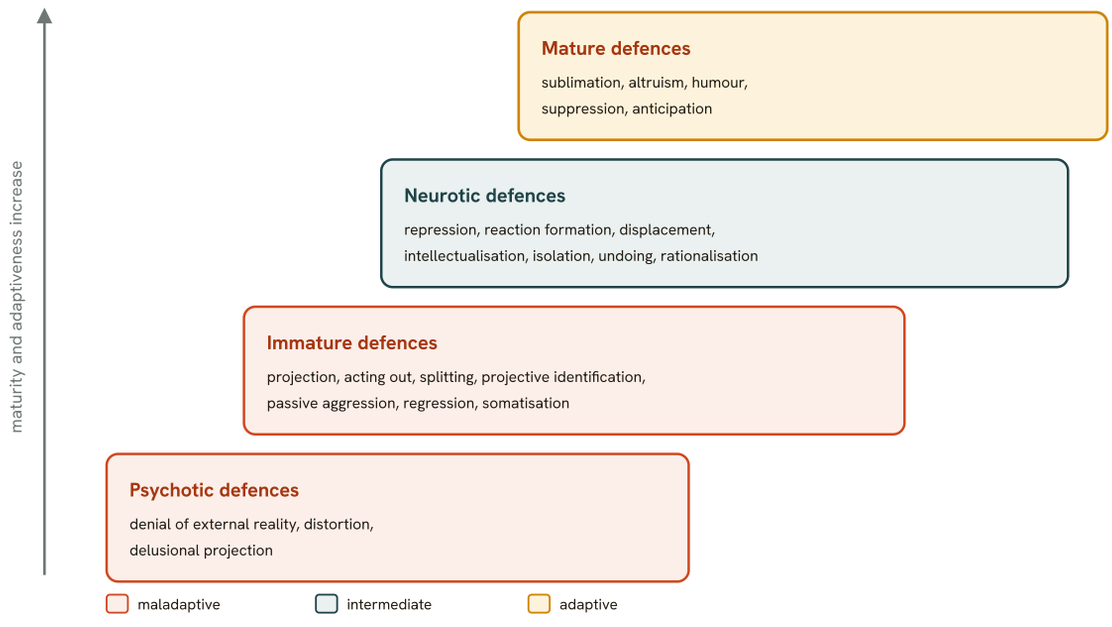


Fig 5.18 Vaillant's hierarchy of defence mechanisms, arranged from the least to the most adaptive. Psychotic defences deny or remake external reality; immature defences externalise conflict onto others; neurotic defences keep the conflict internal and out of awareness; mature defences channel it into something useful or tolerable. The same person uses defences from several levels, but the habitual level predicts functioning and outcome, a finding from Vaillant's long Grant Study cohort. Splitting and projective identification at the immature level are the exam's signature borderline defences.

13.1 Definition and the three founding ideas

The core definition. Defence mechanisms are automatic, unconscious psychological processes, both cognitive and emotional, that protect against anxiety arising from internal conflict or external threat (Kaplan & Sadock 2024). Three features are exam-worthy: they operate *outside awareness* (this separates a defence from a conscious coping strategy), they *distort or exclude* some aspect of internal or external reality to reduce unpleasure, and they change the relationship between the self and the object (Vaillant & Vaillant 1998). Anna Freud's 1936 monograph is the systematising text; her father Sigmund Freud had described repression as the foundational defence, and Melanie Klein later elaborated the earliest, most primitive defences (splitting, projective identification, projection) as characteristic of the paranoid-schizoid position of infancy.

13.2 The evolution of the classification, and the currently accepted frame

From a flat list to a maturity gradient. Anna Freud (1936) gave a flat catalogue of about ten defences with no ordering. The advance that the exam rewards is **Vaillant**'s reconceptualisation of the defences as a developmental *hierarchy of affect regulators*: more sophisticated, more effective defensive strategies emerge across adolescence and adulthood, and a person's defensive level can be scored empirically (Vaillant 1993, *The Wisdom of the Ego*). This fixed the previous scheme's silence on adaptiveness: it tells you which defences are healthy and which are pathological, and it made defence a *measurable* variable in longitudinal research rather than a purely interpretive one. The **currently accepted** operationalisation is Vaillant's four-tier model (psychotic, immature, neurotic, mature), whose adaptiveness ranking DSM-IV carried forward in the more finely graded seven-level *Defensive Functioning Scale* in its appendix. Its *neural correlate* is not a single structure: the automaticity of defence maps onto implicit procedural memory systems that deal with unpleasant affect outside awareness (Gabbard), and the regulation of affect that defences perform is subserved by prefrontal (especially ventromedial and dorsolateral) modulation of limbic structures, notably the amygdala. Mature suppression, for example, recruits deliberate prefrontal control over amygdala-driven affect, whereas psychotic-level denial reflects a failure of reality-testing networks. Name the systems, not a single locus.

HOLD THIS

Vaillant ranks the defences by maturity: psychotic, immature, neurotic, mature. Everyone uses the whole range; the habitual predominant level under stress is what is diagnostic and prognostic, and mature defences predicted better outcomes in the Grant Study.

CLINICAL ANCHOR

Defensive level is part of the psychodynamic formulation and of the mental state's assessment of insight and coping. On the ward, a patient who idealises the morning nurse and vilifies the evening nurse (splitting) signals a borderline-level organisation; a patient who channels grief over a terminal diagnosis into meticulous advance-care planning (anticipation) signals mature functioning and a better prognosis.

13.3 Level 1: Psychotic defences

Psychotic level. These are the most primitive defences, marked by a frank break with reality-testing. They are normal in early childhood and in dreams, but in an adult they signify psychosis. The three classic **psychotic defense** mechanisms:

Denial (of external reality)

Refusal to accept an intolerable external fact, abolishing its very perception. Example: a man with an obvious hemiplegia after a stroke insists nothing is wrong with his arm (anosognosia).

Distortion

Grossly reshaping external reality to fit inner need, including wish-fulfilling delusions and hallucinations. Example: a patient reworks a hostile world into a grandiose belief that he is a chosen prophet.

Delusional projection

Externalising one's own unacceptable impulses as frank persecutory delusions. Example: a patient's own aggressive impulses are experienced as a neighbour's plot to poison him.

13.4 Level 2: Immature defences

Immature level. Common in adolescence, in severe depression, and centrally in the personality disorders. Reality-testing is intact, but the defences are socially undesirable and often externalise distress onto others. This tier includes the Kleinian primitive defences.

Projection

Attributing one's own unacceptable feelings or impulses to another person. Example: a man who resents his wife accuses her of hating him. Core defence of paranoid personality disorder.

Acting out

Discharging an unconscious wish or conflict directly through action rather than reflection, to avoid feeling it. Example: a bereaved adolescent starts fights instead of grieving.

Splitting

Keeping contradictory images of self and others (all-good vs all-bad) rigidly apart because they cannot be integrated. Example: idealising the ward team on Monday and condemning them as cruel on Tuesday. The primary defence of borderline personality disorder (Kaplan & Sadock 2024).

Projective identification

Projecting a part of the self into another, then unconsciously pressuring that person to actually feel or enact it, so the projection is confirmed. Example: a patient projects rage into the therapist and behaves so provocatively that the therapist genuinely becomes angry. Hallmark of borderline organisation; strong driver of countertransference.

Passive aggression

Expressing hostility indirectly through non-compliance, procrastination, or inefficiency. Example: an angry trainee agrees to a task then quietly never completes it.

Regression

Retreating to an earlier developmental level of functioning under stress. Example: a hospitalised adult becomes clingy and demands to be fed.

Somatisation

Converting intrapsychic conflict into bodily symptoms and preoccupation. Example: unexpressed marital distress presents as recurrent unexplained abdominal pain.

Schizoid fantasy

Retreating into an inner world of imagination to resolve conflict and avoid intimacy. Example: a lonely person substitutes an elaborate fantasy life for real relationships (schizoid personality).

13.5 Level 3: Neurotic defences

Neurotic level. Common in everyone under stress and prominent in the neurotic disorders (anxiety, obsessive-compulsive, dissociative, and conversion presentations). They alter the internal experience of a conflict rather than distorting external reality; they are largely private and cause the person, not others, the difficulty.

Repression

The foundational neurotic defence: unconsciously barring an unacceptable idea or feeling from awareness. Example: a woman has no memory of a childhood assault. Distinguish from conscious suppression (mature).

Reaction formation

Transforming an unacceptable impulse into its exact opposite. Example: a person with unconscious hostility becomes exaggeratedly solicitous. Generates obsessive-compulsive personality traits.

Displacement

Redirecting an affect from its true object onto a safer substitute. Example: a man berated by his boss goes home and shouts at his child. Underlies phobia formation in psychodynamic theory.

Intellectualisation

Managing a threatening feeling by engaging with it only in abstract, theoretical terms. Example: a newly diagnosed patient discusses cancer statistics at length but shows no emotion.

Isolation of affect

Separating an idea from the feeling attached to it, so the idea stays in consciousness stripped of emotion. Example: an OCD patient recounts an aggressive intrusive thought flatly, with no distress.

Undoing

Performing an act (often ritualistic) that magically cancels or atones for an unacceptable thought or impulse. Example: a compulsion to wash repeatedly to undo a feared harm. Generates OCD compulsions.

Rationalisation

Offering a plausible, reassuring, but false reason for behaviour whose true motive is unacceptable. Example: a rejected candidate decides the job was beneath him.

Dissociation

A temporary drastic modification of one's sense of identity or consciousness to avoid emotional distress. Example: a person derealises during an assault and later has amnesia for it.

13.6 Level 4: Mature defences

Mature level. The most adaptive tier, characteristic of healthy adults. Mature defences integrate conflicting motives, optimise gratification, and are experienced by others as virtues. They are the

residues that become stable, partly conscious coping styles and mature character traits (Kaplan & Sadock 2024). The five classic **mature defense** mechanisms:

Sublimation
Channelling an unacceptable impulse into a socially valued activity, keeping the aim's energy while altering its object. Example: aggressive drive redirected into competitive surgery or contact sport.

Altruism
Meeting one's own needs vicariously through constructive service to others. Example: a bereaved parent founds a support charity.

Humour
Expressing a distressing feeling openly without discomfort to self or others, giving perspective without denial. Example: a patient jokes gently about his own illness to ease a tense consultation.

Suppression
The conscious or semiconscious decision to postpone attention to a conflict or impulse. Example: a doctor deliberately sets aside personal worry to finish a night shift, then returns to it. The one deliberate defence; contrast with unconscious repression.

Anticipation
Realistic planning for future inner discomfort, rehearsing the affect in advance. Example: rehearsing likely feelings before a difficult conversation, so the emotion is manageable when it arrives.

13.7 Vaillant's Grant Study: the outcome data

The empirical payoff. Vaillant's evidence base was the **Grant Study**, the roughly 60-year prospective longitudinal follow-up of an initially healthy cohort of Harvard undergraduates (Vaillant 1995), part of the Harvard Study of Adult Development. Rating each man's habitual defensive style, Vaillant found that predominant use of *mature* defences (sublimation, altruism, humour, suppression, anticipation) predicted better outcomes across the board: mental and physical health, career and income success, marital stability, and life satisfaction. Predominant *immature* defences predicted the reverse. Two exam-relevant conclusions follow: defensive maturity is not fixed but can advance across the lifespan, and it is one of the stronger psychosocial predictors of successful ageing, independent of the childhood social class the men started from.

13.8 Disorder anchors: which defence flags which condition

The high-yield associations. Examiners test defences most often through their disorder pairings. The table collects the ones that recur in questions.

DEFENCE	VAILLANT LEVEL	CLASSIC DISORDER ASSOCIATION
Splitting	Immature	**Borderline personality disorder (primary defence)**
Projective identification	Immature	**Borderline organisation; drives countertransference**
Projection	Immature	**Paranoid personality disorder; paranoid states**
Schizoid fantasy	Immature	Schizoid personality disorder
Isolation of affect, undoing	Neurotic	**Obsessive-compulsive disorder (compulsions)**
Reaction formation	Neurotic	Obsessive-compulsive personality traits
Displacement	Neurotic	**Phobia formation**
Repression, dissociation	Neurotic	Dissociative and conversion disorders
Denial, distortion, delusional projection	Psychotic	**Psychotic disorders; acute mania (denial)**

The pairings that generate single-best-answer questions: splitting to borderline, projection to paranoid, isolation and undoing to OCD, displacement to phobia.

RULE **Only psychotic-level defences abolish reality-testing.** If a stem preserves reality-testing, the answer is never denial of external reality, distortion or delusional projection.

COMMON TRAP

Do not confuse **suppression** with **repression**. Suppression is *conscious and deliberate* (a mature defence, the only one that is intentional); repression is *unconscious* (a neurotic defence). A stem describing a person who "decided to put the worry out of mind until the exam was over" is suppression, not repression. Likewise, **projection** (immature: attributing your feeling to another) is not **projective identification** (immature: making the other actually feel it).

PEARL

Reality-testing is the fault line between the top and bottom of the ladder. Only the *psychotic* defences abolish reality-testing (denial of external reality, distortion, delusional projection). Every immature, neurotic, and mature defence leaves reality-testing intact and distorts only the internal experience. If a stem preserves reality-testing, the answer is never a psychotic-level defence.

MNEMONIC

Some Adults Have Sensible Anticipation

The five **mature** defences: **S**ublimation, **A**ltruism, **H**umour, **S**uppression, **A**nticipation. These are the ones the Grant Study tied to good outcome, so if a question asks for the "healthiest" defence in a list, pick from this five.

INDIA

Indian teaching frames the defences through their psychodynamic disorder models: isolation of affect, undoing, and displacement generating OCD symptoms; projection generating paranoid presentations; splitting as the borderline defence. For theory-paper answers, pair each defence with its disorder model rather than listing defences in isolation.

GOOD TO REMEMBER

- **Defence mechanism:** automatic unconscious ego process that reduces anxiety from conflict or external threat (Kaplan & Sadock 2024).
- **Anna Freud (1936):** systematised the defences in *The Ego and the Mechanisms of Defence*.
- **George Vaillant (1993):** arranged defences into a four-tier maturity hierarchy of affect regulators; basis of DSM-IV Defensive Functioning Scale.
- **Psychotic defences:** denial of external reality, distortion, delusional projection (reality-testing lost).
- **Immature defences:** projection, acting out, splitting, projective identification, passive aggression, regression, somatisation, schizoid fantasy.
- **Neurotic defences:** repression, reaction formation, displacement, intellectualisation, isolation of affect, undoing, rationalisation, dissociation.
- **Mature defences:** sublimation, altruism, humour, suppression, anticipation (SAHSA).
- **Splitting and projective identification:** primary defences of borderline personality disorder.
- **Projection:** defence of paranoid personality disorder / paranoid states.
- **Isolation of affect and undoing:** defences behind OCD compulsions; displacement behind phobia.
- **Suppression vs repression:** suppression is conscious and mature; repression is unconscious and neurotic.
- **Grant Study (Vaillant 1995):** ~60-year Harvard cohort; predominant mature defences predicted better health, relationships, and successful ageing.

14 · Object relations and the neo-Freudians

The relational turn in psychoanalysis, and the school Paper 1 tests most. After Freud, one large branch of psychoanalysis stopped asking "how does the drive discharge?" and started asking "whose face is the drive reaching for?". That shift is **object relations** theory: the mind is built not out of drives seeking pleasure but out of internalised relationships, the residues of early experience with caregivers laid down as **internal objects** that then govern how the person loves, hates and perceives others for life. Alongside it a looser group, the **neo-Freudian** analysts, kept Freud's method but replaced his biological drive with culture, interpersonal reality and social forces. This topic is high-yield: examiners pair a theorist with a signature concept (Klein and the positions, Winnicott and the transitional object, Fairbairn and object-seeking libido, Kohut and the selfobject, Kernberg and levels of organisation) and they trace the line from these ideas into the borderline-personality therapies now used in the clinic.

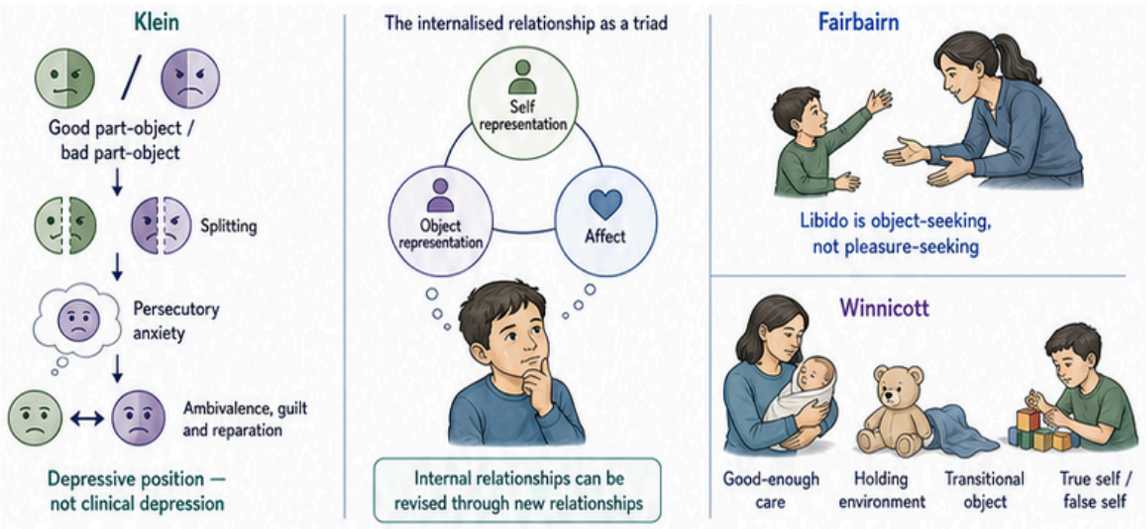


Fig 5.19 Object relations: the internal relational world.

The exam framing follows a clean lineage. Melanie Klein opens the object-relations school by turning Freud's drives inward into a world of good and bad internal objects; the British Independent school (Fairbairn, Winnicott, Balint) makes relationship, not drive, the prime mover; Kohut and Kernberg carry the school into the modern understanding of narcissism and borderline pathology; and Horney, Sullivan and Fromm run in parallel as the socio-cultural neo-Freudians. Mahler (separation-individuation), Adler and Jung belong to the General and Developmental Psychology notes and are only cross-referenced here. Structure below is the standard evolution-of-theories spine: definition, then each theorist in the order the ideas appeared and what each fixed from the last, then the currently accepted synthesis, its neural correlate, and a worked clinical example.

14.1 What "object relations" means

The object is a person, held inside the mind. In psychoanalytic usage the *object* is the target of a drive or a relationship, in practice a person or part of a person (Freud's original term). Object relations theory holds that the infant internalises early relationships as **internal objects**, mental representations of self and other bound together by an affect, and that these internalised self-object-affect units, not the vicissitudes of libido, are the fundamental structures of personality and psychopathology (Kaplan & Sadock 2024). The clinical corollary is that neurosis and character pathology are disturbances in the internal object world, and that the treatment relationship works by offering a new relational experience that can revise those internal objects.

14.2 Melanie Klein: the two positions and the internal object world

Klein turns the drives inward into a world of good and bad objects. Melanie Klein founded the object-relations tradition by taking Freud's late instinct theory, above all the death instinct, and relocating the action into the infant's earliest months (Kaplan & Sadock 2024). Driven to expel intolerable destructive impulses, the infant projects them outward, so the first relationships are with *part-objects*, the gratifying *good breast* and the depriving, persecutory *bad breast*, not yet

combined into a whole mother. Klein described two developmental configurations she called *positions* (recurring modes of experience, not one-off stages).

Paranoid-schizoid position

The earliest mode (roughly the first months). The object is split into an all-good and an all-bad part-object; the dominant anxiety is *persecutory* (fear that the bad object will attack and annihilate the self); the dominant defence is **splitting** together with projection and idealisation. "Schizoid" names the splitting; "paranoid" names the persecutory dread.

Depressive position

The later mode (from around weaning, but revisited lifelong). The infant recognises that the good and bad breast are one whole mother, and that its own aggression has been aimed at the very object it loves and needs. The anxiety shifts from persecution to *guilt and concern*; the developmental work is mourning, reparation and tolerating ambivalence. Reaching and holding the depressive position is, for Klein, the hallmark of psychic maturity.

Two further Kleinian concepts are examinable in their own right. **Projective identification** (elaborated by Klein and extended by Wilfred Bion) is a process in which the subject splits off a part of the self, projects it into another person, and then unconsciously pressures that person to feel and behave in line with the projection, so it is at once a defence, a primitive communication and an interpersonal manoeuvre (Kaplan & Sadock 2024). Bion added the *container-contained* metaphor: the mother (or analyst) absorbs, detoxifies and returns the projected distress in tolerable form for re-internalisation. Klein is regularly criticised for reading all aggression as the death instinct and for dating complex structures such as the superego implausibly early, but her achievement (the internal object world, the two positions, splitting, projective identification) is the foundation of the whole school.

TRAP Klein's **depressive position is a developmental achievement** (whole-object relating, guilt, reparation), not clinical depression.

14.3 Ronald Fairbairn: libido is object-seeking, not pleasure-seeking

The single sentence that defines the school. Ronald Fairbairn made the decisive theoretical break: he held that *libido is object-seeking, not pleasure-seeking* (Kaplan & Sadock 2024). For Freud the infant seeks tension-reducing gratification and the object is merely the means; for Fairbairn the infant seeks the object (the relationship) from the start, and pleasure is the signpost to the object, not the goal. He recast the ego, in Freud a superficial servant of the id, as the core of the psyche, an authentic self that is object-relating from birth. Working chiefly with schizoid patients, he argued their central problem was not controlling dangerous impulses but having an ego capable of relating to objects at all. To manage unsatisfying early relationships the ego *splits* internally, giving Fairbairn's **endopsychic structure**: a central ego relating to an ideal object, a libidinal ego tied to an exciting (tantalising) object, and an anti-libidinal ego (the internal saboteur) tied to a rejecting object. This tripartite internal world of self-and-object pairs is Fairbairn's signature and a direct ancestor of Kernberg's later model.

HOLD THIS

Fairbairn's one line defines the whole school: libido is object-seeking, not pleasure-seeking. The infant seeks the relationship from the start; pleasure is the signpost to the object, not the goal.

14.4 Donald Winnicott: good-enough mothering, the transitional object, the true and false self

The paediatrician-analyst of the Independent school. Donald Winnicott, a paediatrician before he was an analyst and the best-known figure of the British *Independent* (Middle) group, grounded development in the earliest mother-infant relationship (Kaplan & Sadock 2024). His concepts are a cluster examiners love.

Good-enough mother

The mother need not be perfect, only **good-enough**: reliably responsive at first, then failing the infant in small, graduated, tolerable ways so that the child moves from absolute dependence to relative dependence and learns to tolerate frustration. Perfect attunement would in fact impede growth.

Holding environment

The physical and emotional "holding" the mother provides, later a metaphor for the safe, reliable containment the analyst provides in therapy.

Primary maternal preoccupation

The heightened, almost illness-like state of identification with the baby that lets the mother sense and meet its needs in the earliest weeks.

Transitional object

The child's first "not-me" possession (the blanket, the teddy) that stands in for the mother and occupies an intermediate *transitional* space that is neither wholly inner fantasy nor wholly outer reality. It is the developmental bridge from subjective omnipotence to objective reality; Winnicott saw this same intermediate realm of illusion surviving in adult creativity, play, art and religious experience (these are the **transitional** phenomena).

True self and false self

From authentic spontaneous gestures met by good-enough care grows the *true self*. When care is impinging or the child must comply with the mother's needs, a compliant *false self* is built as a protective shell; such patients present as neither neurotic nor psychotic but as subtly unreal, disengaged and compliant.

14.5 The British Independent school and Balint

The middle group that made relationship primary. The *British Independent* (or Middle) group, which formed to sit between Anna Freud's ego-psychological Freudians and the Kleinians during the wartime "Controversial Discussions", is the home of Fairbairn, Winnicott and Michael Balint. Balint's contribution is the **basic fault**: a level of pre-verbal, two-person early damage below the reach of ordinary verbal interpretation, where the therapeutic task is empathic recognition rather than interpretation, and he distinguished benign from malignant regression in treatment (Kaplan & Sadock 2024). The common thread of the Independents is that relationship with the object, not drive discharge, is the engine of development, and that the earliest failures are pre-Oedipal and require a relational, not purely interpretive, cure.

14.6 Heinz Kohut: self psychology and the selfobject

The self, not the drive, at the centre. Heinz Kohut founded **self psychology** in the United States after finding classical drive theory unhelpful with narcissistic patients (Kaplan & Sadock 2024). His organising unit is the **selfobject**: another person experienced not as a separate individual but as a part of, and in the service of, the self, providing functions the self cannot yet perform alone. The infant needs selfobject responses across development, and Kohut named three selfobject transferences that reveal these needs in treatment: *mirroring* (the need to be admired and confirmed, met originally by the gleam in the mother's eye and reflecting the child's *grandiose self*), *idealising* (the need to merge with a calm, strong, idealised figure, the *idealised parental imago*), and *twinship*/alter-ego (the need for likeness and belonging). Normal narcissism matures through gradual, non-traumatic disappointments (*optimal frustration* and *transmuting internalisation*) into healthy self-esteem, ambitions and ideals. Pathological narcissism reflects failed **empathic** mirroring and a self left prone to fragmentation, and the treatment is above all empathic, restoring the arrested developmental need rather than interpreting a conflict.

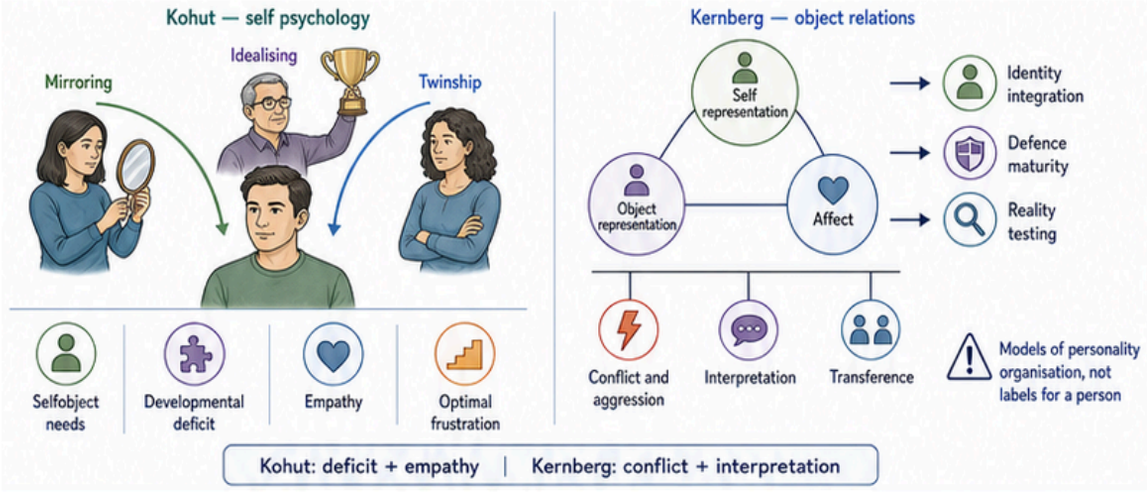


Fig 5.20 Kohut and Kernberg: two models of narcissism.

14.7 Otto Kernberg: the object-relations model of borderline organisation

Drive and object relations, fused, staged by severity. Otto Kernberg integrated ego psychology with object relations and, unlike Kohut, kept libido and aggression inside the model (Kaplan & Sadock 2024). For Kernberg the building blocks of the psyche are *self-representation, object-representation and the affect linking them*, laid down under highly activated early affect states; excess constitutional aggression is central to severe pathology. His most examined contribution is the classification of personality by **levels of personality organisation**, defined by three variables: identity integration, the maturity of defences, and reality testing.

LEVEL	IDENTITY	PREDOMINANT DEFENCES	REALITY TESTING
Neurotic organisation	Integrated (stable sense of self and others)	**Repression-based (repression, reaction formation, intellectualisation)**	Intact
Borderline organisation	**Identity diffusion (contradictory, unintegrated self and object images)**	**Splitting-based (splitting, projective identification, denial, idealisation/devaluation, omnipotence)**	Grossly intact but fragile under stress
Psychotic organisation	Identity diffusion	Splitting-based, more primitive	**Lost**

The two discriminators that place a patient are identity (integrated versus diffuse) and reality testing (intact in neurotic and borderline, lost in psychotic); splitting versus repression separates borderline from neurotic. This is Kernberg's structural interview in one grid.

Borderline *personality organisation* is a broad structural level (identity diffusion + splitting defences + preserved reality testing) that is wider than the DSM/ICD borderline personality *disorder* category and houses much of severe personality pathology. This model is the theoretical engine of transference-focused psychotherapy (below).

14.8 Kohut versus Kernberg on narcissism

The single most-tested contrast in the topic. Both wrote the modern account of narcissism, and they disagree at the root.

FEATURE	KOHUT (SELF PSYCHOLOGY)	KERNBERG (OBJECT RELATIONS)
Status of drive theory	**Discards drive theory; deficit model**	**Retains libido and aggression; conflict model**
Nature of the grandiose self	A fixation of a *normal* archaic infantile self, arrested by empathic failure	A *pathological* structure fusing real self, ideal self and ideal object, saturated with aggression
Core problem	**Developmental *deficit* from failed empathy**	**Defensive structure against oral *aggression* and envy**
Stance in treatment	Empathic, provides missing self-object experience	Interpretive and confrontational; interprets aggression and defences in the transference

Deficit-and-empathy (Kohut) versus conflict-aggression-and-interpretation (Kernberg) is the discriminator line; if a stem contrasts two narcissism theorists, it is testing exactly this.

RULE **On narcissism, Kohut is deficit plus empathy; Kernberg is conflict, aggression and interpretation.** They are opposites, not variants, and a stem contrasting two narcissism theorists is testing exactly this.

14.9 The neo-Freudians: Horney, Sullivan, Fromm

Keep the method, swap the drive for culture and relationships. The **neo-Freudian** analysts accepted Freud's clinical method but replaced his biological instinct theory with social, cultural

and interpersonal determinants.

Karen Horney

Horney located neurosis in disturbed early relationships producing **basic anxiety**, the child's feeling of isolation and helplessness in a potentially hostile world. The child defends against it with one of three *neurotic trends* (interpersonal orientations): *moving toward people* (compliance, seeking love), *moving against people* (aggression, seeking mastery), and *moving away from people* (detachment, seeking self-sufficiency). She is also the first major critic of Freud's *penis envy*, arguing it was a cultural artefact of women's subordinate social position rather than an anatomical inevitability, and she countered it with the notion of male *womb envy*. Her later "tyranny of the should" and the idealised self prefigure cognitive therapy.

Harry Stack Sullivan

Sullivan founded *interpersonal theory*: personality is "the relatively enduring pattern of recurrent interpersonal situations", not an intrapsychic apparatus, so it cannot even be studied apart from relationships. Anxiety originates interpersonally, transmitted from mother to infant, and the self-system develops to avoid it, sorting experience into the *good-me*, *bad-me* and (dissociated, anxiety-laden) *not-me*. He described the *parataxic distortion* (perceiving a present person through the template of a past relationship, an interpersonal reading of transference) and stressed the therapist as a *participant observer*.

Erich Fromm

Fromm, the social and humanistic analyst, sited the person within society and history. In *Escape from Freedom* he argued that modern freedom brings isolation, so people flee it through authoritarianism, destructiveness or automaton conformity. He described non-productive character orientations (receptive, exploitative, hoarding, marketing) versus the healthy *productive* orientation, and set human *existential needs* (relatedness, transcendence, rootedness, identity, a frame of orientation) at the centre of character.

14.10 Currently accepted position

Metabolised, not practised intact. No single one of these systems is used whole today, but the **currently accepted** position is that object relations is the dominant relational framework in contemporary psychodynamic practice, converging with attachment theory (the General and Developmental Psychology notes) and with mentalisation research into a single account: personality is organised around internalised relationships (internal working models / internal objects), and both develop through the caregiver's empathic responsiveness. The practical descendants are two manualised, evidence-based therapies for borderline personality: *transference-focused psychotherapy* (Kernberg and colleagues), which uses the here-and-now transference to integrate split self and object representations, and *mentalisation-based treatment* (Bateman and Fonagy), which grows the capacity to read mental states in self and other, itself rooted in attachment and object-relations thinking (cross-referenced with the personality-disorder and psychotherapy topics). Klein's splitting, Winnicott's holding and Kohut's empathic stance are now common technical vocabulary across the psychodynamic field.

14.11 Neural correlate

Where the internal object world lives, tentatively, in the brain. These are psychological constructs, so any *neural correlate* is a mapping onto systems rather than a discovery within the theories, and it should be held loosely. The *neurobiology* most relevant is the social-cognition and attachment circuitry: representing self and other and reading their mental states engages the *medial prefrontal cortex*, *temporoparietal junction* and *posterior superior temporal sulcus* (the mentalising network that underwrites Fonagy's reflective function). The affect that binds each internal object is carried by the *amygdala* and *anterior cingulate cortex*, and the attachment and selfobject-seeking pull rests on the *oxytocin* and endogenous-opioid systems and mesolimbic reward pathways. Splitting and identity diffusion in borderline organisation track onto impaired prefrontal (especially ventromedial and dorsolateral) regulation of an over-reactive amygdala, the same amygdala-prefrontal imbalance described in the borderline-personality neurobiology. The claim is only that internalised-relationship constructs plausibly ride on the brain's social-cognitive, affective and attachment systems, not that Klein or Kohut localised anything.

14.12 Worked example: one patient through the school

A young woman with unstable relationships and self-harm. Take a patient who idealises a new partner one week and vilifies them the next, cannot say who she is when alone, and cuts herself after feeling abandoned. *Klein:* she is operating largely in the paranoid-schizoid position, splitting each person into all-good and all-bad and using projective identification, drawing others into the very rage she fears. *Winnicott:* a history of impinging, non-attuned early care fostered a false-self compliance masking an unmet true self, and the therapeutic frame must first provide reliable holding. *Kohut:* she seeks selfobjects to steady a fragile self and fragments when mirroring fails, so an empathic stance is primary. *Kernberg:* she shows borderline personality organisation, identity diffusion plus splitting defences with intact reality testing, and transference-focused work would name the alternating idealised and persecutory object relations as they appear with the therapist to integrate them. *Fonagy:* her deficit is mentalising under attachment stress, the explicit target of mentalisation-based treatment. Each lens reads the same clinical picture; together they are the object-relations account of borderline pathology and its treatment.

CLINICAL ANCHOR

Splitting is the concept that unites the school with the ward. The patient who divides the team into all-good and all-bad staff, and the borderline patient who idealises then devalues the same clinician, is displaying Klein's paranoid-schizoid splitting operationalised in Kernberg's borderline organisation. Recognising it (and not taking a side in the split) is the practical yield of this entire topic.

COMMON TRAP

Do not confuse the paired opposites. Klein's *depressive position* is a developmental achievement (whole-object relating, guilt, reparation), not clinical depression. Kohut is *deficit + empathy*; Kernberg is *conflict + aggression + interpretation*, they are opposites on narcissism, not variants. Fairbairn's line is "libido is object-seeking, not pleasure-seeking" (do not swap it onto Winnicott). And keep the neo-Freudians (Horney, Sullivan, Fromm), who replace the drive with culture and interpersonal reality, separate from the object-relations analysts (Klein, Fairbairn, Winnicott), who keep an internal-world focus.

MNEMONIC

"Kids Fear Weird Bosses; Kohut Knows Kids"

The object-relations line in order, then the two moderns: **K**lein (positions, splitting), **F**airbairn (object-seeking libido, endopsychic structure), **W**innicott (good-enough, transitional object, true/false self), **B**alint (basic fault); then **Kohut** (selfobject, mirroring) and **K**ernberg (levels of organisation). For Horney's three trends: "toward, against, away".

GOOD TO REMEMBER

- **Object relations:** mind built from internalised relationships (internal objects), not drive discharge; object = a person or part-person.
- **Melanie Klein:** founder of the school; part-objects (good/bad breast); death-instinct basis.
- **Paranoid-schizoid position:** earliest mode; splitting + persecutory anxiety; all-good vs all-bad part-objects.
- **Depressive position:** whole-object relating; guilt, concern, mourning, reparation; a maturational achievement, not clinical depression.
- **Projective identification:** Klein (extended by Bion); split-off part projected into another who is pressured to enact it; Bion's container-contained.
- **Ronald Fairbairn:** "libido is object-seeking, not pleasure-seeking"; endopsychic structure (central ego / libidinal ego + exciting object / anti-libidinal ego (internal saboteur) + rejecting object); schizoid focus.
- **Donald Winnicott:** good-enough mother; holding environment; primary maternal preoccupation; transitional object and transitional phenomena; true self and false self.
- **Michael Balint:** the basic fault (pre-verbal two-person deficit); benign vs malignant regression.
- **British Independent (Middle) school:** Fairbairn, Winnicott, Balint; relationship over drive; pre-Oedipal focus.
- **Heinz Kohut:** self psychology; the selfobject; mirroring, idealising and twinship transferences; grandiose self and idealised parental imago; optimal frustration and transmuting internalisation; empathy central; discards drive theory (deficit model).
- **Otto Kernberg:** integrates object relations with drive theory (conflict model); self-representation + object-representation + affect; levels of personality organisation (neurotic / borderline / psychotic) defined by identity, defences (repression vs splitting), reality testing; borderline personality organisation; engine of transference-focused psychotherapy.
- **Kohut vs Kernberg:** deficit + empathy vs conflict + aggression + interpretation; normal-archaic vs pathological grandiose self.
- **Karen Horney:** basic anxiety; three neurotic trends (moving toward, against, away from people); critique of penis envy (cultural, not anatomical); womb envy.
- **Harry Stack Sullivan:** interpersonal theory; personality as recurrent interpersonal situations; good-me / bad-me / not-me; parataxic distortion; participant observer.
- **Erich Fromm:** social/humanistic psychoanalysis; Escape from Freedom; character orientations (receptive, exploitative, hoarding, marketing, productive); existential needs.
- **Clinical reach:** transference-focused psychotherapy (Kernberg) and mentalisation-based treatment (Bateman and Fonagy) for borderline personality; converges with attachment theory (the General and Developmental Psychology notes).

15 · Personality theories: trait, type, humanistic and positive psychology

The century-long argument about what a person *is*, told as a sequence of theories each fixing the last one's blind spot. **Personality** is the enduring, characteristic pattern of thinking, feeling and behaving that distinguishes one individual from another and shows consistency across time and situations. The exam does not want a single definition; it wants the four grand traditions and how they answer the same question differently: the **psychodynamic** tradition (personality as the outcome of unconscious conflict and defence, covered under Freud, Jung, Adler on the General and Developmental Psychology notes), the **trait** tradition (personality as a profile of measurable stable dimensions), the **humanistic** tradition (personality as the striving of a whole self toward growth), and the **social-cognitive** tradition (personality as learned expectancies and constructs

interacting with situations). Before all four sat the ancient **type theories**, which sorted people into discrete boxes rather than placing them on continua (Kaplan & Sadock 2024; Companion to Psychiatric Studies).

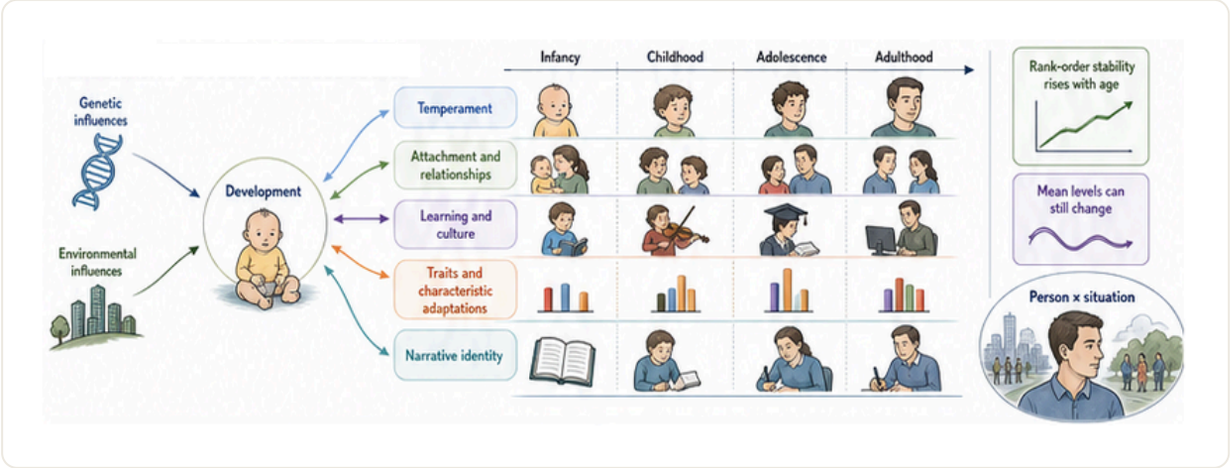


Fig 5.21 Personality develops across levels.

Structure your answer as an evolution. Type theories gave way to the trait tradition when Allport, Eysenck and Cattell showed that people are better described by where they fall on continuous dimensions than by which box they occupy; the trait tradition converged, via the lexical hypothesis and factor analysis, on the **Big Five** or five-factor model, which is the currently accepted descriptive consensus; the humanistic tradition (Rogers, Maslow, and the existential strand of May and Frankl) rose as a "third force" reacting against both the determinism of psychoanalysis and the mechanism of behaviourism; Kelly's personal construct theory bridged into social cognition; the person-situation debate raised by Mischel was resolved into interactionism; and behavioural genetics and the neuroscience of traits supplied the biological correlate. Positive psychology (Seligman, Csikszentmihalyi) is the newest layer, routed here from the General and Developmental Psychology notes, studying what makes life worth living rather than what makes it go wrong (Kaplan & Sadock 2024).



Fig 5.22 The five-factor model (OCEAN), the current descriptive consensus in trait theory. Each factor is a continuous dimension, not a type: a person sits somewhere along all five at once. The poles shown are the extremes; most people cluster near the middle. The model is robust across cultures and languages and is measured by the NEO-PI-R. Neuroticism is the factor most tied to psychopathology, and low conscientiousness and low agreeableness track with externalising problems.

	Psychodynamic	Trait	Type	Humanistic	Social-cognitive
unit	unconscious conflict	stable dimensions	discrete categories	the self, growth	person x situation
human nature	driven, determined	consistent traits	born a type	free, self-actualising	agentic, learning
assessed by	projectives, free association	NEO-PI, 16PF, EPQ	typologies	self-report, Q-sort	behavioural, self-efficacy
key figures	Freud, Klein	Allport, Eysenck	Sheldon, Kretschmer	Rogers, Maslow	Bandura, Mischel

Fig 5.23 The five traditions of personality theory compared. Each answers the same questions differently: what the basic unit of personality is, whether the person is determined or free, how personality is measured, and who its founders are. The trait tradition (now the Big Five) describes personality; the psychodynamic explains its unconscious roots; the type tradition sorts people into kinds; the humanistic centres the self and growth; the social-cognitive puts behaviour at the meeting of person and situation.

15.1 Type theories: the ancient precursor

Sorting people into boxes. A **type theory** proposes a small number of discrete, qualitatively distinct categories into which every person falls; you are one type or another, not more or less of a dimension. The oldest is the humoral scheme of **Hippocrates** and later **Galen**, which linked four bodily humours to four temperaments: blood to the *sanguine* (cheerful, sociable), black bile to the *melancholic* (gloomy, anxious), yellow bile to the *choleric* (irritable, driven), and phlegm to the *phlegmatic* (calm, slow). The word "temperament" preserves this idea of a mixture (Latin *temperare*, to mix). Two twentieth-century constitutional type theories are examinable:

Kretschmer correlated body build with temperament and psychosis-proneness (the lean *asthenic/leptosomic* build with schizoid/schizophrenic tendency, the rounded *pyknic* build with cyclothymic/manic-depressive tendency, plus the *athletic* and *dysplastic* types), and **Sheldon** restated this as three **somatotypes**, the soft rounded *endomorph* (viscerotonic, sociable, comfort-loving), the muscular *mesomorph* (somatotonic, assertive, energetic), and the thin fragile *ectomorph* (cerebrotonic, restrained, introverted). The fatal weakness of every type theory, and the reason the trait tradition replaced it, is that real people do not fall into clean boxes; the correlations were weak and the categories forced continuous variation into false dichotomies (Kaplan & Sadock 2024).

TRAP Type theories are **categories** (you are a type); trait theories are **dimensions** (you have a score on a continuum). The whole point of the trait tradition was to replace the former with the latter.

15.2 Allport: the founding of the trait tradition

What Allport fixed: continua instead of boxes, and the individual restored. Gordon **Allport** (his classic monograph appeared in 1937) is credited with making personality a scientific discipline and with defining the **trait** as a generalised, enduring neuropsychic disposition to render many stimuli functionally equivalent and to initiate consistent forms of behaviour (Kaplan & Sadock 2024). His enduring exam-content is a three-tier hierarchy of traits by how pervasive they are in a life:

Cardinal trait

A single dominant disposition so ruling that almost every act can be traced to it; rare, and it can lend its bearer's name to a quality (Machiavellian, narcissistic). Most people have none.

Central traits

The five to ten general characteristics that describe a person's core and would head a good reference letter (honest, anxious, sociable); the building blocks of personality.

Secondary traits

Situation-specific, peripheral dispositions and preferences (a dislike of crowds, a food preference) shown only in particular contexts and not central to the person.

Allport's second lasting contribution is the **idiographic** versus **nomothetic** distinction. The nomothetic approach studies universal dimensions on which everyone can be ranked and compared (the strategy of Eysenck, Cattell and the Big Five); the idiographic approach studies the unique organisation of the single individual, insisting that the pattern of one person cannot be fully captured by scores on group-derived dimensions. Allport championed the idiographic view while conceding the value of both, and the tension he named still runs through the field (Companion to Psychiatric Studies).

15.3 Eysenck: the biologically grounded trait model

What Eysenck fixed: few dimensions, each with a physiological cause. Hans **Eysenck** insisted that a good trait theory must be parsimonious and biologically anchored, and used factor analysis to reduce personality to a small number of higher-order dimensions with a causal substrate

(Eysenck & Eysenck 1975; Shorter Oxford Textbook of Psychiatry). His **PEN model** has three superfactors:

Extraversion to introversion (E)

Sociability, liveliness and stimulation-seeking versus quiet reserve. Eysenck's causal claim: introverts have *higher* baseline cortical arousal (an ascending reticular activating system set higher), so they seek less external stimulation; extraverts are chronically under-aroused and seek stimulation to compensate.

Neuroticism to stability (N)

Emotional lability, anxiety and reactivity versus calm and even-temperedness, grounded in the reactivity of the limbic/autonomic (visceral brain) system.

Psychoticism (P)

A dimension of tough-mindedness, aggression, impulsivity, coldness and non-conformity (not psychosis itself), added later and the weakest of the three empirically.

What Eysenck left debated: three dimensions proved too few. Many robust traits (notably conscientiousness and agreeableness) do not reduce cleanly to E, N and P, and the arousal theory of extraversion has only partial empirical support. His enduring win is the idea that traits are dimensions with a neurobiology, which the modern field kept (Kaplan & Sadock 2024).

15.4 Cattell: the factor-analytic 16PF

What Cattell fixed: let the data, not the theorist, decide the number of traits. Raymond **Cattell** took the opposite tack to Eysenck's parsimony. Starting from the thousands of trait words in the dictionary (the lexical raw material) and from behavioural and questionnaire data, he applied factor analysis to distil the many observable **surface traits** (clusters of correlated behaviour) down to the smaller set of underlying **source traits** that generate them. He arrived at sixteen source-trait factors and built the **16PF** (Sixteen Personality Factor Questionnaire) to measure them (Companion to Psychiatric Studies). What Cattell left debated was granularity: sixteen factors were too many to be stable and several were intercorrelated, so higher-order analysis of his own factors kept collapsing them into a handful of broad dimensions. That collapse pointed straight at five, and the field's centre of gravity shifted from sixteen to five.

> **PEARL**
>
> Read Eysenck and Cattell as two answers to one question, "how many traits?" Eysenck went top-down and biological and said three; Cattell went bottom-up and statistical and said sixteen. The Big Five sits between them and is where higher-order factoring of Cattell's sixteen and expansion of Eysenck's three both converge. That convergence, from opposite methods, is why the five-factor solution is so trusted.

15.5 The lexical hypothesis and the Big Five (five-factor model)

The currently accepted descriptive consensus. The **lexical hypothesis** holds that the personality differences most important in human affairs have become encoded as single words in every natural language, so factor-analysing the trait lexicon should reveal the true basic dimensions. Independent lexical and questionnaire programmes across the 1980s and 1990s

repeatedly recovered the same five broad orthogonal factors, the **Big Five** or **five-factor** model, remembered by the acronym **OCEAN** (Kaplan & Sadock 2024; Tasman Psychiatry).

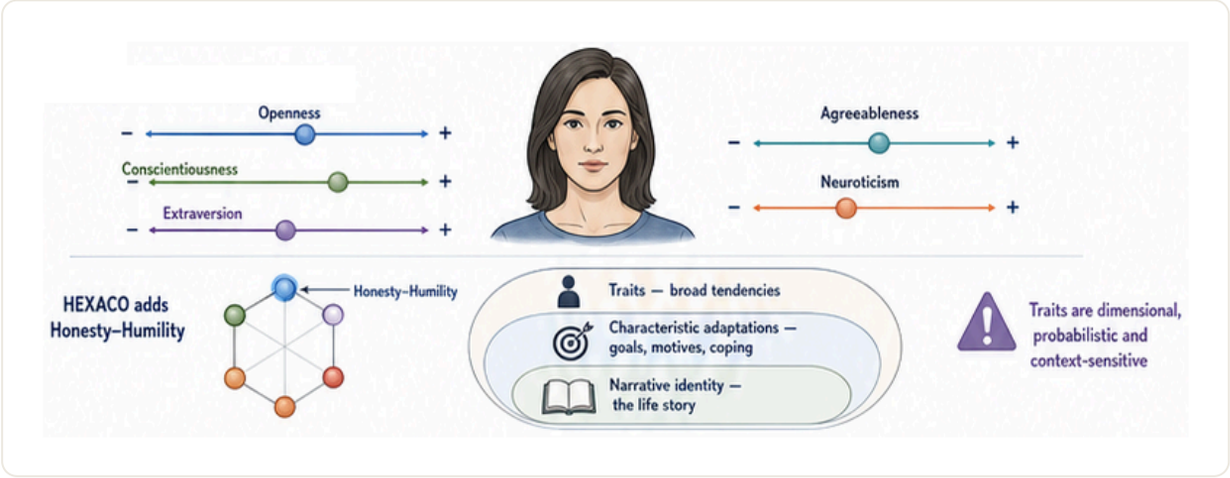


Fig 5.24 Dimensional trait structure.

FACTOR (OCEAN)	HIGH POLE	LOW POLE / CONTRAST
O: Openness to experience	Imaginative, curious, unconventional, aesthetically sensitive, open to ideas and feelings	**Closedness: practical, conventional, prefers routine and the familiar**
C: Conscientiousness	Organised, dependable, self-disciplined, goal-directed, deliberate	**Impulsivity: careless, disorderly, spontaneous, undisciplined**
E: Extraversion	Sociable, assertive, energetic, positive-emotion-prone, stimulation-seeking	**Introversion and detachment: reserved, quiet, solitary**
A: Agreeableness	Trusting, cooperative, warm, altruistic, compliant	**Antagonism: suspicious, competitive, callous, oppositional**
N: Neuroticism	Anxious, moody, emotionally reactive, prone to negative affect	**Emotional stability: calm, even-tempered, resilient**

The discriminator that makes the five-factor model examinable: each factor is a bipolar continuum, not a type, and the five are broadly independent, so a person is a *profile* of five scores. Costa and McCrae's NEO-PI further divides each domain into six narrower facets (thirty facets in all).

HOLD THIS

The Big Five (OCEAN) is the currently accepted descriptive consensus, measured by the NEO-PI. Eysenck (three, PEN) and Cattell (sixteen, 16PF) converge on it from opposite methods; neuroticism is the factor most tied to psychopathology.

Costa and McCrae's NEO-PI. The standard instrument for the five-factor model is the **NEO-PI** (originally the NEO Personality Inventory, later NEO-PI-R, and the brief NEO-FFI), developed by Paul Costa and Robert McCrae, which scores the five domains and their thirty facets and carries strong evidence of reliability, cross-cultural replicability and temporal stability across the adult lifespan (Kaplan & Sadock 2024; Companion to Psychiatric Studies; Tasman Psychiatry). Its clinical footprint is direct: the DSM-5 Section III alternative model of personality disorders

reframes pathology as maladaptive variants of five domains (negative affectivity, detachment, antagonism, disinhibition, psychoticism) that map onto the maladaptive poles of the five-factor model, high neuroticism, low extraversion, low agreeableness, low conscientiousness, and high openness respectively (Kaplan & Sadock 2024).

15.6 The humanistic tradition: Rogers and the self

The "third force" reacting against determinism. The **humanistic** psychologists rejected both the psychoanalytic view of the person as driven by unconscious conflict and the behaviourist view of the person as a bundle of conditioned responses, and put in their place a person who is fundamentally growth-oriented, free and striving toward fulfilment. Carl **Rogers** built the most complete humanistic personality theory, his *self theory* (Wolberg, Technique of Psychotherapy; Kaplan & Sadock 2024). Its core constructs:

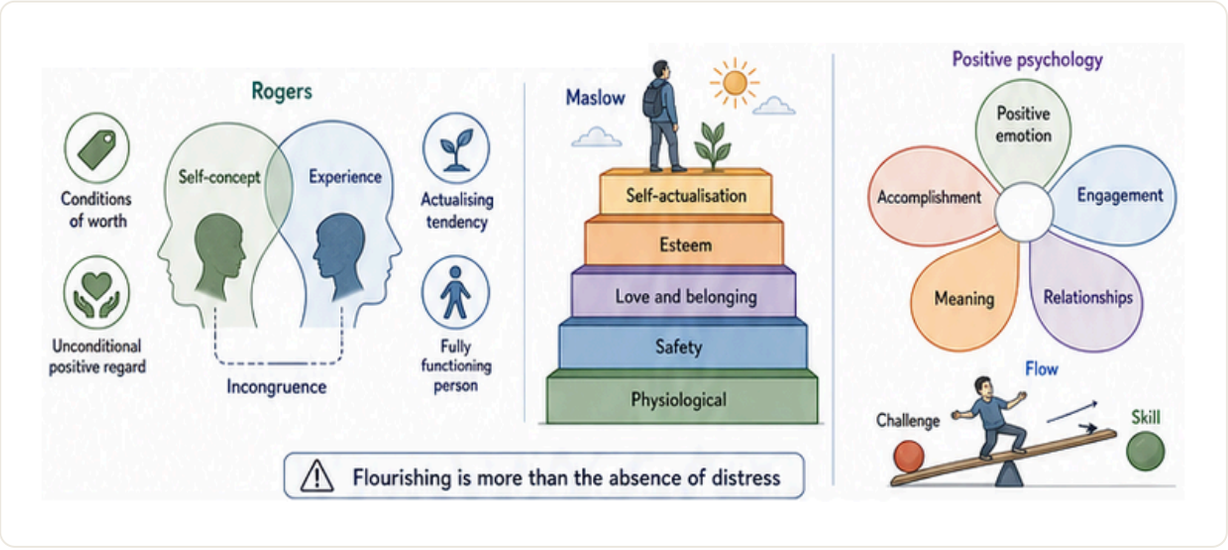


Fig 5.25 Humanistic and positive psychology.

Self-concept

The organised, consistent set of perceptions and beliefs a person holds about themselves; the "real self". Rogers also posits an "ideal self", the person one wishes to be.

Congruence and incongruence

The degree of match between self-concept and actual experience (and between real and ideal self). High congruence underlies psychological health; incongruence, the gap between them, generates anxiety and defensiveness.

Conditions of worth

Internalised standards, learned from others' conditional acceptance, that make self-regard contingent on meeting demands; the developmental source of incongruence.

Unconditional positive regard

Acceptance and valuing of a person without conditions attached. Received in childhood it builds congruence; supplied by the therapist it is the healing condition of client-centred therapy.

Fully functioning person

Rogers' endpoint of healthy development: someone open to experience, living existentially in the present, trusting their own organismic valuing, free and creative.

Driving all of it is a single motivational assumption, the **actualising tendency**, an innate push of every organism toward maintenance, growth and the realisation of its potential. Personality, for Rogers, is what happens to that tendency under the conditions of worth a life imposes (Kaplan & Sadock 2024).

15.7 Maslow and the existential strand

Self-actualisation as a personality endpoint. Abraham **Maslow**, the other pillar of humanism, is examined here for his **hierarchy of needs** (physiological, safety, love and belonging, esteem, and at the summit **self-actualisation**, the realisation of one's full potential) and for his study of self-actualised people as the model of the healthy personality, marked by accurate perception of reality, autonomy, spontaneity, deep relationships, peak experiences and a problem-centred rather than self-centred focus. Where the trait theorists describe the average person, Maslow described the flourishing one, and made growth, not the reduction of tension, the organising motive of personality (Kaplan & Sadock 2024).

The **existential** strand is humanism's darker, European cousin, concerned with freedom, responsibility, meaning and the confrontation with death, isolation and meaninglessness. Rollo **May** brought existential thought into American psychology, framing anxiety as the normal accompaniment of freedom and the awareness of non-being, and distinguishing normal from neurotic anxiety. Viktor **Frankl**, a psychiatrist and Holocaust survivor, founded logotherapy on the "will to meaning", the thesis that the primary human drive is to find meaning, and that meaning can be found even in unavoidable suffering; his *Man's Search for Meaning* is the movement's touchstone. The existentialists fixed a gap in Rogers and Maslow by taking seriously the tragic givens of existence that a purely growth-optimistic humanism underplayed (Kaplan & Sadock 2024).

15.8 Kelly's personal construct theory: the social-cognitive bridge

The person as scientist. George Kelly's **personal construct** theory casts each person as an intuitive scientist who construes the world through a personal repertoire of bipolar dimensions (constructs such as kind-cruel, safe-dangerous) and uses them to anticipate events; behaviour follows from the anticipations these constructs generate, and psychological problems arise when a person's construct system fits the world poorly and resists revision. Because it locates personality in learned cognitive structures interacting with situations rather than in fixed traits or unconscious drives, it forms the bridge from the humanistic tradition into the social-cognitive one (Bandura's reciprocal determinism and self-efficacy, Rotter's locus of control, Mischel's cognitive-affective processing), which is developed under the learning-theory topics (Kaplan & Sadock 2024).

15.9 Currently accepted: the Big Five and the resolution of the person-situation debate

The consensus and the crisis it survived. The **currently accepted** descriptive framework for normal personality is the Big Five / five-factor model: five broad, replicable, cross-culturally recovered dimensions that subsume the earlier trait models and now anchor the dimensional reframing of personality disorder in DSM-5 Section III and ICD-11 (Kaplan & Sadock 2024). It

reached this status only after surviving the **person-situation debate**. In 1968 Walter **Mischel** observed that trait scores predict behaviour in any single situation only modestly (the "personality coefficient" ceiling of roughly 0.30) and argued that situations, not traits, drive behaviour. The debate was resolved into **interactionism**: traits predict behaviour well when it is *aggregated* across many situations (a person high in conscientiousness is reliably more conscientious on average), while situations determine the specific act; person and situation interact, and Mischel's own later cognitive-affective processing system (CAPS) model captured the stable *if-then* behavioural signatures that reconcile cross-situational variability with genuine personality consistency.

15.10 The behavioural-genetic and neural correlate of traits

Where the dimensions live in the biology. Twin and adoption studies converge on a **heritability** of the major personality traits of roughly 40 to 50 per cent (often quoted as the 40 to 60 per cent band across studies and traits), with the remaining variance dominated by the *nonshared* environment and shared-family environment contributing little; large genome-wide association studies have so far failed to pin the heritability to reliable common variants, the classic missing-heritability picture (Tasman Psychiatry; Kaplan & Sadock 2024). For the **neural correlate**, two frameworks are examinable. Jeffrey **Gray**'s reinforcement-sensitivity theory reinterprets Eysenck's axes as two motivational systems: a behavioural inhibition system (BIS, septo-hippocampal and amygdalar, sensitive to punishment and novelty, underlying anxiety and mapping toward high neuroticism/introversion) and a behavioural activation system (BAS, mesolimbic dopaminergic, sensitive to reward, underlying impulsivity and mapping toward extraversion). Colin **DeYoung**'s cybernetic/personality-neuroscience programme links each Big Five domain to systems: extraversion to dopaminergic reward circuitry, neuroticism to the amygdala and threat/defensive systems, conscientiousness to lateral prefrontal cortex, openness to dopaminergic cognitive-exploration circuits, and agreeableness to social-cognition and theory-of-mind networks. The through-line is that traits are moderately heritable dimensions with identifiable neurotransmitter and circuit substrates, exactly the biological grounding Eysenck first demanded (Kaplan & Sadock 2024).

40-50%	~0.30	5
HERITABILITY OF MAJOR TRAITS	MISCHEL'S PERSONALITY COEFFICIENT CEILING	FACTORS (OCEAN), 30 FACETS

CLINICAL ANCHOR

A worked example that ties the traditions together. A reserved, meticulous, easily-worried patient can be read at every level: a *type* theorist would once have called him a melancholic or an ectomorph; the *trait* reading is low extraversion, high conscientiousness, high neuroticism on the NEO-PI; Eysenck would flag high N and high introvert arousal; Gray would locate an overactive behavioural inhibition system; the *humanistic* reading asks what conditions of worth made his self-worth contingent on flawless performance and where his self-concept is incongruent with his experience; Kelly would map the rigid bipolar constructs (perfect-worthless) through which he anticipates evaluation. None is wrong; each tradition answers a different clinical question, and a good formulation uses more than one.

15.11 Positive psychology (delta): Seligman, PERMA, strengths and flow

The science of what goes right, routed here from the General and Developmental Psychology notes. **Positive psychology**, launched as a formal programme by Martin **Seligman** during his 1998 American Psychological Association presidency, studies positive emotions, positive traits and enabling institutions, the conditions under which people and communities flourish, as a deliberate counterweight to psychology's traditional focus on disorder. It sits naturally beside the humanistic tradition (both centre growth and the healthy person) but insists on empirical measurement (Kaplan & Sadock 2024).

PERMA

Seligman's five-element model of well-being: **P**ositive emotion, **E**ngagement, **R**elationships (positive), **M**eaning, and **A**ccomplishment. Well-being is the flourishing that comes of building all five, not merely the absence of distress.

Character strengths and virtues

Peterson and Seligman's classification (the deliberate "un-DSM") of 24 measurable character strengths grouped under six universal virtues (wisdom, courage, humanity, justice, temperance, transcendence), assessed by the VIA Inventory of Strengths.

Flow

Mihaly **Csikszentmihalyi**'s state of complete absorption in an intrinsically rewarding activity, in which challenge and skill are well matched and self-consciousness and the sense of time drop away; the experiential core of engagement.

Authentic happiness / well-being theory

Seligman's earlier "authentic happiness" scheme (pleasant, engaged and meaningful lives) which he later broadened into the multi-element well-being theory captured by PERMA.

COMMON TRAP

Do not confuse the neighbouring frameworks. Type theories are *categories* (you are a type); trait theories are *dimensions* (you have a score on a continuum), and the whole point of the trait tradition was to replace the former with the latter. Eysenck (three factors, PEN) is not Cattell (sixteen factors, 16PF), and neither is the Big Five (five factors, NEO-PI), though all three are trait/nomothetic models. Maslow's self-actualisation is the *top of a need hierarchy*; Rogers' fully functioning person is the *outcome of unconditional positive regard and congruence*, the two are humanistic cousins but not the same construct. And positive psychology (Seligman, an empirical programme with instruments) is not the same as humanism (Rogers/Maslow, a mid-century philosophical movement), even though they share the growth agenda.

INDIA

The five-factor structure replicates in Indian samples but is not culturally exhaustive: indigenous work has argued for additional or reweighted dimensions, and constructs such as the trigunas of Samkhya (sattva, rajas, tamas) have been operationalised into scales as an emic model of personality. For the exam, know that the Big Five is the accepted *universal* descriptive framework while accepting that lexical studies in different languages recover locally salient factors on top of it.

MNEMONIC

OCEAN and PERMA

The Big Five is **OCEAN**: **O**penness, **C**onscientiousness, **E**xtraversion, **A**greeableness, **N**euroticism. Positive-psychology well-being is **PERMA**: **P**ositive emotion, **E**ngagement, **R**elationships, **M**eaning, **A**ccomplishment. For the traditions in order, remember "Boxes, then Dimensions, then Selves, then Scientists": type theories (boxes), trait theories (dimensions), humanism (the self), social-cognitive/constructs (the person as scientist).

GOOD TO REMEMBER

- **Personality:** enduring characteristic pattern of thought, feeling and behaviour, consistent across time and situations.
- **Four grand traditions:** psychodynamic (unconscious conflict), trait (measurable dimensions), humanistic (the striving whole self), social-cognitive (learned expectancies and constructs); type theories are the ancient precursor.
- **Type theories:** Hippocrates/Galen four humours and temperaments (sanguine, melancholic, choleric, phlegmatic); Kretschmer (asthenic-schizoid, pyknic-cyclothymic, athletic); Sheldon somatotypes (endomorph-viscerotonic, mesomorph-somatotonic, ectomorph-cerebrotonic).
- **Allport:** cardinal, central and secondary traits; the idiographic (unique individual) versus nomothetic (universal dimensions) distinction; founder of trait psychology.
- **Eysenck:** biologically grounded PEN model, extraversion-introversion (introverts higher cortical arousal), neuroticism (limbic reactivity), psychoticism; three superfactors.
- **Cattell:** factor-analytic model, surface traits versus source traits, sixteen source factors measured by the 16PF.
- **Big Five / five-factor model (OCEAN):** Openness, Conscientiousness, Extraversion, Agreeableness, Neuroticism; the currently accepted descriptive consensus, measured by Costa and McCrae's NEO-PI (five domains, thirty facets).
- **Rogers (self theory):** self-concept, real versus ideal self, congruence-incongruence, conditions of worth, unconditional positive regard, the fully functioning person, actualising tendency.
- **Maslow:** hierarchy of needs topped by self-actualisation as the healthy-personality endpoint.
- **Existential strand:** May (anxiety as accompaniment of freedom and non-being) and Frankl (logotherapy, the will to meaning).
- **Kelly:** personal construct theory, the person as intuitive scientist; the bridge into social cognition.
- **Person-situation debate:** Mischel (1968) challenged trait predictiveness (the ~0.30 personality coefficient); resolved into interactionism (traits predict aggregated behaviour, situations predict the specific act).
- **Heritability of traits:** roughly 40 to 60 per cent, remainder mostly nonshared environment; GWAS missing-heritability picture.
- **Neuroscience of traits:** Gray's BIS/BAS (septo-hippocampal/amygdalar punishment system; mesolimbic-dopaminergic reward system); DeYoung's personality neuroscience (extraversion-dopamine reward, neuroticism-amygdala threat, conscientiousness-prefrontal, openness-dopaminergic exploration, agreeableness-social cognition).
- **Positive psychology (Seligman, 1998):** PERMA (positive emotion, engagement, relationships, meaning, accomplishment); character strengths and virtues (24 strengths, 6 virtues, VIA inventory); flow (Csikszentmihalyi); well-being theory.

Special topics

16 · Psychology of rape

The **psychology of rape** is a named Paper-1 essay topic, and it is examined as a piece of applied social and forensic psychology rather than as a description of an offence. It sits on these notes because it draws together attribution, aggression, attitudes and the psychology of the survivor. The register throughout is clinical and sober: this is doctor-facing revision, the material concerns both the person who offends and the person who is harmed, and it is written without case detail or sensationalism.

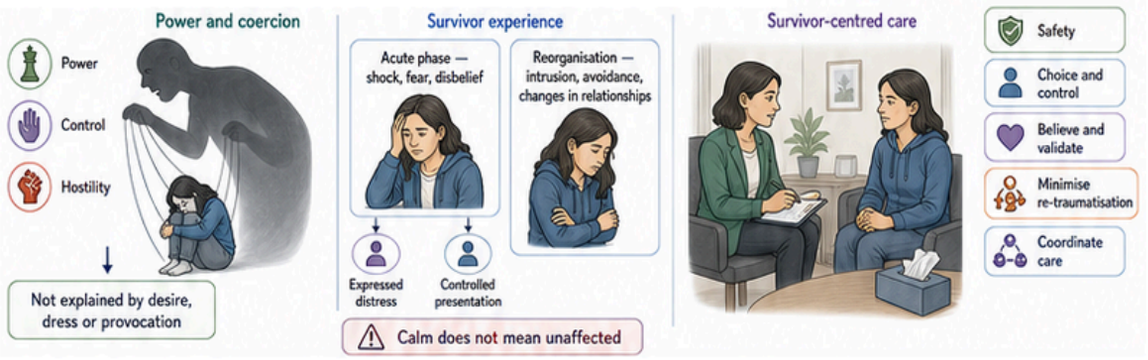


Fig 5.26 Sexual violence: power, trauma and survivor-centred care.

Two bodies of theory are examined. The first is the psychology of the offender, where the central shift over the last half century has been away from the idea that **rape** is driven by sexual desire and toward understanding it as an act of power and aggression expressed through sex. The second is the psychology of the survivor of **sexual assault**, where the key construct is the patterned psychiatric response first described as the rape trauma syndrome. The clinician also needs the framework for a trauma-informed response and an outline of the Indian medico-legal setting.

16.1 The reframing: power and aggression, not lust

The foundational idea, from Nicholas Groth's clinical work with offenders (Men Who Rape, 1979), is that rape is the sexual expression of power and anger, not primarily the aggressive expression of sexuality. Sexuality is the medium; power, control and hostility are the motives. This reframing matters clinically and forensically because it predicts that rape is poorly explained by sexual deprivation or provocation, and it displaces the rape myths that treat the act as a misunderstanding about sex. It also aligns the offence with the general aggression literature of these notes rather than with the paraphilias, although a minority of offenders do have a paraphilic (sexual sadism) or antisocial profile.

RULE **Rape is the sexual expression of power and anger, not primarily lust (Groth).** Sexuality is the medium; power, control and hostility are the motives, which is why the act is poorly explained by sexual deprivation or provocation.

16.2 Offender typologies

Groth (1979) himself described *three* patterns by the motive that predominates: **anger rape**, **power rape** and **sadistic rape**. The four-type scheme below is the later investigative elaboration associated with Hazelwood and the FBI, which splits Groth's power category into reassurance and assertive subtypes (a split Knight's typology also uses) and his anger category into retaliatory and excitation subtypes. Cite the three when asked for Groth and the four when asked for the investigative typology. Either way it is a clinical heuristic, not a diagnosis, and most real offenders are heterogeneous, so the caveats matter as much as the categories.

TYPE	PREDOMINANT MOTIVE	CHARACTER
Power-reassurance	**Restoring a fragile sense of masculine adequacy**	The least overtly aggressive; scripted, seeks reassurance; the commonest type
Power-assertive	**Asserting dominance and entitlement**	Uses force to control; sees the act as a right, not a fantasy
Anger-retaliatory	**Punishing and degrading, revenge on women**	Impulsive, high gratuitous violence, brief and brutal
Anger-excitation (sadistic)	**Eroticised suffering**	The rarest and most dangerous; planned, ritualised; overlaps sexual sadism and psychopathy

The four investigative offender types (Hazelwood and the FBI, elaborating Groth's three) by predominant motive; power types outnumber anger types, and the sadistic type is rare but carries the greatest risk.

16.3 Risk factors and the limits of prediction

The best-supported model of the propensity to sexual aggression is Malamuth's confluence model, which locates risk at the meeting of two paths: hostile masculinity (adversarial, controlling attitudes toward women, hostility, and a need to dominate) and an impersonal, non-intimate orientation to sex. Attitudinal factors (acceptance of rape myths, hostility) and situational disinhibitors (alcohol, peer norms) raise risk. The clinically honest point for the exam is that prediction at the level of the individual is weak: these are group-level associations, and no profile identifies an offender with useful accuracy.

TRAP Groth's typologies and the confluence-model risk factors are **group-level associations, not an individual-prediction tool**; no profile identifies an offender with useful accuracy.

16.4 Rape myths and their harm

Rape myths are false but widely held beliefs about rape, offenders and survivors that serve to deny, minimise or justify sexual aggression: that survivors provoke it by dress or conduct, that only strangers commit it, that a real rape leaves visible injury, that a delayed or calm disclosure means it did not happen. Rape myth acceptance is measurable and predicts victim-blaming attitudes. Its harm is concrete: it suppresses disclosure, colours the medical and legal response, and inflicts the second injury of not being believed. Recognising these myths is part of a competent clinical stance.

16.5 The survivor: the rape trauma syndrome

Ann Burgess and Lynda Holmstrom described the rape trauma syndrome in 1974 as the patterned psychological response to sexual assault, in two phases. The acute or disorganisation phase (days to weeks) brings shock, fear, disbelief, and either an expressed style (overt distress) or a controlled style (a composed, flat presentation that can be misread as unaffected). The reorganisation phase (months and longer) brings the intrusive and avoidant symptoms, phobias, sexual difficulties, and changes to lifestyle and relationships. In modern terms this maps onto acute stress and post-traumatic stress disorder, with high rates of comorbid depression, sexual dysfunction and substance use; suicidality must be assessed.

HOLD THIS

The rape trauma syndrome (Burgess and Holmstrom 1974) runs in two phases: acute or disorganisation, then reorganisation. The acute phase can present as an expressed (overt distress) or a controlled (composed, flat) style, and a calm presentation must not be misread as unaffected.

16.6 Secondary victimisation and a trauma-informed response

Secondary victimisation, the second injury, is the further harm done by the responses of services, family and the legal process: disbelief, blame, intrusive questioning, and loss of control over the process. The clinical antidote is a trauma-informed response whose principles are safety first, restoring choice and control to the survivor, believing and validating, minimising re-traumatisation during examination, and coordinating the medical, psychological and forensic tasks so the survivor is not made to repeat the account needlessly.

16.7 The Indian medico-legal frame

INDIA

In India the clinical care of a survivor of sexual assault is governed by consent, the guidance that treatment is not conditional on reporting to police, and the post-2013 reforms that discredited and moved away from the so called two-finger test as unscientific and degrading. Offences against minors are dealt with under the Protection of Children from Sexual Offences (POCSO) Act. The statutory detail belongs to the the forensic psychiatry chapter and is cross-referenced there; what belongs here is the clinical stance: examine with consent, document carefully, treat injuries and prophylaxis, and provide trauma-informed psychological care.

GOOD TO REMEMBER

- **Reframing (Groth 1979):** rape is power and anger expressed through sex, not lust.
- **Offender types:** Groth's own three are anger, power and sadistic rape; the investigative four (Hazelwood/FBI) are power-reassurance (commonest), power-assertive, anger-retaliatory and anger-excitation or sadistic (rarest, most dangerous).
- **Risk (Malamuth confluence model):** hostile masculinity plus impersonal sex; individual prediction is weak.
- **Rape myths:** false beliefs that deny or justify assault; raise victim-blaming, suppress disclosure.
- **Rape trauma syndrome (Burgess and Holmstrom 1974):** acute or disorganisation phase (expressed or controlled style) then reorganisation phase; maps onto ASD and PTSD with depression and sexual dysfunction.
- **Second injury:** secondary victimisation by services and the process; countered by a trauma-informed response.
- **India:** consent-based care, care not conditional on reporting, the two-finger test abandoned (2013), POCSO for minors.

17 · The Indian therapy relationship: guru-chela and expressed emotion

Two constructs from the Paper-1 syllabus carry a distinct Indian weight, and both concern the relationships around the patient rather than the patient alone. The first is the **guru-chela** relationship, an Indian model of the therapeutic bond. The second is **expressed emotion**, the

measurable emotional atmosphere of the family, whose Indian findings are part of why outcomes for serious mental illness have been better in the developing world.

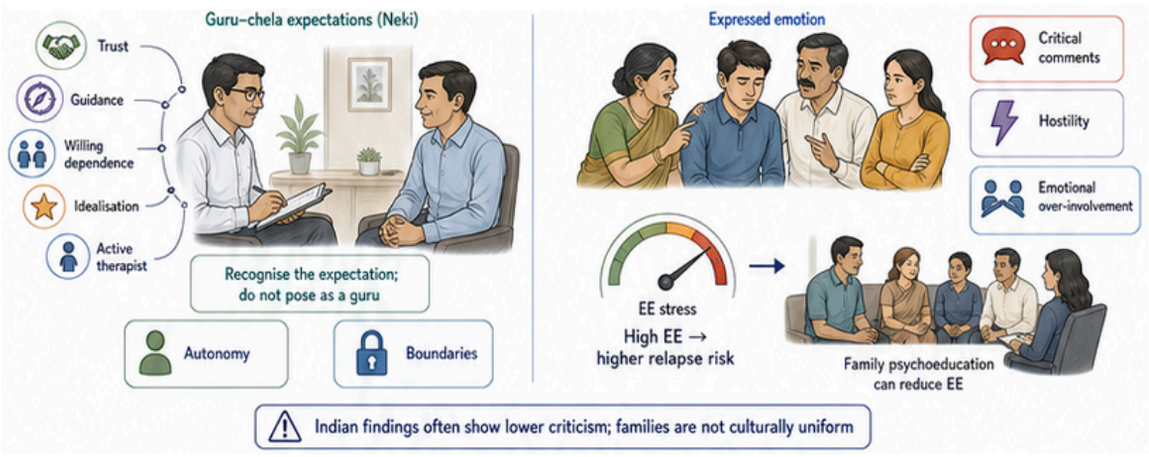


Fig 5.27 Indian therapy relationship and expressed emotion.

17.1 The guru-chela relationship (Neki)

J. S. Neki, in a classic pair of papers (Guru-Chela relationship: the possibility of a therapeutic paradigm, American Journal of Orthopsychiatry, 1973), argued that the Western contractual, autonomy-valuing therapeutic alliance is a poor cultural fit for many Indian patients, and that the indigenous guru-shishya (guru-chela) ideal offers a better paradigm. In that ideal the disciple approaches the teacher with trust, reverence and a willing dependence; the guru accepts responsibility for the disciple's growth, offers guidance and a model to identify with, and the bond is warm, hierarchical and personal rather than contractual and time-limited.

17.2 Why it matters for practice

The guru-chela frame reframes several things the Western model treats as problems. Dependence, which the autonomy-valuing model tends to pathologise, is here a legitimate and even therapeutic stage; idealisation of the therapist is expected rather than interpreted away at once; and the therapist is permitted to be more active, directive and personally invested. Neki's point is not that the therapist should pose as a guru, but that Indian patients often enter treatment with guru-chela expectations, and that understanding this improves engagement and the handling of transference and dependence. Its risks are the risks of any hierarchical bond: fostering excessive dependence, and blurring boundaries if the therapist accepts the idealisation uncritically.

RULE **In the guru-chela paradigm (Neki), dependence and idealisation are legitimate therapeutic stages, not pathology to interpret away.** The point is not to pose as a guru but to recognise the expectations Indian patients often bring.

17.3 Expressed emotion: the construct

Expressed emotion is a measure of the emotional climate a relative shows toward a patient, developed by George Brown and Michael Rutter and assessed through the Camberwell Family

Interview. It has three key components: critical comments (the count of critical remarks), hostility (generalised rejection of the person, not just their behaviour), and emotional over-involvement (over-protective, self-sacrificing, dramatic concern). A household is rated high in expressed emotion if a relative exceeds threshold on criticism or hostility, or shows marked over-involvement.

HOLD THIS

Expressed emotion (Brown and Rutter; Camberwell Family Interview) has three components: critical comments, hostility and emotional over-involvement. Indian families tend to show lower expressed emotion, part of the better outcome of schizophrenia in the developing world.

17.4 Expressed emotion and relapse

The robust finding, replicated across many samples, is that patients with schizophrenia who return to a high expressed emotion household relapse at markedly higher rates than those returning to a low expressed emotion household (Vaughn and Leff), and the effect extends to mood disorders and other conditions. The mechanism is understood as a chronic stressor acting on a vulnerable individual, a family-level instance of the diathesis-stress model. The practical payoff is that family psychoeducation which lowers expressed emotion reduces relapse: Leff and colleagues (1982) showed a controlled social intervention in families cut the relapse rate, which is why lowering expressed emotion is a target of family work.

RULE **High expressed emotion predicts relapse; lowering it (through family psychoeducation) reduces relapse.** It acts as a chronic stressor on a vulnerable individual, a family-level instance of diathesis-stress.

17.5 Expressed emotion in India

INDIA

The cross-cultural expressed emotion research, including the WHO Determinants of Outcome study and Indian work by Wig, Leff and colleagues in Chandigarh, found that Indian families tend to show lower expressed emotion than Western families, with fewer critical comments and less over-involvement. This lower family criticism is one of the cultural factors invoked to explain the better course and outcome of schizophrenia reported in the developing world (cross-ref the cross-cultural outcome material). It is a clean example of a social variable, the family's emotional tone, shaping the course of a biological illness.

GOOD TO REMEMBER

- **Guru-chela (Neki 1973):** an Indian therapeutic paradigm of trust, reverence and willing dependence on the therapist-as-guru, against the Western contractual, autonomy-valuing alliance.
- **Clinical use:** dependence and idealisation are legitimate stages, the therapist is more active; the risk is over-dependence and boundary blurring.
- **Expressed emotion (Brown and Rutter; Camberwell Family Interview):** critical comments, hostility, emotional over-involvement.
- **High expressed emotion predicts relapse** in schizophrenia (Vaughn and Leff) and mood disorders; a family-level diathesis-stress effect.
- **Family psychoeducation** that lowers expressed emotion reduces relapse (Leff et al 1982).
- **India:** Indian families tend to show lower expressed emotion (Wig, Leff), part of the better outcome of schizophrenia in the developing world.

Discriminate

18 · Discriminate: the paradigms side by side

The examinable skill of these notes is discrimination: not reciting one theory, but telling it apart from its neighbours. Three comparisons carry most of the marks. The first tells the learning paradigms apart, the second tells the personality models apart, and the third tells the social-thinking biases apart. Read each table down the discriminator column, the shaded cell, because that is where the distinguishing feature sits.

18.1 The learning paradigms: classical, operant and social learning

The three paradigms are often confused because all three are about learning, but they differ in what is associated, what the learner does, and the therapy each produces. The move across them is also historical, from a passive organism forming stimulus associations to an active, cognitive one learning by watching.

FEATURE	CLASSICAL (PAVLOV)	OPERANT (SKINNER)	SOCIAL LEARNING (BANDURA)
What is learned	**A stimulus predicts a stimulus (CS predicts US)**	**A behaviour predicts a consequence**	**A behaviour and its outcome, by watching a model**
Key relationship	CS paired with US, before the response	Reinforcement or punishment, after the response	Vicarious reinforcement of the model
The learner	Passive, reflexive (involuntary responses)	Active, emitting behaviour (voluntary responses)	Cognitive: attends, retains, expects
Signature study	Salivating dogs; Little Albert	The Skinner box; schedules of reinforcement	The Bobo doll
Therapy it yields	Systematic desensitisation, exposure, aversion	Token economy, contingency management, ABA	Modelling, skills training, self-efficacy work

Classical learns what predicts what before a reflex; operant learns what a voluntary action earns; social learning learns by watching a model, with cognition in the middle.

HOLD THIS

Classical is a stimulus predicting a stimulus before an involuntary reflex; operant is a voluntary behaviour shaped by its consequence; social learning is behaviour acquired by watching a model. The learner moves from passive to active to cognitive across the three.

18.2 The personality models: five traditions

The five traditions answer the same questions differently: what the basic unit of personality is, whether the person is determined or free, how personality is measured, and what the main criticism is. Type theories sort people into kinds; trait theories place them on continuous dimensions; the psychodynamic explains the unconscious roots; the humanistic centres the self and growth; the social-cognitive puts behaviour at the meeting of person and situation.

TRADITION	UNIT OF ANALYSIS	ASSESSED BY	MAIN CRITICISM
Psychodynamic	**Unconscious conflict and drives**	Projective tests, free association	Hard to test, over-interprets
Trait	**Stable dimensions (the Big Five)**	Questionnaires (NEO-PI, 16PF, EPQ)	Describes rather than explains
Type	**Discrete categories or kinds**	Typologies, body build	Loses individual variation
Humanistic	**The self, growth, meaning**	Self-report, Q-sort	Vague, optimistic, hard to measure
Social-cognitive	**Person interacting with situation**	Behavioural measures, self-efficacy scales	Underweights emotion and biology

The five personality traditions compared on their unit of analysis, their assessment method and their main criticism; the trait tradition, now the Big Five, is the descriptive consensus.

18.3 The social-thinking biases

Four social-cognitive phenomena from these notes are easily confused because each describes a distortion in how people think about themselves and others. The one-line signature tells them apart.

BIAS	SIGNATURE
Fundamental attribution error	**Over-attributing others' behaviour to disposition, underrating the situation**
Self-serving bias	**Taking credit for success (internal), blaming the situation for failure (external)**
Cognitive dissonance	**Changing an attitude to resolve the tension of inconsistent cognitions**
Group polarisation	**A group's average view becoming more extreme after discussion**

Four social-thinking distortions distinguished by their signatures; the first two are attribution biases, the third an attitude-change process, the fourth a group effect.

COMMON TRAP

The high-yield mix-ups: classical conditioning is before the reflex and involuntary, operant is after a voluntary act; negative reinforcement strengthens behaviour (it removes something aversive) and must not be called punishment, which weakens it; Eysenck's model is a few broad biologically grounded dimensions (PEN) while Cattell's is sixteen factors (16PF); and in object relations, Klein is the paranoid-schizoid and depressive positions while Kohut is self psychology and the selfobject.

Apply and consolidate

19 · Apply: four psychology vignettes

Four short worked vignettes turn this theory into the reasoning the exam actually rewards. Each gives a brief presentation, then the psychological reasoning, then the read. The tool changes each time, so that the discriminator carries the answer.

RULE **In an applied vignette, name the process, not the personality.** Identify the domain and its mechanism (a bias, a conditioning contingency, a Big Five profile, a group effect), and the discriminator carries the mark.

19.1 An attribution read

CLINICAL ANCHOR

Case: a vignette in attribution. A final-year student fails an examination and tells you the paper was unfair and the examiners had it in for him, while explaining that his distinctions in earlier years were down to his own ability. Reasoning: the failure is attributed to external, unstable, uncontrollable causes and the successes to internal, stable ones. This asymmetry, taking credit for success and externalising failure, is the self-serving bias. His readiness to see the examiners' intent rather than the situation is the fundamental attribution error turned on others. The read: a self-serving attributional style, which protects self-esteem but, if rigid, blocks the learning that a more balanced, effort-based attribution would allow.

19.2 A learning and behaviour-therapy plan

CLINICAL ANCHOR

Case: a vignette in conditioning. A woman develops an intense fear of lifts after being trapped in one during a power cut; she now avoids lifts entirely and her world is shrinking. Reasoning: the trapping (an aversive unconditioned stimulus) was paired with the lift (now a conditioned stimulus), so the lift alone evokes a conditioned fear response, classical conditioning; the avoidance is then maintained by operant negative reinforcement, because escaping the lift removes the anxiety and so is reinforced. The read: a specific phobia by conditioning. The matching treatment follows the paradigm: graded exposure or systematic desensitisation to extinguish the conditioned fear, and blocking the avoidance so the negative reinforcement cycle cannot maintain it.

19.3 A personality-model read

CLINICAL ANCHOR

Case: a vignette in personality. A colleague is described as anxious and easily stressed, meticulous and dutiful, reserved in company, and quick to imagine worst cases. Reasoning on the Big Five: high neuroticism, high conscientiousness, low extraversion, with average openness and agreeableness. A trait clinician stops there and predicts behaviour from the profile. A psychodynamic clinician asks what unconscious conflict the meticulousness defends against (obsessional defences against anxiety). A humanistic clinician asks whether the person is living in line with their own values or an introjected standard, and where the conditions of worth lie. The read: the same person, formulated three ways, showing how the tradition you choose determines the questions you ask.

19.4 A group-influence read

CLINICAL ANCHOR

Case: a vignette in social influence. A junior doctor on a ward round privately doubts a senior's plan but says nothing, and later, in an unanimous team, finds the group settling on a riskier plan than any member would have chosen alone. Reasoning: the silence in the face of an authority is obedience and normative conformity; the unanimous team suppressing dissent and drifting to a more extreme, poorly examined decision is groupthink and group polarisation. The read: name the processes rather than the personalities, and note the antidotes, inviting dissent, a devil's advocate, and anonymous input, that break the pressure toward uniformity.

HOLD THIS

The same case reads differently in each tradition, and the exam rewards saying which lens you are using. A trait reading, a psychodynamic reading and a humanistic reading of one person ask different questions; none is wrong.

20 · Consolidate: mnemonic, retrieval and synthesis

This chapter closes with three consolidation devices: a master mnemonic that chains the whole of it, a set of answer-free retrieval prompts to test yourself against, and a synthesis map to redraw from memory. Use them actively; reading them is not the same as recalling them.

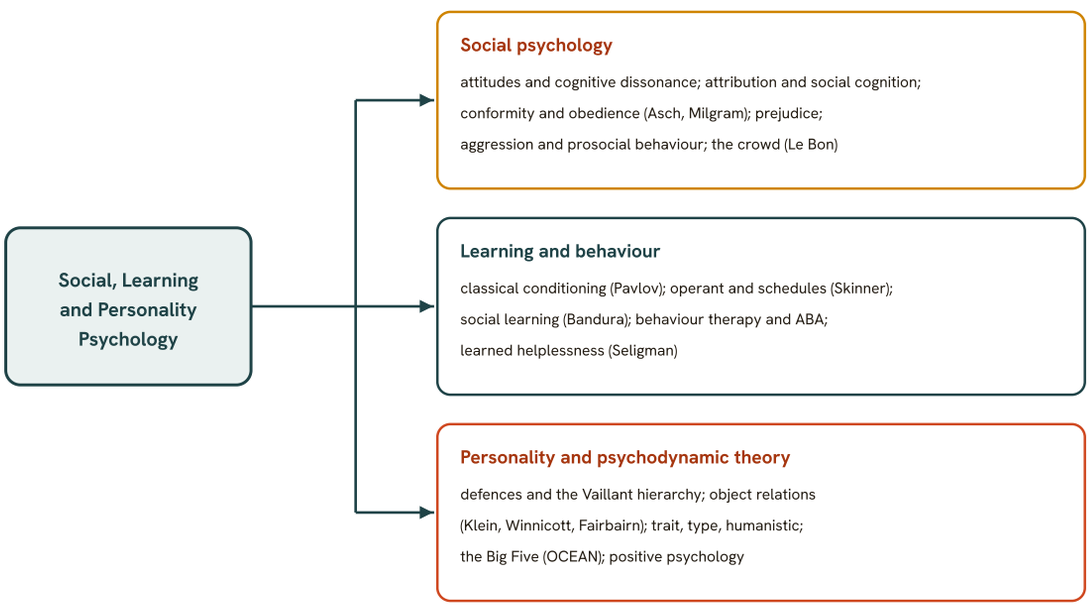


Fig 5.28 The whole synthesis map. Redraw it from memory as a recall check: the three domains branch from the centre, and under each you should be able to reconstruct its topics and their key figures. If a branch comes back thin, that is the section to revisit. Social psychology is how people are moved by others; learning and behaviour is how conduct is shaped and reshaped; personality is how the whole person is theorised.

20.1 The master mnemonic

MNEMONIC

Walk three lands: SOCIAL, LEARNING, PERSONALITY

This chapter is three lands, S, L, P (Social, Learning, Personality). In the SOCIAL land, six phenomena in the line **"Any Argument Gets People Awfully Cross"**: **A**ttitudes (and cognitive dissonance), **A**ttribution (and its biases), **G**roup dynamics (conformity, obedience), **P**rejudice, **A**ggression (and prosocial behaviour), **C**rowd. In the LEARNING land, **"Clever Owls Study Behaviour"**: **C**lassical conditioning, **O**perant conditioning (and schedules), **S**ocial learning (Bandura), **B**ehaviour therapy (with ABA and learned helplessness). In the PERSONALITY land, **"Defend Our Persons"**: **D**efences (the Vaillant hierarchy), **O**bject relations (and the neo-Freudians), **P**ersonality theories (trait, type, humanistic, positive). Two Paper-1 extras hang off the map: the named schools Meyer and Rank, and the two clinical relationships, the psychology of rape and the Indian pair guru-chela and expressed emotion.

HOLD THIS

The whole chapter is three lands: Social, Learning, Personality (SLP). Social psychology is how people are moved by others, learning is how conduct is shaped and reshaped, personality is how the whole person is theorised.

20.2 Retrieval practice

RETRIEVAL PRACTICE, COVER THE ANSWERS

Use active recall: cover the answers you know from this material and try to produce each of these from memory before checking back. This is retrieval practice, and it is worth more than rereading.

1. State Festinger's cognitive dissonance theory and the counter-intuitive result of the induced-compliance study.
2. Name Kelley's three covariation criteria and two attribution biases.
3. Distinguish normative from informational influence, and name the Asch, Milgram and Zimbardo studies.
4. Give Allport's four conditions for the contact hypothesis to reduce prejudice.
5. Contrast the frustration-aggression hypothesis with the social-learning account of aggression.
6. Put the four crowd theories in order from Le Bon to the social identity model.
7. Define the unconditioned and conditioned stimulus and response, and describe extinction and spontaneous recovery.
8. Draw the reinforcement-versus-punishment matrix and name the four schedules with their response patterns.
9. List Bandura's four subprocesses of observational learning and the four sources of self-efficacy.
10. Match three behaviour-therapy techniques to their parent learning paradigm, and state the learned-helplessness reformulation.
11. Name the four levels of Vaillant's defence hierarchy with one mechanism each.
12. Distinguish Klein, Winnicott and Fairbairn in one line each.
13. Give Eysenck's PEN dimensions, Cattell's model and the five factors of OCEAN.
14. State what positive psychology added, and what PERMA stands for.
15. Explain the guru-chela paradigm and the three components of expressed emotion.

20.3 The synthesis map

Finally, redraw the whole synthesis map from memory. Start from the title at the centre, branch to the three domains, and under each branch reconstruct its topics and their key figures: the social phenomena and their theorists, the learning paradigms and the therapies they yield, and the personality strands ending in the Big Five. If a branch comes back thin when you redraw it, that is the section to revisit. The map is not decoration; rebuilding it from memory is the last and best retrieval check for this material.

Previously asked

Real long-essay (25-mark) and short-note (10-mark) questions from the social, learning and personality-psychology territory, most-recent year shown.

LONG ESSAYS (25 MARKS)

Q Discuss the theories of learning and the various types of learning, classical conditioning, and its applied aspect. MUHS 2017

- Discuss defence mechanisms. Discuss their relevance in mental illness. MUHS 2015
- Describe the role of genetic, biological and psychological factors in the development of personality. MUHS 2018
- Describe the basic principles and clinical application of behaviour therapy. MUHS 2012
- Describe the clinical assessment of personality. MUHS 2013
- Discuss the risk factors for aggression. Describe the investigation and management of an aggressive patient. MUHS 2015

SHORT NOTES (10 MARKS)

- Attitudes. MUHS 2017
- Operant conditioning. MUHS 2017
- Humanistic theories in personality. MUHS 2018
- Etiology of aggression. MUHS 2018
- Psychology of aggression and its clinical implications. MUHS 2019

This territory is examined reliably: learning and conditioning with its applied aspect (behaviour therapy), defence mechanisms, the theories of personality and the clinical assessment of personality, and the psychology of aggression recur as both essays and short notes. Prepare each so you can give the core theory, the named figures and one clinical link in a few lines. The acute evaluation and management of the aggressive or violent patient is developed on the the emergency psychiatry chapter; personality and intelligence testing (MMPI, Rorschach, the Wechsler scales) is taken up on the Psychometry, Assessment and Descriptive Psychopathology notes; and Freud's models, Adler and Jung are covered in full on the General and Developmental Psychology notes. The psychology of rape, the guru-chela therapeutic relationship and expressed emotion are named in the Paper 1 syllabus and are covered here.

Quick review

The whole picture in one table	
THE THREE DOMAINS	After the General and Developmental Psychology notes's general functions and developmental theorists, this material covers the social person (moved by others), the learned person (behaviour shaped by consequence and models) and the theorised person (personality). Freud's models, Adler and Jung are on the General and Developmental Psychology notes; the psychological tests are on the Psychometry, Assessment and Descriptive Psychopathology notes.
ATTITUDES AND DISSONANCE	Attitude as affect, behaviour and cognition; the attitude-behaviour gap. Change: the Yale persuasion approach, Festinger's cognitive dissonance (inconsistent cognitions drive change; induced compliance, effort justification), Bem's self-perception, and the elaboration likelihood model (central versus peripheral route). Currently accepted: dual-process persuasion.
ATTRIBUTION AND SOCIAL COGNITION	Heider's naive psychology, Jones-Davis correspondent inference, Kelley's covariation (consensus, distinctiveness, consistency), Weiner's dimensions (locus, stability, controllability). The biases: fundamental attribution error, actor-observer asymmetry, self-serving bias, just-world, false consensus. Culture shifts the default (individualist versus collectivist).
GROUP DYNAMICS	Sherif norm formation; Asch conformity (normative versus informational); Milgram obedience (agentic state, situational factors); Zimbardo roles (with the modern critique). Group decisions: polarisation, groupthink (Janis), social facilitation (Zajonc), social loafing, minority influence (Moscovici).
PREJUDICE	Stereotype (cognition), prejudice (affect), discrimination (behaviour). Allport; the authoritarian personality; realistic conflict (Robbers Cave); social identity theory (Tajfel, minimal groups, in-group favouritism). Reduction: the contact hypothesis and its conditions (equal status, common goals, cooperation, institutional support).
AGGRESSION AND PROSOCIAL	Aggression: instinct and ethology, the frustration-aggression hypothesis (Dollard-Miller, Berkowitz), social learning (Bandura, Bobo doll), biological substrates. Prosocial: the bystander effect and diffusion of responsibility (Latane and Darley), empathy-altruism (Batson) versus social exchange and reciprocity (Trivers), kin selection.
CROWD AND MOB	Le Bon (contagion, suggestibility, the group mind) to deindividuation (anonymity, reduced self-awareness) to emergent-norm theory (Turner and Killian) to the social identity model of crowds (Reicher: crowds act on a shared identity, not mindless contagion). Relevance: mass hysteria, panic, riots.

The whole picture in one table	
CLASSICAL CONDITIONING	Pavlov: US, UR, CS, CR; acquisition, extinction, spontaneous recovery, generalisation, discrimination, higher-order. Watson's Little Albert (conditioned fear). Rescorla-Wagner (learning as prediction and surprise; blocking); preparedness and taste aversion (Garcia). Clinical: phobia, conditioned nausea, cue reactivity.
OPERANT CONDITIONING	Thorndike's law of effect to Skinner: the reinforcement-versus-punishment matrix (positive and negative each), primary and secondary reinforcers, Premack; shaping, chaining, extinction burst, stimulus control. The four schedules: fixed and variable, ratio and interval; variable ratio gives the highest, most extinction-resistant rate.
SOCIAL LEARNING	Bandura: observational learning and modelling (attention, retention, reproduction, motivation), vicarious reinforcement, the Bobo doll. Reciprocal determinism (person, behaviour, environment). Self-efficacy and its four sources (mastery, vicarious, persuasion, physiological state). The cognitive turn away from radical behaviourism.
BEHAVIOUR THERAPY, ABA, LEARNED HELPLESSNESS	From classical conditioning: systematic desensitisation (Wolpe), exposure and flooding, aversion. From operant: token economy, contingency management, applied behaviour analysis (functional analysis, ABC). Seligman's learned helplessness (uncontrollable aversive events, the attributional reformulation) as a model of depression.
MEYER, RANK AND THE MAP	The three depth-psychology schools (Freud psychoanalysis, Adler individual psychology, Jung analytical psychology) are on the General and Developmental Psychology notes. these notes add the two Paper-1-named figures: Adolf Meyer's psychobiology (the reaction-type view, the life chart, his influence on formulation) and Otto Rank (birth trauma, will therapy, the break from Freud).
DEFENCES AND VAILLANT	The full catalogue by Vaillant's maturity levels: psychotic (denial of reality, distortion, delusional projection), immature (projection, acting out, splitting, projective identification, passive aggression, regression), neurotic (repression, reaction formation, displacement, intellectualisation, isolation, undoing), mature (sublimation, altruism, humour, suppression, anticipation).
OBJECT RELATIONS AND NEO-FREUDIANS	Klein (paranoid-schizoid and depressive positions, projective identification), Winnicott (good-enough mother, transitional object, holding, true and false self), Fairbairn (libido is object-seeking); Kernberg (levels of personality organisation) and Kohut (self psychology, the selfobject). Neo-Freudians: Horney, Sullivan, Fromm.
PERSONALITY THEORIES	Type theories (humours, Sheldon, Kretschmer) to trait (Allport; Eysenck PEN; Cattell 16PF) to the Big Five consensus (OCEAN, NEO-PI) to humanistic (Rogers self theory, unconditional positive regard; Maslow; existential) to Kelly's constructs. The

The whole picture in one table	
	person-situation debate resolves into interactionism. Then positive psychology (Seligman, PERMA, character strengths, flow).
PSYCHOLOGY OF RAPE	Rape as an act of power and aggression more than sexuality (Groth); offender typologies (with their caveats) and the limits of prediction; rape myths and their harm. Survivor side: the rape trauma syndrome (Burgess and Holmstrom), PTSD and sequelae, the second injury of secondary victimisation, and trauma-informed care. The Indian medico-legal frame is on the forensic psychiatry chapter.
GURU-CHELA AND EXPRESSED EMOTION	Neki's guru-chela model: an Indian, culturally rooted therapeutic relationship built on guidance, dependence and idealisation, contrasted with the Western contractual alliance. Expressed emotion (Brown and Rutter; the Camberwell Family Interview): critical comments, hostility and over-involvement predict relapse; Indian families tend to show lower expressed emotion.
DISCRIMINATE AND APPLY	Tell the learning paradigms apart (classical versus operant versus social learning), the personality models apart (psychodynamic, trait, type, humanistic, social-cognitive), and the social biases apart (fundamental attribution error, self-serving, dissonance, group polarisation). Apply by reading an attribution, choosing a behaviour-therapy technique, placing a person on the Big Five, and naming a group process.

References

This chapter is grounded in the standard texts (the source hierarchy below); each theory is attributed to its primary source, and landmark, contested or numeric claims carry a PubMed-verified journal citation. Every PMID here was checked against PubMed this build, and citations whose identifier did not resolve to the cited paper were corrected or dropped.

SOURCE HIERARCHY (TEXTBOOKS)

1. Boland R, Verduin ML, eds. Kaplan & Sadock's Comprehensive Textbook of Psychiatry. 11th ed. Philadelphia: Wolters Kluwer; 2024. [primary depth bar; the psychological sciences, personality, behavioural science]
2. Sadock BJ, Sadock VA, Ruiz P. Kaplan & Sadock's Synopsis of Psychiatry. 11th ed. Philadelphia: Wolters Kluwer; 2015. [theories of personality; contributions of the social sciences]
3. Geddes JR, Andreasen NC, Goodwin GM, eds. New Oxford Textbook of Psychiatry. 3rd ed. Oxford: Oxford University Press; 2020. [social and behavioural science chapters]
4. Tasman A, Kay J, Lieberman JA, First MB, Riba MB, eds. Psychiatry. 4th ed. Chichester: Wiley-Blackwell; 2015. [social psychology; learning theory; personality]
5. Johnstone EC, Owens DGC, Lawrie SM, et al, eds. Companion to Psychiatric Studies. 8th ed. Edinburgh: Churchill Livingstone/Elsevier; 2010. [psychology of learning, personality and groups]

FOUNDATIONAL PRIMARY SOURCES

1. Festinger L. A Theory of Cognitive Dissonance. Stanford: Stanford University Press; 1957. And Festinger L, Carlsmith JM. Cognitive consequences of forced compliance. J Abnorm Soc Psychol. 1959;58:203-210. [dissonance; the one-dollar, twenty-dollar study]

2. Hovland CI, Janis IL, Kelley HH. Communication and Persuasion. New Haven: Yale University Press; 1953. And Petty RE, Cacioppo JT. The elaboration likelihood model of persuasion. Adv Exp Soc Psychol. 1986;19:123-205. [attitude change; central and peripheral routes]
3. Heider F. The Psychology of Interpersonal Relations. New York: Wiley; 1958. And Kelley HH. Attribution theory in social psychology. Nebraska Symposium on Motivation. 1967;15:192-238. [attribution]
4. Asch SE. Opinions and social pressure. Sci Am. 1955;193:31-35. And Milgram S. Obedience to Authority. New York: Harper & Row; 1974. And Janis IL. Victims of Groupthink. Boston: Houghton Mifflin; 1972. [conformity, obedience, groupthink]
5. Allport GW. The Nature of Prejudice. Reading, MA: Addison-Wesley; 1954. And Tajfel H, Turner JC. An integrative theory of intergroup conflict. In: The Social Psychology of Intergroup Relations. Monterey: Brooks/Cole; 1979. [prejudice; social identity theory]
6. Dollard J, Doob LW, Miller NE, Mowrer OH, Sears RR. Frustration and Aggression. New Haven: Yale University Press; 1939. And Latane B, Darley JM. The Unresponsive Bystander. New York: Appleton-Century-Crofts; 1970. [aggression; the bystander effect]
7. Le Bon G. The Crowd: A Study of the Popular Mind. London: T. Fisher Unwin; 1896. (Psychologie des foules, Paris, 1895.) And Reicher SD. The St. Pauls riot. Eur J Soc Psychol. 1984;14:1-21. [crowd psychology; the social identity model]
8. Pavlov IP. Conditioned Reflexes. Oxford: Oxford University Press; 1927. And Watson JB, Rayner R. Conditioned emotional reactions. J Exp Psychol. 1920;3:1-14. [classical conditioning; Little Albert]
9. Thorndike EL. Educational Psychology, Vol II: The Psychology of Learning. New York: Teachers College; 1913. And Skinner BF. The Behavior of Organisms. New York: Appleton-Century; 1938. [law of effect; operant conditioning and schedules]
10. Bandura A, Ross D, Ross SA. Transmission of aggression through imitation of aggressive models. J Abnorm Soc Psychol. 1961;63:575-582. And Bandura A. Self-efficacy: toward a unifying theory of behavioral change. Psychol Rev. 1977;84:191-215. [social learning; the Bobo doll; self-efficacy]
11. Wolpe J. Psychotherapy by Reciprocal Inhibition. Stanford: Stanford University Press; 1958. And Ayllon T, Azrin NH. The Token Economy. New York: Appleton-Century-Crofts; 1968. [systematic desensitisation; the token economy]
12. Freud A. The Ego and the Mechanisms of Defence. London: Hogarth Press; 1936. And Vaillant GE. Ego Mechanisms of Defense. Washington DC: American Psychiatric Press; 1992. [the defences; the maturity hierarchy]
13. Klein M. The Psycho-Analysis of Children. London: Hogarth Press; 1932. And Winnicott DW. Playing and Reality. London: Tavistock; 1971. And Fairbairn WRD. Psychoanalytic Studies of the Personality. London: Tavistock; 1952. [object relations]
14. Kernberg OF. Borderline Conditions and Pathological Narcissism. New York: Jason Aronson; 1975. And Kohut H. The Analysis of the Self. New York: International Universities Press; 1971. [levels of organisation; self psychology]
15. Eysenck HJ, Eysenck SBG. Manual of the Eysenck Personality Questionnaire. London: Hodder & Stoughton; 1975. And McCrae RR, Costa PT. Personality in Adulthood. New York: Guilford Press; 1990. [the PEN model; the five-factor model]
16. Rogers CR. Client-Centered Therapy. Boston: Houghton Mifflin; 1951. And Maslow AH. A theory of human motivation. Psychol Rev. 1943;50:370-396. [the humanistic tradition]
17. Meyer A. The Life Chart. In: Contributions to Medical and Biological Research. New York: Paul B. Hoeber; 1919. And Rank O. The Trauma of Birth. London: Kegan Paul; 1929. (Das Trauma der Geburt, Vienna, 1924.) [psychobiology; birth trauma]
18. Groth AN. Men Who Rape: The Psychology of the Offender. New York: Plenum Press; 1979. [the offender typology]
19. Leff J, Vaughn C. Expressed Emotion in Families. New York: Guilford Press; 1985. [the expressed-emotion construct and its outcome studies]

JOURNAL REFERENCES (PUBMED-VERIFIED, 16)

1. Weiner B. An attributional theory of achievement motivation and emotion. Psychol Rev. 1985;92(4):548-73. PMID: 3903815.
2. Gilbert DT, Malone PS. The correspondence bias. Psychol Bull. 1995;117(1):21-38. PMID: 7870861.

3. Pettigrew TF, Tropp LR. A meta-analytic test of intergroup contact theory. J Pers Soc Psychol. 2006;90(5):751-83. PMID: 16737372.
4. Corrigan PW, Watson AC. Understanding the impact of stigma on people with mental illness. World Psychiatry. 2002;1(1):16-20. PMID: 16946807.
5. Berkowitz L. Frustration-aggression hypothesis: examination and reformulation. Psychol Bull. 1989;106(1):59-73. PMID: 2667009.
6. Anderson CA, Bushman BJ. Human aggression. Annu Rev Psychol. 2002;53:27-51. PMID: 11752478.
7. Bandura A, Ross D, Ross SA. Imitation of film-mediated aggressive models. J Abnorm Soc Psychol. 1963;66:3-11. PMID: 13966304.
8. Caspi A, McClay J, Moffitt TE, et al. Role of genotype in the cycle of violence in maltreated children. Science. 2002;297(5582):851-4. PMID: 12161658.
9. Hamilton WD. The genetical evolution of social behaviour. I. J Theor Biol. 1964;7(1):1-16. PMID: 5875341.
10. Abramson LY, Seligman ME, Teasdale JD. Learned helplessness in humans: critique and reformulation. J Abnorm Psychol. 1978;87(1):49-74. PMID: 649856.
11. Maier SF, Seligman ME. Learned helplessness at fifty: insights from neuroscience. Psychol Rev. 2016;123(4):349-67. PMID: 27337390.
12. Schultz W. Dopamine reward prediction error coding. Dialogues Clin Neurosci. 2016;18(1):23-32. PMID: 27069377.
13. Engel GL. The need for a new medical model: a challenge for biomedicine. Science. 1977;196(4286):129-36. PMID: 847460.
14. McEvedy CP, Beard AW. Royal Free epidemic of 1955: a reconsideration. Br Med J. 1970;1(5687):7-11. PMID: 5411611.
15. Burgess AW, Holmstrom LL. Rape trauma syndrome. Am J Psychiatry. 1974;131(9):981-6. PMID: 4415470.
16. Neki JS. Guru-chela relationship: the possibility of a therapeutic paradigm. Am J Orthopsychiatry. 1973;43(5):755-66. PMID: 4742816.

PART VI

VI

Psychometry, Assessment & Descriptive Psychopathology

The instruments of the examination: the tests that measure the mind, and the language that describes it.

01 Psychometry and assessment

02 Descriptive psychopathology

03 Discriminate

04 Apply and consolidate

Psychometry, Assessment & Descriptive Psychopathology

Four sections · 22 topics · one complete notes document

This is a mixed set of notes that pairs the two things every clinical encounter rests on: the formal tools that measure the mind, and the descriptive vocabulary that names what is found in it. The first band is **psychometry and assessment**, a catalogue of psychological tests organised by what each one measures: how a test earns its reliability and validity, the intelligence scales and the deviation IQ with its classification bands, the projective techniques (Rorschach, TAT and the rest), the objective personality inventories (MMPI, 16PF, NEO), the neuropsychological batteries, the adaptive and developmental scales, the clinician-rated symptom scales, and the Indian adaptations that make all of this usable in an Indian clinic. The second band is **descriptive psychopathology**, the phenomenology that Jaspers, Fish and Sims built, split into the domains of mental life: disorders of perception, of thought, of the experience of self, of emotion, of memory, of consciousness and of movement, together with the delusions and the Schneiderian first-rank symptoms. The examinable skill is precision on both fronts: choosing the right test for a referral question, and telling one sign apart from its near-neighbour.

HOW TO USE THESE NOTES

The two bands read differently by design. In the **test catalogue**, every test ends with a **Good to remember** box giving its author, what it measures and how it is scored, so the reference facts sit apart from the reasoning. In **descriptive psychopathology**, every named sign carries a crisp one-line definition, most often in a definition list, because in phenomenology the exam rewards the exact word. Figures declare one axis and code it in colour and shape: **petrol** marks structure and the neutral case, **coral** marks pathology or the point of discrimination, and **marigold** highlights the answer. Two boundaries are worth stating up front: these notes cover the intelligence *tests* while the General and Developmental Psychology notes cover the intelligence *theories* and the mechanism-evolution of hallucination theories; the psychometric reliability and validity here are distinct from the research reliability and validity in the Research Methods, Biostatistics & Epidemiology notes. This is doctor-facing revision, so the full clinical vocabulary is used.

Psychometry and assessment

1 · Orientation: the taxonomy of psychological tests

The General and Developmental Psychology and the Social, Learning & Personality Psychology notes cover the **psychology of the normal mind**: how it learns, remembers, feels, and is measured in the abstract. Paper I now turns to the two working instruments of the clinical examination. This band, Band A, takes up the first instrument, the **test** that measures the mind and puts a number on a construct we cannot see directly. Band B takes up the second instrument, the descriptive language that names what the mind is doing when it goes wrong. Hold the pair in

view: one instrument quantifies, the other describes, and the mature psychiatrist reaches for both.

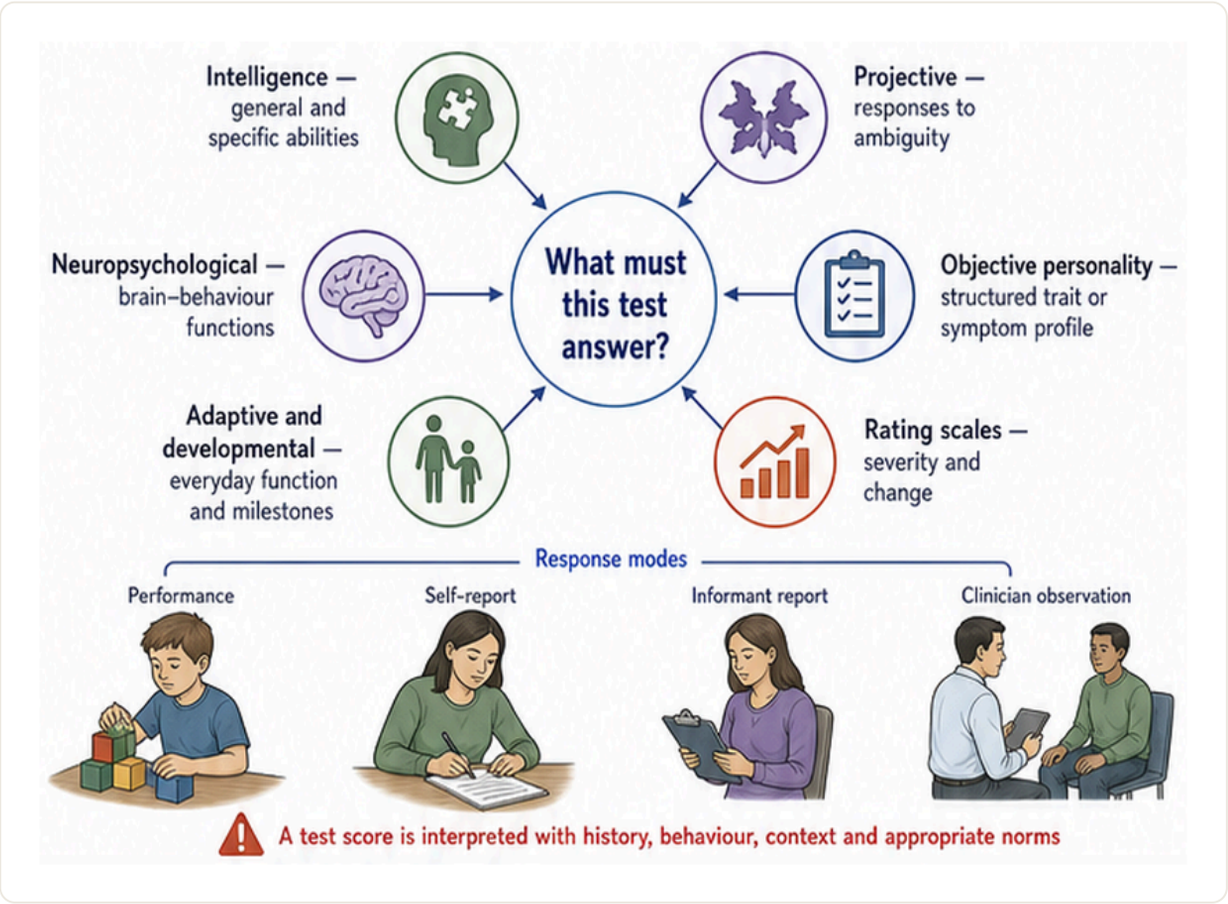


Fig 6.1 Test selection starts with the clinical question.

Why this opens these notes. Examiners love the map before the detail. Before you can defend the Wechsler indices or the Rorschach scoring, you must be able to place any named test into its family and say in one line what that family measures. The taxonomy is the skeleton on which every test in this band hangs, so learn it first and the individual catalogue entries will have somewhere to sit. This is the schema for Band A: a six-family map of **psychometry** that you can redraw from memory.

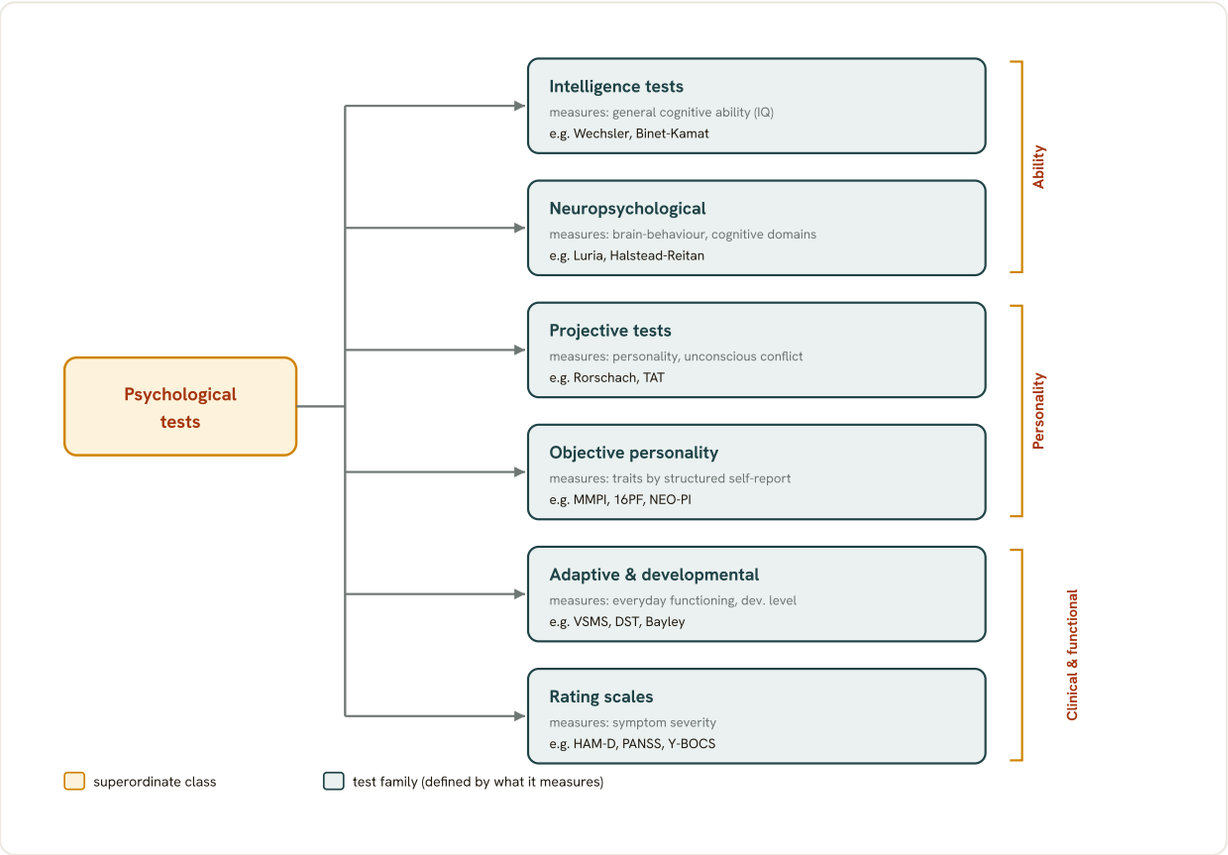


Fig 6.2 The taxonomy of psychological tests. Every test belongs to a family defined by what it measures: the ability tests (intelligence and neuropsychological), the personality tests (projective and objective), and the clinical and functional tests (adaptive-developmental scales and symptom rating scales). Reading a referral question is largely a matter of deciding which family it points to. The families, not the individual instruments, are the unit to learn first.

1.1 What a psychological test is

Definition. A psychological test is an **objective and standardised sample of behaviour**, from which we infer the level of some underlying construct (an ability, a trait, a symptom). The classic phrasing, traceable to Anastasi and repeated across texts, is that a test is a standardised sample of behaviour (Kaplan & Sadock 2024). Two words carry the weight. *Sample*: the test elicits only a slice of behaviour, never the whole person. *Standardised*: administration, scoring, and interpretation follow a fixed procedure against established norms, so that the score means the same thing across testers and occasions.

Psychometry

The science of measuring psychological constructs; the theory and practice of constructing, administering, and interpreting standardised mental tests (Kaplan & Sadock 2024).

Standardisation

Fixing the administration and scoring procedure, and referencing scores to a representative normative sample, so results are comparable.

Norm

The distribution of scores in a defined reference population, against which an individual score is read (percentile, standard score, quotient).

Reliability

The consistency of a test's measurement (test-retest, internal, inter-rater).

Validity
Whether the test measures the construct it claims to (content, criterion, construct validity).

Every catalogue entry in this band is an attempt to hang reliability and validity on a real instrument. A test with poor psychometric properties, however clever its theory, is clinically worthless; structured scales were developed precisely to improve the poor psychometric properties of unstructured clinical judgement (Kaplan & Sadock 2024).

1.2 The test-taxonomy map: six families

The map. Bind each family to a single question, "what does it measure?", and to one anchoring example. Learn this list cold; the rest of these notes is detail poured into these six containers.

Intelligence test
Measures general cognitive ability, expressed as an intelligence quotient (IQ). Anchor: Binet-Kamat, and the Wechsler scales (WAIS for adults, WISC for children).

Projective
Measures personality structure and unconscious conflict by asking the person to impose meaning on an ambiguous stimulus. Anchor: the Rorschach inkblots and the Thematic Apperception Test (TAT).

Objective personality
Measures personality traits and psychopathology through structured, self-report questionnaires with fixed-choice items and empirical scoring. Anchor: MMPI, 16PF, NEO-PI.

Neuropsychological
Measures brain-behaviour relationships across discrete cognitive domains (attention, memory, language, visuospatial, executive function). Anchor: the Luria-Nebraska and Halstead-Reitan batteries.

Adaptive and developmental scales
Measures everyday real-world functioning and developmental level rather than abstract ability. Anchor: Vineland Social Maturity Scale (VSMS), Developmental Screening Test (DST), Bayley Scales.

Rating scale
Measures the severity of a symptom cluster over time, rated by clinician or by patient. Anchor: HAM-D (depression), PANSS (psychosis).

RULE **Name the question, then the family, then the test.** Every test belongs to a family defined by one question - what does it measure - so place the referral before you reach for an instrument.

CLINICAL ANCHOR

The families answer different clinical questions. "How bright is this child?" is an intelligence-test question. "What is the structure of this personality?" is a projective or objective-personality question. "Is this cognitive complaint organic?" is a neuropsychological question. "Can this adult live independently?" is an adaptive-scale question. "Has this depression improved on treatment?" is a rating-scale question. Name the question first, then the family, then the test.

1.3 The intelligence test family

What it measures. General cognitive ability, summarised as an IQ. Population IQ approximates a normal distribution with a mean of 100 and a standard deviation of 15 points (New Oxford Textbook of Psychiatry). An **intelligence test** samples reasoning, comprehension, working memory, and processing speed, typically across separate verbal and performance (or nonverbal) subtests, and yields a global score plus a profile of strengths and weaknesses.

Anchors. The Binet-Kamat Test of Intelligence is the widely used Indian adaptation (Kamat) of the Stanford-Binet, standardised on Indian children. The Wechsler scales split by age: the Wechsler Adult Intelligence Scale (WAIS) and the Wechsler Intelligence Scale for Children (WISC). DSM-5 shifted the definition of intellectual disability so that severity is graded on **adaptive functioning**, not on the IQ number alone; the IQ locates the deficit, but adaptive behaviour grades its severity.

1.4 The projective family

What it measures. Personality organisation and unconscious conflict. The **projective** hypothesis holds that when a stimulus is deliberately ambiguous, the person must draw on their own inner world to give it meaning, and so "projects" their private structure onto the blot or the picture (Kaplan & Sadock 2024). Projective tests are, by design, indirect and unstructured, in contrast to the direct fixed-choice format of objective tests.

Anchors. The Rorschach presents a fixed series of symmetrical inkblots and asks what they might be; responses are coded, not read for content alone. Modern scoring uses the Exner Comprehensive System and, more recently, R-PAS (Rorschach Performance Assessment System), which put the method on a firmer psychometric footing. The Thematic Apperception Test (TAT), developed within Murray's personology, presents ambiguous interpersonal scenes and asks the person to tell a story; the recurring themes are taken to reveal needs and conflicts. (The plates and cards themselves are copyrighted and are described here in words only, never reproduced.)

1.5 The objective personality family

What it measures. Personality traits and psychopathology, through structured self-report. An **objective personality** test uses fixed-choice items (true-false or Likert), empirically or factor-analytically derived scales, and fixed scoring rules, so that interpretation does not depend on the tester's inference in the way a projective does.

Anchors. The MMPI (Minnesota Multiphasic Personality Inventory) is the archetype: an empirically keyed inventory yielding clinical scales plus validity scales that detect response distortion (the current forms are the MMPI-2, MMPI-2-RF, and MMPI-3). Cattell's 16PF measures sixteen primary personality factors by factor-analytic method. The NEO-PI, now NEO-PI-R, operationalises the Five Factor Model (FFM: neuroticism, extraversion, openness, agreeableness, conscientiousness), which is the dominant trait framework in modern personality science.

COMMON TRAP

Do not confuse the two personality routes. The **projective** and the **objective personality** families both measure personality, but by opposite logic: projectives use ambiguous stimuli and inferential scoring to reach the unconscious; objective tests use structured self-report and fixed scoring to map traits. "Objective" refers to the scoring being standardised and examiner-independent, not to the truth being more objective.

1.6 The neuropsychological family

What it measures. Brain-behaviour relationships. A **neuropsychological** assessment profiles performance across discrete cognitive domains, attention, memory, language, visuospatial ability, and executive function, to detect, localise, and characterise cerebral dysfunction, often where structural imaging is normal (Kaplan & Sadock 2024).

Anchors. The Luria-Nebraska Neuropsychological Battery derives from Luria's qualitative approach, systematised into scored scales. The Halstead-Reitan Battery is the classic fixed, quantitative battery yielding an impairment index. Both aim to answer whether a cognitive complaint reflects organic dysfunction and, if so, which systems are affected.

1.7 The adaptive and developmental family

What it measures. Everyday functioning and developmental level, not abstract ability. **Adaptive** scales quantify how well a person meets the practical demands of daily life (self-care, communication, social participation); developmental scales place an infant or young child on a normative developmental trajectory.

Anchors. The Vineland Social Maturity Scale (VSMS), in India the Malin adaptation, yields a social age and a social quotient from an informant interview and is a mainstay in the assessment of intellectual disability. The Developmental Screening Test (DST) and the Bayley Scales of Infant Development assess the developmental level of infants and young children. This family carries extra weight now that DSM-5 grades intellectual disability by adaptive functioning: the adaptive scale, not the IQ test, sets the severity.

1.8 The rating scale family

What it measures. The severity of a defined symptom cluster, quantified so it can be tracked across time and across patients. A **rating scale** is scored either by a clinician (observer-rated) or by the patient (self-rated), and its value lies in serial measurement: charting response to treatment.

Anchors. The Hamilton Depression Rating Scale (HAM-D) is a clinician-rated measure of depression severity. The Positive and Negative Syndrome Scale (PANSS) is a clinician-rated measure of symptom severity in schizophrenia. Rating scales are the instruments the DSM-5 itself now advocates as "emerging measures" to improve routine care (Kaplan & Sadock 2024).

1.9 The two axes of classification

Tests can be sorted along two independent axes, and a good answer names both. The first axis is **by what is measured**, which gives the six families above collapsed into three broad purposes:

ability (intelligence, neuropsychological), personality (projective, objective), and adaptive or symptom-severity (adaptive/developmental scales, rating scales). The second axis is **by structure**, the format of the instrument, cutting across purpose.

STRUCTURAL CONTRAST	ONE POLE	OTHER POLE
Stimulus and scoring	**Objective: fixed-choice items, standardised scoring**	**Projective: ambiguous stimulus, inferential scoring**
Persons tested at once	Individual: one examiner, one subject	Group: many subjects simultaneously
Task medium	Verbal: language-based items	Performance: manipulative or nonverbal tasks
Timing constraint	Power: no time pressure, difficulty graded	Speed: many easy items against the clock

The structural axis cuts across the content axis: an intelligence test can be individual, verbal or performance, and power-type; an objective personality test is group-administrable and objective.

HOLD THIS

A psychological test is a standardised sample of behaviour, placed by what it measures then how it is structured. Six families (I-PONAR), two axes: content (ability, personality, adaptive) and structure (objective vs projective, individual vs group, verbal vs performance).

MNEMONIC

"I Prefer Only Neat And Reliable tests"

The six families in order: **I**ntelligence, **P**rojective, **O**bjective personality, **N**europsychological, **A**daptive/developmental, **R**ating scales. Carry this master mnemonic through the whole set of notes; every catalogue entry that follows lands in one of these six slots.

PEARL

The taxonomy in one line: a psychological test is a **standardised sample of behaviour**, and every test is placed by asking *what it measures* (ability, personality, adaptive function, or symptom severity) and then *how it is structured* (objective or projective; individual or group; verbal or performance).

GOOD TO REMEMBER

- **Psychometry:** the science of measuring psychological constructs; a psychological test is a standardised sample of behaviour (Anastasi; Kaplan & Sadock 2024).
- **Intelligence test family:** measures general cognitive ability (IQ, mean 100, SD 15); examples Binet-Kamat, Wechsler WAIS (adults) and WISC (children).
- **Projective family:** measures personality and unconscious conflict via ambiguous stimuli and inferential scoring; examples Rorschach (Exner / R-PAS scoring), Thematic Apperception Test (TAT, Murray).
- **Objective personality family:** measures traits and psychopathology via structured self-report and fixed scoring; examples MMPI (validity plus clinical scales), 16PF (Cattell), NEO-PI-R (Five Factor Model).
- **Neuropsychological family:** measures brain-behaviour relationships across cognitive domains; examples Luria-Nebraska, Halstead-Reitan (impairment index).
- **Adaptive and developmental family:** measures everyday functioning and developmental level; examples Vineland Social Maturity Scale (social quotient), Developmental Screening Test (DST), Bayley Scales.
- **Rating scale family:** measures symptom severity over time, clinician or self rated; examples HAM-D (depression), PANSS (schizophrenia).
- **Two axes of classification:** by what is measured (ability vs personality vs adaptive) and by structure (objective vs projective; individual vs group; verbal vs performance).
- **Master mnemonic:** "I Prefer Only Neat And Reliable tests" = Intelligence, Projective, Objective personality, Neuropsychological, Adaptive/developmental, Rating scales.
- **Currency:** Rorschach now scored by Exner / R-PAS; NEO-PI-R operationalises the FFM; DSM-5 grades intellectual disability by adaptive functioning, not the IQ number alone; first-rank symptoms are no longer treated as pathognomonic.

2 · Test construction: reliability, validity, standardisation

This is the grammar of every test in these notes. Before you can catalogue the Wechsler, the MMPI or the Rorschach, you have to know what makes any psychometric instrument trustworthy. The examiner treats **test construction** as a set of properties a test must demonstrate before it can be used in the clinic or the courtroom: it must be standardised, reliable and valid, and it must be practical. Every other test in these notes is scored against these four yardsticks, so this topic is the lens for the whole set.

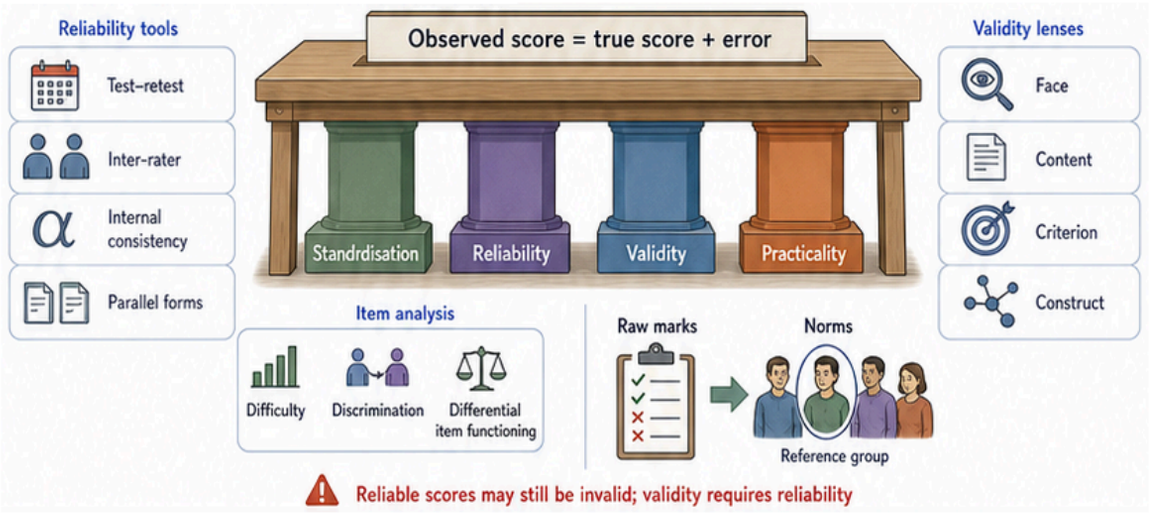


Fig 6.3 Building a sound psychological test.

The single most-tested idea is the relationship between the two central properties. **reliability** is consistency (does the test give the same answer twice?); **validity** is accuracy (does it measure what it claims?). A test can be reliable without being valid, but it can never be valid without first being reliable (Kaplan & Sadock 2024). A crucial boundary to hold: the **reliability** and **validity** taught here are *psychometric* properties of a measurement instrument. They are distinct from the *research* reliability and validity (sensitivity, specificity, internal and external validity of a study design) taught in the Research Methods, Biostatistics & Epidemiology notes. Same words, different domain: keep them in separate drawers.



Fig 6.4 Reliability versus validity, the classic marksman analogy. Reliability is how tightly the shots cluster (consistency); validity is whether they land on the bull (accuracy). The two are independent: a test can be reliable but not valid (tight but off target, the dangerous case of a precise wrong answer), valid but not reliable (centred on average but scattered), neither, or both. The one dependency is that validity requires reliability, so a good test must be reliable first, then shown to be valid.

2.1 The four properties of a good test

Standardisation, reliability, validity, practicality. A psychometrically sound test must satisfy four requirements (Kaplan & Sadock 2024; Companion to Psychiatric Studies). **standardisation** (American: **standardization**) means the test is administered and scored in a uniform, fixed way for every examinee, and that a person's raw score is interpreted against **norms** from a representative reference sample. **reliability** is the consistency, or reproducibility, of the scores. **validity** is the degree to which the test actually measures the construct it claims to measure. *Practicality* (utility) is the down-to-earth fourth property: the test must be reasonable in cost, administration time, ease of scoring and acceptability to the patient. A perfectly reliable and valid instrument that takes six hours and a doctorate to score has little clinical utility. Reliability and validity are the two that carry marks; standardisation is their precondition; practicality is what decides bedside use.

2.2 Standardisation: uniform procedure and a reference sample

Two limbs, procedure and norms. **standardisation** has two components (Kaplan & Sadock 2024). The first is *uniform administration and scoring*: the same instructions, materials, time limits, order of items and scoring rules for every examinee, so that any difference in scores reflects the person and not the testing conditions. This is why a standardised test comes with a manual that scripts the examiner. The second is the derivation of **norms**: the test is given to a

large, carefully selected *standardisation (normative) sample* that represents the population the test is meant for, and the distribution of scores in that sample becomes the yardstick against which any individual's raw score is read. An individual's score is meaningless in isolation; it acquires meaning only as a position within the normative distribution.

2.3 Norms and derived scores: percentiles, z-scores, T-scores, standard scores

Turning a raw score into a position. Because a raw score has no inherent meaning, tests convert it into a *derived score* that locates the person on the normal curve (Kaplan & Sadock 2024). A *percentile rank* states the percentage of the reference sample the person scored at or above (a percentile of 84 means the person did better than 84 percent of the normative sample). A *z-score* expresses the raw score as the number of standard deviations above or below the mean (mean 0, standard deviation 1). A *T-score* is a rescaled standard score with a mean of 50 and a standard deviation of 10; it is the currency of the MMPI, where a T-score of 65 or above (1.5 standard deviations up) flags a clinically significant elevation. *Standard scores* more broadly are any transformation onto a fixed mean and standard deviation, the Wechsler IQ scale (mean 100, standard deviation 15) being the best-known example. Norms are almost always stratified: *age norms* are essential wherever performance changes with age (intelligence, memory, and the neuropsychological tests, where a 70-year-old is compared with age peers, not with 20-year-olds), and *education norms* matter because education strongly shapes cognitive test performance (Kaplan & Sadock 2024).

PEARL

The four derived scores are just four ways of saying the same sentence, "how far from average is this person?" Percentile answers in rank, z in raw standard deviations, T on a 50/10 scale, and the Wechsler standard score on a 100/15 scale. Memorise the two clinical anchors: MMPI T-score of 65 is the clinical cut-off, and a Wechsler IQ of 100 is dead average with each 15 points being one standard deviation.

50 / 10	65	100 / 15
T-SCORE MEAN / SD	MMPI CLINICAL CUT-OFF (T)	WECHSLER IQ MEAN / SD

2.4 Reliability: consistency of measurement

Does the test give the same answer twice? **reliability** is the consistency, stability or reproducibility of test scores (Kaplan & Sadock 2024). It is quantified by a *reliability coefficient*, a correlation running from 0 (no consistency) to 1.0 (perfect consistency); as a rough rule most instruments need a coefficient of about 0.80 or higher to be useful (Kaplan & Sadock 2024). A companion statistic is the *standard error of measurement* (SEM), which uses the reliability coefficient and the test's standard deviation to put a confidence band around any single obtained score: with an SEM of 3, a person scoring 90 today would score within 87 to 93 about 68 percent of the time on a retest, and within 84 to 96 about 95 percent of the time (Kaplan & Sadock 2024). The smaller the SEM, the more reliable the test. There are four ways reliability is estimated, and the examiner expects all four by name.

2.5 The four types of reliability

Temporal, inter-scorer, internal and parallel. Each type answers "consistent across what?" (Kaplan & Sadock 2024).

Test-retest reliability

Temporal stability: the same test given to the same people on two occasions, and the scores correlated. It answers whether the score holds across time. A coefficient of about 0.80 is expected if the two administrations are within roughly two weeks and the trait is stable. The main threat is the *practice (carry-over) effect*.

Inter-rater (inter-scorer) reliability

Agreement between different scorers rating the same performance. It answers whether the score is independent of who marks it, and is critical for any test with judgement in scoring (projectives, structured interviews, clinician rating scales). For categorical judgements it is quantified by *Cohen's kappa*, which corrects agreement for chance.

Internal consistency

Whether all the items measure the same single construct, estimated from one administration. The *split-half* method correlates one half of the test (often odd items) with the other (even items), corrected upward by the *Spearman-Brown formula*. The definitive index is *Cronbach's alpha*, the average of all possible split-half correlations, expressing the mean inter-item correlation; a higher value means a more internally consistent scale.

Parallel (alternate) forms reliability

Two equivalent versions of the same test, built from different items measuring the same construct, are administered and correlated. It answers whether two forms are interchangeable, and is the way to sidestep the practice effect of test-retest. A coefficient of about 0.90 or higher is expected.

2.6 Validity: the test measures what it claims

Accuracy, not mere consistency. **validity** is the degree to which a test measures what it purports to measure (Kaplan & Sadock 2024). A bathroom scale that always reads five kilograms heavy is perfectly reliable but invalid: it is consistent, but consistently wrong. Validity is not a single number but a body of evidence, gathered in four traditional forms of ascending rigour: **face validity**, **content validity**, **criterion validity** (with concurrent and predictive subtypes) and **construct validity** (with convergent and discriminant evidence). The first two are judged by inspection; the second two are established statistically.

2.7 The four types of validity

Face, content, criterion, construct. These run from superficial to deep (Kaplan & Sadock 2024; Companion to Psychiatric Studies).

Face validity

Whether the items *appear*, on the surface and to a lay reader, to measure what the test claims. It is the weakest form, a matter of appearance not evidence; a depression item that asks "do you feel sad?" has high face validity. High face validity aids acceptability but can also make a test easy to fake.

Content validity

Whether the items adequately sample the whole domain of the construct, judged by expert review. A test of depression with content validity covers mood, cognition, and the somatic and vegetative

features, not mood alone. It concerns the representativeness of the item pool, not appearance.

Criterion validity

Whether the test correlates with an external, independent criterion (a gold standard). It has two subtypes by timing. *Concurrent validity*: the test and the criterion are measured at the same time (a new depression scale correlated against a structured diagnostic interview done today). *Predictive validity*: the test predicts a criterion measured in the future (an aptitude test predicting later job performance; a suicide-risk scale predicting later attempts).

Construct validity

Whether the test measures the abstract theoretical construct it targets, the most demanding and encompassing form. It is built from a pattern of evidence: *convergent validity* (the test correlates highly with other measures of the same construct) and *discriminant (divergent) validity* (it does *not* correlate with measures of unrelated constructs). Together these show the test tracks its construct and nothing else.

> **COMMON TRAP**
>
> Do not confuse *discriminant validity* (a construct-validity concept: the test should not correlate with unrelated constructs) with a test's ability to *discriminate between groups* (e.g. depressed vs non-depressed patients), and do not confuse either with the item *discrimination index* in item analysis. Three different "discriminate" ideas at three different levels: the item, the group, and the construct.

2.8 Item analysis: difficulty and discrimination indices

How individual items earn their place. Before a test is finalised, each candidate item is evaluated statistically, a process called **item analysis** (Kaplan & Sadock 2024; Companion to Psychiatric Studies). The *item difficulty index* (p) is the proportion of examinees who answer an item correctly (or endorse it): a value near 1.0 means almost everyone passes it (too easy), near 0 means almost nobody does (too hard); items of moderate difficulty (around 0.5) usually carry the most information and spread people out best. The *item discrimination index* asks how well a single item separates high scorers from low scorers on the test as a whole: a good item is one that high total-scorers tend to get right and low total-scorers tend to get wrong (a positive point-biserial correlation between the item and the total score). An item that everyone passes, or that good and poor overall performers pass equally, discriminates poorly and is dropped. Item analysis is how the item pool is pruned so the finished test is reliable and valid.

2.9 Test bias and item bias (differential item functioning)

When a test is unfair to a subgroup. **test bias** exists when a test systematically over- or under-estimates the true standing of an identifiable group (defined by culture, language, sex, education or socioeconomic status), so that scores mean different things across groups (Kaplan & Sadock 2024). The item-level form is *differential item functioning* (DIF): a single item behaves differently for two groups *matched on the underlying trait*, so that equally able members of the two groups have unequal odds of answering it correctly for reasons unrelated to the construct (a vocabulary item that assumes a culturally specific term is the classic example). Detecting and removing biased items is central to fair test construction, and it is a major reason culture-fair and non-verbal tests were developed. In the Indian setting this is not academic: many Western

instruments must be translated, culturally adapted and re-normed on Indian samples before their scores can be trusted.

INDIA

A Western test cannot simply be handed to an Indian patient. The standard requirement is translation with *back-translation*, cultural adaptation of items, and fresh **standardisation** on a representative Indian sample to generate local **norms**; without local norms an imported test carries built-in **test bias**. This is why Indian adaptations and indigenously developed tests (for intelligence, adaptive behaviour and personality) exist, and why examiners expect you to name re-standardisation as the fix for cross-cultural bias.

2.10 The reliability-validity relationship: the bullseye

Reliable but not valid; valid requires reliable. This is the highest-yield single point of the topic. The two properties are asymmetrically linked: a test *can* be reliable without being valid, but it *cannot* be valid without being reliable (Kaplan & Sadock 2024). The standard teaching device is the target (bullseye) analogy. Imagine four archers, each firing several arrows. *Reliable but not valid*: the arrows land tightly clustered but off to one side, consistent yet wrong (a scale that always reads five kilos heavy). *Neither*: the arrows scatter all over, inconsistent and off-target. *Reliable and valid*: the arrows land tightly clustered on the bullseye, consistent *and* accurate, the goal of test construction. The logic of the asymmetry: reliability sets a ceiling on validity, because a test that cannot even agree with itself has no stable signal to correlate with any external criterion. So reliability is necessary but not sufficient for validity. This is exactly why we establish reliability first and validity second.

RULE **A test can be reliable without being valid, but never valid without first being reliable.** Reliability caps validity, so it is established first.

HOLD THIS

Reliability is consistency; validity is accuracy; validity requires reliability. Four types of each: reliability (test-retest, inter-rater, internal, parallel); validity (face, content, criterion, construct).

MNEMONIC

"Reliable = the same; Valid = the truth."

Reliability asks "same answer twice?" (consistency); validity asks "the right answer?" (accuracy). Pair it with the bullseye: tight cluster off-centre is reliable-not-valid; tight cluster on the bullseye is reliable-and-valid. You cannot hit the bullseye consistently (valid) without first grouping your arrows tightly (reliable).

2.11 Keeping psychometric and research reliability/validity apart

The same two words in two different examinations. Because the identical terms recur in the research-methodology paper, students routinely conflate them, and examiners probe the boundary. In *psychometrics* (this topic), **reliability** means score consistency (test-retest, inter-rater, internal consistency, parallel forms) and **validity** means the test measures its construct

(face, content, criterion, construct). In *research methodology* (covered in the Research Methods, Biostatistics & Epidemiology notes), the same words carry different technical meanings: the reliability and validity of a diagnostic test are expressed through *sensitivity* and *specificity*, and the validity of a *study* splits into internal validity (are the causal inferences sound within the study?) and external validity (do the findings generalise?). Do not import one framework into the other. When a psychometry question says "validity," it wants face/content/criterion/construct; the sensitivity-and-specificity and study-design material belongs to the Research Methods, Biostatistics & Epidemiology notes.

CLINICAL ANCHOR

These are not abstractions. When you read that the MMPI-2 has clinical utility, you are being told it was standardised on a large normative sample, has adequate test-retest and internal consistency reliability, and has criterion and construct validity against independent diagnoses (Kaplan & Sadock 2024). When a rating scale you use in the ward has published "good reliability and validity", it means precisely: raters agree (inter-rater), the scale is internally consistent, and its scores track true symptom severity. The four properties are the questions to ask of any instrument before you trust its number.

GOOD TO REMEMBER

- **Four properties of a good test:** standardisation, reliability, validity, practicality (utility).
- **Standardisation:** uniform administration and scoring + norms from a representative reference sample (age and education norms).
- **Derived scores:** percentile (rank in sample); z-score (mean 0, SD 1); T-score (mean 50, SD 10; MMPI clinical cut-off ~65); Wechsler standard score (mean 100, SD 15).
- **Reliability** = consistency of measurement; reliability coefficient 0 to 1.0, useful if ~0.80+; SEM puts a confidence band around a score.
- **Test-retest reliability:** temporal stability; same test twice, scores correlated; threatened by practice effect.
- **Inter-rater reliability:** agreement between scorers; categorical agreement corrected for chance by Cohen's kappa.
- **Internal consistency:** items measure one construct; split-half (Spearman-Brown corrected) and Cronbach's alpha (mean inter-item correlation).
- **Parallel (alternate) forms reliability:** two equivalent forms correlated; avoids the practice effect; expected ~0.90+.
- **Validity** = the test measures what it claims; body of evidence, not one number.
- **Face validity:** items appear to measure the construct (weakest; appearance only).
- **Content validity:** items adequately sample the whole domain (expert review).
- **Criterion validity:** correlation with an external criterion; concurrent (same time) and predictive (future).
- **Construct validity:** measures the theoretical construct; convergent (correlates with same-construct measures) and discriminant (does not correlate with unrelated ones).
- **Item analysis:** item difficulty index (p, proportion passing) and item discrimination index (separates high from low total-scorers).
- **Test bias / item bias:** systematic unfairness to a subgroup; item-level form is differential item functioning (DIF); fixed by re-standardisation and local norms.
- **Reliability-validity relationship:** a test can be reliable but not valid; validity requires reliability (bullseye analogy; reliability caps validity).
- **Cross-reference:** psychometric reliability/validity (here) are distinct from research reliability/validity (sensitivity, specificity, study internal/external validity) taught in the Research Methods, Biostatistics & Epidemiology notes.

3 · Intelligence tests and the deviation IQ

Intelligence **tests** are the oldest and most psychometrically mature instruments in clinical psychology, and they are the workhorse of any assessment for intellectual disability, developmental delay, or a suspected drop in premorbid ability. This topic is about the *tests* and about how a raw score becomes an IQ. The competing *theories* of what intelligence actually is (Spearman's g, Thurstone's primary abilities, Gardner's multiple intelligences, Sternberg's triarchic model, and the Cattell-Horn-Carroll or CHC hierarchy) belong to the General and Developmental Psychology notes; here we cross-refer to them and do not re-teach them.

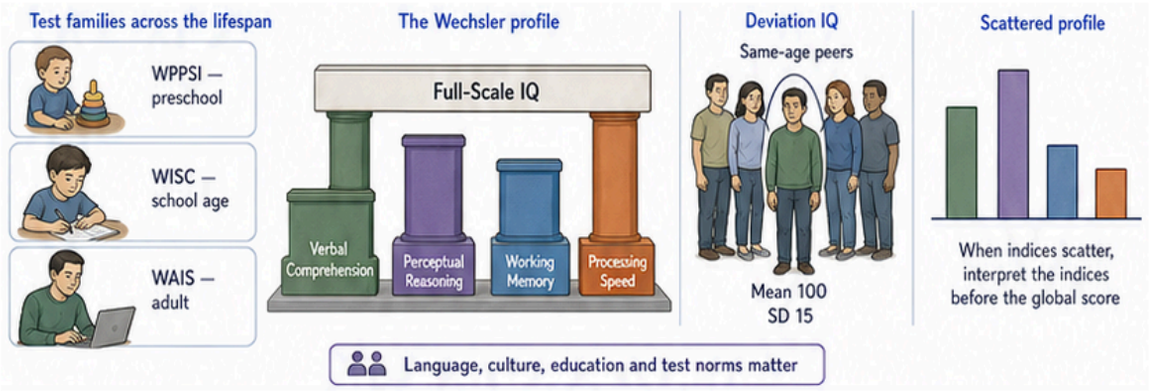


Fig 6.5 Intelligence testing: interpret the profile, not only FSIQ.

The single most examined idea here is the shift from the old **ratio IQ** to the modern **deviation iq**. Examiners love the exact **classification** bands and the intellectual-disability severity ranges, so verify and memorise them cold. A second favourite is the internal architecture of the **wechsler** scales: which subtests feed which of the four index scores, and how those roll up into the full-scale iq.

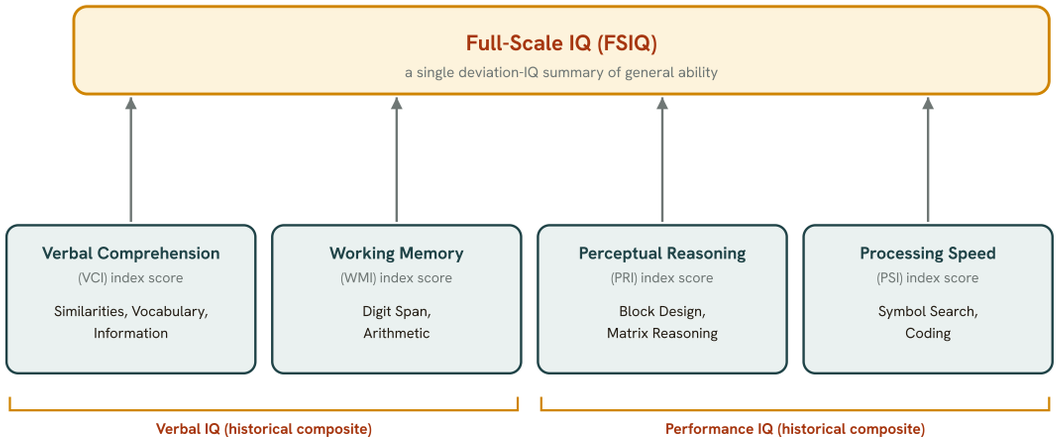
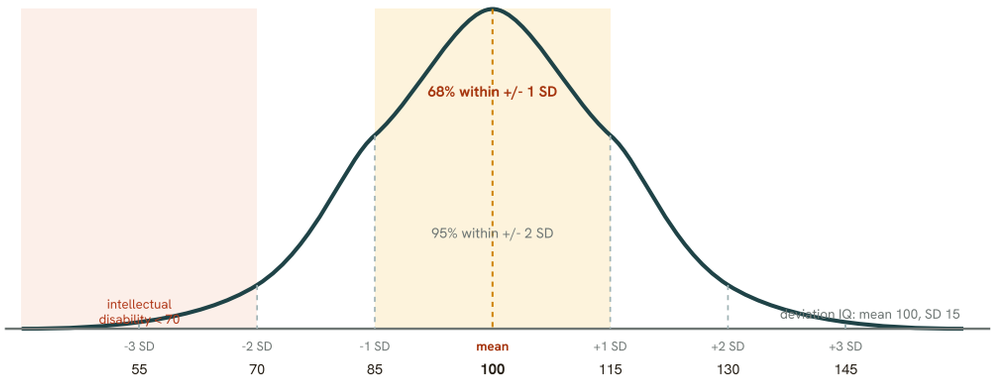


Fig 6.6 Structure of the Wechsler scales (WAIS for adults, WISC for children). Individual subtests aggregate into four index scores (verbal comprehension, working memory, perceptual reasoning, processing speed), which in turn combine into the single Full-Scale IQ. The older Wechsler editions summed the subtests into a Verbal IQ and a Performance IQ instead; the four-index structure is the modern replacement, and a wide split between indices is itself clinically informative.



Classification bands

Very superior 130 and above
Superior 120 to 129
High average 110 to 119
Average 90 to 109
Low average 80 to 89
Borderline 70 to 79

Intellectual disability (ICD-10 IQ, approx.)

Mild 50 to 69
Moderate 35 to 49
Severe 20 to 34
Profound below 20

DSM-5 and ICD-11 grade severity by adaptive functioning, not the IQ score alone.

Fig 6.7 The deviation IQ and its classification bands. IQ is scaled as a standard score with a mean of 100 and a standard deviation of 15, so about 68 per cent of people score within one SD (85 to 115) and about 95 per cent within two SD (70 to 130). The bands read off the distribution: average is 90 to 109, and the intellectual-disability range begins below 70. The ICD-10 severity bands are given by approximate IQ, but current systems (DSM-5, ICD-11) grade severity by adaptive functioning rather than the IQ number alone.

3.1 The intelligence quotient: ratio versus deviation

Ratio IQ. The original **intelligence quotient** was a ratio. William Stern proposed dividing mental age by chronological age; Lewis Terman popularised multiplying by 100 to remove the decimal, giving IQ = (mental age / chronological age) x 100. A child of mental age 10 and chronological age 8 scores 125. The ratio worked well in childhood but breaks down in adulthood, because mental age plateaus while chronological age keeps rising, so the ratio drifts spuriously downward after the late teens.

Deviation IQ. David Wechsler solved this by abandoning the ratio for a standard score. The **deviation iq** expresses where an individual sits on the normal distribution of same-age peers, scaled to a mean of 100 and a standard deviation of 15 (New Oxford Textbook of Psychiatry). It is not a quotient at all despite the retained name: it is a normalised z-score rescaled so that average is 100 and one SD is 15 points. This is the IQ used by every modern scale.

COMMON TRAP

Not every test uses SD 15. The historic Stanford-Binet (Form L-M) used an SD of 16, and Cattell's culture-fair test used SD 24. A "borderline" cut of two SDs therefore lands at 68 on an SD-16 test but at 70 on the modern SD-15 scales. Always ask which SD the norms use before interpreting a score near a cut-off.

RULE **The deviation IQ is a rescaled z-score, not a quotient.** Wechsler abandoned the ratio (mental age over chronological age) because it drifts spuriously downward in adulthood.

3.2 The Stanford-Binet lineage

Binet-Simon (1905). Alfred **binet** and Theodore Simon built the first practical intelligence test in Paris in 1905, commissioned to identify schoolchildren needing special education. It introduced the concept of mental age: a graded ladder of age-normed tasks a child of a given age should be able to pass.

Stanford-Binet (Terman). Lewis Terman at Stanford University revised and re-standardised the scale for American use, publishing the **stanford-binet** in 1916. This is the version that carried Stern's ratio IQ into wide clinical use. The scale is now in its fifth edition (SB5), which has itself moved to a deviation IQ with mean 100 and SD 15 and yields verbal and nonverbal domain scores plus a full-scale composite.

Indian adaptation (Binet-Kamat). The Stanford-Binet was adapted for Indian use as the Binet-**kamat** Test of Intelligence (V. V. Kamat), a locally normed verbal-loaded scale still used in some Indian centres for children (also recorded as the Binet-Simon or Binet-Kamath test).

3.3 The Wechsler family of scales

One design, three age bands. David **wechsler** built a single conceptual instrument spanning the lifespan, split into three age-banded versions. All use the deviation iq (mean 100, SD 15).

WAIS

Wechsler Adult Intelligence Scale; ages 16 to 90 (current edition WAIS-IV, and WAIS-V). The **wais** tradition began with the Wechsler-Bellevue scale of 1939 and is the "gold standard" adult measure (Companion to Psychiatric Studies).

WISC

Wechsler Intelligence Scale for Children; ages roughly 6 to 16. The **wisc** gives a profile of specific verbal and perceptual abilities plus a full-scale IQ.

WPPSI

Wechsler Preschool and Primary Scale of Intelligence; the youngest band, from about 2 years 6 months to 7 years 7 months (Kaplan and Sadock 2024).

3.4 Verbal and performance subtests: the historic dichotomy

The classic split. The original Wechsler design summed a set of subtests into a **verbal** IQ and a **performance** IQ, then combined both into a Full-Scale IQ (Companion to Psychiatric Studies). Verbal subtests (Vocabulary, Similarities, Arithmetic, Digit Span, Information, Comprehension) tap crystallised, language-loaded ability. Performance subtests (Picture Completion, Block Design, Matrix Reasoning, Digit Symbol coding, Picture Arrangement) tap fluid, visuospatial, timed ability. A large verbal-minus-performance discrepancy was traditionally read as a clue to lateralised deficit or to a specific learning disorder, though this interpretation is now used cautiously.

CLINICAL ANCHOR

In a suspected dementia or acquired brain injury, a preserved verbal score with a collapsed performance/processing-speed score is the classic pattern: crystallised knowledge (Vocabulary, Information) is relatively "hold" and resists decline, while timed fluid tasks are "don't-hold" and fall early. The verbal-performance gap is a quick bedside heuristic before formal neuropsychology.

3.5 The four index scores and the full-scale IQ

Modern structure. From WAIS-IV and WISC-IV onward the verbal/performance dichotomy was superseded by four factor-analytically derived **index score** composites, each a standard score with mean 100 and SD 15 (Companion to Psychiatric Studies). They aggregate into the overall **full-scale iq**.

Verbal Comprehension Index (VCI)

Crystallised verbal reasoning and acquired knowledge; core subtests such as Similarities, Vocabulary, Information.

Perceptual Reasoning Index (PRI)

Fluid, nonverbal, visuospatial reasoning; core subtests such as Block Design, Matrix Reasoning, Visual Puzzles. (Called Perceptual Organisation in older editions; Visual Spatial plus Fluid Reasoning in WAIS-V.)

Working Memory Index (WMI)

Attention, mental control, and manipulation of information; Digit Span, Arithmetic, Letter-Number Sequencing.

Processing Speed Index (PSI)

Speed of simple visual scanning and graphomotor output; Coding (Digit Symbol), Symbol Search.

Full-Scale IQ. The full-scale iq is the best single summary of general ability (a proxy for Spearman's g, see the General and Developmental Psychology notes). It is a weighted composite of the index scores. When the four indices scatter widely, clinicians are taught to interpret the individual indices rather than the full-scale iq, because a single global number then masks a clinically important profile.

PEARL

Of the perceptual subtests, Matrix Reasoning has the highest correlation with overall full-scale iq (Companion to Psychiatric Studies), which is why abbreviated screeners like the WASI use Vocabulary plus Block Design plus Matrix Reasoning plus Similarities to estimate FSIQ in about 30 minutes.

3.6 Deviation IQ classification bands

The Wechsler descriptive bands. These are the standard qualitative **classification** labels attached to deviation-iq ranges (mean 100, SD 15). They are among the most commonly asked exact-recall facts in the assessment paper.

DEVIATION IQ RANGE	DESCRIPTIVE CLASSIFICATION
130 and above	Very superior / gifted
120 to 129	Superior
110 to 119	High average
90 to 109	Average
80 to 89	Low average
70 to 79	Borderline
Below 70	Extremely low / intellectual disability range

Standard Wechsler deviation-IQ descriptive bands; the average band 90 to 109 captures roughly the middle 50 percent of the population, and 70 is two standard deviations below the mean.

100 MEAN DEVIATION IQ	**15** ONE STANDARD DEVIATION	**70** 2 SD BELOW (ID RANGE BEGINS)	**130** 2 SD ABOVE (VERY SUPERIOR)

3.7 Intellectual disability severity: ICD-10 IQ ranges

The four ICD-10 severity levels. Below an IQ of about 70 (two SDs below the mean), ICD-10 subdivides intellectual disability into four severity levels by approximate IQ range. Learn these exact bands.

SEVERITY	APPROXIMATE ICD-10 IQ RANGE	ROUGH MENTAL AGE
mild	**50 to 69**	9 to under 12 years
moderate	**35 to 49**	6 to under 9 years
severe	**20 to 34**	3 to under 6 years
profound	**below 20**	under 3 years

ICD-10 (F70 to F73) intellectual disability severity by approximate IQ; the mental-age figures are the ICD-10 clinical descriptions. Mild covers the large majority of cases.

COMMON TRAP

DSM-5 and ICD-11 no longer set severity by the IQ number. They classify intellectual disability severity by *adaptive functioning* across the conceptual, social, and practical domains, because it is adaptive skill (not the test score) that determines the level of support a person needs. IQ still establishes the diagnosis (roughly two SDs below the mean, about 65 to 75 allowing for measurement error), but severity is graded on adaptive functioning. Do not quote IQ cut-offs as the DSM-5 severity criterion.

HOLD THIS

The IQ locates the deficit; adaptive functioning grades its severity. Deviation IQ (mean 100, SD 15) replaced the ratio IQ; ICD-10 still bands severity by IQ (mild 50-69, moderate 35-49, severe 20-34, profound below 20), but DSM-5 and ICD-11 grade by adaptive function.

3.8 Culture-fair and nonverbal tests

Raven's Progressive Matrices. The best-known culture-fair test is Raven's Progressive Matrices (J. C. Raven, 1938). It is a purely nonverbal test of inductive reasoning and eductive ability: the person completes a visual matrix pattern by choosing the missing piece from options. Because it needs no reading, spoken instructions, or culture-specific knowledge, it is used across language and educational backgrounds and loads heavily on Spearman's g (see the General and Developmental Psychology notes). Versions include Coloured Progressive Matrices (young children and adults with impairment), Standard Progressive Matrices, and Advanced Progressive Matrices (high-ability adults).

Other performance tests. Where language or literacy is a barrier, performance and form-board tests are used: Seguin Form Board, Gesell's Drawing Test, and, in India, Bhatia's Battery of Performance Tests of Intelligence (a widely used indigenous nonverbal battery for Indian populations).

INDIA

For Indian assessment, the go-to instruments are the Binet-Kamat Test (verbal, child), Bhatia's Battery of Performance Tests of Intelligence (nonverbal, for low-literacy and rural populations), Malin's Intelligence Scale for Indian Children (MISIC, an Indian adaptation of the WISC), and the Seguin Form Board (a quick performance screen). Raven's Matrices are also widely used because they are language-free.

3.9 Strengths, limits, and clinical use

Strengths. Modern intelligence tests are the most rigorously standardised, reliable (IQ-scale reliabilities of about 0.88 to 0.97 for the WAIS), and well-normed instruments in psychology, with excellent predictive validity for academic and occupational outcomes.

Limits. A single global IQ can obscure a scattered profile; scores are affected by motivation, sensory or motor impairment, language, education, and test-taking familiarity; the Flynn effect (secular rise in raw scores over decades) means norms date and old norms inflate scores, so a test must be re-normed periodically. Verbal-loaded tests disadvantage those from different linguistic or cultural backgrounds, which is exactly why culture-fair tests such as Raven's exist.

TRAP The Flynn effect means old norms inflate scores, so an outdated test over-estimates IQ; a scale must be periodically re-normed, and a score is only as current as the norms behind it.

MNEMONIC

"VW-PP" ages: Very-young WPPSI, then WISC-child, then WAIS-adult

WPPSI (preschool, ~2.5 to 7) then WISC (child, ~6 to 16) then WAIS (adult, 16+). The three Wechsler bands stack across the lifespan in that order.

GOOD TO REMEMBER

- **Ratio IQ (Stern, Terman):** measures childhood ability as (mental age / chronological age) x 100; scored as that ratio; breaks down in adulthood.
- **Deviation IQ (Wechsler):** measures ability relative to same-age peers; scored as a standard score with mean 100 and SD 15; used by all modern scales.
- **Binet-Simon (Binet and Simon, 1905):** first practical intelligence test; introduced mental age; graded age-normed tasks.
- **Stanford-Binet (Terman, 1916; now SB5):** measures general ability across the lifespan; SB5 scores as deviation IQ (mean 100, SD 15) with verbal and nonverbal domains.
- **Binet-Kamat (V. V. Kamat, India):** measures child intelligence, verbal-loaded; Indian adaptation of the Stanford-Binet, scored as an IQ.
- **WAIS (Wechsler):** measures adult intelligence (16 to 90); scored on four index scores and a full-scale iq (mean 100, SD 15).
- **WISC (Wechsler):** measures child intelligence (~6 to 16); same index-score and full-scale iq scoring.
- **WPPSI (Wechsler):** measures preschool intelligence (~2 yr 6 mo to 7 yr 7 mo); deviation-IQ scoring.
- **Four index scores:** Verbal Comprehension, Perceptual Reasoning, Working Memory, Processing Speed, each mean 100 / SD 15, combining into the full-scale iq.
- **Raven's Progressive Matrices (Raven, 1938):** measures nonverbal inductive reasoning and g; culture-fair, language-free; scored by percentile against norms.
- **Bhatia's Battery / MISIC / Seguin Form Board (India):** measure nonverbal or child intelligence; indigenous or adapted Indian performance tests.
- **Wechsler classification bands:** very superior/gifted 130 and above, superior 120 to 129, high average 110 to 119, average 90 to 109, low average 80 to 89, borderline 70 to 79.
- **ICD-10 intellectual disability by IQ:** mild 50 to 69, moderate 35 to 49, severe 20 to 34, profound below 20.
- **DSM-5 and ICD-11 shift:** intellectual disability severity is now graded by adaptive functioning, not the IQ score alone.

4 · The Rorschach inkblot test

The **rorschach** inkblot test is the archetypal projective test of personality: an ambiguous, unstructured stimulus onto which the examinee is presumed to project fundamental features of their personality organisation and thought process. **Why it matters.** It is the single most examined projective instrument, sits at the centre of the objective-versus-projective divide, and is a favourite viva vehicle for the psychometric reliability-and-validity debate. Exam answers must couple the classic clinical description (Hermann Rorschach, the 10 cards, the two administration phases) with the modern scoring standard (the Exner Comprehensive System, its successor R-PAS) and the honest caveat that its psychometric standing is contested (Kaplan & Sadock 2024).

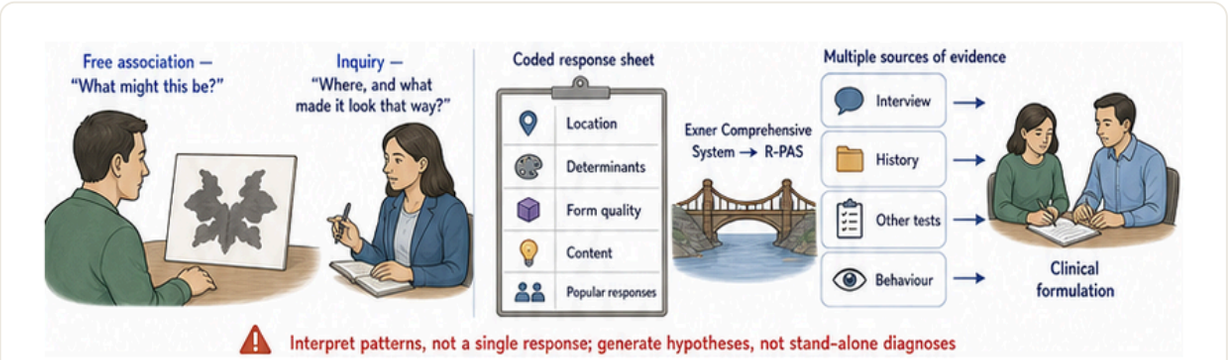


Fig 6.8 Rorschach: standardised performance, cautious inference.

Frame it as a catalogue entry: who built it and when, what construct it measures, how it is administered, how it is scored, what it is used for clinically, and where it is strong and weak. The **inkblot** is deliberately ambiguous, the projective hypothesis holding that when the stimulus is minimally structured the response is shaped by the respondent's own needs, conflicts and characteristic mode of perceiving reality (Kaplan & Sadock 2024).

4.1 Origin, authorship and what it measures

Author and date. Hermann Rorschach, a Swiss psychiatrist, published the test in his monograph *Psychodiagnostik* in 1921 (Kaplan & Sadock 2024, Tasman 2015); he died the following year, before the method matured. **Construct measured.** A projective assessment of whole-personality organisation, perceptual-associative processing and, clinically, thought process and reality testing, rather than any single narrow trait. It is a broad-band instrument: unlike an objective inventory that indexes one dimension (for example depression), the **rorschach** aims to sample personality as a whole and to expose latent, less consciously controlled aspects that the examinee cannot easily fake, because they do not know how a given response will be scored (Kaplan & Sadock 2024).

4.2 The stimulus material: describing the ten cards without reproducing them

The plates. There are 10 symmetrical **inkblot** cards, produced by dropping ink on paper and folding it so the two halves mirror each other, which is why every blot is bilaterally symmetrical. By convention the cards are labelled with Roman numerals I to X. This note describes them in words only and does not reproduce the copyrighted plates. In composition they divide into three groups.

Achromatic cards (5)

Cards I, IV, V, VI and VII are rendered in black and grey only, with no colour; several are large solid masses that commonly evoke whole-figure percepts such as a bat or a butterfly.

Black-and-red cards (2)

Cards II and III combine black-grey with prominent red detail; the red areas frequently draw colour-based and, on Card III, paired-human-figure responses.

Fully chromatic cards (3)

Cards VIII, IX and X are multicoloured; Card X in particular is broken into many separate coloured elements and tends to pull numerous discrete detail responses.

The 10 blots split 5 achromatic, 2 black-and-red, 3 fully chromatic; described in words only, plates never shown.

4.3 Administration: the free-association phase then the inquiry phase

Administration proceeds in two sequential phases, kept deliberately standardised with minimal examiner interaction so as not to contaminate the response (Kaplan & Sadock 2024).

Free-association (response) phase

Each card is handed over in turn with the open instruction, in effect "What might this be?" The examinee says whatever the blot could be; the examiner records the responses verbatim and notes any rotation of the card. This is also called the response-proper phase.

Inquiry phase

After all 10 cards are done, the examiner goes back through them and asks the examinee to point out where each percept was seen and what about the blot made it look that way. The inquiry supplies the information needed to code location and determinants; it is a clarification, not an invitation to give fresh responses.

Whole administration and scoring typically takes about 1.5 to 2 hours (Kaplan & Sadock 2024).

RULE **Free-association first, inquiry second, and the inquiry only clarifies.** The inquiry codes location and determinants; it never harvests fresh percepts.

4.4 Scoring systems: from Beck and Klopfer to the Exner Comprehensive System

The historical fragmentation. Several rival scoring-and-interpretation systems grew up around the test (K&S counts six), the best known being those of Samuel Beck and Bruno Klopfer, whose incompatible coding conventions long undermined comparability. **The modern standard.** John E. **exner** integrated the defensible elements of the earlier systems into the **exner** Comprehensive System, which became the most widely used and accepted framework and is the version examiners should name as current (Kaplan & Sadock 2024). Scoring yields a structural summary, an elaborate set of ratios and indices computed from the coded responses, and can generate dozens of variables.

4.5 The core scoring dimensions in the Exner Comprehensive System

Every response is coded along several axes. The location says which part of the blot was used, the determinants say what perceptual feature drove the percept, form quality says how well it fits the blot, content says what was seen, and popularity says how ordinary the percept is.

Location: Whole (W)

The response uses the entire blot; high W with good form can indicate integrative, organising capacity.

Location: Common detail (D)

The response uses a commonly selected, obvious sub-area of the blot.

Location: Unusual detail (Dd)

The response uses a small or rarely chosen area; a heavy load of Dd can signal an idiosyncratic, over-particular way of scanning the field.

Determinant: Form (F)

The percept is driven purely by the shape or contour of the blot; the workhorse determinant.

Determinant: Human movement (M)

The percept attributes human-like movement or action; M is linked interpretively to inner life, fantasy and ideation.

Determinant: Colour (C)

The percept is driven by the chromatic colour of the blot; colour determinants are read against form as an index of affect and its regulation.

Determinant: Shading

The percept is driven by the grey-black shading or texture of the blot; shading responses are tied interpretively to anxiety and dysphoric affect.

Form quality

How faithfully the percept matches the actual contour of the area used; good form quality reflects intact reality testing, poor form quality (distorted or arbitrary fit) is a key marker of disturbed perception.

Content

The category of what was seen, coded into classes such as human, animal, anatomy, nature and so on; the spread and any morbid or aggressive content carry interpretive weight.

Popular (P) responses

Percepts given so frequently by the normative population that they are catalogued as popular; a normal number of P responses reflects conventional, shared perception, a paucity suggests the examinee is not seeing the world as most people do.

Five coding axes of the Exner system: location, determinants, form quality, content, popularity.

4.6 R-PAS: the newer successor system

The Rorschach Performance Assessment System (R-PAS), introduced in 2011 by Meyer, Viglione, Mihura, Erard and Erdberg, is the newer successor to the **exner** Comprehensive System. It was built to retain only variables with defensible empirical support, to use internationally normed reference data, and to standardise the number of responses per card (a brief prompt for a second response, and a limit that discourages excessive responding), addressing the older systems' vulnerability to variable response productivity. For exams, cite it as the current evidence-driven refinement while the **exner** system remains the reference point most texts still teach (Kaplan & Sadock 2024).

4.7 Clinical uses and interpretation

Interpretation reads the structural summary as a whole rather than any single response. Its principal clinical yields:

Thought disorder and deviant verbalisation

Bizarre, illogical or contaminated verbalisations during the response phase (for example fusing two incompatible percepts, or reasoning arbitrarily from the blot) are read as formal thought disorder; the test has an established role in indexing disordered thinking, and in India the Rorschach Ink Blot Test is listed among instruments used to assess thought disorder (Clinical Methods in Psychiatry, AIIMS 2024).

Reality testing via form quality

Poor form quality, percepts that do not fit the contour used, is the direct operational marker of impaired reality testing.

Indices suggestive of psychosis

The Exner system carries a composite perceptual-thinking index built from form-quality and special-score variables that is used as a screen suggestive of psychotic-level disturbance; a positive index supports, but does not by itself diagnose, psychosis.

Depression and dysphoria

A depression-related index and a preponderance of shading and morbid content are read as pointing toward depressive affect.

Suicide risk

The system includes a suicide constellation, a cluster of variables interpreted as flagging heightened self-destructive risk that warrants clinical corroboration.

In all cases the finding is a hypothesis to be integrated with history, interview and other data, never a stand-alone diagnosis (Kaplan & Sadock 2024).

4.8 Strengths, limits and the psychometric controversy

Strengths. It is the most widely used and best-researched projective device, with a large interpretive literature; the standardised two-phase administration and the Exner structural summary imposed much-needed rigour; and its indirect, hard-to-fake nature can surface material that self-report inventories miss (Kaplan & Sadock 2024). **Limits and the controversy.** The central caveat is psychometric: the reliability and validity of several Rorschach variables are contested. Critics argue that many indices lack demonstrated validity and that scoring is labour-intensive and examiner-dependent; the debate has been sharp enough that some clinical-psychology programmes dropped formal Rorschach coursework, though many retain it (Kaplan & Sadock 2024). The honest exam position: a rich clinical instrument in experienced hands whose variable-by-variable psychometrics remain uneven, which is exactly what R-PAS was designed to tighten.

CLINICAL ANCHOR

Position the **rorschach** as adjunctive, not diagnostic. It is most defensibly used to generate hypotheses about thought process and reality testing (via poor form quality and deviant verbalisations) that are then cross-checked against the mental-state examination and history. A positive perceptual-thinking or suicide-constellation index prompts closer clinical evaluation; it never substitutes for it.

COMMON TRAP

Do not say the **rorschach** "diagnoses schizophrenia" or "measures IQ". It is a projective personality and thought-process measure, not a diagnostic or intelligence test. Also avoid crediting the modern scoring to Rorschach himself: the coding standard is the **exner** Comprehensive System (with R-PAS the successor), not the 1921 original. And never collapse the two phases: free-association first, inquiry second, and the inquiry only clarifies, it does not harvest new percepts.

MNEMONIC

"LoDeF-CoP" for the Exner coding axes

Location (W, D, Dd), **De**terminants (F, M, C, shading), **F**orm quality, **Co**ntent, **P**opular. Five axes: where they looked, what drove it, how well it fits, what it was, and how ordinary it is.

FEATURE	EXNER COMPREHENSIVE SYSTEM	R-PAS
Author / year	John E. Exner, 1974 onward	**Meyer and colleagues, 2011**
Design goal	Integrate rival systems into one standard	**Keep only empirically supported variables**
Response productivity	Not tightly controlled per card	**Standardised with prompts and a response limit**
Norms	Largely US-based reference data	**International reference data**
Exam status	The system most texts still teach	The current evidence-driven successor

Exner set the modern standard; R-PAS is the newer successor built to tighten its psychometrics.

HOLD THIS

Rorschach 1921, ten symmetrical inkblots, free-association then inquiry, scored on Exner's Comprehensive System (R-PAS the successor). A projective measure of thought process and reality testing, adjunctive only, with contested psychometrics.

GOOD TO REMEMBER

- **Author / year:** Hermann Rorschach, Swiss psychiatrist, *Psychodiagnostik*, 1921.
- **What it measures:** projective personality organisation, perceptual-associative process, and clinically thought process plus reality testing (broad-band, not one trait).
- **The stimulus:** 10 symmetrical inkblot cards, labelled I to X; composition = 5 achromatic (black-grey), 2 black-and-red, 3 fully chromatic. Described in words only, never reproduced.
- **Administration:** free-association (response-proper) phase first, then the inquiry phase (points out where and what made it look so); about 1.5 to 2 hours total.
- **Scoring, modern standard:** the Exner Comprehensive System, superseding the older Beck and Klopfer systems (K&S counts six historical systems).
- **Exner coding axes:** LOCATION (Whole W, common detail D, unusual detail Dd); DETERMINANTS (form F, human movement M, colour C, shading); FORM QUALITY; CONTENT categories; POPULAR responses.
- **Successor system:** R-PAS (Rorschach Performance Assessment System, Meyer et al. 2011), empirically pruned variables, international norms, controlled response count.
- **Clinical uses:** thought disorder and deviant verbalisations; reality testing via form quality; a perceptual-thinking index suggestive of psychosis; depression and suicide-constellation indices; always a hypothesis, integrated with history and interview.
- **Controversy:** contested reliability and validity of several variables; labour-intensive, examiner-dependent scoring; some programmes dropped it, many retain it.
- **Hard rule:** not a diagnostic test, not an intelligence test; adjunctive only.

5 · Projective tests: TAT, SCT, draw-a-person

Where the Rorschach uses inkblots, this family of **projective** tests hands the subject other kinds of ambiguous, unstructured material and watches what gets built from it. The governing idea is the **projective hypothesis**: when a stimulus is deliberately vague and the response space is almost unlimited, the person has nothing external to lean on, so their reply is shaped from within, and unconscious needs, conflicts and personality organisation are read off the material as if it were a screen onto which they are projected (Kaplan & Sadock 2024). Instructions are general, the scoring key is hidden, and the response is broad-band, meaning the whole personality is sampled rather than one narrow trait.

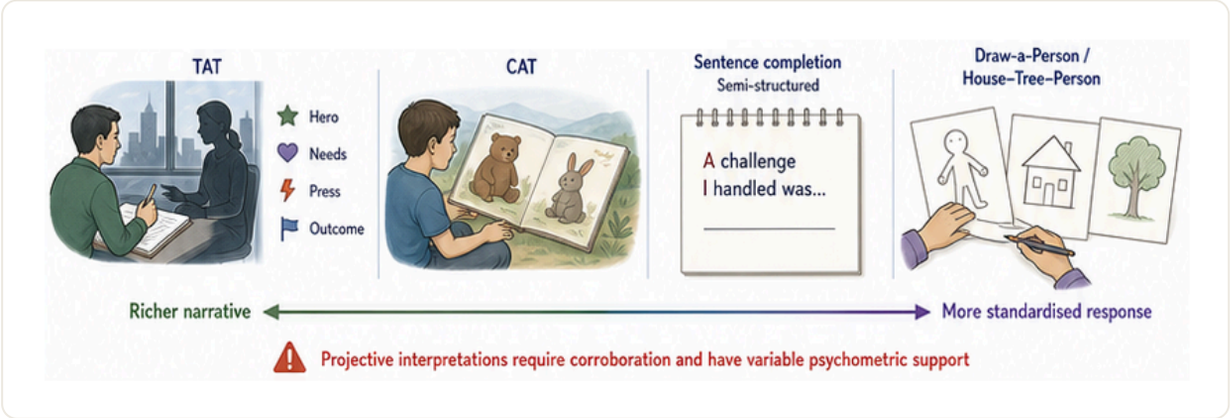


Fig 6.9 The projective family: structure changes the response.

Exam framing. Paper I loves this cluster as a compare-and-list station: name the test, its author and year, what it uses as stimulus, and what is scored. Examiners also probe the projective-versus-objective contrast and the reliability weakness. Treat each test below as a catalogue entry: stimulus, administration, scoring, use, and limits.

5.1 The projective hypothesis and what "projective" means

The core assumption. A **projective** test presents an ambiguous, unstructured stimulus for which there is no correct answer, so the subject must impose structure. That imposed structure, on the projective hypothesis, reflects fundamental aspects of personality: needs, drives, conflicts and characteristic defences (Kaplan & Sadock 2024). The term traces to psychoanalytic projection but is used descriptively here, not as a claim that projection (the defence) is the only mechanism. Because the subject does not know how responses will be scored, projective techniques are considered harder to fake than self-report inventories.

5.2 Thematic Apperception Test (TAT): stimulus and administration

What it is. The **thematic apperception** test (**tat**; Morgan and Murray, first described 1935; developed at the Harvard Psychological Clinic) is a story-telling projective. The full set is a series of ambiguous black-and-white picture cards depicting people, singly or in groups, in emotionally suggestive but unresolved situations. The classic Card 1 shows a young boy seated before a violin. Card 16 is a blank card. Cards carry age and sex codes so a tailored subset is chosen; a subject is typically shown around 20 cards over two sessions, though shorter clinical administrations of

about 10 cards are common. (HARD rule for this note: the cards themselves are copyrighted and are described in words only, never reproduced.)

The task. For each card the subject tells a story: what is happening now, what led up to it, what the figures are thinking and feeling, and how it will end. The dramatic, invented narrative is the raw data.

5.3 TAT scoring and interpretation: needs, press, the hero

Murray's framework. The **tat** is interpreted through Murray's personology. The interpreter identifies the **hero** (the figure the subject identifies with), the hero's **needs** (internal motivational forces such as need for achievement, affiliation, aggression), and the environmental **press** (forces acting on the hero from outside). The clinician tracks recurrent **themes** and **conflicts** across stories, the emotional tone, and how tensions are resolved (Kaplan & Sadock 2024). Formal quantitative systems exist (for example Bellak's scheme and the Defense Mechanisms Manual), but everyday clinical use is largely qualitative and impressionistic, which is exactly why interpreter dependence is a stated weakness.

5.4 Children's Apperception Test (CAT) and other apperception variants

The child analogue. The Children's Apperception Test (**cat**; Bellak, 1949) is the developmental analogue of the **tat** for younger children, classically ages 3 to 10. Its original version replaces human figures with anthropomorphic **animal figures** in familiar situations (feeding, toileting, sibling rivalry), on the assumption that young children project more readily onto animals; a human-figure form (CAT-H) was added later. Related child instruments include the Roberts Apperception Test for Children. The task and interpretive logic mirror the adult **tat**: tell a story, then read needs, conflicts and defences.

> **CLINICAL ANCHOR**
>
> The **tat** earns its keep when self-report is guarded or when object-relational and motivational material is the question, for example probing interpersonal conflict in a personality-disorder assessment. It is a supplement within a battery, never a standalone diagnostic test, and it does not yield a diagnosis.

5.5 Sentence completion tests (SCT): the semi-structured bridge

What it is. A **sentence completion** test (**sct**) presents a series of incomplete sentence stems ("My greatest fear...", "My father...") that the subject finishes with the first response that comes to mind. Because the stem constrains the response, the **sct** is the most direct of the projectives and is often called a **bridge between projective and objective** methods: more structured than the **tat**, less structured than a true-false inventory (Kaplan & Sadock 2024).

Named forms. The best-validated version is the **Rotter Incomplete Sentences Blank** (Rotter and Rafferty, 1950), 40 stems scored on a 7-point scale for degree of conflict or maladjustment to yield an overall adjustment index. The Sacks Sentence Completion Test (SSCT) samples defined attitude areas (family, sex, interpersonal relationships, self-concept). Stem counts vary widely across versions, from under 10 to over 75.

Strengths and limits. Quick, easy to administer and score, and flexible; but it is transparent, so it is easy to fabricate or deceive, and most versions other than Rotter's lack established reliability and validity data.

TRAP The sentence completion test is the most structured, most transparent projective, so it is the easiest to fake; only Rotter's Incomplete Sentences Blank has solid reliability and validity data.

5.6 Draw-a-Person test (Machover): projective figure drawing

What it is. The **draw-a-person** test in its projective form (Machover, 1949) asks the subject to draw a person, then to draw a person of the opposite sex. The drawing is interpreted qualitatively as a body-image projection: placement, size, line quality, omissions, distortions and the treatment of specific features are read for anxiety, conflict and self-concept. This is the interpretive descendant of Goodenough's earlier work, but Machover's use is clinical-projective rather than developmental.

Caveat. Machover-style sign interpretation of the **draw-a-person** has notoriously poor psychometric support; single-sign readings ("large eyes mean paranoia") are not defensible and it survives as a rapport-builder and hypothesis-generator, not as a validated instrument.

5.7 Goodenough-Harris Draw-a-Man and House-Tree-Person

Developmental use. The Goodenough Draw-a-Man test (Florence Goodenough, 1926), revised as the **Goodenough-Harris Drawing Test** (Harris, 1963), scores a child's human-figure drawing for the number of correct body details and proportions to yield a nonverbal estimate of intellectual maturity or mental age. Here the same act of drawing a person is used as a **developmental and screening** measure of cognitive maturity, not as a projective probe, a distinction examiners like to test.

House-Tree-Person. The House-Tree-Person test (Buck, 1948) extends the projective drawing idea: the subject draws a house, a tree and a person, each read for aspects of the self and the perceived environment, followed by an inquiry phase.

CLASSIC TRAP The same act of drawing a person is a projective probe in Machover's Draw-a-Person (body image, self-concept) but a developmental estimate of intellectual maturity in the Goodenough-Harris Draw-a-Man - same drawing, opposite purpose.

5.8 Other projective techniques worth naming

Rosenzweig Picture-Frustration Study (P-F Study; Rosenzweig, 1945)

Cartoon strips show one figure frustrating another; the subject supplies the frustrated figure's reply. Scored for direction of aggression (extrapunitive, intropunitive, impunitive) and type of reaction, giving a semi-projective measure of reaction to frustration.

Word-association test

The examiner reads a standard list of stimulus words; the subject gives the first word that comes to mind. Reaction-time delays, blocks and unusual associations flag emotionally charged complexes. Associated with Jung's complex work and the Kent-Rosanoff standardised list.

Object-sorting test (Goldstein-Scheerer)

The subject sorts objects or blocks into groups and explains the principle; concrete, idiosyncratic or overinclusive sorting is used to demonstrate concrete thinking and conceptual (formal thought) disturbance, classically in schizophrenia.

5.9 Projective versus objective tests: the trade-off

The contrast. Objective personality tests (self-report inventories such as the MMPI) use structured items with fixed scoring; **projective** tests use ambiguous stimuli with open scoring. Each buys something at a cost.

DIMENSION	PROJECTIVE TESTS	OBJECTIVE TESTS
Stimulus	Ambiguous, unstructured	Structured, fixed items
Data	**Rich, idiographic, whole-personality**	Narrow, quantified traits
Fakeability	**Harder to fake (scoring hidden)**	Easier to fake or fake-good
Reliability and validity	**Often low; interpreter-dependent**	Generally higher, normed
Scoring	Subjective, needs trained interpreter	Objective, can be automated

Projectives trade psychometric rigour for depth and resistance to faking; objective tests trade richness for reliability.

HOLD THIS

The projective family runs from least to most structured: TAT tells tales, SCT stops at a stem, DAP draws the self. All are broad-band, hard to fake, and low in reliability; all generate hypotheses, none names a diagnosis.

COMMON TRAP

Do not call any projective a "diagnostic test". None of them names a diagnosis; they generate hypotheses about personality and conflict that must be integrated with history and the rest of a battery. Equally, do not conflate the projective **draw-a-person** (Machover) with the developmental Goodenough-Harris Draw-a-Man: same drawing, opposite purpose (personality projection versus cognitive maturity).

MNEMONIC

TAT tells Tales; SCT Stops Short; DAP Draws the self

The **tat** elicits full narrative **tales** (least structured, richest), the **sct** stops the subject at a sentence **stem** (semi-structured bridge), and the **draw-a-person** reads the **self** off a figure drawing. Order of increasing structure: TAT to SCT.

INDIA

Indian residency texts (AIIMS Clinical Methods in Psychiatry 2024) list the Rorschach, **tat**, the Draw-a-Person test (DAPT) and the **sentence completion** test (often the Sacks form, SSCT) as the standard projective battery, and note that formal projective testing is done by a clinical psychologist, not at the bedside by the psychiatrist.

GOOD TO REMEMBER

- **Projective hypothesis:** ambiguous unstructured stimuli let the subject project unconscious needs, conflicts and personality onto the material.
- **Thematic Apperception Test (TAT):** Morgan and Murray, 1935 (Harvard) / measures needs, press, motives and interpersonal conflict via story-telling / scored by identifying the hero, needs, press, recurrent themes and conflicts (Murray's personology).
- **Children's Apperception Test (CAT):** Bellak, 1949 / child analogue of the TAT (ages ~3 to 10) / same needs-and-conflict interpretation, uses animal figures (CAT-A; human form CAT-H).
- **Sentence Completion Test (SCT):** semi-structured stems, a bridge between projective and objective / measures attitudes, conflicts and adjustment / Rotter Incomplete Sentences Blank (Rotter and Rafferty 1950), 40 stems, 7-point conflict scoring, overall adjustment index; Sacks SSCT samples attitude areas.
- **Draw-a-Person (Machover, 1949):** projective figure drawing / measures body image, self-concept, anxiety and conflict / qualitative sign interpretation of the drawn figure (weak psychometrics).
- **Goodenough-Harris Draw-a-Man (Goodenough 1926; Harris 1963):** developmental and screening measure / estimates intellectual maturity / scored by counting correct body details and proportions for a mental-age estimate.
- **House-Tree-Person (Buck, 1948):** projective drawing of house, tree and person / self and perceived environment / qualitative interpretation plus an inquiry phase.
- **Rosenzweig Picture-Frustration Study (Rosenzweig, 1945):** cartoon frustration scenes / reaction to frustration / scored for direction of aggression (extrapunitive, intropunitive, impunitive) and reaction type.
- **Word-association test (Jung; Kent-Rosanoff list):** respond with first word to a stimulus word / detects emotionally charged complexes / scored on reaction time, blocks and atypical associations.
- **Object-sorting test (Goldstein-Scheerer):** sort and explain groupings / conceptual (formal-thought) disturbance / concrete or overinclusive sorting flags concrete thinking, classically in schizophrenia.
- **Projective vs objective:** projectives are rich and harder to fake but low in reliability and interpreter-dependent; objective inventories are reliable and normed but narrow and easier to fake.

6 · Objective personality tests: MMPI, 16PF, NEO

An **objective personality** test is a structured self-report inventory built from a fixed set of items (usually true or false, or a Likert scale), scored by a standardised key against published norms, so that the same protocol yields the same profile in any hands. This is the defining contrast with projective testing: the stimulus is unambiguous, the response format is closed, and the scoring is mechanical, not interpretive. The tests taught here **operationalise** the trait theories of personality you met in the Social, Learning & Personality Psychology notes, translating abstract dimensions (Cattell's source factors, Eysenck's superfactors, the Big Five) into measurable scale scores.

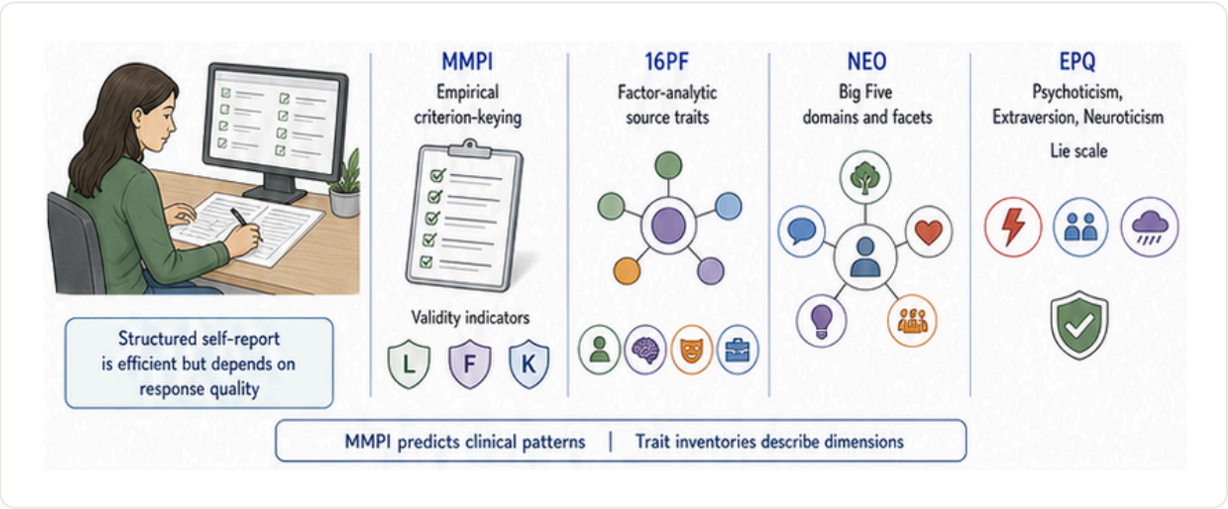


Fig 6.10 Objective personality tests: different construction logics.

For the exam, master three things per test: **who built it and on what logic** (empirical criterion-keying versus factor analysis), **what construct it measures**, and **how it detects faking**. The MMPI validity scales are the classic answer to the central weakness of self-report, that respondents can distort. Kaplan and Sadock (2024) and the Companion to Psychiatric Studies remain the depth bar.

6.1 What makes a test "objective"

Fixed stimulus, fixed scoring. Objective tests are typically pencil-and-paper (or digital) instruments made of specific items that yield numerical scores and profiles amenable to statistical analysis (Kaplan & Sadock 2024). Because administration and scoring are standardised, inter-rater variance is near zero: two clinicians scoring the same answer sheet get the same T-scores. Reliability and validity are formally established against a normative base, and it is the size and representativeness of those norms that largely determine a test's usefulness. This is the whole appeal, and the whole vulnerability, of the class: rigour bought at the price of relying on what the person is willing to report.

6.2 The MMPI: empirical criterion-keying

Origin and logic. The **mmpi** (Minnesota Multiphasic Personality Inventory) was developed by Starke R. Hathaway and J. Charnley McKinley at the University of Minnesota, the original version published in 1943 (Hathaway & McKinley 1943; Kaplan & Sadock 2024). Its innovation was **empirical criterion-keying**: items were retained on a clinical scale not for face content but because they were answered differently by a criterion group (say, diagnosed depressives) than by normal controls. The test is deliberately descriptive and atheoretical, not derived from psychoanalytic theory. It is a true-or-false questionnaire for persons aged 16 and over, coded into multiple scales. A scoring innovation was the T-score, a standardised metric with mean set at 50 and standard deviation at 10, which let scores be compared across scales; a T-score at or above 65 (MMPI-2) is the usual clinical cut-off.

6.3 MMPI validity scales: detecting response style

The faking detectors. The MMPI's signature contribution is a set of **validity scales** that gauge how the person approached the test before any clinical scale is trusted. The three classical ones:

L (Lie scale)

Items describing minor human faults most people admit; high L means the person claims implausible virtue ("naive" or unsophisticated faking-good).

F (Infrequency)

Items endorsed by fewer than 10 percent of the normative sample; high F suggests over-reporting, random responding, severe pathology, or "faking bad".

K (Correction)

A subtler defensiveness index; detects a guarded, image-managing test-taking set, and a fraction of K is statistically added back to several clinical scales to correct for under-report.

Later forms add F-family scales (Fb, Fp) and consistency indices (VRIN, TRIN). The L, F, K configuration ("validity triangle") is read first: an invalid protocol voids clinical interpretation.

RULE **Read L, F, K before any clinical scale.** An invalid protocol (implausible virtue, over-reporting or defensiveness) voids the whole profile, so the validity triangle comes first.

6.4 MMPI clinical scales and code types

Ten basic clinical scales. The original MMPI carries ten numbered clinical scales, each empirically keyed to a criterion group. Scales are read by number and by two-point **code type** (the two highest scales), for example a 2-7 code (depression plus psychasthenia) suggesting anxious-depressive presentation.

NO.	SCALE	WHAT HIGH SCORES FLAG
1	Hs Hypochondriasis	Bodily preoccupation, somatic complaints
2	D Depression	Dysphoria, pessimism, low morale
3	Hy Hysteria	Conversion tendency, denial, stress-related somatics
4	Pd Psychopathic deviate	Rebelliousness, antisocial traits, poor authority relations
5	Mf Masculinity-femininity	Interest patterns departing from gender-role norms
6	Pa Paranoia	Suspiciousness, sensitivity, ideas of reference
7	Pt Psychasthenia	Anxiety, obsessions, doubt, tension
8	Sc Schizophrenia	Bizarre ideation, alienation, unusual experience
9	Ma Hypomania	Elevated energy, expansive-ness, overactivity
0	Si Social introversion	Withdrawal, shyness, low sociability

The ten clinical scales in order; scales 5 and 0 are non-clinical dimensions, the other eight index symptom domains.

6.5 MMPI-2, the RF and MMPI-3

The modern versions. The MMPI-2 (1989) re-standardised the instrument on a large contemporary US normative sample and added content and supplementary scales while keeping the ten clinical scales for continuity. The MMPI-2-RF (Restructured Form) is a shorter psychometric revision built around Restructured Clinical (RC) scales that strip out a shared "demoralisation" factor. The MMPI-3, the newest edition, brings new norms and updated items (Kaplan & Sadock 2024). All remain heavily used in clinical, forensic, and employment-screening contexts, and the validity indicators carry into every version.

6.6 The 16PF: Cattell's source factors

Factor-analytic architecture. The **16pf** (Sixteen Personality Factor Questionnaire) was devised by Raymond B. Cattell (1905 to 1998) in 1949, on a factor-analytic logic that contrasts sharply with the MMPI's criterion-keying (Kaplan & Sadock 2024). Starting from the **lexical hypothesis** (that every trait important in social life becomes encoded in language), Cattell reduced Allport and Odbert's roughly 4,000 trait terms to 35 variable clusters, factored them, and derived sixteen primary **source factors** (for example warmth, reasoning, emotional stability, dominance, vigilance, apprehension, tension). These sixteen primaries themselves load onto five second-order "global" factors that broadly anticipate the Big Five. The 16PF measures normal-range personality rather than psychopathology, so it is used in occupational, counselling, and research settings more than in psychiatric diagnosis.

6.7 The NEO-PI-R: operationalising the Big Five

The five-factor instrument. The **neo-pi** (NEO Personality Inventory, current research forms the NEO-PI-R and NEO-PI-3) was built by Paul T. Costa Jr and Robert R. McCrae as the operationalisation of the **five-factor model** (FFM), the Big Five (Costa & McCrae; Kaplan & Sadock 2024). The five domains carry the mnemonic OCEAN: Openness to experience, Conscientiousness, Extraversion, Agreeableness, Neuroticism. Crucially the neo pi is hierarchical: each of the five domains is measured by six specific **facet** scales (thirty facets in total), so Neuroticism decomposes into anxiety, angry hostility, depression, self-consciousness, impulsiveness, and vulnerability. This facet-level resolution gives a more detailed personality portrait than a domain score alone and is why the FFM and the neo pi now dominate trait research and inform dimensional models of personality pathology.

> **CLINICAL ANCHOR**
>
> DSM-5's alternative model of personality disorders (Section III) maps disorders onto FFM-derived trait domains, and the PID-5 is its self-report instrument. When an examiner asks how modern classification links to trait psychology, the neo pi and the Big Five are the bridge: categories reframed as extreme standings on continuous dimensions.

6.8 The Eysenck Personality Questionnaire: the PEN model

Three superfactors. Hans Eysenck argued that Cattell's sixteen factors were needlessly complex and non-replicable, and reduced personality to a small number of higher-order dimensions. The

eysenck personality questionnaire (**epq**), developed by Hans and Sybil Eysenck (Eysenck & Eysenck 1975), measures the **PEN model**: Psychoticism, Extraversion, and Neuroticism, with a Lie scale for response validity. Extraversion and Neuroticism were his original two superfactors (from the earlier EPI); Psychoticism (tough-mindedness, hostility, non-conformity) was added later. Eysenck grounded these biologically, tying introversion to higher cortical arousal and easier conditioning. The epq is a brief, dimensional, heavily biologically-anchored instrument, and its E and N reappear inside the Big Five.

6.9 Criterion-keyed versus factor-analytic construction

Two ways to build a personality test. The exam loves this contrast, because it explains why the MMPI looks and behaves so differently from the 16PF and neo pi.

FEATURE	EMPIRICAL CRITERION-KEYING (MMPI)	FACTOR-ANALYTIC (16PF, NEO, EPQ)
Item selection	Retained if it discriminates a criterion group	**Retained if it loads on a statistical factor**
Guiding aim	Predict clinical group membership	**Describe the structure of normal traits**
Face validity	Often low (item content may seem unrelated)	**Usually high (items obviously tap the trait)**
Typical use	Psychopathology, forensic, screening	**Normal-range description, research, occupational**

The MMPI predicts; the trait inventories describe. Different logic, different clinical niche.

HOLD THIS

The MMPI was built by empirical criterion-keying (it predicts group membership); the 16PF, NEO and EPQ were built by factor analysis (they describe trait structure). The MMPI's validity scales are the answer to the fakeability of all self-report.

6.10 Objective versus projective assessment

Strengths and the fakeability problem. Against projective techniques (Rorschach, TAT), objective tests offer clear advantages: standardised administration, established reliability and validity, representative norms, quantified scores, and objectivity of scoring. Their central weakness is transparency: because items are structured and often obvious, respondents can consciously distort ("fake good" for custody or employment, "fake bad" for compensation or disability). This is precisely the gap the MMPI validity scales close, and why any credible objective inventory now carries validity or consistency indices. Projective tests trade the norms and objectivity away in exchange for being harder to fake and richer on unconscious material, a trade-off the personality-theory discussion in the Social, Learning & Personality Psychology notes frames.

COMMON TRAP

Do not call the 16PF or NEO-PI-R "projective" because they concern personality: they are firmly objective (structured, keyed, normed). Projective means an ambiguous stimulus (inkblot, picture) with an open response. Also do not confuse the MMPI's empirical criterion-keying with factor analysis: the MMPI was NOT built by factor analysis, the 16PF, NEO and EPQ were.

MNEMONIC

"Lie, Frequency, correction" then "OCEAN" and "PEN"

MMPI validity trio in order = **L, F, K**. Big Five (neo pi) domains = **OCEAN** (Openness, Conscientiousness, Extraversion, Agreeableness, Neuroticism). Eysenck (epq) = **PEN** (Psychoticism, Extraversion, Neuroticism).

PEARL

Everything reduces to a family tree of extraversion and neuroticism. Eysenck's E and N (epq) are two of Cattell's second-order factors and two of the Big Five domains; the neo pi simply adds Openness, Agreeableness and Conscientiousness. If asked "what unifies the trait inventories", the answer is that they are competing resolutions of the same underlying factor space.

GOOD TO REMEMBER

- **Objective personality test:** structured self-report, fixed items, standardised key, norm-referenced scoring; contrast with projective (ambiguous stimulus, open response).
- **MMPI / MMPI-2 / MMPI-3:** Hathaway and McKinley (1943); measures psychopathology and response style; empirical criterion-keying; scored as T-scores (mean 50, SD 10), clinical cut-off T at or above 65.
- **MMPI validity scales:** L (lie, faking-good), F (infrequency, over-reporting/faking-bad), K (correction, defensiveness); read before clinical scales.
- **MMPI ten clinical scales:** 1 Hypochondriasis, 2 Depression, 3 Hysteria, 4 Psychopathic deviate, 5 Masculinity-femininity, 6 Paranoia, 7 Psychasthenia, 8 Schizophrenia, 9 Hypomania, 0 Social introversion; interpreted by two-point code types.
- **16PF:** Raymond Cattell (1949); measures sixteen normal-range source factors derived by factor analysis (lexical hypothesis); occupational and research use.
- **NEO-PI-R / NEO-PI-3:** Costa and McCrae; operationalises the five-factor model (OCEAN); scored as five domains, each with six facets (thirty facets total).
- **Eysenck Personality Questionnaire (EPQ):** Hans and Sybil Eysenck (1975); measures the PEN dimensions (Psychoticism, Extraversion, Neuroticism) plus a Lie scale; biologically anchored.
- **Construction logic:** MMPI = empirical criterion-keying (predicts group membership, low face validity); 16PF, NEO, EPQ = factor-analytic (describe trait structure, high face validity).
- **Fakeability:** the core limit of objective tests; addressed by validity/consistency scales (the MMPI L, F, K being the exemplar).

7 · Neuropsychological testing

This is where psychometry meets the brain. A **neuropsychological** test is a standardised task whose performance is known to depend on a particular brain structure or pathway, so that a deficit on the task can be read as a statement about the integrity of that system. The discipline

studies **brain-behaviour relationships**, and a neuropsychological assessment can be thought of as an elaboration and refinement of the cognitive component of the mental status and neurological examination (Kaplan & Sadock 2024). No single instrument covers the brain; instead a battery samples five cognitive domains: **attention**, **memory**, **language**, **visuospatial function** and **executive function**.

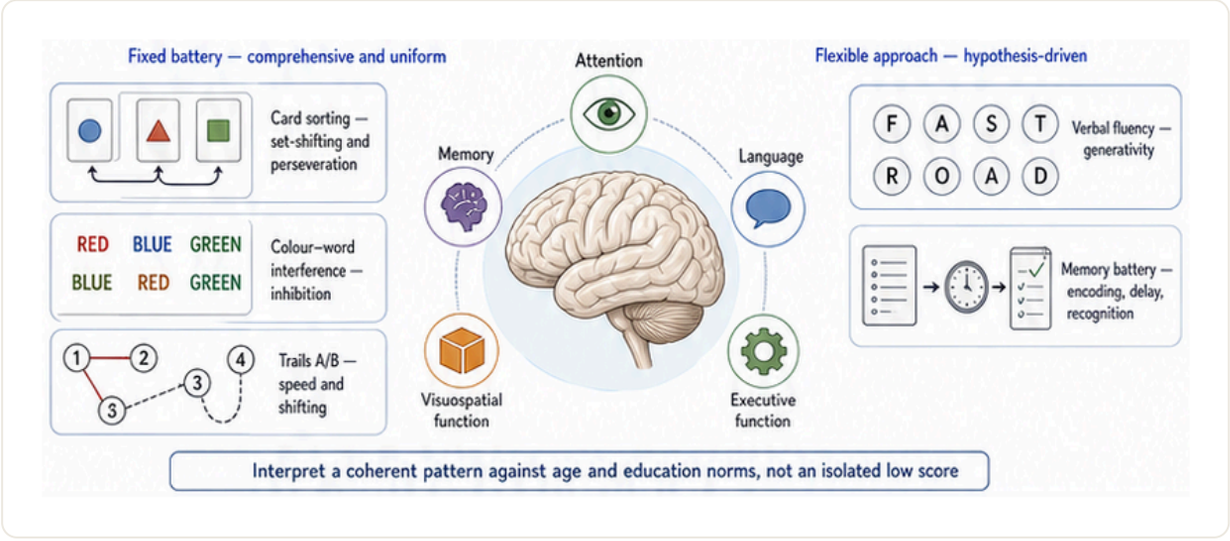


Fig 6.11 Neuropsychological assessment: test the pattern.

For the examiner this topic sits at the junction of psychometry and organic psychiatry, so it is examined twice: once as a catalogue of named batteries and tests (who built them, what construct each measures, how each is scored), and once as a clinical tool (dementia work-up, traumatic brain injury, lesion localisation, capacity and rehabilitation). Two organising axes run through the whole topic: the *fixed battery versus flexible hypothesis-driven* distinction in approach, and the frontal/executive tests, which carry the most marks because they are the hardest deficits to catch at the bedside.

7.1 The two approaches: fixed batteries versus the flexible, hypothesis-driven method

Standard battery or tailored investigation. Neuropsychological assessment is organised along two philosophies (Kaplan & Sadock 2024). The *fixed (standard) battery* approach administers the same comprehensive, pre-set set of tests to every patient regardless of the referral question; its strength is a uniform, quantitative, normed profile that will not miss an unexpected deficit, and its weakness is length and inefficiency. The **halstead-reitan** battery and the **luria**-Nebraska battery are the two classic fixed batteries. The *flexible, hypothesis-driven* (or process) approach, historically exemplified by **luria** and formalised as the Boston Process Approach by Edith Kaplan, selects and sequences tests to test a specific hypothesis about the patient's complaint, honing in efficiently on the suspected area of impairment and describing the *quality* of errors, not just the score; its shortcoming is that it may overlook an unexpected deficit outside the hypothesis (Kaplan & Sadock 2024). Most modern clinical neuropsychology is flexible-hypothesis-driven, drawing tests from many batteries.

RULE **Diagnose impairment on a pattern consistent with a known condition, never on isolated low scores.** A long battery yields some low scores even in healthy people, read against age and education norms.

7.2 The Halstead-Reitan battery: a fixed battery for organicity

Category Test, Tactual Performance Test, Impairment Index. The **halstead-reitan** Neuropsychological Test Battery (HRNTB) is the prototype fixed battery, assembled by Ward Halstead and extended by Ralph Reitan, with versions for adults and children (Kaplan & Sadock 2024). It is a compilation of tests designed to detect and localise brain damage and to lateralise it. Three components are named repeatedly:

Category Test

A test of abstraction and concept formation: the patient works out an organising principle for sets of stimuli using the examiner's right/wrong feedback. It is regarded as one of the most sensitive single measures of general brain dysfunction in the battery, and, like the Wisconsin, it taps the ability to benefit from feedback and shift strategy (Kaplan & Sadock 2024).

Tactual Performance Test (TPT)

A blindfolded test of visuospatial and tactile-kinaesthetic function: the patient places blocks into a form-board by touch alone, then draws the board from memory. It yields measures of tactile problem-solving, and of incidental memory and localisation (Kaplan & Sadock 2024).

Halstead Impairment Index

The summary score of the battery: the proportion of the core tests on which the patient falls in the brain-damaged range, running from 0.0 (none impaired) to 1.0 (all impaired). A higher index means more pervasive dysfunction; it is the battery's single global organicity figure.

Other HRNTB components (finger tapping, grip strength, the rhythm and speech-sounds perception tests, and double simultaneous stimulation) provide the lateralising and sensory-motor data.

7.3 The Luria-Nebraska Neuropsychological Battery: qualitative Luria, quantified

A fixed battery built on Luria's qualitative approach. The **luria**-Nebraska Neuropsychological Battery (LNNB), developed by Golden and colleagues, took the qualitative, syndrome-analytic bedside method of the Russian neuropsychologist A. R. **luria** and repackaged it as a standardised, quantitatively scored fixed battery (Kaplan & Sadock 2024). It is organised into clinical scales spanning motor, rhythm, tactile, visual, receptive speech, expressive speech, writing, reading, arithmetic, memory and intellectual processes, each generating a score referenced to norms; it thus tries to preserve **luria**'s functional-systems logic while giving the actuarial, normed output that the pure qualitative method lacks. A Children's Revision exists for younger patients.

7.4 Frontal and executive testing: the highest-yield cluster

Why the frontal lobe needs special tests. Executive functions (planning, initiation, self-monitoring, inhibition of inappropriate responses, set-shifting, and control of complex problem-solving) are the most vulnerable to frontal and frontal-subcortical damage and the least reliably caught by a routine mental status examination, so a distinct set of tests targets them (Kaplan &

Sadock 2024). A patient can score normally on the MMSE and intelligence testing yet be grossly dysexecutive. The examiner expects the following tests by name and by the specific process each isolates.

7.5 The Wisconsin Card Sorting Test: set-shifting and perseveration

The signature test of frontal set-shifting. The **wisconsin card** Sorting Test (WCST) is the classic measure of set-shifting and cognitive flexibility, functions compromised by frontal lobe damage (Companion to Psychiatric Studies). In the standard version four target cards (one red triangle, two green stars, three yellow crosses, four blue circles) are laid out, and the patient sorts a deck of 128 response cards under them by an unstated rule (colour, form or number), learning the rule only from the examiner's right/wrong feedback. After ten consecutive correct sorts the rule is silently changed and the patient must shift to the new principle (Companion to Psychiatric Studies). Performance is scored as the number of categories achieved and, critically, the percentage of *perseverative errors*, the hallmark of frontal dysfunction, where the patient keeps applying the old rule after it has stopped working. Nelson's modified WCST uses 48 rather than 128 cards to reduce the distress of continuous negative feedback. Schizophrenia patients perform poorly, with reduced dorsolateral prefrontal activation on imaging during the task (Tasman 2015).

HOLD THIS

The Wisconsin Card Sorting Test is the signature test of set-shifting, and perseverative errors are its frontal marker. It is not a memory test - the patient keeps applying a rule after it has stopped working.

7.6 The Stroop test: response inhibition and interference

Naming the ink, not the word. The **stroop** test measures response inhibition and the ability to suppress a prepotent, automatic response, an executive function anchored in frontal control (Companion to Psychiatric Studies). The patient must name the ink colour of a colour word while the word itself spells a different colour (for example the word printed in green ink); reading the word is automatic and must be inhibited to name the ink, so the *interference* condition is slower and more error-prone than neutral naming. The size of this **stroop** interference effect indexes the integrity of inhibitory control. The paradigm generalises: the *emotional Stroop*, in which emotionally salient words slow colour-naming, is widely used in anxiety research.

7.7 The Trail Making Test: attention and set-shifting in two parts

Part A speed, Part B shifting. The **trail making** test requires rapid and efficient integration of attention, visual scanning and cognitive sequencing (Kaplan & Sadock 2024). It has two parts. *Part A* asks the patient to connect numbered circles (1-2-3...) in order as fast as possible, indexing visual attention, scanning and psychomotor speed. *Part B* requires alternating between numbers and letters (1-A-2-B-3-C...), adding a set-shifting and working-memory load to the same visuomotor task; the B-minus-A difference isolates the executive shifting cost from simple speed. A markedly prolonged or failed Part B is a sensitive, quick sign of executive impairment.

7.8 Verbal fluency and the Frontal Assessment Battery

Generativity and a bedside frontal screen. *Verbal fluency* tests measure the capacity to self-generate words to a rule against the clock, a frontally mediated generative function (Companion to Psychiatric Studies). *Phonemic (letter) fluency*, also called the Controlled Oral Word Association Test (COWAT), asks for as many words as possible beginning with F, A and S, sixty seconds per letter; *semantic (category) fluency* asks for exemplars of a category such as animals. Impaired letter fluency follows left or bilateral frontal damage. The *Frontal Assessment Battery* (FAB), the broader bedside frontal lobe test battery, is a short six-item screen (conceptualisation and similarities, lexical/verbal fluency, motor programming via the Luria hand sequence, conflicting instructions, a go/no-go for inhibitory control, and prehension behaviour) scored out of a maximum of 18, with lower scores indicating greater frontal dysfunction; it is sensitive to frontal lobe damage and useful where a full battery is impractical (Clinical Methods in Psychiatry AIIMS 2024).

EXECUTIVE TEST	CORE PROCESS ISOLATED	KEY SCORE
Wisconsin Card Sorting Test	**Set-shifting; perseveration**	Categories achieved; % perseverative errors
Stroop test	**Response inhibition of a prepotent response**	Interference effect (time/errors)
Trail Making Test A and B	**Attention/speed (A); set-shifting (B)**	Time to complete; B minus A
Verbal fluency (COWAT)	**Generativity/self-activation**	Words in 60 seconds (F, A, S; or a category)
Frontal Assessment Battery	**Global frontal/executive screen**	Total out of 18

The frontal/executive tests, each mapped to the one process it best isolates and its scoring currency.

128	18	0.0-1.0
WCST RESPONSE CARDS (NELSON: 48)	FAB MAXIMUM (LOWER IS WORSE)	HALSTEAD IMPAIRMENT INDEX (HIGHER IS WORSE)

MNEMONIC

"SWIFT" frontal tests: Stroop, Wisconsin, Interference, Fluency, Trails.

Five executive tests to name: **S**troop (inhibition), **W**isconsin card sort (set-shifting/perseveration), **I**nterference is what both Stroop and the emotional Stroop measure, **F**luency (verbal/COWAT, plus the FAB), and **T**rails A and B. If asked for one bedside frontal test, say FAB (out of 18); if asked for the signature perseveration test, say Wisconsin.

7.9 The Bender Gestalt test: visuoconstruction and an organicity screen

Copying nine designs. The **bender** Gestalt test (the Bender Visual-Motor Gestalt Test, Lauretta Bender) asks the patient to copy a set of nine geometric designs, sampling visuoconstruction and visuomotor integration (Clinical Methods in Psychiatry AIIMS 2024). Distortions, rotations, perseveration and integration failures in the reproductions are read as soft signs of organic (especially parietal and constructional) impairment, so historically it served as a quick *organicity screen*; it is also used as a low-stress opening task to build rapport before more demanding testing.

A recall administration (drawing the designs from memory) adds a visual-memory element. In modern practice it is a screen and a projective adjunct, not a stand-alone diagnostic instrument.

7.10 Memory testing: the Wechsler Memory Scale

The reference memory battery. The **neuropsychological** assessment of memory is anchored by the Wechsler Memory Scale (WMS), a comprehensive set of subtests measuring attention, encoding, retrieval and recognition of both verbal and visual material, with immediate recall and delayed retention and excellent age-stratified norms up to about age 90 (Kaplan & Sadock 2024). It was co-normed with the Wechsler Adult Intelligence Scale so that memory can be compared directly against intellectual level, which is how a disproportionate memory deficit (as in an amnestic syndrome or early dementia) is demonstrated. Complementary list-learning tests (the California Verbal Learning Test, the Rey Auditory Verbal Learning Test, the Hopkins Verbal Learning Test) dissect encoding versus retrieval, and the Rey-Osterrieth Complex Figure tests non-verbal (visual) memory and construction.

7.11 Clinical uses and Indian batteries

What the referral actually asks. Neuropsychological testing is requested for five broad purposes (Kaplan & Sadock 2024; Clinical Methods in Psychiatry AIIMS 2024): the *dementia work-up* (detecting early impairment, profiling the deficit, and distinguishing dementia from pseudodementia and from normal ageing); *traumatic brain injury* (documenting deficits, guiding return-to-work and medicolegal opinion); *lesion localisation* (a profile of preserved and impaired functions points to frontal, temporal, parietal or lateralised pathology, complementing imaging); *capacity assessment* (financial, testamentary and treatment-decision capacity in the elderly and the brain-injured); and *rehabilitation* (baseline profiling and serial re-testing to track recovery or decline, which is why parallel-form and repeatable batteries exist). A key cautionary principle: because a long battery yields many scores, some low scores occur even in healthy people, so a diagnosis of impairment rests on a *pattern* consistent with a known condition, interpreted against age and education norms, not on isolated abnormal scores (Kaplan & Sadock 2024).

INDIA

Western neuropsychological tests must be adapted and re-normed before use here, and three indigenous batteries are examinable. The *PGI Battery of Brain Dysfunction* (PGI-BBD; Pershad and Verma 1990) gives a global organicity score and comprises five subscales: the PGI Memory Scale, the Bhatia Short Battery, a Verbal Adult Intelligence Scale, the Nahor-Benson test of perceptual acuity, and the Bender Visual Motor Gestalt Test. The *NIMHANS Neuropsychology Battery* (Rao, Subbakrishna and Gopukumar 2004) is a comprehensive, domain-wise Indian battery. The *AIIMS Comprehensive Neuropsychological Battery in Hindi* (Gupta et al 2000) applies **luria**-Nebraska principles with ten basic scales plus secondary scales. The *PGI Memory Scale* is the workhorse memory instrument in Indian practice.

CLINICAL ANCHOR

A 68-year-old man referred for capacity after a stroke might show, on a flexible battery: MMSE 23/30, FAB 9/18, Trail Making Test B unable to complete, and perseverative errors on the Wisconsin Card Sorting Test (a pattern drawn from a real medicolegal report structure). Read together this is a dysexecutive, frontally weighted profile with preserved gross orientation, exactly the picture that supports impaired financial capacity while a bare MMSE would have under-called it. This is why executive tests are added when capacity is the question.

COMMON TRAP

Do not equate the Halstead *Impairment* Index (0.0 to 1.0, proportion of core tests in the brain-damaged range) with a fluency or FAB *score*, and do not call the Wisconsin a memory test: it is set-shifting. Also remember the direction of scoring differs: on the FAB a *lower* score is worse, whereas a *higher* Halstead Impairment Index is worse. And screening tests (MMSE, Bender Gestalt) are not interchangeable with a full neuropsychological battery; they under-call deficits in the educated and over-call them in the poorly schooled.

GOOD TO REMEMBER

- **Two approaches:** fixed (standard) battery (uniform, comprehensive, cannot miss the unexpected but long) versus flexible hypothesis-driven/process approach (Luria, Boston Process Approach of Edith Kaplan; efficient but can miss the unexpected).
- **Halstead-Reitan battery (Halstead 1947; Reitan):** measures presence, severity and lateralisation of brain damage; scored via the *Halstead Impairment Index* (0.0 to 1.0, proportion of core tests in the impaired range). Named components: Category Test, Tactual Performance Test.
- **Category Test:** abstraction/concept formation from feedback; one of the most sensitive single measures of general brain dysfunction in the Halstead-Reitan battery.
- **Tactual Performance Test:** blindfolded form-board placement by touch; scores tactile problem-solving plus incidental memory and localisation.
- **Luria-Nebraska Neuropsychological Battery (Golden, from A. R. Luria):** measures brain dysfunction across motor, tactile, rhythm, speech, reading, writing, arithmetic, memory and intellectual clinical scales; standardised quantitative scores on each scale.
- **Wisconsin Card Sorting Test:** measures set-shifting and cognitive flexibility; 128 cards sorted to an unstated shifting rule; scored as categories achieved and percentage of *perseverative errors* (frontal marker). Nelson modification uses 48 cards.
- **Stroop test (Stroop 1935):** measures response inhibition of a prepotent response; scored as the interference effect (extra time/errors when naming ink colour of an incongruent colour word).
- **Trail Making Test A and B:** measures attention, scanning and psychomotor speed (A) and set-shifting/working memory (B); scored as time to complete, with B minus A isolating the executive shifting cost.
- **Verbal fluency (COWAT):** measures generativity/self-activation; scored as number of words in 60 seconds to letters F, A, S (phonemic) or to a category such as animals (semantic).
- **Frontal Assessment Battery (FAB):** measures global frontal/executive function at the bedside; six subtests; scored out of a maximum of 18 (lower is worse).
- **Bender Gestalt test (Lauretta Bender):** measures visuoconstruction/visuomotor integration; scored by distortions, rotations, integration failures in copying nine designs; classic organicity screen and rapport-builder.
- **Wechsler Memory Scale (WMS):** measures attention, encoding, retrieval and recognition of verbal and visual material, immediate and delayed; age-stratified index scores, co-normed with the WAIS for direct comparison against IQ.
- **Five domains sampled:** attention, memory, language, visuospatial function, executive function.
- **Clinical uses:** dementia work-up, traumatic brain injury, lesion localisation, capacity assessment, rehabilitation (with serial/parallel-form re-testing).
- **Indian batteries:** PGI Battery of Brain Dysfunction (Pershad and Verma 1990; global organicity score, five subscales); NIMHANS Neuropsychology Battery (Rao et al 2004); AIIMS Comprehensive Neuropsychological Battery in Hindi (Gupta et al 2000; Luria-Nebraska principles).
- **Interpretation rule:** diagnose impairment on a *pattern* consistent with a known condition against age/education norms, never on isolated low scores.

8 · Adaptive behaviour and developmental scales

Intelligence testing tells only half the story of a child's functioning. The other half is **adaptive behaviour**: the conceptual, social and practical skills a person actually deploys in everyday life. Since DSM-5, adaptive functioning sits alongside measured IQ as a co-equal, indeed *defining*, axis of intellectual disability, and severity is graded by the adaptive domains rather than by an IQ band alone (Kaplan & Sadock 2024). A parallel family of **developmental scales** extends the same

logic downward to infants and preschoolers who are too young for a formal IQ test: they yield a developmental quotient by comparing observed milestones against age norms. This topic is a high-yield catalogue for Paper I: examiners want author, construct, and scoring for each instrument.

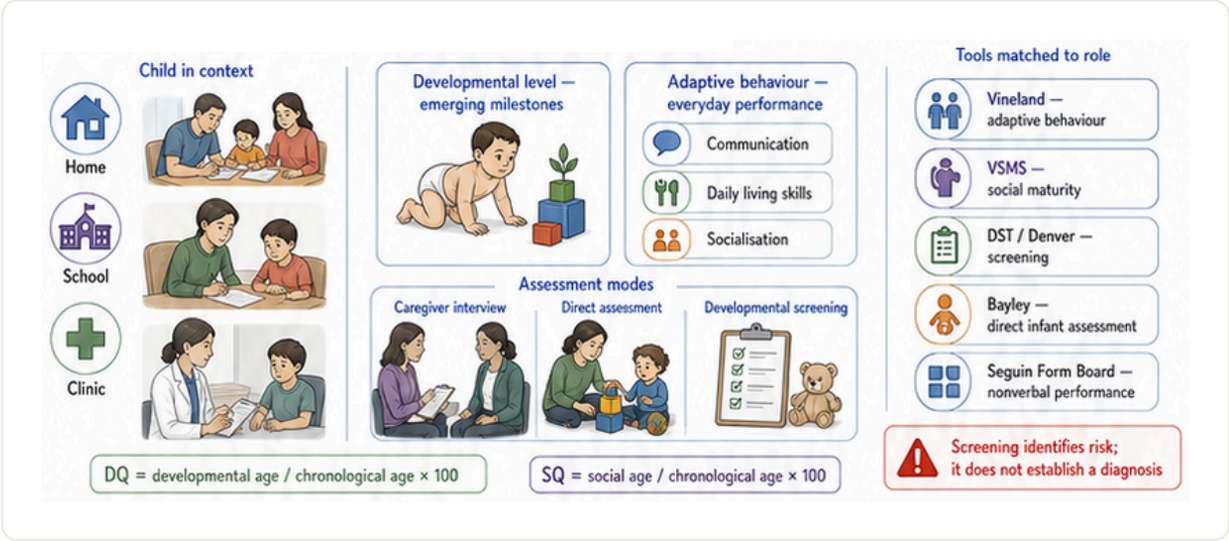


Fig 6.12 Adaptive behaviour and developmental assessment.

Two derived scores anchor the whole area. A **social quotient (SQ)** is social age divided by chronological age times 100, exactly parallel to the classic ratio IQ (mental age over chronological age). A **developmental quotient (DQ)** is developmental age over chronological age times 100. Both read like an IQ: 100 is average, and a score well below expectation flags delay. Note the required British and American spellings both appear in the literature: "adaptive behaviour" (Shorter Oxford, IPS) and "adaptive behavior" (the Vineland's own American title).

8.1 Adaptive behaviour as a construct

Definition. Adaptive behaviour is the person's ability to meet the responsibilities of social, personal, occupational and interpersonal life appropriate to their age, sociocultural setting and education. It is operationalised across three broad clusters.

Conceptual skills

Language, literacy, money, time, number and self-direction.

Social skills

Interpersonal skills, social responsibility, following rules, gullibility, avoiding victimisation.

Practical skills

Activities of daily living, occupational skills, healthcare, travel, safety and use of money in the real world.

CLINICAL ANCHOR

A diagnosis of intellectual disability requires a deficit in *both* general intellectual functioning *and* adaptive behaviour, with onset in the developmental period (DSM-5). An IQ below 70 alone does not make the diagnosis; equally, IQ near 70 with grossly deficient adaptive skills still counts. Under DSM-5 the severity label (mild to profound) is set by the adaptive domains, not the IQ number.

8.2 Vineland Adaptive Behavior Scales

The vineland (VABS). The modern flagship of adaptive assessment, now in its third edition (Vineland-3, Sparrow, Cicchetti and Saulnier). It is a caregiver or teacher informant instrument, age-normed from birth into late adult life (Kaplan & Sadock 2024).

Format

Semi-structured caregiver interview or rating form; self-report, parent and teacher versions exist.

Domains scored

Four adaptive domains: communication, daily living skills, socialisation, and motor skills (Shorter Oxford, Table 17.6), rolling up to an Adaptive Behavior Composite.

Extra domain

An optional maladaptive behaviour index captures internalising and externalising problems.

Clinical use

Diagnosing and grading intellectual disability, autism spectrum disorder, and tracking developmental progress over time.

> **PEARL**
>
> The vineland is one of the few instruments that spans birth to roughly age 90, which is why it is the reference adaptive scale in both child and adult intellectual-disability work. It is informant-based, so it can be completed even when the patient cannot be tested directly.

8.3 Vineland Social Maturity Scale (VSMS)

The vsms. The direct ancestor of the vineland, devised by Edgar Doll (the original Vineland Social Maturity Scale). It is the Indian workhorse: an informant scale that yields a **social age (SA)** and, from it, a **social quotient (SQ)**. An Indian adaptation (Malin) is a recognised instrument for issuing disability certificates for persons with intellectual disability in India (Clinical Methods in Psychiatry, AIIMS 2024).

Author

Edgar A. Doll (Vineland Training School, New Jersey).

What it measures

Social maturity across areas such as self-help (general, eating, dressing), self-direction, occupation, communication, locomotion and socialisation.

Scoring

Items credited by informant report give a social age; SQ = social age / chronological age times 100.

> **INDIA**
>
> The vsms is heavily used in Indian practice for the disability-certification workup, often paired with a performance IQ test. A worked medico-legal example from the AIIMS Clinical Methods text describes a patient with a mental age of about three years and a social quotient of 20 on VSMS, used to support a finding of incapacity to manage her person and property (AIIMS 2024).

8.4 The developmental screening test (DST, Bharat Raj)

The developmental screening test. The DST is an Indian instrument developed by J. Bharat Raj (NIMHANS). It is a caregiver-report screen that plots achieved milestones against age to yield a developmental age, and from it a developmental quotient. It is popular in Indian child-psychiatry and developmental-paediatric clinics because it is quick, informant-based and locally normed.

Author

J. Bharat Raj (Indian instrument).

What it measures

Overall development from infancy through early childhood, screening for developmental delay.

Scoring

Yields a developmental age; developmental quotient (DQ) = developmental age / chronological age times 100.

> **COMMON TRAP**
>
> Do not confuse the two "DST"s. In this topic DST is the developmental screening test (Bharat Raj). Elsewhere in psychiatry DST means the dexamethasone suppression test, a neuroendocrine probe of the HPA axis. Read the context.

8.5 Denver Developmental Screening Test

The denver (DDST). The most widely known international developmental screen (Frankenburg and Dodds). It screens, rather than diagnoses, sorting children into normal, questionable or abnormal so that delayed children are referred onward.

What it measures

Four sectors: gross motor, fine motor-adaptive, language, and personal-social development.

Age range

Birth to about six and a half years (Shorter Oxford; Indian developmental-milestone charts are modelled on the DDST sectors).

Scoring

Each item is placed as pass, fail or refusal against the age at which most children achieve it; a pattern of delays flags an abnormal screen for further assessment.

8.6 Bayley Scales of Infant Development

The bayley. The gold-standard directly administered developmental assessment for infants and toddlers (Nancy Bayley; now the Bayley Scales of Infant and Toddler Development, currently Bayley-4). Unlike informant screens, a trained assessor elicits and scores the child's own responses (Kaplan & Sadock 2024).

What it measures

Cognitive, language, motor, social-emotional and adaptive-behaviour scales.

Age range

Approximately 16 days to 42 months (Bayley-4, Kaplan & Sadock 2024).

Scoring

Standard tasks scored on mental, motor and behaviour scales, compared with norms; comprehensive developmental quotients / composite indices are derived.

PEARL

Bayley is a performance-based assessment, not a screen: it is used when a rigorous, examiner-administered estimate of infant development is needed (early diagnosis, research, tracking preterm or at-risk infants).

8.7 Gesell Developmental Schedules

The gesell. Arnold Gesell's developmental schedules (Yale Child Study Center) were the foundational normative maps of infant development, and remain a byword for milestone-based assessment. Gesell's data underlie many later schedules.

What it measures

Developmental landmarks across four fields: motor, adaptive, language, and personal-social behaviour.

Age range

Roughly 8 weeks (4 weeks in some editions) to 3.5 years, by direct observation of responses to standard toys and situations (Kaplan & Sadock 2024).

Scoring

Observed behaviour is matched to normative milestones to give a developmental age and a developmental quotient.

A simplified derivative, the Gesell Drawing Test (GDT), asks the child to copy geometric forms of increasing difficulty and gives a rapid rough estimate of mental age; it is used in Indian practice as a quick performance measure (AIIMS 2024).

8.8 Seguin Form Board

The seguin form board. A classic nonverbal performance test (traced to Edouard Seguin, who pioneered educational methods for the intellectually disabled; the Seguin Form Board Test, SFBT). The child fits solid geometric blocks into matching recesses as fast as possible.

What it measures

Visuomotor speed, eye-hand coordination and nonverbal problem-solving; a culture-fair, language-free estimate of intelligence.

Best use

Young children and language-impaired, deaf, or non-verbal individuals, where verbal tests are invalid.

Scoring

Timed: the shortest (best) of three trials is converted, via norms, to an estimate of mental age.

CLINICAL ANCHOR

The seguin form board is the go-to quick nonverbal screen in an Indian intellectual-disability workup when language is a barrier, often alongside the vsms and a Bhatia or Raven's performance measure.

8.9 How DQ and SQ are derived and read

The shared arithmetic. Every ratio quotient in this area follows one template: an age equivalent (mental, social or developmental) is divided by the actual chronological age and multiplied by 100.

QUOTIENT	NUMERATOR	FORMULA	INSTRUMENTS
IQ (ratio)	Mental age	**MA / CA times 100**	Stanford-Binet, Binet-Kamat
Social quotient (SQ)	Social age	**SA / CA times 100**	vsms
Developmental quotient (DQ)	Developmental age	**DA / CA times 100**	dst, gesell, bayley

All three ratio quotients share the same age-over-age arithmetic; only the numerator (the kind of age) differs.

RULE **Every ratio quotient is an age-equivalent over chronological age, times 100.** Only the numerator changes: mental age gives IQ, social age gives SQ, developmental age gives DQ; 100 is average throughout.

Reading the score. A quotient of 100 is average. Because a young child's chronological age is small, a modest lag in age-equivalent produces a proportionally low quotient, so early delay shows up clearly. In intellectual disability, SQ and DQ track roughly with IQ and are used both to support the diagnosis and to grade severity when a full IQ test is impractical.

8.10 Choosing the right tool: screen vs assessment, informant vs performance

Two axes organise the catalogue. First, screen versus full assessment: DDST and DST are screens that sort children for referral, whereas bayley and the vineland are full assessments that quantify functioning. Second, informant-report versus direct performance: vsms, vineland, DST and DDST rely on caregiver report or observation, whereas bayley, gesell and the seguin form board require the child's own elicited performance.

INSTRUMENT	TYPE	SCORE YIELDED	AGE BAND
vineland (VABS-3)	Informant, full assessment	**Adaptive Behavior Composite (4 domains)**	Birth to ~90 y
vsms	Informant	**Social age, SQ**	Birth to adulthood
developmental screening test (dst)	Informant screen (India)	**Developmental age, DQ**	Infancy to early childhood
denver (DDST)	Observation screen	**Normal / questionable / abnormal**	Birth to ~6.5 y
bayley	Performance, full assessment	**Cognitive/motor/language indices, DQ**	~16 days to 42 mo
gesell	Observation schedule	**Developmental age, DQ**	~8 wk to 3.5 y
seguin form board	Nonverbal performance	**Mental age (timed)**	Young / language-impaired

A single decision grid: pick a screen to triage, a full assessment to quantify, and a nonverbal test when language cannot be used.

HOLD THIS

Adaptive behaviour is now a co-defining axis of intellectual disability, not a supplement. The VSMS gives a social quotient and is the Indian disability-certification workhorse; screens (DST, Denver) triage, full assessments (Bayley, Vineland) quantify.

MNEMONIC

"Very Very Direct Doctors Believe Good Seguin"

vineland, vsms, developmental screening test (dst), denver (DDST), bayley, gesell, seguin: the seven scales of this topic in a single string.

GOOD TO REMEMBER

- **Adaptive behaviour / adaptive behavior:** conceptual, social and practical everyday skills; a co-defining axis of intellectual disability alongside IQ (DSM-5). Both spellings are in use.
- **vineland (VABS-3):** Sparrow, Cicchetti and Saulnier. Measures adaptive functioning in four domains (communication, daily living skills, socialisation, motor skills) plus a maladaptive index. Informant/caregiver interview, birth to ~90 y; yields an Adaptive Behavior Composite.
- **vsms (Vineland Social Maturity Scale):** Edgar Doll. Measures social maturity (self-help, self-direction, occupation, communication, locomotion, socialisation). Scores social age, then SQ = SA / CA times 100. Indian disability-certification workhorse.
- **developmental screening test (dst):** J. Bharat Raj (Indian instrument). Screens overall development; scores a developmental age and DQ = DA / CA times 100.
- **denver (DDST):** Frankenburg and Dodds. Screens gross motor, fine motor-adaptive, language and personal-social sectors; birth to ~6.5 y; scores normal / questionable / abnormal.
- **bayley (Scales of Infant and Toddler Development):** Nancy Bayley. Examiner-administered; cognitive, language, motor, social-emotional and adaptive scales; ~16 days to 42 months; yields developmental indices / DQ.
- **gesell (Developmental Schedules):** Arnold Gesell. Direct observation of motor, adaptive, language and personal-social behaviour; ~8 weeks to 3.5 y; yields developmental age and DQ. Gesell Drawing Test (GDT) is the rapid drawing-copy derivative.
- **seguin (form board):** Edouard Seguin. Nonverbal, timed block-fitting test of visuomotor speed and nonverbal intelligence; for young or language-impaired individuals; best of three trials gives an estimated mental age.
- **Social quotient (SQ):** social age / chronological age times 100.
- **Developmental quotient (DQ):** developmental age / chronological age times 100. Both read like an IQ (100 = average).

9 · Symptom rating scales

A symptom **rating scale** is a standardised instrument that turns a clinical construct (depression, mania, psychosis, obsessions) into a number, so that severity can be quantified, tracked over time, and compared across patients and studies. This topic is a working catalogue organised *by clinical domain*, so that when the exam (or the ward) asks "which scale for this?" you can reach for the right one and say what it measures, who rates it, and roughly how it scores. The point of a rating scale is not diagnosis but the **measurement of severity and change**: it converts the impressionistic "the patient seems a bit better" into a defensible delta on a validated metric (Kaplan & Sadock 2024).

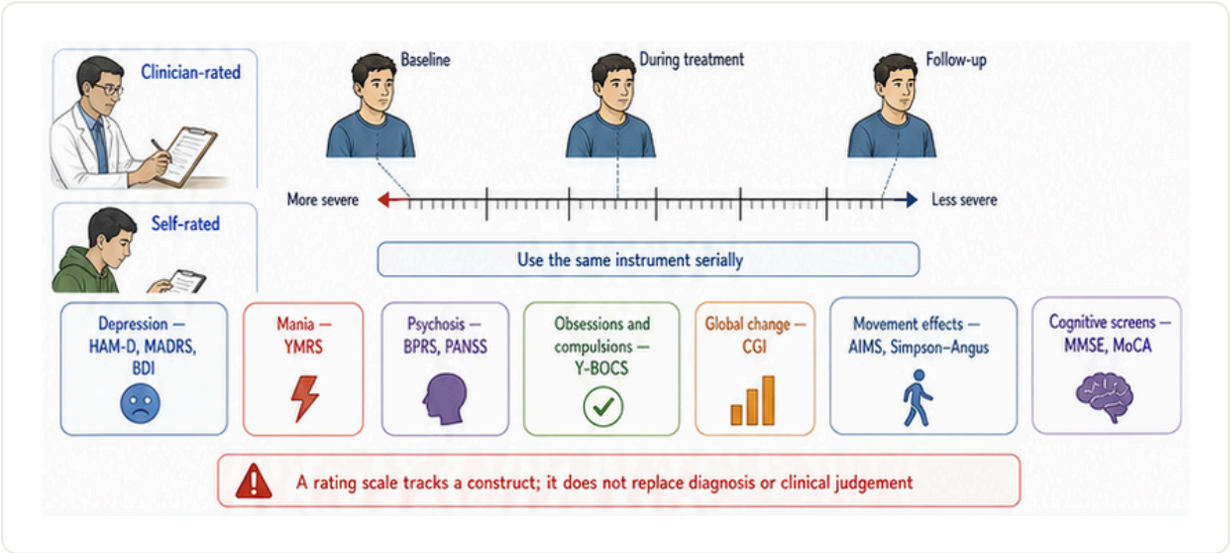


Fig 6.13 Rating scales: measure severity and change.

The single most examined distinction here is **clinician-rated** versus **self-rated**. A clinician-rated (observer-rated) scale is scored by a trained rater from a clinical interview and observation; a self-rated scale is a questionnaire the patient completes. Examiners also love the exact "signature" facts of the flagship scales: item counts, the number of items in the two Hamilton depression forms, and which scales are the standard primary outcome measures in drug trials. A fuller rating-scale catalogue (including screening, functional, quality-of-life and paediatric instruments) is developed in a Paper III chapter; here we stay with the core clinician-rated symptom scales by disorder.

9.1 What a rating scale is for: severity, response, remission

Three clinical jobs. A symptom rating scale is used to (1) quantify baseline severity, (2) track change with treatment, and (3) define trial endpoints. In trials, a fixed cut in the total score defines *response* (conventionally a 50 percent reduction from baseline) and a low absolute score defines *remission* (Kaplan & Sadock 2024). This is why the same scales that clinicians use to monitor a patient are also the primary outcome measures of the randomised controlled trials that licensed the drugs.

Scale versus diagnosis. A rating scale measures a construct on a continuum; it does not, by itself, make a categorical diagnosis. A cut-off can be applied to a continuous scale to flag likely caseness (for example an MMSE below 24 suggesting dementia, or a HAM-D above a threshold suggesting a depressive episode), but the diagnosis remains a clinical, criterion-based judgement (Kaplan & Sadock 2024).

9.2 Clinician-rated versus self-rated

Clinician-rated (observer-rated)

A trained clinician scores the item from a semi-structured interview plus observation. Strength: robust where insight is poor (mania, psychosis, cognitive disorders) and it captures observed signs. Cost: rater time, and the need for training to keep inter-rater reliability high. Examples: HAM-D, MADRS, YMRS, BPRS, PANSS, Y-BOCS.

Self-rated (self-report)

The patient completes a questionnaire; quick, cheap, captures the inner experience directly. Cost: depends on insight, literacy and honesty, and is vulnerable to response bias. Examples: Beck Depression Inventory (BDI), PHQ-9, GAD-7.

CLINICAL ANCHOR

The choice tracks insight. For a manic or floridly psychotic patient a self-rated scale is nearly useless (the patient does not experience the elevated mood or the delusion as pathological), so the clinician-rated YMRS or PANSS is used. For an ambulant patient with depression, a self-rated BDI or PHQ-9 can be posted to the waiting room and is a reasonable severity screen; but the trial-grade severity metric remains the clinician-rated HAM-D or MADRS.

RULE **Where insight is poor, use a clinician-rated scale.** Mania and psychosis need observer-rated instruments (YMRS, PANSS) because the patient does not experience the state as pathological.

9.3 Depression: HAM-D, MADRS and the Beck Depression Inventory

Hamilton Depression Rating Scale. The **hamilton** Depression Rating Scale (**ham-d**, also HDRS; Hamilton 1960) is the historic gold-standard clinician-rated depression scale and, for six decades, the most common primary outcome in antidepressant trials. The original published form has 17 items; a widely used extended form adds items to make 21 (Tasman describes it as a 17 to 21 item clinician scale with a strong focus on somatic and neurovegetative symptoms). It is scored by the clinician from interview; higher total means more severe depression. Its criticised feature is exactly that somatic loading, which can inflate scores in the physically ill.

Montgomery-Asberg Depression Rating Scale. The MADRS (**montgomery** and Asberg 1979) is a 10-item clinician-rated depression scale, explicitly designed to be *sensitive to change* and therefore favoured as a trial outcome; it is lighter on somatic items than the HAM-D, which makes it useful when physical illness would otherwise confound a depression score. Each item is rated 0 to 6.

Beck Depression Inventory. For contrast, the Beck Depression Inventory (BDI; Beck, current BDI-II 1996) is the classic *self-rated* depression questionnaire: 21 self-report items, quick, and weighted toward the cognitive symptoms of depression (guilt, worthlessness, pessimism). It is a severity and screening tool, not a diagnostic instrument, and shows the self-rated trade-off: convenient and patient-centred, but dependent on insight and honesty.

COMMON TRAP

Do not conflate the HAM-D and the BDI as interchangeable "depression scales". They differ on the axis examiners test: the HAM-D and MADRS are **clinician-rated** (the standard trial outcome measures), the BDI is **self-rated**. And the two Hamilton forms are 17 and 21 items, not "the 17-item Hamilton" alone.

9.4 Mania: the Young Mania Rating Scale

YMRS. The Young Mania Rating Scale (**ymrs**; Young, Biggs, Ziegler and Meyer 1978) is the standard clinician-rated mania severity scale and the usual primary outcome in acute-mania

trials. It has 11 items; seven items are scored 0 to 4 and four items (irritability, speech, thought content, and disruptive-aggressive behaviour) are given double weight and scored 0 to 8, so that harder-to-rate but clinically salient items are not lost. Because manic patients characteristically lack insight, a self-rated mania scale is unreliable, which is precisely why the observer-rated YMRS is the field standard.

9.5 Psychosis: BPRS and PANSS

Brief Psychiatric Rating Scale. The **bprs** (Overall and Gorham 1962) is one of the oldest clinician-rated scales for overall psychiatric symptom severity in psychotic and severe illness. The classic form has 18 items, each rated 1 to 7, covering a broad spread (anxiety, depression, hostility, hallucinatory behaviour, unusual thought content, blunted affect). It is quick and remains widely used to track global change, but it assesses positive and disorganised symptoms far better than it assesses negative symptoms.

Positive and Negative Syndrome Scale. The **panss** (Kay, Fiszbein and Opler 1987) is the modern flagship clinician-rated schizophrenia scale and the standard primary outcome in antipsychotic trials. It has 30 items, each rated 1 to 7, structured into three subscales: a 7-item Positive scale, a 7-item Negative scale, and a 16-item General Psychopathology scale. Its virtue over the BPRS is the explicit, separately scored negative-symptom dimension, which matters because positive and negative symptoms respond differently to treatment.

FEATURE	BPRS	PANSS
Items	18	**30 (7 positive, 7 negative, 16 general)**
Item anchor	1 to 7	1 to 7
Negative symptoms	**poorly captured**	separate 7-item negative subscale
Rating	clinician-rated	clinician-rated
Typical role	quick global severity	**standard antipsychotic-trial outcome**

BPRS versus PANSS: both clinician-rated and anchored 1 to 7, but the PANSS is larger and scores the negative syndrome on its own subscale, which is why it displaced the BPRS as the trial standard in schizophrenia.

17 / 21 HAM-D ITEM FORMS	**11** YMRS ITEMS (4 DOUBLE-WEIGHTED)	**30** PANSS ITEMS (7+7+16)	**0-40** Y-BOCS TOTAL (10 ITEMS)

9.6 Negative and positive symptoms: SANS and SAPS

The Andreasen pair. Before the PANSS folded both dimensions into one instrument, Nancy Andreasen built two dedicated clinician-rated scales for schizophrenia (Andreasen). The **rating scale** pair is the SANS (Scale for the Assessment of Negative Symptoms) and the SAPS (Scale for the Assessment of Positive Symptoms). The SANS rates affective flattening, alogia, avolition-apathy, anhedonia-asociality, and attentional impairment; the SAPS rates hallucinations,

delusions, bizarre behaviour, and formal thought disorder. They remain the reference standard specifically for detailed negative-symptom research.

9.7 Obsessive-compulsive disorder: the Y-BOCS

Yale-Brown Obsessive Compulsive Scale. The **y-bocs** (Goodman and colleagues 1989) is the standard clinician-rated OCD severity scale and the primary outcome in OCD trials. It has 10 items (five rating obsessions, five rating compulsions), each scored 0 to 4, giving a total of 0 to 40. Crucially it rates the *severity* of obsessions and compulsions (time occupied, interference, distress, resistance, control) independently of their *content*, which is captured separately on a symptom checklist. That form-over-content design is what makes it usable across the very different symptom pictures of OCD.

9.8 The global scale and the anxiety scale: CGI and HAM-A

Clinical Global Impression. The CGI (Clinical Global Impression; Guy 1976, from the NIMH battery) is the great pragmatic all-rounder: a clinician-rated global judgement independent of any single disorder. It has two brief parts, CGI-Severity (how ill is the patient now, rated 1 = normal to 7 = among the most extremely ill) and CGI-Improvement (how much has the patient changed since baseline, 1 = very much improved to 7 = very much worse). Its simplicity is its strength: it is applied across every disorder and is a near-universal secondary outcome in trials.

Hamilton Anxiety Rating Scale. The HAM-A (Hamilton 1959) is the standard clinician-rated anxiety severity scale, with 14 items covering psychic and somatic anxiety, each rated 0 to 4. It is the anxiety counterpart to the HAM-D and, like it, is a common trial outcome. Its self-rated primary-care counterpart is the GAD-7.

9.9 Drug-induced movement disorders: AIMS and Simpson-Angus

AIMS. The Abnormal Involuntary Movement Scale (AIMS; NIMH, Guy 1976) is the standard clinician-rated scale for tardive dyskinesia in patients on antipsychotics. It has 12 items, each rated 0 to 4: ten cover the abnormal movements themselves (by body region, plus global severity), and two concern dental status that can mimic dyskinesia. It requires a brief structured motor examination, not just an interview. Mild dyskinetic movements in two body areas, or moderate movements in one, on adequate neuroleptic exposure, suggest tardive dyskinesia (Kaplan & Sadock 2024).

Simpson-Angus Scale. The Simpson-Angus Scale (Simpson and Angus 1970) is the clinician-rated scale for antipsychotic-induced *parkinsonism* and other extrapyramidal side effects. It has 10 items rated 0 to 4, strongly weighted to rigidity, plus one akathisia item; the score is reported as the mean of the items, with about 0.3 as the upper limit of normal, and it correlates with antipsychotic dose (Kaplan & Sadock 2024). Akathisia specifically is more often rated on the dedicated Barnes Akathisia Rating Scale.

PEARL

Remember the movement scales as a triad: AIMS for tardive dyskinesia, Simpson-Angus for drug-induced parkinsonism, and the Barnes Akathisia Rating Scale for akathisia. All three are clinician-rated and all three need you to *examine* the patient, not just ask questions.

9.10 Cognitive screens: MMSE and MoCA

Mini-Mental State Examination. The MMSE (Folstein, Folstein and McHugh 1975) is the classic clinician-administered cognitive screen, scored out of 30, sampling orientation, registration, attention and calculation, recall, language and construction. A score below 24 is the traditional cut suggesting cognitive impairment (Kaplan & Sadock 2024), though education and age shift the threshold. It is a screen for global cognitive impairment, not a diagnostic or a detailed neuropsychological test.

Montreal Cognitive Assessment. The MoCA (Nasreddine and colleagues 2005) is also scored out of 30 but is more sensitive than the MMSE to *mild* cognitive impairment and to executive and visuospatial deficits, because it adds trail-making, clock-drawing, abstraction and more demanding delayed recall. A one-point education adjustment is added for fewer than 12 years of schooling. It is now often preferred where early or subtle impairment is the question.

INDIA

The MMSE and MoCA are language and literacy loaded, so raw Western cut-offs misclassify illiterate or non-English-speaking Indian patients. Validated Indian and vernacular adaptations are used instead, such as the Hindi Mental State Examination (HMSE) and other locally normed cognitive screens; the Addenbrooke's Cognitive Examination (ACE-III) also has Indian-language versions. Always screen cognition in a language the patient is literate in, and apply local norms rather than the standard cut of 24 or 26.

HOLD THIS

A rating scale measures severity and change, not diagnosis. Know the flagship per domain: depression HAM-D/MADRS, mania YMRS, psychosis PANSS, OCD Y-BOCS, cognition MMSE/MoCA (both out of 30, MMSE cut below 24).

MNEMONIC

"Doctors Manage Patients' Obsessive Complaints" → Depression, Mania, Psychosis, OCD, plus the Cognitive and movement screens

Walk the domains in order and name the flagship clinician-rated scale for each: Depression (HAM-D, MADRS), Mania (YMRS), Psychosis (BPRS, PANSS), OCD (Y-BOCS), then the add-ons: global (CGI), anxiety (HAM-A), movement (AIMS, Simpson-Angus), cognition (MMSE, MoCA). Self-rated contrast: BDI for depression.

GOOD TO REMEMBER

- **Clinician-rated vs self-rated:** clinician-rated (HAM-D, MADRS, YMRS, BPRS, PANSS, Y-BOCS) is scored by a trained rater and is robust when insight is poor; self-rated (BDI, PHQ-9, GAD-7) is a patient questionnaire, quick but insight-dependent.
- **HAM-D / HDRS (Hamilton 1960):** rates depression severity; clinician-rated; 17-item and extended 21-item forms; historic trial gold standard, somatically loaded.
- **MADRS (Montgomery & Asberg 1979):** rates depression severity, designed to be sensitive to change; clinician-rated; 10 items each 0 to 6; less somatic than HAM-D.
- **Beck Depression Inventory (Beck; BDI-II 1996):** rates depression severity; self-rated; 21 items; cognitively weighted; screening not diagnostic.
- **YMRS (Young et al 1978):** rates mania severity; clinician-rated; 11 items (four double-weighted); standard acute-mania trial outcome.
- **BPRS (Overall & Gorham 1962):** rates overall psychotic/psychiatric severity; clinician-rated; 18 items each 1 to 7; weak on negative symptoms.
- **PANSS (Kay, Fiszbein & Opler 1987):** rates schizophrenia symptoms; clinician-rated; 30 items each 1 to 7; positive (7), negative (7), general (16) subscales; standard antipsychotic-trial outcome.
- **SANS / SAPS (Andreasen):** rate negative and positive symptoms of schizophrenia respectively; clinician-rated; reference standard for negative-symptom research.
- **Y-BOCS (Goodman et al 1989):** rates OCD severity; clinician-rated; 10 items (5 obsessions, 5 compulsions) each 0 to 4, total 0 to 40; rates severity independent of content.
- **CGI (Guy 1976, NIMH):** global severity and improvement; clinician-rated; CGI-Severity and CGI-Improvement each 1 to 7; disorder-independent, near-universal trial secondary outcome.
- **HAM-A (Hamilton 1959):** rates anxiety severity; clinician-rated; 14 items each 0 to 4; psychic and somatic anxiety; self-rated counterpart GAD-7.
- **AIMS (NIMH, Guy 1976):** rates tardive dyskinesia; clinician-rated with motor exam; 12 items each 0 to 4.
- **Simpson-Angus (Simpson & Angus 1970):** rates antipsychotic-induced parkinsonism/EPS; clinician-rated exam; 10 items each 0 to 4, reported as item mean.
- **Barnes Akathisia Rating Scale:** rates akathisia; clinician-rated; completes the movement-scale triad with AIMS and Simpson-Angus.
- **MMSE (Folstein et al 1975):** screens global cognition; clinician-administered; scored out of 30; traditional impairment cut below 24.
- **MoCA (Nasreddine et al 2005):** screens cognition; clinician-administered; out of 30; more sensitive to mild cognitive impairment; +1 point if under 12 years of schooling.

10 · Indian test adaptations

Every imported Western test carries a hidden assumption: that the person taking it grew up inside the culture, language and schooling the norms were built on. In India that assumption fails, so the standard batteries were not merely translated but **adapted** and re-normed. This topic is the India-context marker for the whole assessment band. It is the single reliable place an examiner can drop a "name the Indian adaptation and who standardised it" question, and it recurs in the ID, disability-certification and neuropsychology sections. Teach each instrument as a catalogue entry: what Western parent it adapts, who built it, the language or population it serves, and where its Indian norms come from.

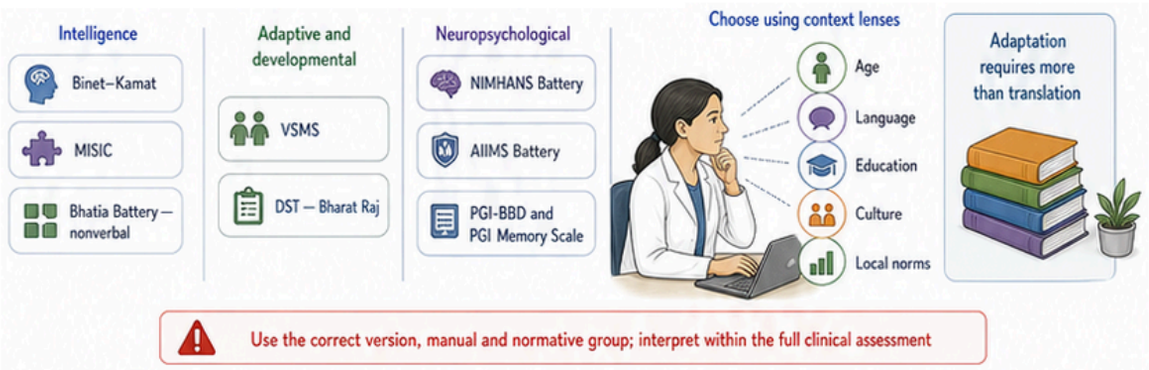


Fig 6.14 Indian psychological tests: match norms to context.

Why adaptation is not optional. Four forces make a foreign test invalid in an Indian testee, and each is a separable exam point. First, **norms**: a raw score only becomes an IQ or a percentile against a reference sample, and an American or British sample mis-scores an Indian child. Second, **language**: verbal items translated word-for-word lose difficulty gradation and cultural sense; Indian adaptations rebuild the verbal ladder in Kannada, Marathi, Hindi and other languages. Third, **cultural fairness**: item content (foods, festivals, idioms, pictures) must map onto the testee's world or it measures acculturation, not ability. Fourth, **literacy** and schooling: much of India is rural and non-literate, so performance (non-verbal) tests that do not depend on reading or writing become essential rather than optional (AIIMS Clinical Methods 2024).

INDIA

The through-line for the whole test band: for almost every Western instrument on this exam there is a locally standardised Indian counterpart, and the examiner wants the pair. Intelligence: Stanford-Binet becomes the **binet-kamat** test (BKT); WISC becomes **malin**'s Intelligence Scale for Indian Children (MISIC); Bhatia's Battery of Performance Tests of Intelligence serves illiterate and rural testees. Adaptive/developmental: the Vineland Social Maturity Scale gains an Indian adaptation with **indian norms**, and the Developmental Screening Test (DST) is India's own screener. Neuropsychology: the NIMHANS Neuropsychological Battery, the AIIMS Comprehensive Neuropsychological Battery, the PGI Battery of Brain Dysfunction (PGI-BBD) and the PGI Memory Scale are all Indian-normed.

10.1 Binet-Kamat Test (BKT): the Indian Stanford-Binet

What it adapts. The **binet-kamat** test (**bkt**) is V. V. Kamat's Indian adaptation of the Stanford-Binet (itself the Terman revision of the Binet-Simon scale). Kamat standardised it in the 1930s on Indian children, originally in Kannada and Marathi, giving India its first indigenous individually administered intelligence scale (AIIMS Clinical Methods 2024; also recorded as the Binet-Simon or Binet-Kamat test).

What it measures and how it scores. Like its parent it is a verbally loaded, age-graded scale that yields a mental age and, classically, a ratio IQ = (mental age / chronological age) x 100. Items are arranged by age level; testing runs from a basal age (all items passed) up to a ceiling age (all items failed), and mental age is summed across the ladder. It suits children and lower ability ranges and

is still used in some Indian centres, though it is verbal-heavy and therefore language and schooling dependent.

10.2 Malin's Intelligence Scale for Indian Children (MISIC): the Indian WISC

What it adapts. **misic** is **malin**'s Intelligence Scale for Indian Children, Arthur J. Malin's Indian adaptation of the Wechsler Intelligence Scale for Children (WISC). It is the workhorse individual IQ test for Indian children in clinical and disability settings (AIIMS Clinical Methods 2024).

What it measures and how it scores. It preserves the Wechsler architecture: a set of verbal subtests and a set of performance subtests, each yielding scaled scores that combine into a Verbal IQ, a Performance IQ and a Full-Scale IQ (deviation-IQ scoring). AIIMS Clinical Methods notes that its objective subtests, for example the arithmetic and digit-span items, are borrowed for bedside cognitive testing in adults too, which shows how embedded MISIC is in Indian practice.

10.3 Bhatia's Battery of Performance Tests of Intelligence: for illiterate and rural populations

What it adapts and why it matters. Bhatia's Battery of Performance Tests of Intelligence (C. M. Bhatia, 1955) is a purely non-verbal, performance-based intelligence test built specifically so that illiterate, poorly schooled and rural Indian testees can be assessed fairly, because it needs no reading or writing (AIIMS Clinical Methods 2024). It is India's canonical answer to the literacy problem.

What it measures and how it scores. It is administered as a set of performance subtests, classically the Koh's Block Design, the Alexander Pass-Along, the Pattern Drawing, the Immediate Memory (digit span) and the Picture Construction tests. Each is timed and scored, and the summed performance yields an intelligence estimate. Its non-verbal, culture-reduced format is exactly why it also survives as a sub-scale inside the PGI Battery of Brain Dysfunction (as the "Bhatia Short Battery").

RULE **For low-literacy or rural Indian testees, reach for a non-verbal performance test.** Bhatia's Battery, the Seguin Form Board and Raven's Matrices bypass the reading and language barrier that invalidates verbal-loaded scales.

10.4 Vineland Social Maturity Scale: Indian adaptation and Indian norms

What it adapts. The Vineland Social Maturity Scale (VSMS) is Doll's American measure of adaptive behaviour; its Indian adaptation with **indian norms** (A. J. Malin, Nagpur adaptation) is the standard Indian adaptive-behaviour instrument and is central to certifying intellectual disability in India (AIIMS Clinical Methods 2024).

What it measures and how it scores. It is an informant-based interview (parent or caregiver, not the child directly) covering everyday social competence across domains such as self-help, self-direction, occupation, communication, locomotion and socialisation. Scored items across age levels give a **social age**, which divided by chronological age and multiplied by 100 gives a **social quotient** (SQ). Because DSM-5 and modern ID definitions anchor severity to adaptive functioning rather than IQ alone, an adaptive measure like VSMS is now essential, not supplementary.

10.5 Developmental Screening Test (DST, Bharat Raj): India's developmental screener

What it is. The Developmental Screening Test (DST) is Jayanti Bharat Raj's Indian developmental screening instrument, built to give a quick developmental quotient in Indian infants and young children as an indigenous alternative to Western screeners like the Denver Developmental Screening Test.

What it measures and how it scores. It screens developmental attainments from early infancy through childhood, converting the pass pattern into a **developmental age** and a developmental quotient (developmental age / chronological age x 100). It is a screening tool, flagging children who need fuller assessment, not a diagnostic IQ test.

> **COMMON TRAP**
>
> Do not confuse the three quotient families that all look like "age over age times 100": intelligence gives an *IQ* (mental age, from BKT or MISIC), adaptive behaviour gives a *social quotient* (social age, from VSMS), and screening gives a *developmental quotient* (developmental age, from DST). Also, in exams "DST" almost always means the Developmental Screening Test here, not the Dexamethasone Suppression Test from the biology chapter: read the stem.

10.6 NIMHANS Neuropsychological Battery: the Indian normative neuropsychology battery

What it is. The **nimhans neuropsychological** battery (Rao, Subbakrishna and Gopukumar, 2004) is the Indian normative neuropsychology battery developed and standardised at NIMHANS, Bengaluru. The adult form comprises 19 neuropsychological tests, adapted largely from the Halstead-Reitan tradition, with Indian norms for ages 16 to 65 years (AIIMS Clinical Methods 2024).

What it measures and how it scores. It is modular: the clinician selects tests by referral question rather than running all 19. It samples attention and visual search, psychomotor speed and perseveration, working memory, executive and visuospatial planning, block design and object assembly, and verbal memory (its Rey Auditory Verbal Learning Test is a standard verbal-memory measure). There are companion versions: the NIMHANS Neuropsychological Battery for children (Kar et al., 2004, ages 5 to 15, probing frontal, parieto-occipital and temporal function) and the NIMHANS Neuropsychological Battery for Elderly (NNB-E, Tripathi et al., 2013). Each subtest is scored against the Indian norms; deficits are read for localisation and lateralisation.

10.7 AIIMS Comprehensive Neuropsychological Battery

What it is. The AIIMS Comprehensive Neuropsychological Battery in Hindi, adult form (Gupta et al., 2000), applies the principles of the Luria-Nebraska Neuropsychological Battery to an Indian, Hindi-medium population (AIIMS Clinical Methods 2024).

What it measures and how it scores. It contains 160 items organised into ten basic scales plus secondary scales spanning motor, tactile, visual, receptive and expressive speech, reading, writing, arithmetic, memory and intellectual processes, rolling up into left-hemisphere, right-hemisphere and total-score scales. It is used to detect lateralisation (right versus left lesion) and localisation of brain lesions. Indian norms cover ages 15 to 80 years; its length is its main limitation.

10.8 PGI Battery of Brain Dysfunction (PGI-BBD) and the PGI Memory Scale

What it is. The PGI Battery of Brain Dysfunction (Pershad and Verma, 1990) is the Chandigarh (PGIMER) battery for detecting and quantifying organic brain damage in Indian adults (AIIMS Clinical Methods 2024).

What it measures and how it scores. PGI-BBD combines five sub-scales: the PGI Memory Scale, the Bhatia Short Battery, the Verbal Adult Intelligence Scale, the Nahor-Benson test of perceptual acuity and the Bender Visual Motor Gestalt Test. It yields a global dysfunction score built from 19 variables, with norms for ages 20 to 59 years; a cut-off above 20 signifies organic brain damage, which is why it is heavily used in disability certification (for example in traumatic brain injury). The **PGI Memory Scale** is its embedded memory instrument and is also used standalone: it tests immediate and delayed recall across items such as paired and unpaired associations, logical memory and recognition, and its items (like digit span) are borrowed for bedside memory testing.

CLINICAL ANCHOR

The India disability-certification chain runs through these adaptations. For an intellectual-disability certificate you pair an Indian IQ test (MISIC or BKT) with the VSMS Indian adaptation for adaptive behaviour; for an organicity or head-injury certificate you reach for the PGI-BBD (cut-off above 20) or the NIMHANS Neuropsychological Battery. Knowing which instrument certifies what is the practical, and examinable, payoff of this whole topic.

10.9 The Indian adaptations at a glance

INDIAN TEST	ADAPTS / PARENT	LANGUAGE / POPULATION
Binet-Kamat Test (BKT), V. V. Kamat	**Stanford-Binet**	Kannada, Marathi; Indian children
MISIC, A. J. Malin	**Wechsler WISC**	Indian children; verbal + performance
Bhatia's Battery of Performance Tests, C. M. Bhatia (1955)	**performance/non-verbal intelligence**	**illiterate and rural populations**
VSMS Indian adaptation, A. J. Malin	**Vineland (Doll) adaptive behaviour**	informant-based; Indian norms; ID certification
Developmental Screening Test (DST), Bharat Raj	**developmental screening (cf. Denver DDST)**	Indian infants and young children
NIMHANS Neuropsychological Battery (Rao et al., 2004)	**Halstead-Reitan tradition**	Indian norms, ages 16 to 65
AIIMS Comprehensive Battery (Gupta et al., 2000)	**Luria-Nebraska**	Hindi; ages 15 to 80; 160 items
PGI-BBD and PGI Memory Scale (Pershad and Verma, 1990)	**organicity battery + memory scale**	Indian adults, ages 20 to 59

The India-context pairing table: each Western instrument on the exam has a locally standardised Indian counterpart, and the examiner wants the pair with its author and population.

HOLD THIS

For almost every Western test the examiner wants the Indian pair with its author. Stanford-Binet to Binet-Kamat (Kamat); WISC to MISIC (Malin); adaptive to VSMS Indian norms; organicity to the PGI-BBD (cut-off above 20) or the NIMHANS battery.

GOOD TO REMEMBER

- **Binet-Kamat Test (BKT):** V. V. Kamat's Indian adaptation of the Stanford-Binet; verbal, age-graded; yields mental age and ratio IQ; standardised in Kannada and Marathi for Indian children.
- **MISIC:** A. J. Malin's Intelligence Scale for Indian Children; adapts the Wechsler WISC; verbal and performance subtests giving Verbal, Performance and Full-Scale IQ; workhorse child IQ test in India.
- **Bhatia's Battery of Performance Tests of Intelligence:** C. M. Bhatia (1955); purely non-verbal performance battery (Koh's Block Design, Alexander Pass-Along, Pattern Drawing, Immediate Memory, Picture Construction) for illiterate and rural populations; timed subtests summed to an IQ estimate.
- **VSMS Indian adaptation:** A. J. Malin's Indian adaptation of the Vineland Social Maturity Scale with Indian norms; informant-based adaptive behaviour; social age gives a social quotient (SQ); used for ID certification.
- **Developmental Screening Test (DST):** Bharat Raj; Indian developmental screener for infants and young children; developmental age gives a developmental quotient; a screener, not a diagnostic IQ test.
- **NIMHANS Neuropsychological Battery:** Rao, Subbakrishna and Gopukumar (2004); Indian normative neuropsychology battery from NIMHANS; 19 adult tests from the Halstead-Reitan tradition; norms 16 to 65; modular, scored for localisation and lateralisation; child (Kar et al., 2004) and elderly (NNB-E, 2013) versions exist.
- **AIIMS Comprehensive Neuropsychological Battery:** Gupta et al. (2000); Hindi adult form on Luria-Nebraska principles; 160 items, ten basic scales; detects lateralisation and localisation; Indian norms ages 15 to 80.
- **PGI Battery of Brain Dysfunction (PGI-BBD):** Pershad and Verma (1990); five sub-scales (PGI Memory Scale, Bhatia Short Battery, Verbal Adult Intelligence Scale, Nahor-Benson, Bender-Gestalt); global score from 19 variables; norms 20 to 59; cut-off above 20 signifies organic brain damage; used for disability certification.
- **PGI Memory Scale:** Indian memory instrument (immediate and delayed recall, paired and unpaired associations, logical memory, recognition); used standalone and as the memory sub-scale of PGI-BBD.
- **Why adapt at all:** norms, language, cultural fairness, literacy; a foreign test's norms mis-score an Indian testee, so instruments are re-standardised, re-languaged and, for non-literate testees, made non-verbal.

Descriptive psychopathology

11 · Orientation: descriptive psychopathology and the domains of mental life

Before any diagnosis is named, the psychiatrist must be able to *describe* what is wrong with precision. That skill has a name and a lineage. **Descriptive psychopathology** is the objective, theory-free description of abnormal states of mind, limited to conscious experience and

observable behaviour, avoiding preconceived aetiological ideas (Sims 2015, Shorter Oxford 2018). Its aim, in Jaspers's phrase, is to elucidate the essential qualities of morbid mental experiences and so to understand each patient's illness from the inside. It has been called the fundamental professional skill of the psychiatrist, and for the exit examination it is the spine of the whole signs-and-symptoms section: every later topic (perception, thought, self, mood, memory, consciousness, motor) is a chapter within this one framework.

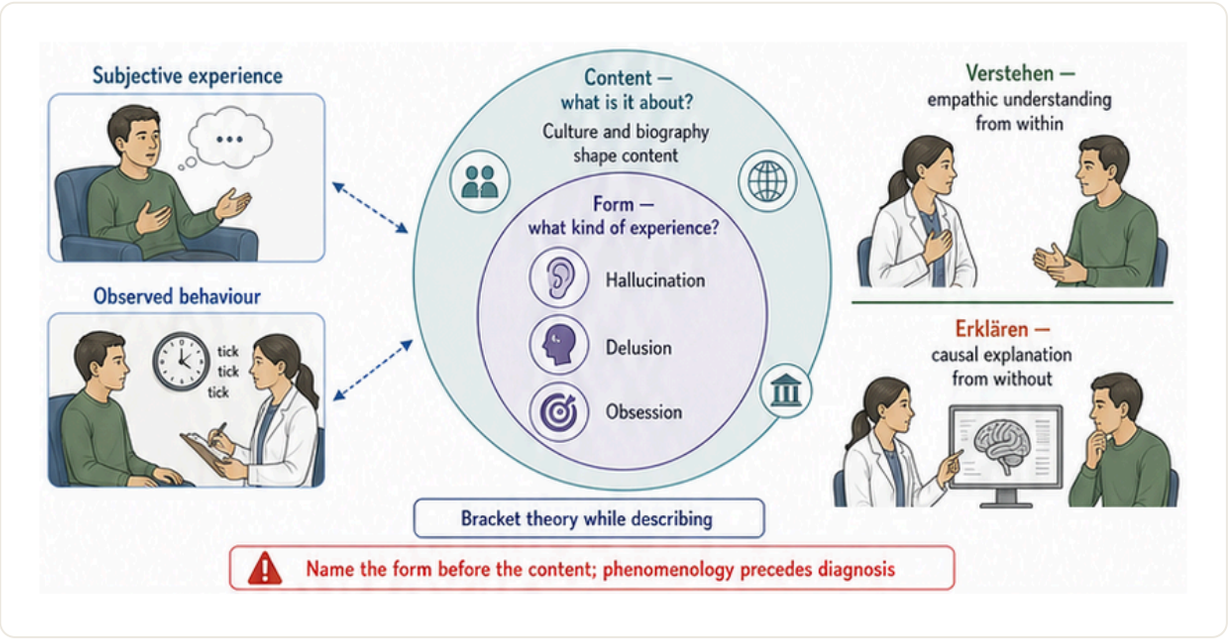


Fig 6.15 Describe the phenomenon before explaining it.

Why this opener matters. Band B of these notes is a map, not a list. This topic lays the map: it fixes the **method** (empathic description, form over content), names the three canonical texts you cite in a viva (jaspers, fish, sims), and gives the domains-of-mental-life scaffold onto which every individual sign hangs. Learn the scaffold once here and the rest of these notes becomes filling in cells.

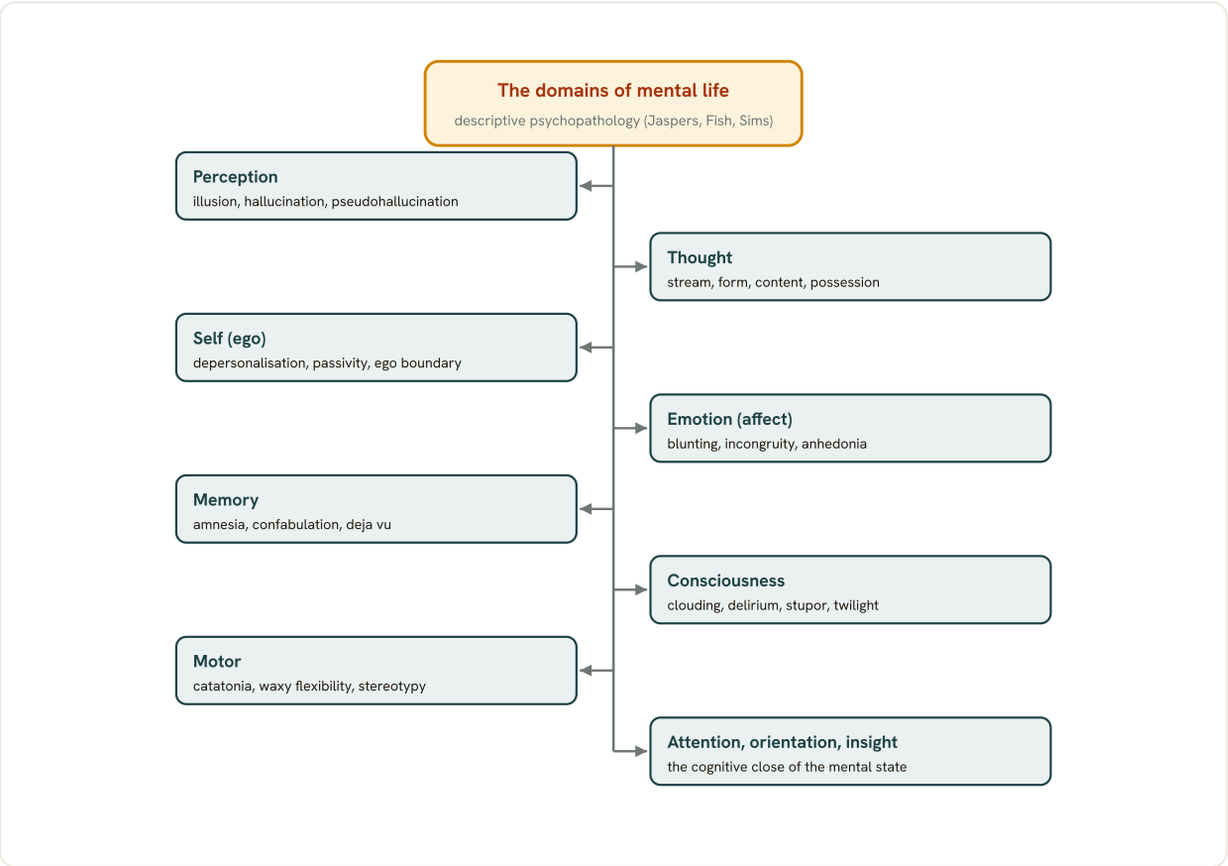


Fig 6.16 The domains of descriptive psychopathology, the map that organises the whole of Band B. Each branch is a domain of mental life with its characteristic disorders; naming a sign precisely means first knowing which domain it belongs to. The delusions and the Schneiderian first-rank symptoms sit within the thought branch and are drawn out separately. Following Jaspers, the aim throughout is to describe the form of an experience before interpreting its content.

11.1 Descriptive psychopathology defined

The definition to memorise. Descriptive psychopathology is the objective description of abnormal states of mind, avoiding as far as possible preconceived ideas or theories, and limited to the description of conscious experiences and observable behaviour (Sims 2015). Two commitments are load-bearing. First, it is *atheoretical*: it describes the phenomenon, it does not explain its cause. Second, it stays with what is *conscious and observable*: it does not appeal to unconscious mechanisms (that is the province of psychoanalytic or experimental psychopathology, not descriptive). This is why a Freudian reading of a persecutory delusion as projection is explanation, not description, and sits outside the descriptive method.

TRAP Reading a persecutory delusion as unconscious projection is explanation (erklaren), not description; descriptive psychopathology stays with conscious, observable experience and appeals to no unconscious mechanism.

11.2 Phenomenology and the two words for the same skill

Phenomenology. In clinical psychiatry, **phenomenology** is used almost synonymously with descriptive psychopathology: the careful study and categorisation of subjective mental experiences as the patient reports them. Strictly the terms are not identical (phenomenology carries additional philosophical meanings from Husserl and from Jaspers's own borrowing, per

Berrios 1992), but in exam and ward usage, phenomenology = the description of inner experience, one wing of descriptive psychopathology, the other wing being the observation of behaviour and signs.

11.3 Jaspers, and the method of empathic understanding

Jaspers 1913. Karl Jaspers, psychiatrist and philosopher, published *Allgemeine Psychopathologie* (General Psychopathology) in 1913; it remains the most complete account of the subject (Jaspers 1913, cited in Sims 2015). His central methodological gift was the distinction between two modes of grasping a mental state.

Verstehen (understanding)

Grasping a mental state *from within* by empathy: how one experience meaningfully arises from another (grief follows loss, jealousy follows infidelity). The tool of descriptive psychopathology.

Erklaren (explanation)

Grasping a phenomenon *from without* by causal, natural-science laws (a lesion, a neurotransmitter, a genetic mechanism). The tool of the biological sciences.

A phenomenon that empathy can enter is *understandable*; one that empathy cannot bridge (the primary delusion, the true first-rank symptom) is *un-understandable* and points to process, not development. This verstehen versus erklaren axis is Jaspers's signature and a recurring viva question.

11.4 The clinical distillations: Fish and Sims

From Jaspers to the ward. Jaspers is dense and philosophical; two English texts translated him into daily clinical use, and these are the ones you name.

fish, Clinical Psychopathology

Frank Fish's concise British manual (carried forward by Casey and Kelly, Fish 2007); the standard signs-and-symptoms crib for trainees, tightly organised by domain.

sims, Symptoms in the Mind

Andrew Sims's *Symptoms in the Mind: An Introduction to Descriptive Psychopathology* (Sims 2015); the fuller, more readable exposition, now the reference text most cited in Indian and UK postgraduate exams.

Cite the trio in one breath in a viva: the method is Jaspers, the crib is Fish, the exposition is Sims.

11.5 Form versus content: the central Jasperian rule

The single most examinable distinction. For any abnormal experience, separate its **form** from its **content**.

Form

The *kind* of experience, its structure: is this a hallucination, a delusion, an obsession, a thought that is being withdrawn? The form is what defines the sign and carries diagnostic weight. Phenomenology attends to form.

Content

What the experience is *about*: the theme, its subject matter (persecution by neighbours, guilt over a sin, a voice saying a particular sentence). Content is shaped by culture and biography; it is clinically interesting but does not by itself fix the diagnosis.

Worked example: two patients each believe the neighbours are poisoning them. Same content. If patient A holds it as an unshakeable false belief arising fully formed and un-understandably, the form is a delusion; if patient B has heard a voice announce it, the form is an auditory hallucination with secondary belief. The form differs, and it is the form that guides you. Sims's formulation is the one to quote: descriptive psychopathology is concerned with form, the phenomenologist deliberately brackets the content (Sims 2015).

RULE **Name the form before the content.** The form (hallucination, delusion, obsession) carries the diagnostic weight; the content (its theme) is culture-shaped and does not fix the diagnosis.

COMMON TRAP

Chasing content and missing form. A candidate who lists what the delusion is about, but cannot say whether the experience is a delusion, a hallucination or an overvalued idea, has described nothing diagnostically useful. Always name the form first, then the content.

11.6 Why phenomenology precedes diagnosis

The sign is the unit. Diagnosis in psychiatry is the recognition of a pattern of signs and symptoms. You cannot recognise a pattern you cannot first describe. A precisely elicited sign (a third-person auditory hallucination, thought insertion, a mood-congruent delusion) is the atomic unit of the mental state examination; the diagnosis is the molecule assembled from those atoms against operational criteria. As the New Oxford text puts it, phenomenology precedes diagnosis, reflecting both observed signs and the subjective experience of the person (New Oxford 2020). Precision at the level of the single sign is therefore not pedantry; it is what makes the diagnosis defensible. Reverse the order (decide the diagnosis, then relabel the phenomena to fit) and you confabulate.

11.7 The domains of mental life: the map

The scaffold. Descriptive psychopathology partitions abnormal experience into domains that mirror the structure of the mental state examination. Learn these headings as the master index for the whole set of notes; each expands into its own topic.

disorders of perception

Abnormalities of sensory experience: illusions, hallucinations across modalities, pseudohallucinations, and perceptual distortions.

disorders of thought

Abnormalities of thought stream, form (formal thought disorder), possession (insertion, withdrawal, broadcast) and content (delusions, overvalued ideas, obsessions).

disorders of self

Disturbance of the awareness of one's own existence and boundaries: depersonalisation, derealisation, and the disorders of ego boundary and agency (the Ich-Storungen).

disorders of emotion

Abnormalities of affect and mood: their intensity, quality, congruity, stability and reactivity (blunting, incongruity, lability, anhedonia, elation, depression).

disorders of memory

Abnormalities of registration, retention and recall: the amnesias, confabulation, and the false-recognition phenomena (deja vu, jamais vu).

disorders of consciousness

Abnormalities of the level and clarity of awareness: clouding, delirium, stupor, and the twilight and oneiroid states.

disorders of motor function

Abnormalities of movement and volition, chiefly the catatonic phenomena: stupor, negativism, waxy flexibility, echopraxia, mannerisms and stereotypies.

Two cross-cutting domains complete the map. **Attention and orientation** (the capacity to focus and to know time, place and person) is the substrate the whole examination sits on; a patient disoriented in time cannot give a reliable account of anything else. **Insight and judgement** (awareness of being ill, of the need for treatment, and the ability to act sensibly on that awareness) is graded, not all-or-none, and closes the mental state examination.

MNEMONIC

Please Think Slowly, Every Memory Counts Meaningfully, And Insightfully

Perception, Thought, Self, Emotion, Memory, Consciousness, Motor, then Attention/orientation and Insight/judgement. The seven core "disorders of ..." domains plus the two cross-cutting ones, in mental-state-examination order.

11.8 The domains against the mental state examination

Same map, two uses. The domains are also the running order of the MSE, so learning them once serves both the phenomenology paper and the clinical long case.

DOMAIN	MSE HEADING IT FEEDS	ANCHOR SIGN
disorders of perception	**Perception**	Third-person auditory hallucination
disorders of thought	**Thought (stream, form, content, possession)**	Thought insertion; primary delusion
disorders of self	**Perception / thought (ego boundary)**	Depersonalisation
disorders of emotion	**Mood and affect**	Blunted or incongruous affect
disorders of memory	**Cognition**	Confabulation
disorders of consciousness	**Consciousness / sensorium**	Clouding, delirium
disorders of motor function	**Behaviour and motor**	Waxy flexibility
Attention and orientation	**Cognition**	Disorientation to time

DOMAIN	MSE HEADING IT FEEDS	ANCHOR SIGN
Insight and judgement	**Insight**	Denial of illness

The domains of mental life read top-to-bottom are, near enough, the running order of the mental state examination.

HOLD THIS

The method is Jaspers, the crib is Fish, the exposition is Sims; describe form by empathy, do not explain content by cause. Phenomenology precedes diagnosis: the precisely named sign is the unit of the mental state examination.

PEARL

The domains in one line: perception, thought, self, emotion, memory, consciousness, motor, plus attention/orientation and insight, described by form not content, understood by empathy not explained by cause.

CLINICAL ANCHOR

In a viva, when asked "how would you describe this patient's symptoms", do not lead with a diagnosis. Lead with the method: "I would describe the phenomena in each domain, attending to the form of each experience rather than its content, in the order perception, thought, self, emotion, memory, consciousness and motor." You have then shown you own the framework, which scores before any single sign is named.

INDIA

For the Indian postgraduate exams, Sims's *Symptoms in the Mind* and Fish's *Clinical Psychopathology* are the two most-cited phenomenology texts, and the AIIMS *Clinical Methods in Psychiatry* (2024) reproduces the same Jasperian framework. Content of delusions is often culture-shaped (possession, black magic, the evil eye); describe the form precisely and record the content faithfully without pathologising a culturally normative belief.

GOOD TO REMEMBER

- **Descriptive psychopathology:** objective, theory-free description of abnormal states of mind, limited to conscious experience and observable behaviour (Sims 2015).
- **Phenomenology:** the study and categorisation of subjective mental experience; in clinical use, near-synonymous with descriptive psychopathology.
- **jaspers, General Psychopathology (1913):** the founding text; the method of empathic understanding.
- **Verstehen versus erklaren:** understanding a state from within by empathy versus explaining it from without by cause; understandable versus un-understandable phenomena.
- **fish, Clinical Psychopathology (2007):** the concise British signs-and-symptoms crib, domain-organised.
- **sims, Symptoms in the Mind (2015):** the fuller exposition; the most-cited exam reference.
- **Form:** the kind or structure of an experience (hallucination, delusion, obsession); phenomenology attends to form; form carries diagnostic weight.
- **Content:** what the experience is about (its theme); culture- and biography-shaped; not diagnostic by itself.
- **Phenomenology precedes diagnosis:** a precisely elicited sign is the unit of the mental state examination; the diagnosis is assembled from signs.
- **disorders of perception:** illusions, hallucinations, pseudohallucinations, perceptual distortions.
- **disorders of thought:** stream, form, possession, content (delusions, obsessions, overvalued ideas).
- **disorders of self:** depersonalisation, derealisation, ego-boundary and agency disturbance.
- **disorders of emotion:** intensity, quality, congruity and stability of affect and mood.
- **disorders of memory:** amnesias, confabulation, deja vu and jamais vu.
- **disorders of consciousness:** clouding, delirium, stupor, twilight and oneiroid states.
- **disorders of motor function:** the catatonic phenomena and disorders of volition.
- **Cross-cutting:** attention/orientation (the substrate) and insight/judgement (graded, closes the MSE).

12 · Disorders of perception

Perception is the process by which sensory data reaching the brain are organised and interpreted into a meaningful representation of the outside world; it is more than raw sensation, which is the mere registration of a stimulus at a receptor. The descriptive psychopathology of perception classifies the ways this process goes wrong, and it is a perennial Paper I favourite because the whole subject rewards precise definitions and clean discriminations rather than mechanism. This note gives the **phenomenology** only, in the tradition of Jaspers, Fish and Sims (Jaspers 1913, Fish 2007, Sims 2015): what each abnormality looks like at the level of description, form over content.

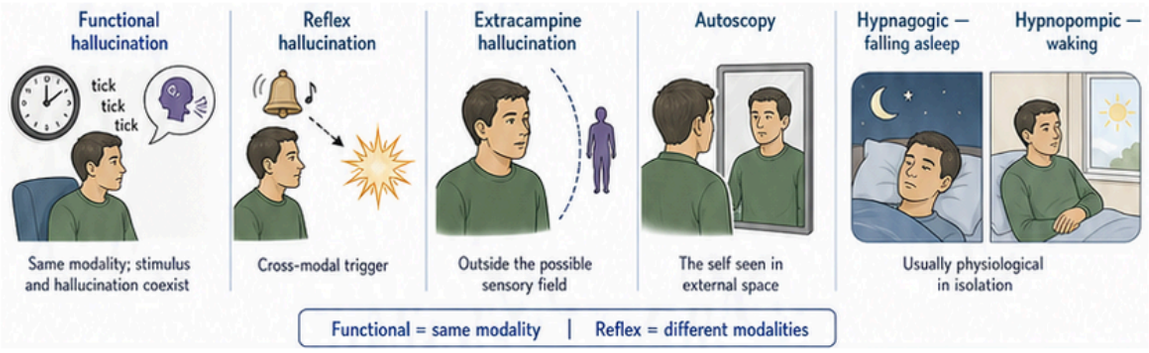


Fig 6.17 Special hallucinatory experiences.

Exam framing. The examiner wants three levels kept apart cleanly: a real percept altered (**sensory distortion**), a real stimulus misperceived (**illusion**), and a percept with no stimulus at all (**hallucination**), plus the halfway house of the **pseudohallucination**. The mechanistic theories of hallucination (perceptual-release, aberrant salience, predictive coding) belong to the General and Developmental Psychology notes and are cross-referred there; here the task is to name and define, ideally with a one-line clinical example each. Aetiology and the theories live in the General and Developmental Psychology notes, not here.

Discriminator	Illusion	Hallucination	Pseudo-hallucination
Real external stimulus?	Yes: a real object misperceived	No stimulus	No stimulus
Where perceived	outer objective space	outer objective space	inner subjective space
Reality status	a real percept, misjudged	full force of a true perception	lacks full substantiality
Insight	corrects on closer attention	absent (believed real)	relatively preserved
Typical setting	fear, inattention; organic states	psychosis; organic states	grief, dissociation, hypnagogic

Fig 6.18 Telling perceptual phenomena apart. An illusion is a real external stimulus misperceived; a hallucination is a percept without any stimulus, experienced in outer objective space with the full force of a true perception and without insight; a pseudohallucination arises in inner subjective space, lacks the full substantiality of a true perception, and leaves insight relatively preserved. The two discriminators that carry most of the work are the locus (outer versus inner space) and the reality status.

12.1 The three-level framework and its discriminators

Orientation. Every perceptual abnormality can be placed by asking four questions: is there a real external stimulus, where is the experience located (objective external space versus inner subjective space), does it carry the full substantiality and involuntary force of a true percept, and is insight retained. A **sensory distortion** and an **illusion** both start from a real stimulus; a

hallucination and a **pseudohallucination** do not. A hallucination sits in external space with full force and insight lost; a pseudohallucination sits in inner subjective space, lacks that substantiality, and insight is relatively preserved (Sims 2015). Hold this scaffold and the named subtypes below slot in without confusion.

RULE **Two discriminators do most of the work: the presence of a real stimulus, and the locus (outer objective versus inner subjective space).** Stimulus separates the illusion; locus separates the hallucination from the pseudohallucination.

12.2 Sensory distortions: a real percept altered

Definition. A **sensory distortion** is a change in the intensity, quality or spatial form of a percept arising from a real external object; the object is correctly identified, but its perceived attributes are altered (Fish 2007).

Hyperaesthesia

Increased intensity of perception: sounds seem unbearably loud, colours vivid; seen in migraine, hangover, some drug states and the heightened arousal of mania and anxiety.

Hypoaesthesia

Decreased intensity of perception: the world seems dull, muffled or grey; seen in delirium, depression (where colours are described as washed out) and clouded consciousness.

Changes in quality

Altered perceived quality, chiefly of colour: chromatopsia, e.g. xanthopsia (yellow vision), chloropsia (green) or erythropsia (red), typically from drugs or toxins such as digitalis or santonin.

Changes in spatial form (dysmegalopsia)

The perceived size or shape of an object is altered. **Micropsia**: objects seem smaller or further away. **Macropsia** (megalopsia): objects seem larger or nearer. Metamorphopsia: objects appear irregular or distorted in shape. Seen in retinal disease, temporal and parietal lesions, migraine (the Alice-in-Wonderland complex) and, rarely, epilepsy.

12.3 Illusions: a real stimulus misperceived

Definition. An **illusion** is a misperception, a false perception, of a real external stimulus, in which the actual object is transformed into a different percept (Sims 2015). Illusions occur in the healthy (a coat on a door mistaken for an intruder in poor light) and are not in themselves pathological; the classical trigger conditions are reduced sensory clarity, inattention and strong affect.

Completion illusion

Depends on inattention: the mind fills in gaps, so misprints are read as the intended word, e.g. "-ook" with the first letter faded is read as "book" when the word was "look". Abolished by attention.

Affect illusion

Arises out of a particular mood state: a bereaved person momentarily "sees" the deceased, or a delirious, frightened patient perceives innocent gestures as threatening.

Pareidolia

Vivid illusions produced without effort, indeed sometimes against will, from ambiguous stimuli, e.g. seeing faces or figures in clouds or in the embers of a fire. Unlike the other two, pareidolia is intensified, not abolished, by attention.

12.4 Hallucinations: definition and modality classification

Definition. A **hallucination** is a percept experienced in the absence of any external stimulus, arising in external objective space, with the full force and substantiality of a true perception, and not subject to conscious manipulation (Jaspers 1913, Fish 2007). It is not a distorted image, not a misperception, and not under voluntary control. Hallucinations are classified first by sensory **modality**.

Auditory (elementary)

Unstructured or rudimentary noises: buzzing, tapping, banging, music, whistling. Common in organic states and early psychosis.

Auditory (second-person)

Voices addressing the patient directly ("you are worthless"). Second-person derogatory or command voices are typical of severe depression; command hallucinations carry risk.

Auditory (third-person)

Voices discussing or arguing about the patient in the third person ("he is a sinner"), or a **running commentary** narrating the patient's actions as they occur. These are Schneiderian first-rank symptoms, classically of schizophrenia.

Visual

From elementary flashes (photopsiae) to fully formed figures and scenes. Prominent, well-formed or Lilliputian visual hallucinations point strongly toward an organic cause: delirium, occipital or temporal lesions, drug intoxication and withdrawal.

Olfactory and gustatory

Hallucinations of smell and taste, often unpleasant (burning rubber, rotten flesh). Raise suspicion of temporal lobe epilepsy (uncinate fits) and other organic disease; also seen in psychotic depression as congruent themes of decay.

Somatic and tactile

Superficial (haptic, on or under the skin), kinaesthetic (of limb movement) and visceral (deep, of the internal organs). **Formication** is the tactile hallucination of insects crawling on or under the skin, classically in cocaine and alcohol-related states ("cocaine bugs") and delirium tremens.

12.5 Special named hallucinations: functional and reflex

Two easily confused stimulus-bound types. Both involve a real stimulus, but the relation differs, and this discrimination is high-yield.

Functional hallucination

A real external stimulus is *necessary* to provoke the hallucination, and the normal perception of that stimulus and the hallucination are experienced *together in the same modality*, e.g. hearing the voice of God only while, and each time, a tap runs or a clock ticks, the running water still heard as running water alongside the voice (Fish 2007). Unlike an illusion, the voice is not an elaboration of the sound; the sound merely triggers a separate hallucination.

Reflex hallucination

A stimulus in one sensory modality triggers a hallucination in a *different* modality, e.g. feeling a cold sensation in the spine on hearing a fingernail scratch a blackboard, or seeing a flash when a bell rings. A pathological cross-modal analogue of synaesthesia.

12.6 Special named hallucinations: extracampine, autoscopy and the physiological types

Extracampine hallucination

A hallucination experienced *outside the limits of the possible sensory field*, e.g. hearing voices talking from another city hundreds of miles away, or seeing a figure standing behind the head, beyond the field of vision. May occur in schizophrenia and in organic and epileptic states; not to be confused with the **extracampine** being merely "distant".

Autoscopy (phantom mirror image)

The experience of seeing oneself and knowing that it is oneself, projected into external space. In *negative* autoscopy the subject looks in a mirror and sees no image; in *internal* autoscopy the subject perceives their own internal organs. Seen in delirium, epilepsy, parietal lesions and dissociative states.

Hypnagogic hallucination

A hallucination (visual, auditory or tactile) occurring while *falling asleep*. A physiological, non-pathological phenomenon in the healthy; when frequent and vivid, part of the narcolepsy tetrad.

Hypnopompic hallucination

The same phenomenon occurring on *waking*. Rarer than hypnagogic; likewise usually of no pathological significance in isolation.

> **COMMON TRAP**
>
> Do not confuse the functional hallucination with the reflex hallucination. In a **functional hallucination** the trigger and the hallucination share the *same* modality and are heard together (tick plus voice, both auditory); in a **reflex hallucination** the trigger is in *one* modality and the hallucination in *another* (sound triggering a bodily sensation). Same modality equals functional; cross modality equals reflex.

12.7 Pseudohallucination: the inner-space percept

Definition (Jaspers). A **pseudohallucination** is a clear and vivid mental image that, although experienced in full consciousness, *lacks the full substantiality of a true perception*, is located in *inner subjective space* rather than external objective space, is known by the subject not to be a real perception, and so leaves insight *relatively preserved* (Jaspers 1913, Sims 2015). It is more vivid and involuntary than ordinary imagery but less concrete and less "real" than a true hallucination, occupying the ground between them.

Two usages, a caution. Kaplan and Sadock note the term is used in two ways: the Jaspersian sense above (perceived within the mind, in inner space), and a looser sense of a hallucination whose reality the patient doubts, for which "partial hallucination" is the better label (Kaplan & Sadock 2024). For the exam, lead with the Jaspers definition. Pseudohallucinations are common in grief, dissociative states and post-traumatic conditions and, unlike true hallucinations, are not by themselves evidence of psychosis.

12.8 Discriminating the four: the summary table

Read down the discriminators. The whole topic reduces to five axes: presence of a real stimulus, locus of the experience, whether it can be controlled, its reality-status, and insight.

AXIS	SENSORY DISTORTION	ILLUSION	PSEUDOHALLUCINATION	HALLUCINATION
Real external stimulus	Yes (percept altered)	Yes (misperceived)	**No**	**No**
Locus	External space	External space	**Inner subjective space**	**External objective space**
Voluntary control	No	No	Involuntary but image-like	No
Reality-status (substantiality)	Real object, altered	Real object, transformed	**Lacks full substantiality**	**Full force of true percept**
Insight	Usually retained	Corrected on attention	**Relatively preserved**	**Typically lost**

The four levels separate cleanly on stimulus, locus, reality-status and insight; the pseudohallucination is the diagnostic pivot between imagery and true hallucination.

HOLD THIS

Illusion = real stimulus misperceived; hallucination = percept without a stimulus, in outer space, full reality, insight lost; pseudohallucination = inner space, lacks full substantiality, insight preserved. A pseudohallucination is not itself evidence of psychosis.

CLINICAL ANCHOR

Modality guides aetiology at the bedside. Third-person and running-commentary auditory voices steer toward schizophrenia; second-person derogatory voices toward severe depression; well-formed visual, olfactory, gustatory and vivid tactile (formication) hallucinations should trigger an *organic* workup (delirium, epilepsy, substance intoxication or withdrawal) before a primary psychotic label. This is descriptive triage, not a diagnosis named to the patient.

PEARL

First-rank auditory hallucinations (third-person voices, running commentary) are *suggestive* of schizophrenia but are **not pathognomonic**: they occur in affective psychosis and organic states, and the modern consensus (reflected in DSM-5 dropping their special weighting) is that they are not diagnostically decisive. Form over content still holds, but form alone does not fix the diagnosis.

MNEMONIC

"FREAK-Hy": the special hallucinations

Functional, **Re**flex, **E**xtracampine, **A**utoscopy, and the physiological **K** (hypnagogic and hypnopompic) plus **Hy**pnopompic, the named non-modality types to rattle off when asked to enumerate special hallucinations.

GOOD TO REMEMBER

- **Perception:** organisation and interpretation of sensory data into a meaningful representation of the world; more than raw sensation.
- **Sensory distortion:** a real percept altered in intensity, quality or spatial form.
- **Hyperaesthesia / hypoaesthesia:** increased / decreased intensity of perception (mania, migraine / delirium, depression).
- **Chromatopsia:** altered colour quality, e.g. xanthopsia (yellow), chloropsia (green), erythropsia (red); usually toxic or drug-induced.
- **Dysmegalopsia:** altered perceived size or shape: micropsia (smaller), macropsia/megalopsia (larger), metamorphopsia (distorted shape).
- **Illusion:** a misperception of a real external stimulus; can be normal.
- **Completion illusion:** gap filled by inattention (misprints read as intended word); abolished by attention.
- **Affect illusion:** misperception driven by mood (bereaved "sees" the deceased).
- **Pareidolia:** effortless vivid illusions from ambiguous stimuli (faces in clouds/fire); intensified by attention.
- **Hallucination:** a percept with no external stimulus, in external space, with full force of a true perception, not controllable; insight typically lost.
- **Auditory (elementary / second-person / third-person / running commentary):** noises / voices to the patient / voices about the patient and narration of actions; third-person and running commentary are Schneiderian first-rank.
- **Visual / olfactory / gustatory hallucinations:** well-formed forms of these raise suspicion of an organic cause (delirium, temporal lobe epilepsy, drugs).
- **Formication:** tactile hallucination of insects on/under the skin ("cocaine bugs"); cocaine, alcohol withdrawal, delirium tremens.
- **Functional hallucination:** a real stimulus is needed and is perceived together with the hallucination in the *same* modality (voice heard as a tap runs).
- **Reflex hallucination:** a stimulus in one modality triggers a hallucination in a *different* modality (sound producing a bodily sensation).
- **Extracampine hallucination:** experienced outside the limits of the possible sensory field (voices from another city; figure behind the head).
- **Autoscopy (phantom mirror image):** seeing oneself, knowing it is oneself; negative and internal variants; organic/dissociative.
- **Hypnagogic / hypnopompic hallucinations:** physiological, on falling asleep / on waking; part of the narcolepsy tetrad; usually non-pathological.
- **Pseudohallucination:** vivid image in inner subjective space, lacking full substantiality, insight relatively preserved (Jaspers); not itself evidence of psychosis.
- **First-rank symptoms:** suggestive of schizophrenia but *not* pathognomonic (occur in affective and organic psychoses; de-weighted in DSM-5).
- **Mechanism note:** theories of hallucination (perceptual-release, aberrant salience, predictive coding) are covered in the General and Developmental Psychology notes, not here.

13 · Disorders of thought

The **disorders of thought** are the descriptive psychopathology of thinking as it is inferred from speech, and they are examined most cleanly when parcelled into four classic headings: disorders of the **stream** (tempo and continuity), of the **form** (the structure and connectedness of thinking), of the **content** (what is being thought), and of the **possession** (the sense of ownership and control of one's own thoughts). This carving is the one used in the classical texts (Fish 2007, Sims

2015) and in Indian teaching (Clinical Methods in Psychiatry, AIIMS 2024), and it is a perennial Paper I favourite because it rewards precise definitions and clean discriminations, not mechanism.

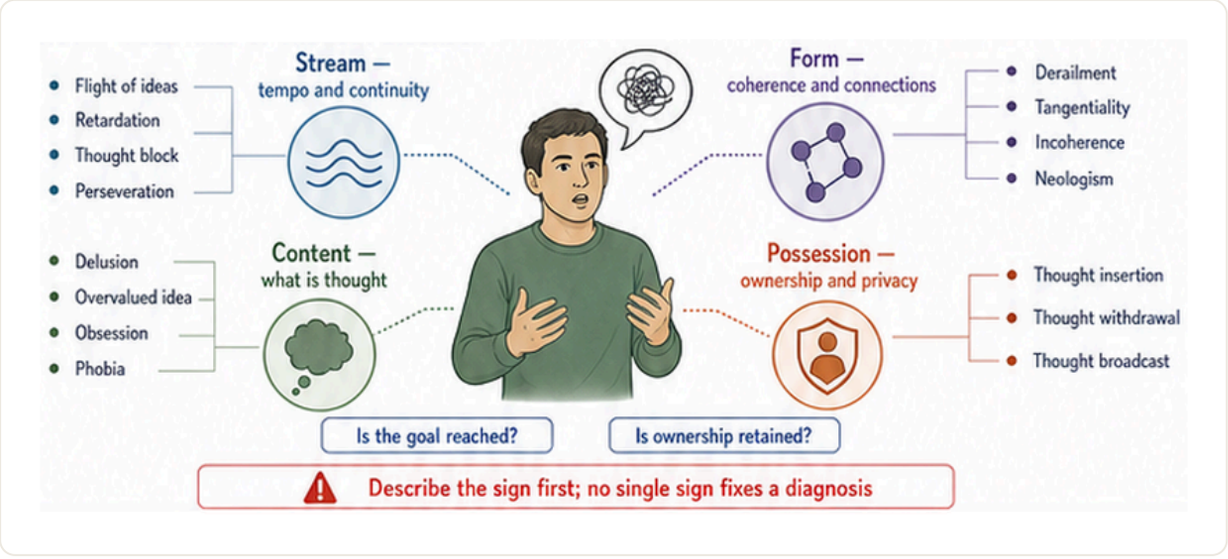


Fig 6.19 Four axes of thought disorder.

Exam framing. Keep the four axes rigidly apart, because the same clinical phenomenon can be pigeonholed differently by different authors (flight of ideas is a disorder of stream to Fish but of form to some; circumstantiality is placed under stream by Sims). Teach the **description**, not the explanation, form over content, in the tradition of Jaspers, Fish and Sims (Jaspers 1913). Thinking is never observed directly: it is read off speech, so a "thought disorder" is always an inference from disordered language. Delusions, though a disorder of content, are dealt with in the next topic and are only signposted here; the possession disorders are the Schneiderian first-rank symptoms and are cross-referred to the perception and first-rank notes.

13.1 The four-axis framework and how to place any sign

Orientation. Any abnormality of thinking can be located by asking four questions in turn: is the *speed or continuity* of the flow abnormal (stream), is the *logical structure and connectedness* lost (form), is *what* is being thought abnormal (content), or is the patient's *sense of owning and controlling* the thought disturbed (possession). Stream is about tempo and flow, form is about coherence, content is about the ideas themselves, possession is about ego-boundary. Hold this scaffold and the named signs below slot in without confusion. A useful cross-cut, from Andreasen's work on the Scale for the Assessment of Thought, Language and Communication (TLC), divides formal disturbances into *positive* (a wealth of disorganised output: derailment, incoherence, neologisms) and *negative* (impoverished output: poverty of speech and of content) (Andreasen 1979).

13.2 Disorders of the stream: tempo of thought

Definition. Disorders of the stream (or tempo) concern the *rate and pressure* of the flow of thought, either accelerated or slowed, independent of whether the logical links are preserved (Fish 2007).

Flight of ideas

The manic acceleration of thinking: a rapid, pressured stream in which thoughts follow each other by *chance associations*, and where the goal of thinking is repeatedly lost. The direction is set by external distraction and by verbal associations such as assonance, alliteration, rhyme, punning and **clang associations** (word choice governed by sound not meaning). Crucially, the links remain *understandable*: one can often follow, or even reverse, the train (Sims 2015). Seen in mania and hypomania.

Prolixity (ordered flight)

A milder, more ordered variant of flight of ideas seen in hypomania: the stream is over-productive and diffuse but the patient does eventually reach the goal.

Retardation / inhibition of thinking

Slowing of the stream: the train of thought is decelerated, the number of ideas reduced, and the patient experiences difficulty in thinking; seen in depression and in retarded psychomotor states.

Poverty of thought

A reduction in the *amount* of thought, reflected as poverty of speech (brief, unelaborated, monosyllabic replies) or poverty of content (adequate quantity of speech conveying little information); a negative disturbance seen in the negative syndrome of schizophrenia and in depression.

13.3 Disorders of the stream: continuity of thought

Definition. These concern *breaks and repetitions* in the continuity of the flow rather than its speed (Companion to Psychiatric Studies).

Thought block

An *abrupt, complete cessation* of the stream of thought, experienced by the patient as the mind suddenly going blank ("snapping off"), with no thought to replace it; on resuming, the patient often takes up a fresh, unrelated topic. When subjectively distressing and unexplained, it carries weight toward schizophrenia. Distinguish from the ordinary "losing one's thread", which is explicable and recoverable.

Perseveration

Senseless persistence of a word, phrase or idea *beyond the point at which it is relevant*, so that once a subject or word is begun the patient repeatedly returns to it despite a change of question. A pointer to organic brain disease, especially dementia and frontal lesions.

Circumstantiality

Speech that is over-inclusive and indirect, laden with tedious and parenthetical detail, so that reaching the goal idea is greatly delayed; but the goal *is eventually reached*. This is the key discriminator from derailment and from tangentiality, where the goal is never reached. Common in obsessional and epileptic personalities, low intelligence, and, at a non-pathological level, in normal "long-winded" people.

13.4 Disorders of the form: formal thought disorder and its concept

Definition. **Formal thought disorder** denotes a disturbance of the *form* of thought: the loss of the normal structure, logical coherence and connectedness of thinking, the "how" or mechanics of thought rather than its content, which makes conversation hard or impossible to follow and is a cardinal feature of schizophrenia (Companion to Psychiatric Studies, AIIMS 2024). Because

thinking is inferred only from language, the terms "thought disorder" and "speech disorder" are sometimes used interchangeably. Healthy thinking has been summarised by the "3 Cs": *constancy*, *clear organisation* and *continuity*; deviation in any is a formal disturbance (AIIMS 2024). Historically several theorists each proposed a core defect, summarised below.

Kraepelin: akataphasia

An early term for the disordered, ungrammatical expression of thought.

Bleuler: loosening of associations

The founding concept: a loosening of the associative threads that normally bind thoughts together, held by Bleuler to be the fundamental disturbance of schizophrenia.

Cameron: asyndesis and overinclusion

Asyndesis is a lack of adequate connections between successive thoughts (a breakdown of the "cementing" links); **overinclusion** is the inability to keep the boundaries of a concept, so that irrelevant material is drawn in.

Goldstein: concrete thinking

Loss of the abstract attitude, so the patient thinks in literal, concrete terms and cannot handle concepts; tested by proverb interpretation and similarities, and seen in schizophrenia and organic states.

Schneider: the five features

Schneider described derailment, substitution, omission, fusion and drivelling as the elementary faults underlying disordered thinking.

13.5 Disorders of the form: the descriptive signs

The named phenomena. These are the signs actually elicited at the bedside, each a way in which the connectedness of speech breaks down (Fish 2007, Sims 2015, Andreasen 1979).

Loosening of associations / derailment

The ideas slip off the track onto another that is obliquely related or wholly unrelated; the "train" of thought leaves the "rails". Meaning is lost gradually over several sentences (contrast word salad, where individual sentences are already meaningless).

Knight's move thinking

A term for a sharp, illogical, oblique transition between ideas, likened to the chess knight that never moves in a straight line; used near-synonymously with severe derailment.

Tangentiality

Replies that veer away from the point and, unlike circumstantiality, *never return* to the goal idea; the answer is obliquely related to the question but never actually answers it.

Word salad (schizophasia, incoherence)

Speech so disordered that it is essentially incomprehensible: words and phrases are strung together with the rules of grammar and syntax ignored, so that even individual sentences carry no meaning.

Neologisms

The invention of genuinely new words with no established meaning (e.g. "tarn-harn"); to be distinguished from *word approximations*, where existing words are used in an idiosyncratic, quasi-logical way ("a menu three times a day" for meals).

Verbigeration

The automatic, stereotyped repetition of words, phrases or sentences "like a scratched record", a verbal stereotypy; differs from echolalia, which repeats another person's words.

Concrete thinking

See Goldstein above: literal, non-abstract interpretation, e.g. giving the literal meaning of a proverb.

Asyndesis

See Cameron above: a lack of the connecting links between one thought and the next.

13.6 Distinguishing the three "goal" disorders

Read down the discriminator column. Circumstantiality, tangentiality and derailment are constantly confused; they separate on two axes, whether the goal is reached and whether the digressions are logically related.



Fig 6.20 Does the speech reach its goal?.

SIGN	REACHES THE GOAL IDEA?	NATURE OF DIGRESSION	TYPICAL SETTING
Circumstantiality	**Yes, eventually (after much detail)**	Over-inclusive but relevant detail	Obsessional / epileptic personality, low IQ
Tangentiality	**No, never returns**	Obliquely related, drifts off	Schizophrenia, organic states
Derailment (loosening)	**No, meaning lost over sentences**	Slips onto unrelated track	Schizophrenia (positive FTD)

The single most reliable discriminator is whether the goal idea is finally reached: only circumstantiality reaches it.

RULE **Does the patient reach the goal?** Circumstantiality reaches it after a long detour; tangentiality and derailment never do - the single cleanest discriminator in the topic.

13.7 Disorders of the content of thought

Definition. Content is *what* the person is thinking; its disorders are the abnormal ideas that dominate the mind, graded by the degree to which they are held with conviction and resisted (Sims 2015). Delusions, the most important, are taught in full in the next topic and are only listed here.

Preoccupations

Ordinary thoughts that recur with abnormal intensity or frequency (worry over illness, somatic preoccupation in depression and anxiety); ego-syntonic and not resisted.

Overvalued idea

A comprehensible, non-delusional belief that comes to dominate the person's life and is pursued with abnormal intensity, but which is not held with true delusional conviction and is not utterly beyond reason (e.g. the litigious querulant, the morbidly jealous, the dysmorphophobic). Sits between a normal belief and a delusion.

Obsessions and compulsions

Obsessions are recurrent, intrusive, unwanted thoughts, images or impulses, recognised as one's own (contrast thought insertion) and as senseless, and actively resisted, generating anxiety; **compulsions** are the repetitive mental or motor acts performed to neutralise them. Ego-dystonic, insight typically retained.

Phobias

Persistent, irrational fears of a specific object or situation, recognised as excessive, leading to avoidance.

Delusions

Fixed, false, unshakeable beliefs out of keeping with the person's culture, held with absolute conviction; the full taxonomy is in the next topic.

13.8 Disorders of the possession of thought

Definition. **Thought possession** is the normal sense that one's thoughts are *one's own* and are *under one's own control*. Its disorders are disturbances of this ownership and boundary: at one pole the obsessional experiences unwanted thoughts as intrusive yet still recognises them as self-generated; at the other, the patient with schizophrenia attributes thoughts to a foreign agency or feels them stolen or made public. This group, "thought alienation", comprises three named signs of disordered thought possession that are *Schneiderian first-rank symptoms* (cross-refer to the first-rank note): thought insertion, thought withdrawal and thought broadcast.

Thought insertion

The experience that thoughts *not one's own* have been placed into one's mind by an external agency; in thought insertion the key is the loss of the sense of *ownership*, the patient insisting the thoughts are alien, not merely unwanted (distinguishing it from an obsession).

Thought withdrawal

The experience, thought withdrawal, that one's thoughts are being *removed or extracted* from the mind by an external force, often reported together with the sudden emptiness of thought block.

Thought broadcast

In thought broadcast the experience is that one's thoughts *escape the confines of the self* and are known to, or shared with, others (broadcast, diffused), without the patient speaking them aloud; a disorder of thought, to be distinguished from the auditory hallucination of *thought echo* (hearing one's own thoughts spoken aloud).

COMMON TRAP

Do not confuse an **obsession** with **thought insertion**. Both are unwanted intrusive thoughts, but the obsessional patient *knows the thought is his own* (ego-dystonic but self-owned) and resists it, whereas in thought insertion *ownership itself is lost*: the patient is convinced the thought was put there by an outside agency. Ownership retained equals obsession; ownership lost equals thought insertion, a first-rank symptom.

HOLD THIS

Thought disorder splits four ways: stream (tempo), form (coherence), content (the ideas), possession (ownership). Two settling questions - is the goal reached? is ownership retained? - resolve most of the confusions.

13.9 Aetiological pointers and the modern caveat

Description first, then the pattern. The signs are defined descriptively, but each carries a soft aetiological steer worth holding at the bedside (Companion to Psychiatric Studies). Flight of ideas, clang associations and pressure of speech point to mania; poverty of thought and content to negative-syndrome schizophrenia or depression; perseveration, stereotypy and echolalia to organic brain disease; positive formal thought disorder (derailment, incoherence, neologisms) and the possession disorders to schizophrenia. But two cautions are decisive for the exam. First, formal thought disorder is *not specific* to any one diagnosis and overlaps heavily across stream and form. Second, the thought-possession signs, though first-rank, are *not pathognomonic* of schizophrenia: they occur in affective and organic psychoses, and the modern consensus, reflected in DSM-5 dropping the special weighting of first-rank symptoms, is that they are not diagnostically decisive. Form over content still holds as a descriptive discipline, but form alone does not fix the diagnosis.

CLINICAL ANCHOR

At the bedside the four axes map onto four bedside manoeuvres: for *stream*, listen to the rate and continuity of spontaneous speech; for *form*, ask an open question and judge whether you can follow the reply; for *content*, ask directly about worries, obsessions and beliefs; for *possession*, ask "are your thoughts your own, can others read or take them, are thoughts put into your mind?". This is descriptive triage, never a diagnosis named to the patient.

PEARL

The cleanest single discriminator in the whole topic: *does the patient reach the goal?* Circumstantiality reaches it after a long detour; tangentiality and derailment never do. And the cleanest boundary sign: *is ownership of the thought retained?* Yes in obsessions, no in thought insertion. Two questions settle most of the confusions examiners set.

MNEMONIC

"S-F-C-P": Stream, Form, Content, Possession

Four axes in order of increasing "depth" from surface tempo to the ego-boundary: **S**tream (speed and flow: flight of ideas, block, perseveration, circumstantiality), **F**orm (coherence: derailment, word salad, neologisms), **C**ontent (the ideas: overvalued ideas, obsessions, delusions), **P**ossession (ownership: insertion, withdrawal, broadcast).

GOOD TO REMEMBER

- **Disorders of thought:** classified into stream (tempo/continuity), form (coherence), content (the ideas) and possession (ownership/control).
- **Flight of ideas:** pressured accelerated stream, chance associations by rhyme, punning and clang, distractible, but links remain understandable; mania.
- **Prolixity:** ordered flight of ideas of hypomania that eventually reaches the goal.
- **Retardation of thinking:** slowed stream, fewer ideas; depression.
- **Poverty of thought:** reduced amount of thought (poverty of speech / of content); negative schizophrenia, depression.
- **Thought block:** abrupt complete cessation of the stream, mind goes blank ("snapping off"); weight toward schizophrenia.
- **Perseveration:** senseless repetition of a word/idea beyond its relevance; points to organic brain disease.
- **Circumstantiality:** over-inclusive, detour-laden speech that *does* eventually reach the goal idea.
- **Formal thought disorder:** loss of the structure and connectedness of thinking, the "how" of thought; cardinal in schizophrenia.
- **Kraepelin:** akataphasia (disordered, ungrammatical expression of thought).
- **Bleuler:** loosening of associations, the fundamental disturbance of schizophrenia.
- **Cameron:** asyndesis (lack of connecting links) and overinclusion (loss of concept boundaries).
- **Goldstein:** concrete thinking (loss of the abstract attitude).
- **Schneider:** derailment, substitution, omission, fusion, drivelling.
- **Derailment / loosening:** ideas slip off the track; meaning lost over sentences.
- **Knight's move thinking:** sharp illogical oblique transitions, like the chess knight; near-synonym of severe derailment.
- **Tangentiality:** replies that veer off and *never* return to the goal (contrast circumstantiality).
- **Word salad (schizophasia):** incomprehensible speech, syntax ignored, individual sentences meaningless.
- **Neologisms:** genuinely new invented words; distinct from word approximations.
- **Verbigeration:** automatic stereotyped repetition of phrases "like a scratched record"; differs from echolalia.
- **Overvalued idea:** comprehensible non-delusional belief dominating life, held short of delusional conviction.
- **Obsessions / compulsions:** intrusive unwanted self-owned thoughts, resisted, ego-dystonic; neutralising acts.
- **Thought possession:** the sense of owning and controlling one's thoughts; its disorders are thought alienation.
- **Thought insertion:** alien thoughts placed in the mind by an external agency; loss of *ownership* (first-rank).
- **Thought withdrawal:** thoughts extracted from the mind by an external force (first-rank).
- **Thought broadcast:** thoughts escape the self and are known to others; a disorder of thought, not thought echo (first-rank).
- **Andreasen (1979):** TLC scale; positive vs negative formal thought disorder.
- **First-rank caveat:** possession disorders are first-rank but *not* pathognomonic; DSM-5 dropped their special weighting.

14 · Understanding delusions

The delusion is the cardinal disturbance of thought content and, for the phenomenologist, the most examined sign in all of descriptive psychopathology. The exam does not want a lay

definition, it wants the Jaspersian architecture: what makes a belief delusional in **form** rather than in content, how the un-understandable **primary delusion** is separated from the merely secondary one, and how the delusion is discriminated from the **overvalued idea** and the obsession. Ground your description in Jaspers (General Psychopathology, 1913), Fish (Clinical Psychopathology) and Sims (Symptoms in the Mind), the three texts every Indian examiner reaches for.

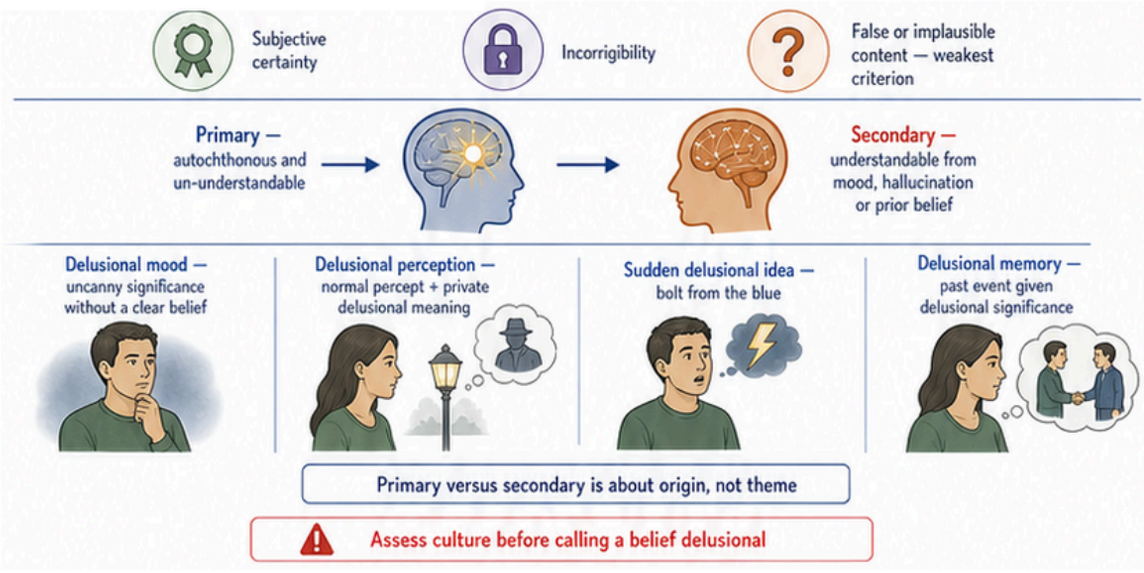


Fig 6.21 Primary delusional experience.

Describe, do not explain. Jaspers' discipline is to characterise the abnormal experience by its form (a fixed belief held with absolute certainty) and to resist the temptation to explain it by its content (why the patient believes he is persecuted). The whole classification below is a classification of form and origin, not of theme alone.

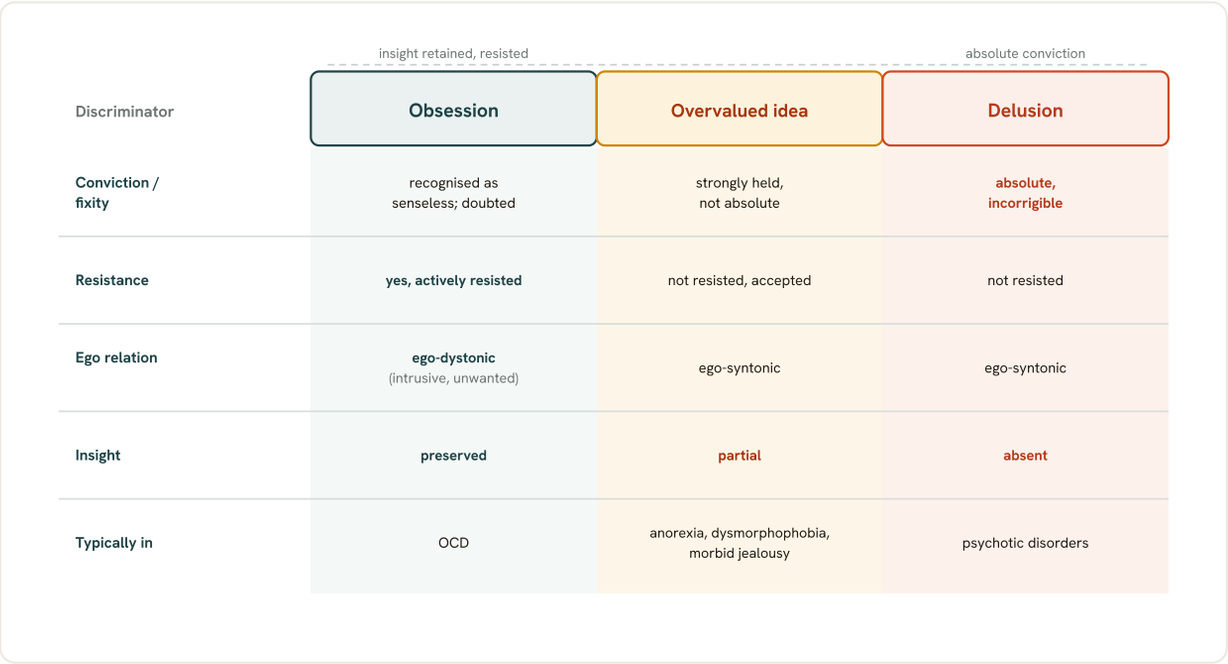


Fig 6.22 Delusion, overvalued idea and obsession compared. Read left to right as a gradient. An obsession is ego-dystonic, resisted, and held with retained insight; a delusion is ego-syntonic, unresisted, held with absolute conviction and no insight; the overvalued idea sits in between, a comprehensible belief held strongly but not with delusional fixity. Insight and resistance fall as conviction rises across the three.

14.1 Definition and the three Jaspersian criteria

Definition. A delusion is a fixed, false belief, held with absolute conviction, out of keeping with the person's social, educational and cultural background, and not amenable to reason or contrary evidence (Sims 2015, Kaplan & Sadock 2024). Jaspers gave three external criteria by which a delusion can be recognised.

Subjective certainty

Held with extraordinary, incorrigible conviction, an absolute subjective certainty far beyond ordinary belief.

Incorrigibility

Not amenable to reason, logic, argument or contrary experience; counter-evidence is re-interpreted rather than accepted.

Impossibility or falsity of content

The content is false, impossible or at least implausible; note this is the weakest criterion, since a delusion may occasionally be true (see the pitfall below).

COMMON TRAP

Falsity is NOT the defining feature. A morbidly jealous man may be delusional even if his wife happens to be unfaithful: the belief is delusional by the disordered mental process that produced it, not by whether it turns out true (Shorter Oxford). The other trap is culture: a belief widely shared within the patient's religious or cultural group is not a delusion, so always ask whether peers hold it.

14.2 The primary versus secondary split (the Jaspersian core)

The distinction is by origin, not theme. Jaspers separated delusions according to whether they can be understood as arising from something else.

primary delusion

Autochthonous (arising from within), it appears suddenly, fully formed, with full conviction and no discernible mental events leading up to it; it is un-understandable, meaning it cannot be derived from mood, personality or another experience. Given great diagnostic weight in schizophrenia.

secondary delusion

Understandable (in Jaspers' sense) as arising out of a preceding morbid experience: a mood state, a hallucination, or a pre-existing delusion. A profoundly depressed woman believes she is worthless; a man who hears accusing voices concludes he is being followed.

Secondary delusions may accumulate and interconnect into a **systematised** delusional system. Primary delusions are recorded only when certainly present, because of their diagnostic force.

RULE **The primary versus secondary split is by origin, not theme.** A primary delusion is un-understandable and autochthonous; a secondary one is understandable as arising from mood, a hallucination or a prior delusion.

14.3 The primary delusional experiences

Not all primary experiences begin as an idea. Jaspers and Schneider described four forms of primary delusional experience; the examiner wants each defined crisply.

delusional mood (Wahnstimmung)

An uncanny, uneasy sense that the world has subtly changed and that something of immense significance is about to be revealed; the patient feels "something sinister is going on but I cannot say what." This delusional atmosphere provides the context from which fully formed delusions crystallise.

delusional perception

A real, normally perceived object is given a private, unwarranted delusional meaning (a curtain drawn, leaves turning yellow, become a signal that he is the chosen one). Schneider's two-memberedness: link one is the normal percept, link two is the delusional significance. A first-rank symptom of schizophrenia.

Sudden delusional idea (autochthonous idea, delusional intuition)

A fully formed delusional belief arrives in a single stage, out of the blue, with no preceding perception, "a bolt from the blue" (distinguished from delusional perception, which has two stages: percept then meaning).

Delusional memory (delusional retrospective falsification)

A delusional significance is attached to a real past event, or an autochthonous delusion is falsely dated to the past so the memory itself is the delusion (Fish).

CLINICAL ANCHOR

A 25-year-old man saw the yellowing leaves of a tree outside his house (a true, normal percept) and was instantly and unshakeably certain this was a signal from God that he alone was the chosen one, with no prior delusional system. That is a textbook delusional perception, not a misinterpretation of a pre-existing belief.

14.4 Delusional perception as a first-rank symptom (and the modern caveat)

First-rank status. Schneider listed delusional perception among the first-rank symptoms of schizophrenia, alongside audible thoughts, voices arguing or commenting, passivity of affect, impulse and volition, somatic passivity, thought insertion, withdrawal and broadcast. It is a disorder of thought, not of perception: the percept is entirely normal, only the meaning is deluded.

PEARL

The currency point examiners reward: first-rank symptoms are NOT pathognomonic of schizophrenia (they occur in mania, psychotic depression and organic states) and neither ICD-11 nor DSM-5 gives them privileged diagnostic weight any longer. Delusional perception remains the phenomenologically purest FRS, but it is a pointer, not proof.

14.5 Delusions classified by content, part one: the affect-linked themes

By theme. Once origin is settled, delusions are catalogued by content. Content carries some diagnostic hint but is defined descriptively.

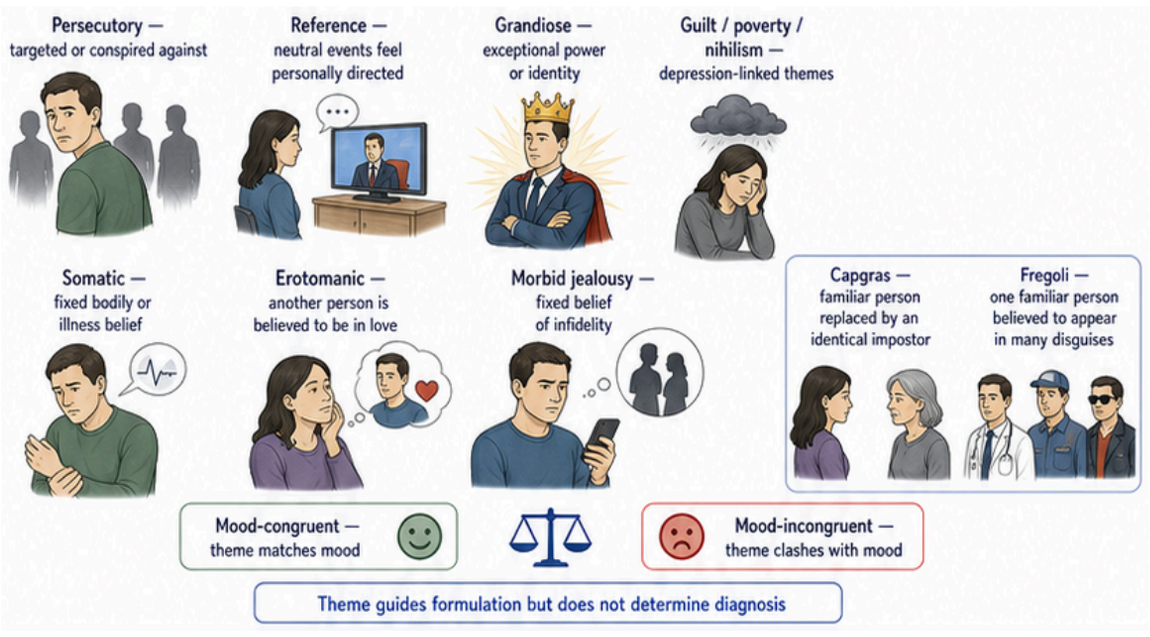


Fig 6.23 Delusions: theme, mood and misidentification.

persecutory (paranoid)

The conviction of being targeted, harassed, cheated, spied on or conspired against; the commonest theme, seen across schizophrenia, affective psychoses and organic states.

Delusion of reference

Events, objects, remarks, gestures or media items (a newspaper article, a stranger's cough) are believed to carry a special personal meaning directed at the patient.

grandiose delusion (expansive)

Inflated belief in one's own power, identity, wealth, talent or special mission; characteristic of mania and of grandiose-type delusional disorder.

Religious delusion

A delusion with religious content (being God, the chosen one, damned); judged delusional only when it exceeds the patient's faith community's shared beliefs.

Delusion of guilt (self-accusation)

Unfounded conviction of having committed a terrible sin or crime deserving punishment; a mood-congruent feature of severe depression.

Nihilistic delusion (Cotard)

Belief that oneself, a body part, others or the world has ceased to exist, is dead, rotting or has no blood or bowels; described by Cotard (1882), typically in severe depression.

14.6 Delusions classified by content, part two: body, love, jealousy, identity, control

Hypochondriacal (somatic) delusion

Fixed false belief of harbouring a serious illness (cancer, AIDS, infestation) despite reassurance; the core of monosymptomatic hypochondriacal psychosis.

Amorous delusion (erotomania, de Clerambault's syndrome)

The unshakeable conviction that another person, usually of higher status, is secretly in love with the patient.

Morbid jealousy (Othello syndrome, delusion of infidelity)

Delusional certainty that the partner is being unfaithful, driving relentless checking and interrogation; carries a real risk of violence.

Capgras delusion (illusion of doubles)

Belief that a familiar person (usually close) has been replaced by an identical impostor or double; a delusional misidentification.

Fregoli delusion

Belief that a familiar persecutor is disguising himself as, or masquerading behind, various strangers the patient meets; the mirror image of Capgras.

Delusions of control and passivity

The patient experiences his own actions, impulses, thoughts or affect as made or controlled by an external agency (passivity phenomena, first-rank symptoms).

COMMON TRAP

Capgras is the double (replacement of the familiar by an impostor); Fregoli is the disguise (a single persecutor hiding inside many strangers). Do not swap them. Both are delusional misidentification syndromes, phenomenological symptoms that can also present as named syndromes.

14.7 The discrimination the exam loves: delusion versus overvalued idea versus obsession

The classic viva triad. An **overvalued idea** (Wernicke 1900, reviewed by McKenna 1984) is a comprehensible, understandable, isolated belief, strongly held with intense affect and pursued beyond the bounds of reason, dominating the person's life, yet not utterly fixed and, crucially, ego-syntonic (the patient does not regard it as senseless and does not resist it). Jaspers held that the overvalued idea differs from a delusion mainly in the level of conviction, and Fish added that the patient with an overvalued idea invariably acts on it, determinedly and repeatedly. The obsession, by contrast, is intrusive, recognised as the patient's own but senseless, ego-dystonic and actively resisted.

FEATURE	DELUSION	OVERVALUED IDEA	OBSESSION
Fixity / conviction	**Absolute, incorrigible**	Strong but not utterly fixed	Doubt present, not fixed
Understandability	Un-understandable (if primary)	**Comprehensible, understandable**	Comprehensible but senseless
Resistance by patient	None	None	**Present, active resistance**
Ego relation	Ego-syntonic	**Ego-syntonic**	Ego-dystonic
Sense of intrusiveness	Absent	Absent	**Present, felt as intrusive**
Insight	Absent	Partial or absent	**Retained (thought is own, senseless)**
Prototype	Persecutory delusion	Anorexia, morbid jealousy	Contamination obsession

The three-way discrimination on fixity, understandability, resistance, ego relation and insight; the diagnostic axis examiners probe most.

HOLD THIS

A delusion is a fixed, false, incorrigible belief out of cultural keeping; falsity is its weakest criterion. The obsession is resisted and ego-dystonic with insight; the overvalued idea is comprehensible and strongly held but not absolutely fixed.

14.8 Mood-congruent versus mood-incongruent delusions

Congruence with prevailing affect. This axis matters for classifying psychotic mood disorder.

Mood-congruent

Content matches the affective state: guilt, nihilism, poverty and hypochondriasis in depression; grandiosity and special powers in mania.

Mood-incongruent

Content is at odds with the mood, for example persecutory or passivity delusions in a euphoric manic patient; their presence carries prognostic and diagnostic weight.

TRAP Mood-incongruent delusional content (a delusion whose theme clashes with the mood) is not the same as incongruity of affect (an emotional display that clashes with the topic) - one is content, the other is expressed affect.

14.9 Bizarre versus non-bizarre delusions

Physical possibility is the axis.

Bizarre delusion

Content is clearly implausible, not understandable to same-culture peers and not derived from ordinary life experience (e.g. one's organs have been removed and replaced without a scar); historically weighted toward schizophrenia.

Non-bizarre delusion

Content, though false, could conceivably occur (being followed, poisoned, deceived by a spouse); the hallmark of delusional disorder.

PEARL

DSM-5 downgraded bizarreness: it removed the old rule that a single bizarre delusion alone met Criterion A for schizophrenia. Inter-rater reliability for judging bizarreness is poor, so lean on the primary versus secondary and delusion versus overvalued idea distinctions, which the examiner scores more reliably.

MNEMONIC

"SII" for the Jaspers triad

Subjective certainty, Incorrigibility, Impossibility (or falsity) of content. For the primary delusional experiences, remember the sequence a patient may pass through: delusional Mood, delusional Perception, sudden delusional Idea, delusional Memory (M-P-I-M).

GOOD TO REMEMBER

- **Delusion (definition):** fixed, false belief, absolute conviction, out of keeping with social and cultural background, not amenable to reason (Jaspers, Sims, K&S).
- **Jaspers' three criteria:** subjective certainty, incorrigibility, impossibility or falsity of content (the last being the weakest).
- **primary delusion:** autochthonous, sudden, fully formed, un-understandable; heavy diagnostic weight in schizophrenia.
- **secondary delusion:** understandable as arising from mood, another experience (hallucination) or a pre-existing delusion; may systematise.
- **delusional mood (Wahnstimmung):** uncanny sense that something significant is about to be revealed; the atmosphere before the delusion crystallises.
- **delusional perception:** real normal percept given a private delusional meaning; Schneider's two-memberedness; a first-rank symptom.
- **Autochthonous (sudden delusional) idea:** fully formed delusion in one stage, "bolt from the blue," no preceding percept.
- **Delusional memory:** delusional meaning attached to a real past event, or delusion falsely dated to the past (Fish).
- **persecutory (paranoid):** conviction of being targeted, harassed or conspired against; commonest theme.
- **Delusion of reference:** ordinary events or remarks carry a special personal meaning aimed at the patient.
- **grandiose delusion:** inflated power, identity, wealth or mission; classic in mania.
- **Religious / guilt / nihilistic:** religious content; unfounded sin deserving punishment; Cotard (1882) belief of non-existence or being dead.
- **Hypochondriacal:** fixed false belief of serious illness despite reassurance.
- **Erotomania (de Clerambault):** conviction a higher-status other secretly loves the patient. **Othello:** delusional infidelity, violence risk.
- **Capgras:** familiar person replaced by an impostor double. **Fregoli:** a persecutor disguised as many strangers.
- **Passivity / control:** actions, impulses, thoughts or affect experienced as externally made; first-rank symptoms.
- **overvalued idea (Wernicke 1900, McKenna 1984):** comprehensible, strongly held, not utterly fixed, ego-syntonic, unresisted, dominates life (anorexia, morbid jealousy).
- **Obsession:** intrusive, recognised as own but senseless, ego-dystonic, resisted, insight retained.
- **Mood congruence:** congruent (guilt, nihilism in depression; grandiosity in mania) versus incongruent (persecution in mania).
- **Bizarre vs non-bizarre:** physically impossible versus conceivable; DSM-5 removed the single-bizarre-delusion shortcut for schizophrenia.
- **Modern caveat:** first-rank symptoms (including delusional perception) are not pathognomonic and lost privileged status in ICD-11 and DSM-5.

15 · Schneiderian first-rank symptoms

The single most examined list in descriptive psychopathology. Kurt **Schneider** (1959), working in the Jaspersian descriptive tradition, proposed a set of **first-rank** symptoms (**symptoms of the first rank**, **Symptome ersten Ranges**) not as a theory of what schizophrenia *is*, but as a pragmatic clinical shortcut: experiences that, when present in the absence of coarse brain disease, let a busy clinician make the diagnosis reliably. He explicitly ranked them above

second-rank symptoms (other hallucinations, perplexity, mood change) and above the affective disorders, so that an organic or schizophrenic picture took precedence over an unmistakable manic or melancholic one (Companion 2010; Sims 2015).

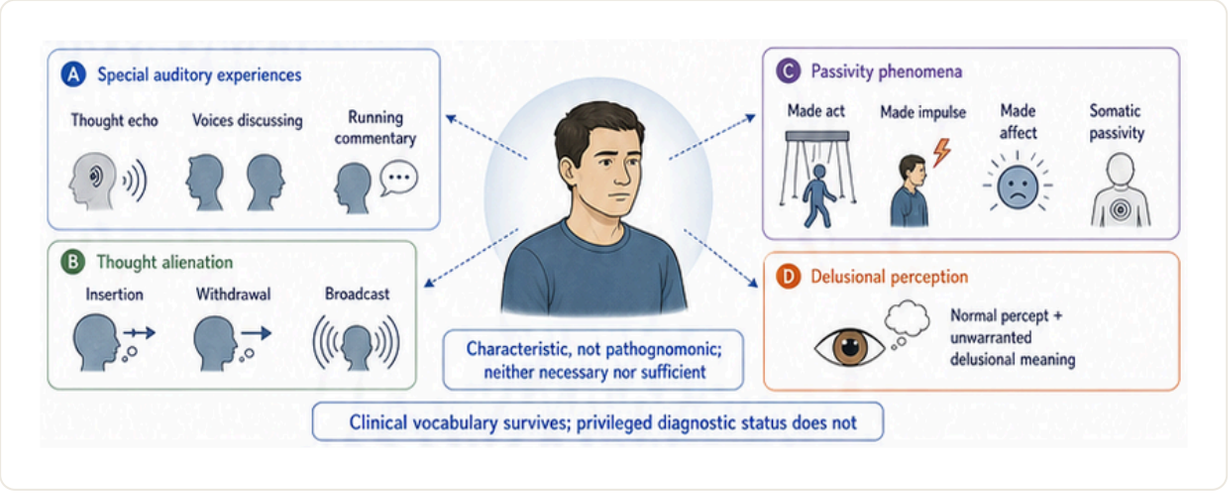


Fig 6.24 Schneiderian first-rank symptoms.

For the exam the two things that must be paired are (1) the crisp list in its classic groups, and (2) the modern caveat. Schneider called them pathognomonic of schizophrenia "except in the presence of coarse brain disease", but they are **not** pathognomonic: they occur in mania and in organic states, and up to a fifth of people with schizophrenia never show any of them (Fish 2007; Kaplan and Sadock 2024). Because of this they have lost their privileged diagnostic status: DSM-5 (2013) removed the rule that a single first-rank symptom sufficed for the A-criterion, and ICD-11 (2022) no longer grants them special weight. Learn them as the highest-yield descriptive vocabulary, not as a diagnostic test.

15.1 What Schneider actually claimed (and the exact caveat)

Pragmatic, not theoretical. Schneider (1959) built the list for reliability, not validity. He wrote that these symptoms are diagnostic of schizophrenia "provided only that there is no question of cerebral disease"; the phrase *coarse brain disease* is the escape clause every examiner wants quoted. He did not believe they revealed a mechanism, only that they were the most characteristic and easily recognised markers available to a clinician at the bedside (Kaplan and Sadock 2024). The three ranks form a hierarchy: first-rank symptoms outrank second-rank symptoms, and both outrank affective symptoms, which is why a Schneiderian diagnostician diagnosed schizophrenia even when florid mood symptoms were present.

RULE **Quote the escape clause: first-rank symptoms point to schizophrenia only "in the absence of coarse brain disease".** Schneider built them for reliability at the bedside, not as a theory of what schizophrenia is.

15.2 Group (a): auditory hallucinations of a special kind

Three specific forms, all auditory. Schneider singled out three verbal auditory hallucinations. These are the ones to define precisely, because "voices" alone is not first-rank.

Thought echo (audible thoughts, Gedankenlautwerden, echo de la pensee)

The patient hears their own thoughts spoken aloud, just before, at the same time as, or immediately after they occur. Example: while washing up, "I thought I must fetch washing powder, and instantly a voice said, I shall go to the market to buy washing powder" (Clinical Methods AIIMS 2024).

Voices arguing or discussing (third person)

Two or more hallucinatory voices discuss or argue about the patient, referring to them in the third person ("he", "she"), as though the patient were absent.

Running commentary

A voice (or voices) gives a running commentary on the patient's actions as they happen: "she is putting salt in excess, she is leaving the food unattended" (Clinical Methods AIIMS 2024).

The discriminator. The form, not the content, is what matters. Third-person and running-commentary voices are first-rank; second-person command hallucinations ("get up and run away") are *not* first-rank, however distressing.

15.3 Group (b): thought alienation

The self-boundary is breached. The unifying experience is loss of the normal sense that one's own thoughts are private and one's own. Cross-refer the thought-disorder topic for fuller treatment; here are the three first-rank members.

Thought insertion

Thoughts that are not the patient's own are experienced as put into their mind by an external agent. The alienness of ownership, not merely the content, is the sign.

Thought withdrawal

The patient experiences thoughts being actively removed or extracted from their mind by an outside force, often felt as a sudden emptying.

Thought broadcast

The patient's thoughts escape and are experienced as accessible to others, as if spoken aloud or transmitted, without any act of speaking. Distinguish from thought echo, which is a hallucination of hearing one's own thoughts, not a leakage to others (Clinical Methods AIIMS 2024).

15.4 Group (c): passivity phenomena (made or influenced experiences)

Volition, impulse, affect and body are hijacked. In **passivity** phenomena the patient experiences their own actions, urges, feelings or bodily sensations as made, imposed or controlled by an external agency: the sense of personal agency (the feeling that "I am the author of this") is lost while the act still occurs. The key words examiners look for are "made" and "as if controlled".

Made act (made volition)

The patient carries out an action but experiences it as not their own; some external agent is felt to be moving them, and they are a passive participant in their own behaviour.

Made impulse (made drive)

An impulse or drive to act is experienced as imposed from outside, so the urge itself is felt to be alien and controlled.

Made affect (made feeling)

Emotions the patient does not own are felt to be produced in them by an external agent: "the feelings are not mine, they are put into me".

Somatic passivity

Bodily sensations (tactile or visceral) are experienced as imposed on the patient by an outside agency, for example a sensation of being irradiated or manipulated from without.

15.5 Group (d): delusional perception

A real percept, an unwarranted delusional meaning. In **delusional perception** a normal, correctly perceived object or event is invested, in a single step, with a highly personal delusional significance that is not logically connected to it: seeing the traffic light turn red and knowing instantly "that means I am the chosen one". Schneider stressed its *two-memberedness* (link one the real perception, link two the abnormal meaning), and Jaspers held it to be *un-understandable*, arising without any mediating inference (Schneider 1959; Jaspers 1913; Sims 2015). It is the only first-rank symptom that is a delusion rather than a hallucination or passivity experience.

15.6 The classic list at a glance

GROUP	FIRST-RANK SYMPTOM	ONE-LINE DESCRIPTION
(a) Special auditory hallucinations	**Thought echo**	Own thoughts heard spoken aloud (audible thoughts, Gedankenlautwerden)
	Voices arguing / discussing	Two or more voices discuss the patient in the third person
	Running commentary	A voice narrates the patient's actions as they happen
(b) Thought alienation	Thought insertion	Alien thoughts put into the mind by an external agent
	Thought withdrawal	Thoughts extracted from the mind by an outside force
	Thought broadcast	Thoughts leak out and are shared with others without speech
(c) Passivity (made) phenomena	**Made act**	Actions experienced as controlled by an external agent
	Made impulse	Urge to act imposed from outside
	Made affect	Emotions felt to be produced by an external agent
	Somatic passivity	Bodily sensations imposed by an outside agency
(d) Delusional perception	Delusional perception	Real percept given an instant, unwarranted delusional meaning

The eleven classic first-rank symptoms in Schneider's four groups; three hallucinatory forms, three thought-alienation experiences, four passivity phenomena, and delusional perception.

HOLD THIS

First-rank symptoms are highly characteristic but not pathognomonic; they are neither necessary nor sufficient for schizophrenia. DSM-5 and ICD-11 removed the single-symptom rule, so learn them as vocabulary, not a diagnostic test.

15.7 Why they are not pathognomonic

They cross diagnostic boundaries. The single most important teaching point. First-rank symptoms are neither necessary nor sufficient for schizophrenia. They occur in affective psychosis: Schneiderian first-rank symptoms have been reported in around 10 to 20% of manic patients, where they are usually short-lived, changing in content within days (Shorter Oxford 2012). They occur in organic states, in psychotic depression, in substance-induced psychosis and in temporal lobe epilepsy. Conversely, up to a fifth of people with schizophrenia never manifest any first-rank symptom (Fish 2007). The International Pilot Study of Schizophrenia (WHO) confirmed they appear cross-culturally but did not establish specificity (Kaplan and Sadock 2024).

10-20%	~20%
OF MANIC PATIENTS SHOW FRS	OF SCHIZOPHRENIA SHOW NONE

15.8 Loss of special status in DSM-5 and ICD-11

The one-symptom rule is gone. Earlier systems (DSM-III onward, ICD-10) gave first-rank symptoms extra diagnostic weight; ICD-10 allowed a single clear first-rank symptom to satisfy criterion. DSM-5 (2013) abolished this: bizarre delusions and first-rank auditory hallucinations no longer count double, and at least two criterion-A symptoms are now required, one of which must be delusions, hallucinations or disorganised speech. ICD-11 (2022) similarly dropped their privileged position, treating them as one class of core symptom among several. The clinical vocabulary survives; the diagnostic shortcut does not.

COMMON TRAP

First-rank symptoms do not equal schizophrenia. The exam favourite: a manic patient with thought insertion and delusional perception is *not* thereby schizophrenic, because around 10 to 20% of manic patients show first-rank symptoms and up to a fifth of people with schizophrenia show none. First-rank symptoms are highly characteristic descriptive markers, but they are not necessary, not sufficient, and not pathognomonic; the diagnosis rests on the whole picture and course, not on ticking a Schneiderian box. Also do not confuse second-person command hallucinations ("run away") with first-rank third-person or running-commentary voices; commands are not first-rank.

MNEMONIC

A B C D: Auditory, Broadcast-of-thought, Controlled (passivity), Delusional perception

The four Schneiderian groups map to A (the three special auditory hallucinations), B (thought alienation, of which broadcast is the memorable member alongside insertion and withdrawal), C (Controlled = the made or passivity phenomena), and D (delusional perception). Within C, remember the four "made" experiences: made act, made impulse, made affect, and somatic passivity.

GOOD TO REMEMBER

- **Kurt Schneider (1959):** proposed first-rank symptoms as pragmatic, reliable markers of schizophrenia "except in the presence of coarse brain disease"; ranked above second-rank and above affective symptoms.
- **Thought echo (Gedankenlautwerden, audible thoughts):** hearing one's own thoughts spoken aloud, just before, during or after thinking them.
- **Voices arguing / discussing:** two or more voices talk about the patient in the third person.
- **Running commentary:** a voice narrates the patient's actions as they occur.
- **Thought insertion:** alien thoughts placed into the mind by an external agent.
- **Thought withdrawal:** thoughts felt to be removed from the mind by an outside force.
- **Thought broadcast:** one's thoughts escape and become accessible to others without speech.
- **Made act (made volition):** passivity of action, experienced as externally controlled.
- **Made impulse:** an urge to act imposed from outside.
- **Made affect:** emotions felt to be produced by an external agent.
- **Somatic passivity:** bodily sensations imposed by an outside agency.
- **Delusional perception:** a real percept given an instant, unwarranted personal delusional meaning (the only first-rank delusion; Jaspers' two-member analysis).
- **Not pathognomonic:** present in around 10 to 20% of manic patients and in organic states; absent in up to a fifth of people with schizophrenia.
- **DSM-5 (2013) and ICD-11 (2022):** removed the single-first-rank-symptom rule; first-rank symptoms lost their special diagnostic weight.

16 · Disorders of the experience of self

The disorders of self are the psychopathology of the "I". The **disorders of self** (the ego disorders, the **Ich-Storungen**) concern not *what* the patient perceives or believes but the very awareness that experience belongs to a self and that this self is bounded off from the world. Every normal act of consciousness carries an implicit sense of ownership: *I* am thinking this thought, *I* am doing this act, *I* am a single person now and the same person I was yesterday, and there is a boundary between what is inside me and what is outside. Jaspers (1913) in *General Psychopathology* and later Scharfetter (1980) analysed this awareness of the self into distinct dimensions; their disorder gives the ego disorders their exam-relevant structure (Sims 2015; Fish 2007).

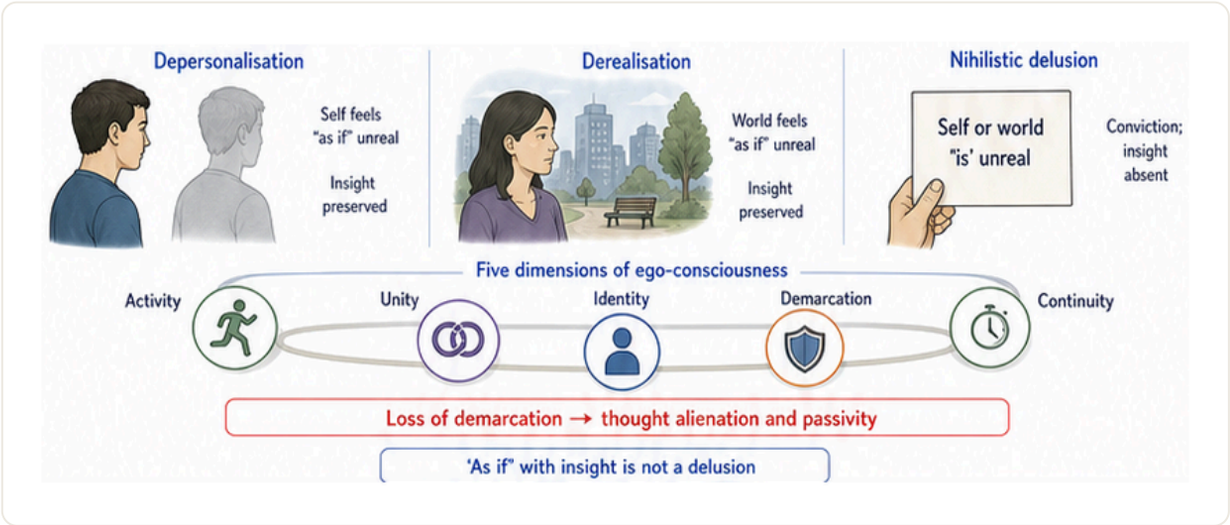


Fig 6.25 Experience of self: 'as if' versus 'is'.

For the exam, three things must be held together. First, the definition of the **ego boundary** and its loss as the substrate of passivity and thought alienation. Second, depersonalisation and derealisation as changes in the *quality* of self-awareness that keep their "as if" character and therefore preserve insight, unlike delusion. Third, the Jaspers to Scharfetter dimensions of ego-consciousness with the disorder that maps onto each. Ground the description, not the explanation: as always in descriptive psychopathology it is the form of the experience that is the sign (Jaspers 1913; Sims 2015).

16.1 Awareness of the self and its boundary with the world

The self is normally felt, not inferred. Healthy self-experience has a taken-for-granted, pre-reflective quality: I do not have to work out that a thought is mine or that my body is me, it is simply given. Jaspers (1913) described this immediate ego-consciousness as the background against which all other experience is set, and Scharfetter (1980) called it the awareness of one's own being (*Ich-Bewusstsein*). The self is also demarcated: there is an experienced membrane between the inner world (thoughts, feelings, will, body) and the outer world of objects and other people. Disorders of self are disturbances of this immediate awareness and of this membrane, and they range from the mild and neurotic (depersonalisation) to the psychotic dissolution of the boundary in schizophrenia (Sims 2015).

16.2 The ego boundary and its loss

The membrane between self and not-self. The **ego boundary** is the experienced line that separates what is self from what is not-self, that marks internal mental events as belonging to me and external percepts as belonging to the world. Paul Federn introduced the concept in the psychoanalytic theory of psychosis, holding that in schizophrenia the ego boundary that ordinarily separates thoughts and feelings from perceptions becomes faulty, so that inner mental content is no longer marked with "ego feeling" as one's own and is experienced instead as external reality (Kaplan and Sadock 2024). When the ego boundary fails, the patient can no longer reliably tell whether an experience arises from within or is imposed from without: this is the common root of thought alienation (insertion, withdrawal, broadcast) and of the passivity phenomena. Schneider spoke of the same clinical picture as a "loss of ego boundaries" (Companion 2010). The

loss of the ego boundary is therefore the descriptive substrate that unifies the most characteristic first-rank experiences of schizophrenia.

16.3 Depersonalisation

A change in the awareness of one's own self. **Depersonalisation** (also spelt **depersonalization** in American texts) is an altered, uncanny change in the quality of self-awareness: the patient feels unreal, detached, cut off from their own experience, as though watching themselves from outside or acting like an automaton or robot going through the motions. Feelings feel deadened or absent, actions feel mechanical, the self feels strange and unfamiliar (Sims 2015). The defining feature is the *"as if"* quality: the patient says they feel *as if* they were unreal, *as if* in a dream, *as if* watching themselves, and they retain full insight that this is a subjective change and not literally true. That preserved "as if" awareness is exactly what separates depersonalisation from a nihilistic delusion, in which the patient believes with delusional conviction that they truly do not exist or have no self (Fish 2007).

Depersonalisation (depersonalization)

An "as if" change in the awareness of one's own self: feeling unreal, detached, watching oneself, emotionally deadened, an automaton, with insight preserved.

Emotional numbing (de-affectualisation)

The component in which affect feels flattened or absent, so the patient complains of feeling nothing, not even grief they know they should feel.

Anaesthesia dolorosa psychica

The painful awareness of the loss of feeling itself: distress precisely because emotions cannot be felt.

16.4 Derealisation

The outer world, not the self, feels unreal. **Derealisation** (also spelt **derealization**) is the outward-facing counterpart: the surrounding world feels unreal, flat, two-dimensional, lifeless, artificial or stage-managed, as if made of cardboard or seen through glass. People may look like actors or automata, familiar places feel strange, colours seem dull and the world seems drained of vitality (Sims 2015). Depersonalisation and derealisation are together classically described as the "as-if" phenomena: they very frequently co-occur, both share the preserved "as if" quality and preserved insight, and both are grouped in DSM-5 as depersonalisation-derealisation disorder. They are non-specific, appearing in anxiety and panic (Roth's phobic-anxiety-depersonalisation syndrome), depression, temporal lobe epilepsy, migraine, fatigue and sleep deprivation, dissociative states, and as an SSRI or benzodiazepine withdrawal symptom, and can occur transiently in healthy people (Kaplan and Sadock 2024).

Derealisation (derealization)

An "as if" change in the awareness of the surrounding world: it feels unreal, flat, lifeless, artificial or stage-set, with insight preserved.

Deja vu / jamais vu (adjacent)

Familiarity phenomena often reported alongside derealisation: the unfamiliar felt as familiar (deja vu) or the familiar felt as strange (jamais vu).

16.5 Depersonalisation versus derealisation versus delusion

FEATURE	DEPERSONALISATION	DEREALISATION	NIHILISTIC DELUSION
What feels unreal	The self ("I feel unreal")	The outer world ("the world feels unreal")	Self or world (asserted as fact)
Quality of experience	**"As if" unreal**	**"As if" unreal**	**"Is" unreal (conviction)**
Insight	Preserved	Preserved	Absent
Reality testing	Intact	Intact	Lost
Typical setting	Anxiety, depression, dissociation, fatigue	Same, often with depersonalisation	Psychotic (Cotard's) depression, schizophrenia

The "as if" quality with preserved insight is the single discriminator that separates depersonalisation and derealisation from a nihilistic (Cotard) delusion, in which unreality is asserted as fact with delusional conviction.

RULE **The "as if" quality with preserved insight is what keeps depersonalisation and derealisation from being delusions.** "I feel as if I am unreal" (insight kept) is not "I do not exist" (Cotard, insight lost).

16.6 The five dimensions of ego-consciousness (Jaspers and Scharfetter)

Jaspers' four formal characteristics, and Scharfetter's fifth. Jaspers (1913) held that immediate ego-consciousness has *four* formal characteristics: activity, unity at any one moment, continuity of identity over time, and demarcation from the environment. Scharfetter then *added a fifth*, **ego vitality**, the awareness of simply being or existing, which Jaspers had folded inside the awareness of activity. The resulting five, each with a characteristic disorder, are the most examinable single frame for the disorders of self. Note the exam trap: the fifth dimension is vitality, not a separate "continuity", because continuity of identity over time is already Jaspers' identity aspect (Sims 2015; Fish 2007).

Awareness of vitality (ego-vitality; Scharfetter's addition)

The awareness of simply being alive and existing. Disorder: the conviction of not existing, of being dead or having no body, which is the core experience of the nihilistic (Cotard) delusion, and the deadened quality of severe depersonalisation.

Awareness of activity (ego-activity)

The sense that I am an active agent, that my mental acts and perceptions are my own doing. Disorder: loss of the sense of agency, and the "acting as an automaton" quality of depersonalisation.

Awareness of unity (ego-consistency, coherence)

The awareness that at any one moment I am a single, undivided person. Disorder: the experience of being split or divided, of two selves or a self fragmenting.

Awareness of identity (ego-identity, continuity of identity over time)

The awareness that I am the same person now that I have always been, persisting as one and the same self from moment to moment and across the lifespan. Disorder: the conviction of having become someone or something else, as in metamorphosis or delusional misidentification of the self, and the sense of being a different self before and after a point.

Awareness of demarcation (ego-boundary)

The awareness of a boundary between self and not-self, between inner and outer world. Disorder: loss of the boundary, giving thought insertion, withdrawal, broadcast and the passivity (made) phenomena.

The bridge to the earlier sections. Depersonalisation is chiefly a disorder of the awareness of activity, shading into vitality when the patient feels dead rather than merely unreal; the nihilistic delusion is the full disorder of vitality; and the loss of the ego boundary is the disorder of the awareness of demarcation and is the substrate of passivity and thought alienation. The scheme thus ties the whole topic together.

HOLD THIS

Loss of the ego boundary (the awareness of demarcation) is the descriptive substrate of passivity and thought alienation. Scharfetter's five dimensions - activity, unity, identity, demarcation, continuity - each carry their own disorder.

16.7 Passivity experiences as loss of the self's demarcation

Agency is retained by the act but lost by the self. In **passivity** experiences the patient carries out an action, feels an emotion, has an impulse or registers a bodily sensation, but experiences it as made, imposed or controlled by an external agency: the act still occurs, yet the sense of being its author is gone. Descriptively this is precisely a failure of the awareness of demarcation, a breach of the ego boundary that no longer walls off the self's own volition, affect and body from outside control (Sims 2015; Fish 2007). The passivity phenomena are Schneiderian first-rank symptoms; cross-refer the first-rank topic for their diagnostic weight and the modern caveat that they are not pathognomonic.

Made act (made volition)

An action is performed but experienced as controlled by an external agent; the patient is a passive vehicle of their own behaviour.

Made impulse (made drive)

The urge or drive to act is felt to be imposed from outside and alien to the self.

Made affect (made feeling)

Emotions are experienced as produced in the patient by an external agency: "these feelings are not mine, they are put into me".

Somatic passivity

Bodily or visceral sensations are experienced as imposed by an outside force, for example being irradiated or manipulated from without.

16.8 Where the ego disorders sit clinically

A spectrum from neurotic to psychotic. The disorders of self span a range. At the mild end, depersonalisation and derealisation are non-psychotic, keep their "as if" quality and preserved insight, and occur in anxiety, depression, dissociation, fatigue and healthy people. At the severe end, loss of the ego boundary, the passivity phenomena and thought alienation are psychotic disorders of the awareness of demarcation and are highly characteristic of schizophrenia. Between them, disorders of the awareness of unity, identity and continuity shade from the

depersonalised "as if I were two people" into frank delusions of being split, transformed or replaced. Reading a report of unreality, the crucial clinical question is always whether the "as if" quality and insight are preserved (depersonalisation or derealisation) or lost (delusion) (Sims 2015; Fish 2007).

CLINICAL ANCHOR

A young woman with panic attacks says: "During the attacks I feel as if I'm not really here, like I'm watching myself from behind a glass wall, and the room looks flat and fake, like a film set." Two signs are present: depersonalisation (watching herself, feeling unreal) and derealisation (the room flat and fake). Both carry the "as if" quality and she knows the change is in her, not in reality, so insight is intact. This is not psychosis; it is the classic anxiety-linked depersonalisation-derealisation picture, and naming it correctly steers you to anxiety management rather than antipsychotics.

COMMON TRAP

Do not confuse depersonalisation with a nihilistic delusion. The patient who says "I feel *as if* I have no self" and knows it is a feeling has depersonalisation with insight; the patient who states as fact "I have no head, I do not exist, my organs have rotted away" and is convinced of it has a nihilistic (Cotard) delusion. The word "unreal" appears in both; the discriminator is the preserved "as if" quality and insight, not the content. Equally, do not label ordinary loss of the ego boundary as merely "poor concentration": thought insertion, withdrawal and broadcast are disorders of the awareness of demarcation, not of attention.

MNEMONIC

V A C I D: Vitality, Activity, Consistency (unity), Identity, Demarcation

The five dimensions of ego-consciousness, Jaspers' four plus Scharfetter's fifth: awareness of Vitality (being alive at all, Scharfetter's addition), Activity (agency), Consistency or unity (single person now), Identity (same person as always, continuous through time), and Demarcation (the ego boundary). Their disorders, respectively: the nihilistic delusion of not existing; depersonalisation of agency; feeling split; feeling transformed into another; and loss of the boundary giving passivity and thought alienation.

GOOD TO REMEMBER

- **Disorders of self (ego disorders, Ich-Storungen):** disturbances of the immediate awareness that experience belongs to a self and that the self is bounded from the world (Jaspers 1913; Scharfetter 1980).
- **Ego boundary (Federn):** the experienced membrane between self and not-self; its loss un-marks inner events as one's own and is the substrate of passivity and thought alienation (Kaplan and Sadock 2024; Schneider's "loss of ego boundaries").
- **Depersonalisation (depersonalization):** an "as if" change in awareness of one's own self, feeling unreal, detached, watching oneself, an automaton, with insight preserved.
- **Derealisation (derealization):** an "as if" change in awareness of the surrounding world, which feels unreal, flat, lifeless or artificial, with insight preserved.
- **"As if" quality:** the defining feature of depersonalisation and derealisation; preserves insight and separates them from nihilistic (Cotard) delusion, where unreality is asserted as fact.
- **Jaspers (1913):** formal analysis of immediate ego-consciousness in General Psychopathology.
- **Scharfetter (1980) five dimensions:** awareness of activity, of unity, of identity, of demarcation (the boundary), and of continuity of the self, each with its own disorder.
- **Awareness of activity:** sense of agency and vitality; disorder = depersonalisation, the automaton quality.
- **Awareness of unity:** being one person at a moment; disorder = feeling split or divided.
- **Awareness of identity:** being the same person one has always been; disorder = feeling transformed into another.
- **Awareness of demarcation:** the self-world boundary (the ego boundary); disorder = thought insertion, withdrawal, broadcast and passivity.
- **Awareness of continuity:** the self persisting through time; disorder = feeling discontinuous, a broken self.
- **Passivity (made) phenomena:** made act, made impulse, made affect and somatic passivity; loss of the self's demarcation; Schneiderian first-rank but not pathognomonic (cross-refer first-rank).
- **Depersonalisation-derealisation:** the classic "as-if" phenomena; non-specific, seen in anxiety, depression, dissociation, epilepsy, migraine, fatigue and SSRI or benzodiazepine withdrawal; grouped as one disorder in DSM-5.

17 · Disorders of emotion and affect

The **disorders of emotion** are the descriptive psychopathology of feeling: how the range, quality, tone, congruity and stability of a person's emotional life go wrong, read purely at the level of what is observed and reported. This is a genuine Paper I long-essay topic in its own right, distinct from the *theories* of emotion (James-Lange, Cannon-Bard, Schachter-Singer), which belong to the General and Developmental Psychology notes and explain the *mechanism* of normal emotion. Here the task is Jaspers, Fish and Sims phenomenology: name the abnormality, define it crisply, and give a clinical example, form over content (Jaspers 1913, Fish 2007, Sims 2015).

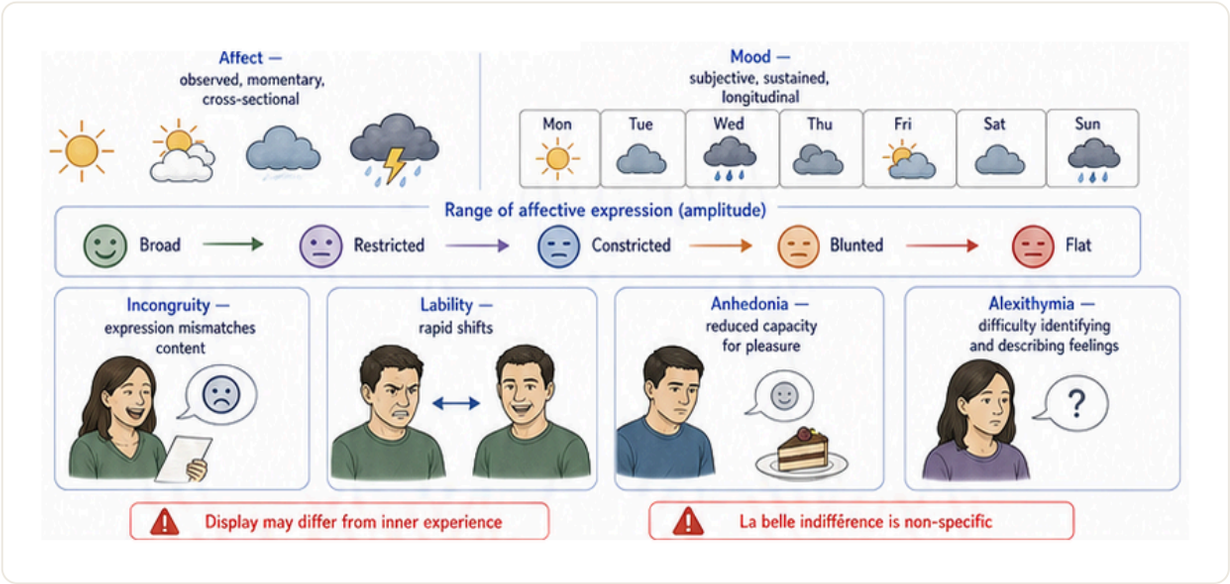


Fig 6.26 Mood, affect and emotional experience.

Exam framing. Examiners reward three things kept clean. First, the vocabulary: **affect** versus **mood** versus emotion, a distinction that anchors the whole answer. Second, the disorders of the *tone or quality* of mood (depression, elation, anxiety, irritability, perplexity, apathy). Third, the disorders of the *expression, congruity and stability* of affect (blunting and flattening, incongruity, lability, and the special signs anhedonia and alexithymia). Weave in the caveat that blunting and incongruity, though characteristic of schizophrenia, are not by themselves pathognomonic and must be judged over the whole interview, not spot-diagnosed (Companion to Psychiatric Studies).

17.1 The vocabulary: affect, mood and emotion

Get the three terms exactly right; the whole answer rests on them. The standard convention (Kaplan & Sadock 2024, Sims 2015) separates the momentary observed expression from the sustained subjective state.

Emotion

The generic term for a complex feeling state with psychic, somatic and behavioural components; a stirred-up condition of the organism. Affect and mood are the two clinical ways of sampling it.

Affect

The *momentary, observed, cross-sectional* emotional expression: the outward, moment-to-moment tone the clinician sees, inferred from face, voice, posture and reactivity during the interview. It is to mood as weather is to climate. Described by quality, range, appropriateness (congruity) and reactivity.

Mood

The *sustained, pervasive, subjective* emotional state, coloured over time and reported by the patient; the emotional climate that persists and biases perception, as against the transient affect. Rated by quality, stability and congruity with thought.

> **PEARL**
>
> **Affect** is weather (what you see now, cross-sectional, observed); **mood** is climate (what is felt over time, sustained, subjective). The examiner's shorthand test: mood is what the patient tells you, affect is what you observe.

HOLD THIS

Affect is the momentary observed expression; mood is the sustained subjective state; the range runs broad to restricted or constricted to blunted to flat. Blunting and incongruity point to schizophrenia but are never a spot diagnosis.

17.2 Disorders of the quality or tone of mood

Abnormal tone. The commonest disorders are of the *quality* of the prevailing mood: it is pathologically lowered, raised, fearful, irritable, bewildered or empty. Each has a phenomenological signature beyond ordinary sadness or happiness.

Depression (lowering of mood)

A sustained lowering of mood below the normal range, often described by the patient as qualitatively distinct from ordinary unhappiness, with a peculiarly painful quality, and accompanied by pessimism, guilt and loss of drive (Companion to Psychiatric Studies).

Dysphoria

An unpleasant, discontented, low-tinged mood, not amounting to full depression; often mixed with irritability and tension.

Elation and euphoria

Elation is an exaggerated feeling of well-being, cheerfulness and expansiveness, classically in mania, felt by the patient as heightened vitality. **Euphoria** is a milder, more static state of contented well-being out of keeping with circumstances; when neurologically driven and shallow it shades into the fatuous cheerfulness (*moria*) of frontal lesions.

Ecstasy

An intense, exalted feeling of rapture, awe or oceanic bliss, often with a sense of union with the world; seen in temporal-lobe epilepsy and some acute psychoses.

Anxiety and fear

Anxiety is an unpleasant affect of apprehensive expectation, an anticipation of threat without a defined object, with somatic accompaniments (palpitation, tremor, restlessness). **Fear** is the same affect directed at a specific, identified danger. Free-floating anxiety is anxiety unattached to any object.

Irritability

A lowered threshold for anger and annoyance, a readiness to react with hostility to minor provocation; prominent in mania, mixed affective states, depression, organic and personality disorders.

Perplexity

A bewildered, puzzled affect in which the patient feels something strange is happening but cannot grasp what, with lowered brows, slowed gaze and a partly open mouth; seen par excellence in puerperal psychosis and in any acute psychotic or organic state (Companion to Psychiatric Studies).

Apathy

A loss of feeling tone, an emotional indifference and absence of concern in which nothing seems to matter; reduced spontaneous action and speech accompany it. Seen in chronic schizophrenia, frontal and subcortical disease, and severe depression; it shades into avolition.

17.3 Blunting and flattening of affect

Loss of the normal range and depth of emotional expression, characteristic of schizophrenia. This is the single most important negative-symptom disturbance of affect, and the exam prizes the flattening-versus-**blunting** distinction, though the two are often used loosely as synonyms (Companion to Psychiatric Studies, Kaplan & Sadock 2024).

Flattening of affect

A reduction in the *range and depth* of emotional expression, with loss of the subtlety and nuance of moment-to-moment emotional interaction; the face is immobile, the voice monotonous, gestures few. Note that such patients may still be highly aroused physiologically despite the flat display.

Blunting of affect

A *coarsening* of emotion with insensitivity to social context, Kraepelin's loss of the delicacy of emotion. Sims described a woman with schizophrenia who, with obvious relish for the sensational effect, took her visitors upstairs to view her mother's corpse, dead two days (Sims 2015).

> **COMMON TRAP**
>
> **Blunting** and flattening carry a strong diagnostic inference (schizophrenia), so the terms must not be used loosely. They should never be a spot diagnosis: judge them across the whole interview, and exclude cultural emotional reserve, a habitually dead-pan personality, dissocial indifference, and above all drug-induced bradykinesia or parkinsonian masking, which mimic flattening (Companion to Psychiatric Studies).

17.4 Restricted and constricted affect

The milder gradations on the range-of-affect spectrum. Below full flattening lie two intermediate degrees, which the examiner expects graded correctly against the normal *broad* or full range (Kaplan & Sadock 2024).

Restricted (constricted) affect

A reduction in the range and intensity of emotional expression that is *less severe than blunting or flattening*: emotion is present and modulated but the amplitude is dampened, the display muted. Kaplan & Sadock define the two terms identically and cross-reference one to the other, so treat them as a single construct and do not grade one as milder than the other.

Broad (full) affect

The normal reference state: a full, appropriately modulated range of emotional expression across the interview. Named only to sit the abnormalities against it.

The full continuum runs: broad (normal) to restricted or constricted to blunted to flat, in order of decreasing range and depth of expression.

17.5 Incongruity of affect

A mismatch between expressed emotion and thought content. This is a discrete sign, not merely reduced affect, and one of the most examined single terms in the topic.

Incongruity of affect

The expressed emotion does not match the thought content or the setting, for example laughing while describing a bereavement, or a patient who, asked about the bandage on her wrist, replied that she had

tried to kill herself that morning and burst out laughing (Fish 2007, Companion to Psychiatric Studies). Also called inappropriate or parathymia. Sudden causeless giggling in chronic schizophrenia is the classic instance.

COMMON TRAP

Do not confuse **incongruity of affect** (form) with mood-incongruent *content* of delusions or hallucinations. Incongruity of affect is a mismatch of the emotional *display* with the topic; mood-incongruent psychotic content is a delusion whose theme is out of keeping with the mood (for example grandiose delusions in depression). Assess incongruity circumspectly: inappropriate laughter may equally signal social awkwardness or embarrassment (Companion to Psychiatric Studies).

17.6 Lability of affect, emotional incontinence and la belle indifference

Disorders of the stability and control of affect. Here the fault is not the tone or congruity but the *regulation*: emotion shifts too readily, discharges without control, or is conspicuously absent where it should be present.

Lability of affect

An abnormal instability of affect with rapid, exaggerated shifts from one emotional state to another (for example tears to laughter within minutes), the swings often shallow and readily provoked. Seen in mania, organic and frontal disorders, and delirium. The exact string **lability** denotes this rapid shifting.

Emotional incontinence

A severe, pathological loss of control over emotional *expression*: the patient weeps or laughs uncontrollably with little or no provoking stimulus and cannot check it, the outburst out of proportion to any felt emotion. The hallmark of pseudobulbar palsy and other organic (vascular, degenerative) states; sometimes called pathological (forced) crying or laughing.

La belle indifference

A striking, inappropriate *lack of concern* about a serious symptom or disability, classically described with conversion (functional neurological) disorder: the patient reports a dramatic deficit such as paralysis or blindness with bland unconcern. Its diagnostic value is now regarded as limited and non-specific.

TRAP La belle indifference is not diagnostic of conversion disorder; its specificity is now regarded as limited, so do not use bland unconcern to clinch a functional label.

17.7 Anhedonia

The loss of the capacity to experience pleasure. A cardinal, high-yield sign that straddles the mood disorders and the negative syndrome of schizophrenia.

Anhedonia

The loss or marked decrease of the capacity to experience pleasure, so that activities once enjoyed no longer give satisfaction (DSM-5-TR 2022, Sims 2015). A core symptom of a major depressive episode (loss of interest or pleasure) and one of the recognised negative symptoms of schizophrenia.

Consummatory anhedonia

Reduced pleasure experienced *in* the moment (in-the-moment liking).

Anticipatory anhedonia

Reduced pleasure *anticipated from* a future reward (wanting); research suggests it is anticipatory more than consummatory pleasure that is chiefly impaired in schizophrenia, where the patient may still enjoy an activity in the moment yet fail to seek it out.

CLINICAL ANCHOR

Anhedonia is one of the two gateway symptoms of a major depressive episode (with depressed mood) and one of the five negative-symptom domains of schizophrenia alongside avolition, asociality, affective blunting and alogia (Kaplan & Sadock 2024). Distinguish it from apathy (loss of feeling and motivation broadly) and from alexithymia (difficulty naming feelings): anhedonia is specifically the failure of *pleasure*.

17.8 Alexithymia

A difficulty identifying and describing one's own feelings. Not a psychotic sign but a trait-like disturbance of emotional awareness, coined by Sifneos (1973), that recurs in psychosomatic and personality contexts.

Alexithymia

A difficulty in identifying and describing one's own feelings, distinguishing feelings from bodily sensations of emotional arousal, coupled with an externally oriented, concrete cognitive style and impoverished fantasy life (Sifneos 1973). Literally "no words for mood".

It is associated with somatoform and psychosomatic presentations, substance use, eating disorders and cluster B personality disorders, and is measured by the Toronto Alexithymia Scale. It is a disorder of emotional *insight and articulation*, not of the experience of pleasure (contrast anhedonia) nor of expressed range (contrast blunting).

17.9 Discriminating the negative-affect signs

The exam's favourite confusion set. Four flat-looking or empty-looking states are constantly conflated. Keep the discriminator column in mind.

SIGN	WHAT IS LOST	DOMAIN OF FAULT	CLASSIC SETTING
Blunting / flattening	**Range and depth of emotional *expression***	Affect (display)	Schizophrenia (negative)
Anhedonia	**Capacity to feel *pleasure***	Hedonic experience	Depression; schizophrenia
Apathy	**Feeling tone and *motivation*; nothing matters**	Drive and feeling	Frontal, chronic schizophrenia
Alexithymia	**Ability to *identify and name* feelings**	Emotional insight	Psychosomatic, cluster B

Ask what is lost: expression (blunting), pleasure (anhedonia), motivation and feeling tone (apathy), or the words for feeling (alexithymia).

RULE **For the flat-looking states, ask what is lost.** Expression is blunting, pleasure is anhedonia, motivation and feeling tone is apathy, and the words for feeling is alexithymia.

MNEMONIC

"A FIB LIkes CAKE"

The core affect abnormalities: **A**ffect vs mood, **F**lattening, **I**ncongruity, **B**lunting; **L**ability, **I**ncontinence (emotional); **C**onstriction/restriction, **A**nhedonia, **K**eeps-calm belle indifference, al**E**xithymia. A memory hook, not a classification.

GOOD TO REMEMBER

- **Emotion:** generic stirred-up feeling state with psychic, somatic and behavioural parts.
- **Affect:** the momentary, observed, cross-sectional emotional expression (weather; what you see).
- **Mood:** the sustained, pervasive, subjective emotional state (climate; what the patient reports).
- **Depression:** sustained lowering of mood, often qualitatively distinct from ordinary sadness.
- **Elation / euphoria:** exaggerated well-being and expansiveness (mania) / milder static contentment out of keeping with circumstances.
- **Anxiety vs fear:** apprehension without an object vs apprehension at a specific identified danger.
- **Irritability:** lowered threshold for anger; prominent in mania, mixed states, depression, organic disease.
- **Perplexity:** bewildered puzzled affect, feels something strange is happening; par excellence in puerperal psychosis.
- **Apathy:** loss of feeling tone and concern, nothing matters; frontal and chronic schizophrenia, severe depression.
- **Flattening of affect:** reduced range and depth of emotional expression; schizophrenia (may be aroused underneath).
- **Blunting of affect:** coarsening of emotion and insensitivity to social context (Kraepelin's loss of delicacy).
- **Restricted / constricted affect:** dampened range milder than flattening; continuum broad to restricted or constricted to blunted to flat.
- **Incongruity of affect:** expressed emotion does not match thought content, e.g. laughing while describing a bereavement.
- **Lability of affect:** rapid, exaggerated, shallow shifts between emotional states; mania, organic, delirium.
- **Emotional incontinence:** uncontrollable weeping or laughing with little stimulus; hallmark of pseudobulbar palsy.
- **La belle indifference:** bland unconcern about a serious deficit; classically conversion disorder (low specificity now).
- **Anhedonia:** loss of the capacity to experience pleasure; core to major depression and a schizophrenia negative symptom.
- **Alexithymia:** difficulty identifying and describing one's own feelings (Sifneos 1973); Toronto Alexithymia Scale.
- **Caveat:** blunting and incongruity are characteristic of schizophrenia but not pathognomonic; judge over the whole interview, not by a single glance.

18 · Disorders of memory and of consciousness

This note teaches two neighbouring domains of descriptive psychopathology that examiners love to pair. The **disorders of memory** classify the ways in which registration, retention and recall break down, and the ways in which the *content* of what is recalled becomes distorted. The **disorders of consciousness** classify the ways in which the clear, alert awareness of self and environment is dimmed, narrowed or fragmented. Both reward the Jasperian discipline of describing form over explanation (Jaspers 1913, Fish 2007, Sims 2015): the task is to name the sign and define it crisply, ideally with a one-line clinical example, not to theorise about mechanism.

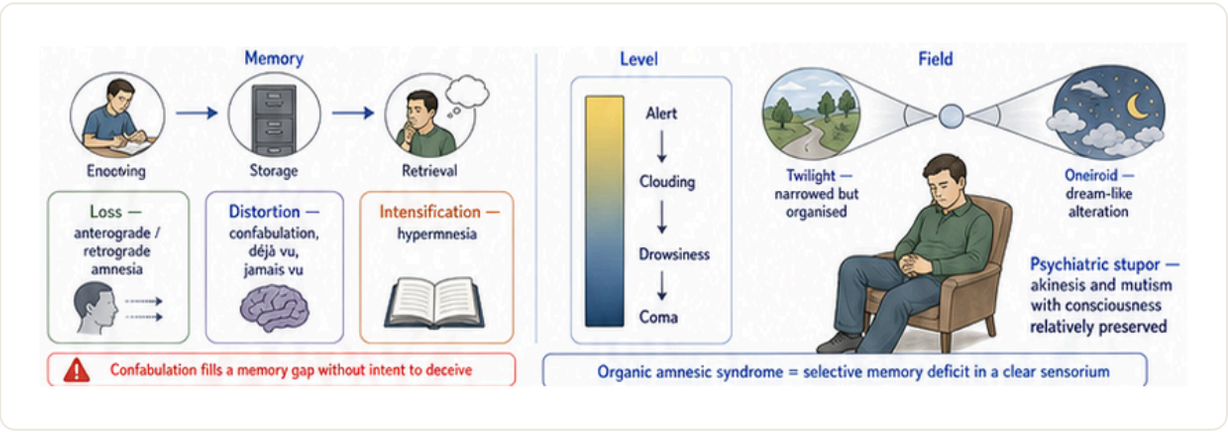


Fig 6.27 Memory and consciousness: loss, distortion, level, field.

Exam framing. Keep the two halves architecturally separate. Under memory, hold apart the loss of memory (the **amnesia**s and the organic amnesic syndrome) from the distortions of recall (the **paramnesia**s, of which **confabulation** is the flagship) and from the rare intensification, **hypermnesia**. Under consciousness, hold apart a change in the *level* of consciousness on the wakefulness continuum (**clouding**, **delirium**, drowsiness, **stupor**, coma) from a change in the *field* of consciousness (the **twilight state** and the **oneiroid** state). The neural basis of memory is developed in the Functional Neuroanatomy & Neurophysiology notes and cross-referred there; Wernicke encephalopathy and the Korsakoff syndrome are developed in full on the organic psychiatry chapter; the acute management of delirium belongs to the emergency psychiatry notes. Here we describe.

18.1 The disorders of memory: the working model

Three processes, three vulnerabilities. Memory is conventionally split into *registration* (encoding of new material), *retention* (storage), and *recall* (retrieval), layered by time-scale into immediate (working) memory, recent (short-term) memory, and remote (long-term) memory (Sims 2015). Bedside testing maps onto this: digit span probes immediate registration, a name-and-address or three-object recall at five minutes probes recent memory and new learning, and autobiographical or general-knowledge questioning probes remote memory. The classic organic profile is a severe deficit of *recent* memory and new learning with immediate memory and, initially, remote memory relatively spared, which is exactly the fingerprint of the organic amnesic syndrome below.

18.2 Amnesia: anterograde and retrograde

Definition. An **amnesia** is a pathological loss of, or gap in, memory. The single most useful division is by the temporal relation of the gap to a causative event, typically a head injury, seizure or other insult (Fish 2007, Sims 2015).

Anterograde amnesia

Loss of memory for events *after* the causative insult: the inability to lay down new memories going forward. In head injury the duration of post-traumatic amnesia (from injury to the return of continuous memory) is a rough index of severity.

Retrograde amnesia

Loss of memory for events *before* the insult, extending back from it. It typically shrinks over time and characteristically obeys Ribot's law, older and better-consolidated memories being the last to go and the first to return.

Lacunar (circumscribed) amnesia

A discrete gap for a bounded period of time, with intact memory on either side; seen after delirium, complex partial seizures and some dissociative states.

18.3 The organic amnesic syndrome (and Korsakoff)

Definition. The *organic amnesic (amnestic) syndrome* is a selective, enduring impairment of recent memory and new learning caused by identifiable brain disease, in *clear consciousness* and without global intellectual decline (ICD-10). The ICD-10 requirements are worth carrying verbatim: recent-memory impairment (anterograde and retrograde amnesia) with defective new learning; preserved immediate recall (normal digit span), attention and consciousness; preserved other intellectual function; and objective evidence of a relevant brain lesion, characteristically of the diencephalic and medial temporal structures (mammillary bodies, fornix, hippocampus, dorsomedial thalamus).

Korsakoff as the prototype. The commonest cause is thiamine deficiency in chronic alcohol dependence: Wernicke encephalopathy is the acute phase and the Korsakoff syndrome the chronic amnesic residue, together the Wernicke-Korsakoff syndrome. Its bedside signature is a dense anterograde amnesia with disorientation to time, relative sparing of remote memory, absent insight, and, in the early phase, **confabulation** filling the memory gap; confabulation tends to fade as the syndrome becomes chronic (Companion to Psychiatric Studies). The neural basis of memory and the full Wernicke-Korsakoff picture are cross-referred to the Functional Neuroanatomy & Neurophysiology notes and the organic psychiatry chapter respectively.

> **COMMON TRAP**
>
> The organic amnesic syndrome is *not* delirium and *not* dementia. It differs from **delirium** by the absence of any disturbance of consciousness or attention, and from dementia by the absence of global intellectual and personality decline. The discriminator to state in the viva is: an isolated, selective memory deficit in a clear sensorium.

HOLD THIS

Split memory into loss (amnesias), distortion (paramnesias, confabulation the flagship) and intensification (hypermnesia); split consciousness into level (clouding to coma) and field (twilight, oneiroid). The organic amnesic syndrome is a selective memory deficit in a clear sensorium.

18.4 Transient global amnesia and psychogenic amnesia

Two abrupt-onset amnesias to contrast. One is organic and self-limiting; one is dissociative and content-selective.

Transient global amnesia

An abrupt, isolated dense anterograde amnesia lasting some hours, in a fully alert middle-aged or older person who remains able to converse and perform complex tasks but repeatedly asks the same orienting questions ("where am I?"). Attributed to transient ischaemia or a spreading-depression-like event in the medial temporal lobes; it resolves within about 24 hours leaving only a permanent gap for the episode, and personal identity is retained throughout (Kaplan & Sadock 2024).

Psychogenic (dissociative) amnesia

A loss of memory, usually retrograde and for emotionally significant autobiographical material, that exceeds ordinary forgetting and is not due to organic disease; classically it spares neutral information and impersonal skills. Onset follows psychological stress or trauma.

Dissociative fugue

Dissociative amnesia *plus* a purposeful journey away from home or work, often with loss of personal identity and sometimes the assumption of a new one; the person appears outwardly normal to observers during the wandering, with a dense amnesic gap for the episode afterwards (Sims 2015).

CLINICAL ANCHOR

Loss of memory for *personal identity* (one's own name) with preserved capacity to lay down new memories points toward a psychogenic or dissociative amnesia; the organic amnesias, by contrast, spare identity and hit new learning. This is the fastest bedside organic-versus-psychogenic discriminator, though it must be held loosely and corroborated.

18.5 The paramnesias I: confabulation and deja vu / jamais vu

Definition. A *paramnesia* is a distortion of recall in which memory is falsified, rather than simply lost; the material recalled is altered, misplaced in time, or fabricated (Fish 2007, Sims 2015).

Confabulation

The filling of a gap in memory with fabricated or misplaced material that the patient accepts as genuine recollection, without intent to deceive; classically in the Korsakoff syndrome. Fish distinguishes *momentary (provoked)* confabulation, elicited by questioning and drawn from real but displaced memories, from *fantastic (spontaneous)* confabulation, an unprompted flow of grandiose or improbable false memories seen with basal-forebrain and frontal involvement.

Deja vu

A false sense of familiarity: the conviction that a genuinely novel experience has been encountered before ("I have been here already"). A common benign experience in the healthy; when frequent,

intense or stereotyped it points toward temporal lobe epilepsy (Kaplan & Sadock 2024). Related variants include deja vecu (already lived through) and deja pense (already thought).

Jamais vu

The converse: a false sense of *un*familiarity, in which a genuinely familiar situation, place or person feels strange and never before encountered. Likewise seen in temporal lobe epilepsy and in dissociative states.

18.6 The paramnesias II: retrospective falsification and delusional memory

Retrospective falsification

The unwitting distortion of a genuine memory as it is recalled, coloured by the person's present mood or beliefs, e.g. a depressed patient recalling a happy past as uniformly bleak, or an anxious one exaggerating past dangers. The event is real; its remembered emotional colouring is falsified.

Delusional memory (retrospective delusion)

The "recollection" of an event that is itself clearly delusional, or a real memory retrospectively invested with delusional significance, e.g. a patient who "remembers" that a teacher slipped LSD into his juice, the memory serving to explain his psychosis (Kaplan & Sadock 2024). A disorder of the content of thought expressed through memory, not a simple mnestic error.

18.7 Hypermnesia

Definition. **Hypermnesia** is an abnormal intensification or exaggeration of the capacity to recall, in which memories are retrieved with unusual vividness, detail or completeness (Sims 2015). It is described in the heightened arousal of mania and some anxiety states, under hypnosis, in eidetic ("photographic") imagery, in certain savant abilities, and around intensely emotionally charged experiences (the vividness of traumatic and flashbulb memories). It is the mirror image of amnesia and, unlike the paramnesias, is a change in quantity rather than accuracy of recall.

18.8 The disorders of consciousness: level on the wakefulness continuum

Definition. **Consciousness** is the awareness of the self and of the environment; the disorders of consciousness are described first as a fall in the *level* of that awareness along a continuum from full alertness to coma (Sims 2015, Fish 2007). The states below are graded points on that single dimension.

Clouding of consciousness

The mildest, most important grade: a state ranging from barely perceptible impairment to definite drowsiness, in which the person reacts incompletely to stimuli, with attention, concentration, memory and orientation impaired to varying degrees and thinking muddled, so that events may be misinterpreted. Clouding of consciousness is the *defining feature of delirium* (Shorter Oxford Textbook of Psychiatry).

Delirium

The acute confusional state: an acute, fluctuating disturbance of consciousness and attention with global cognitive impairment, disorientation, perceptual disturbance (illusions, misinterpretations, visual hallucinations), a disturbed sleep-wake cycle and altered psychomotor activity, on an organic basis. Its management is cross-referred to the emergency psychiatry notes.

Drowsiness and somnolence

Pathological sleepiness in which the patient readily lapses into sleep but is rousable to a degree; the related state Fish terms *torpor* couples drowsiness with slow thinking and a narrowed range of perception.

Coma

The deepest grade: unrousable unresponsiveness with no purposeful reaction even to strong (painful) stimuli, gradable by remaining reflex responses and EEG activity (Shorter Oxford Textbook of Psychiatry).

18.9 Stupor: the special case

Definition (the psychiatric usage). In psychiatry, **stupor** is a state of akinesis and mutism, the patient immobile, silent and unresponsive, yet with *relative preservation of consciousness*: the eyes are typically open and follow objects, or if closed are held shut against opening, reflexes are normal and posture is maintained (Shorter Oxford Textbook of Psychiatry). This is a critical and examinable divergence from neurological usage, where "stupor" implies an actual impairment of consciousness intermediate between drowsiness and coma. The psychiatric stupor is a disorder of the expression of consciousness, not necessarily of its level.

TYPE OF STUPOR	SETTING / CAUSE	DISCRIMINATING FEATURES
Organic stupor	Structural or metabolic brain disease (e.g. diencephalic or brainstem lesion, hepatic, hypoglycaemic)	**Consciousness genuinely impaired; neurological signs, abnormal EEG**
Catatonic stupor	Schizophrenia, mood disorder, organic catatonia	**Waxy flexibility, negativism, mutism, posturing; may alternate with excitement**
Depressive stupor	Severe (psychotic) depression, the extreme of retardation	**Preceded by profound retardation and low mood; recall of the period afterwards**
Dissociative stupor	Psychological trauma or conflict	**Follows stress; normal muscle tone and reflexes; no organic cause found**

Four stupors to separate: only organic stupor carries a true impairment of consciousness; the psychiatric stupors preserve it, and the discriminator is context plus the accompanying signs.

CLASSIC TRAP In psychiatry, stupor is akinesis and mutism with consciousness relatively preserved (eyes open, follow objects); in neurology it implies impaired consciousness between drowsiness and coma - the same word, opposite implication for the sensorium.

18.10 The disorders of consciousness: changes in the field

Definition. Beyond a simple dimming of level, consciousness may be *narrowed* or *qualitatively altered*, a change in the *field* rather than the level of awareness (Fish 2007, Sims 2015).

Twilight state

An abnormal state of narrowed but relatively *coherent* consciousness: awareness is constricted to a limited set of ideas and affects, yet the patient may still perform complex, apparently purposeful

actions, of which there is subsequent amnesia. Classically seen in epilepsy (postictal and complex partial states), and in dissociative and organic (e.g. anticholinergic) conditions. Emotional outbursts or violent acts may occur during a twilight state (Kaplan & Sadock 2024; Clinical Methods in Psychiatry, AIIMS 2024).

Oneiroid state

A dream-like alteration of consciousness in which vivid, often fantastic scenic (dream-like) imagery unfolds and the patient is partly detached from the real environment while not asleep; primary and secondary process thinking intermingle. If prolonged, an oneiroid state may merge into a twilight state (Shorter Oxford Textbook of Psychiatry; Kaplan & Sadock 2024). Described in catatonic and cycloid psychoses and in some organic and toxic states.

PEARL

Distinguish a change in *level* from a change in *field*. Clouding, drowsiness and coma dim the overall brightness of consciousness; the **twilight state** and the **oneiroid** state leave a preserved brightness but distort its *content and range*, narrowing the field (twilight) or filling it with dream-imagery (oneiroid). The historical Ganser syndrome, with its twilight state, approximate answers (vorbeireden) and clouded consciousness, sits at the boundary of the two (Kaplan & Sadock 2024).

MNEMONIC

Level = "CD-DS-C"; Field = "TO"

The *level* continuum in order of deepening: **C**louding, **D**rowsiness, **D**elirium (as the clinical syndrome of clouding), **S**tupor (psychiatric sense, level preserved), **C**oma. The *field* changes: **T**wilight (narrowed, coherent) and **O**neiroid (dream-like).

GOOD TO REMEMBER

- **Disorders of memory:** split into loss of memory (amnesias, organic amnesic syndrome), distortions of recall (paramnesias), and intensification (hypermnesia).
- **Anterograde amnesia:** loss of memory for events *after* the insult (failure of new learning); post-traumatic amnesia duration indexes head-injury severity.
- **Retrograde amnesia:** loss of memory for events *before* the insult; shrinks with time, obeys Ribot's law (recent memories most vulnerable).
- **Organic amnesic syndrome:** selective recent-memory and new-learning deficit in clear consciousness, no global intellectual loss (ICD-10); diencephalic / medial-temporal lesions.
- **Korsakoff syndrome:** chronic amnesic residue of thiamine deficiency in alcohol dependence; dense anterograde amnesia, disorientation, absent insight, early confabulation (cross-refer the Functional Neuroanatomy & Neurophysiology notes and the organic psychiatry chapter).
- **Transient global amnesia:** abrupt isolated anterograde amnesia for hours in an alert older person, repeated questioning, identity retained, resolves in ~24 h.
- **Psychogenic (dissociative) amnesia:** retrograde loss of emotionally significant autobiographical memory after stress; spares neutral facts and skills.
- **Dissociative fugue:** dissociative amnesia plus purposeful travel and loss of / new personal identity, with amnesia for the episode.
- **Paramnesia:** a distortion (falsification) of recall rather than a loss.
- **Confabulation:** filling a memory gap with fabricated / misplaced material accepted as true, without intent to deceive; classic in Korsakoff; momentary (provoked) vs fantastic (spontaneous) types.
- **Deja vu:** false sense of familiarity with a novel experience; frequent/stereotyped forms suggest temporal lobe epilepsy.
- **Jamais vu:** false sense of unfamiliarity with a genuinely familiar situation; temporal lobe epilepsy, dissociation.
- **Retrospective falsification:** a real memory unwittingly distorted by present mood or belief.
- **Delusional memory:** recollection of a clearly delusional event, or a real memory invested with delusional meaning; a content-of-thought disorder.
- **Hypermnesia:** abnormal intensification of recall (mania, hypnosis, eidetic imagery, flashbulb / traumatic memories); the mirror of amnesia.
- **Disorders of consciousness:** split into change of *level* (wakefulness continuum) and change of *field* (narrowing / qualitative alteration).
- **Clouding of consciousness:** incomplete reaction to stimuli with impaired attention, memory and orientation; the defining feature of delirium.
- **Delirium:** acute, fluctuating clouding with global cognitive impairment and perceptual disturbance on an organic basis (management cross-referred to the delirium and neurocognitive notes).
- **Drowsiness / somnolence (torpor):** pathological rousable sleepiness with slow thinking and narrowed perception.
- **Coma:** unrousable unresponsiveness with no purposeful response to strong stimuli; graded by reflexes and EEG.
- **Stupor (psychiatric):** akinesis and mutism with *relative preservation of consciousness* (eyes open, follow objects); differs from neurological usage (which implies impaired consciousness). Types: organic, catatonic, depressive, dissociative.
- **Twilight state:** narrowed but relatively coherent consciousness with complex purposeful acts and later amnesia; epilepsy, dissociation, organic states.
- **Oneiroid state:** dream-like consciousness with vivid scenic imagery while awake; catatonic / cycloid psychoses, organic-toxic states; may merge into a twilight state if prolonged.

19 · Disorders of movement, and attention, orientation and insight

This note closes the phenomenology by teaching two remaining domains. The **disorders of motor** function are the abnormalities of movement and behaviour that arise from a disordered mind rather than from a primary neurological lesion, with the catatonic syndrome at their centre. Then come the cognitive domains that complete the mental state examination: attention, orientation, insight and judgement. As throughout descriptive psychopathology, the discipline is to name the sign and define it crisply (Fish 2007; Sims 2015).

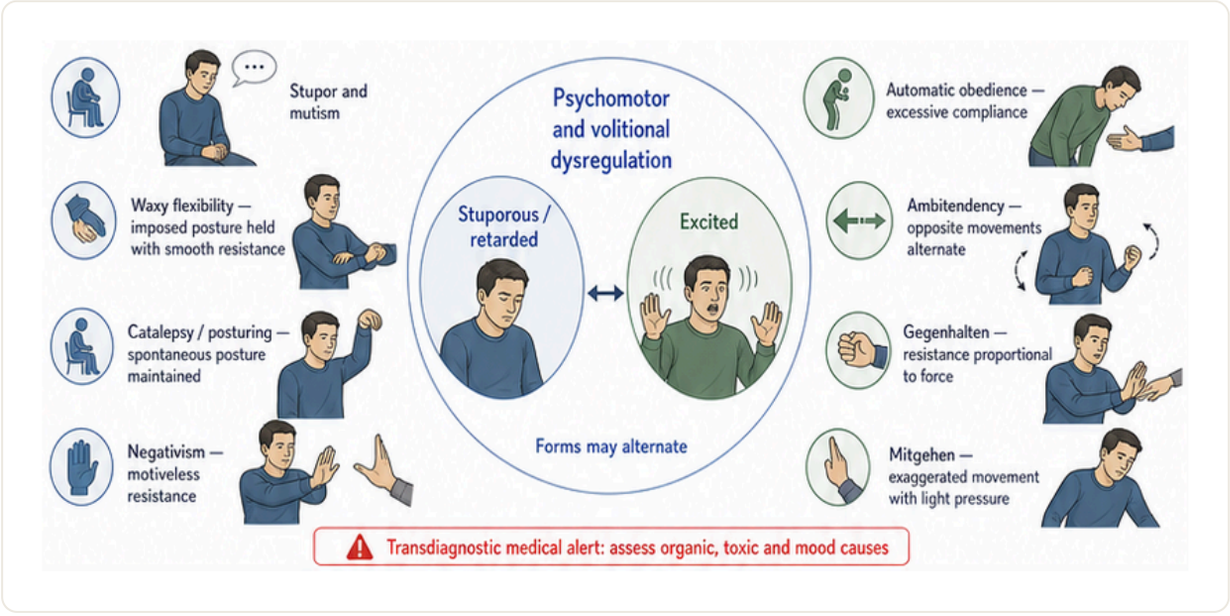


Fig 6.28 Catatonia: dysregulation of will and movement.

Exam framing. Catatonia is high-yield and returns as a short note (its clinical features), so learn the signs precisely and be able to separate the abnormal movements from one another. The cognitive close of the mental state, attention, orientation, insight and judgement, is where a candidate loses easy marks by treating insight as all-or-none; the examiner wants it graded. The acute treatment of catatonia (benzodiazepines, ECT) belongs to the emergency psychiatry notes and is cross-referred.

19.1 Catatonia: the syndrome

Definition. **Catatonia** is a syndrome of psychomotor dysregulation in which the normal relationship between will and movement is disturbed, producing a striking combination of motor, volitional and behavioural signs (Kahlbaum 1874; Fish 2007). It was long tied to schizophrenia but is now recognised as transdiagnostic: it complicates mood disorders (especially severe depression and mania) more often than schizophrenia, and it arises from many organic and toxic causes (encephalitis, metabolic derangement, autoimmune disease). It can present as a retarded/stuporous form or an excited form, and the two may alternate. Modern operationalised criteria (DSM-5, and rating instruments such as the Bush-Francis catatonia rating scale) require a threshold number of signs from the list below (Bush and colleagues 1996).

19.2 The catatonic signs

The named signs, each defined. The examiner rewards the ability to define each sign in a line, so hold the list precisely (Fish 2007; Sims 2015).

Stupor

Akinesis and mutism with relative preservation of consciousness (the catatonic stupor; cross-refer the consciousness topic).

Waxy flexibility (cerea flexibilitas)

The limbs, when moved by the examiner, hold their new posture with a smooth, even, plastic resistance, like bending a wax rod; the imposed posture is then maintained.

Catalepsy and posturing

The spontaneous maintenance of an abnormal, often uncomfortable posture for long periods; psychological pillow, in which the head is held raised off the bed as if on a pillow, is the classic example.

Negativism

A motiveless resistance to all instructions or attempts to be moved, and the doing of the opposite of what is asked; a disorder of the will, not simple non-cooperation.

Mutism and automatic obedience

Mutism is a verbal near-absence with no organic cause. Automatic obedience is the opposite pole to negativism: the patient carries out every instruction like an automaton, even harmful ones.

Ambitendency

An alternation between opposite movements arising from a conflict of will, for example repeatedly half-extending and withdrawing the hand when asked to shake hands.

Gegenhalten and mitgehen

Gegenhalten (opposition) is a resistance to passive movement proportional to the force applied; mitgehen (going along) is an excessive, exaggerated movement of a body part in response to the lightest pressure ("anglepoise lamp" sign).

Excitement

The excited form: purposeless, frenzied, agitated motor activity not influenced by external stimuli, which may become a life-threatening malignant catatonia.

> **COMMON TRAP**
>
> Waxy flexibility, negativism and automatic obedience look contradictory but coexist in the same syndrome because all three are disorders of the *will*: catatonia is a dysregulation of volition, so a patient may resist one instruction (negativism) yet passively hold an imposed posture (waxy flexibility) minutes later. Do not expect internal consistency.

19.3 Abnormal movements and behaviours

The repetitive and imitative signs. Beyond the catatonic core, descriptive psychopathology names several disorders of purposeful movement, and the exam turns on telling them apart (Fish 2007; Sims 2015).

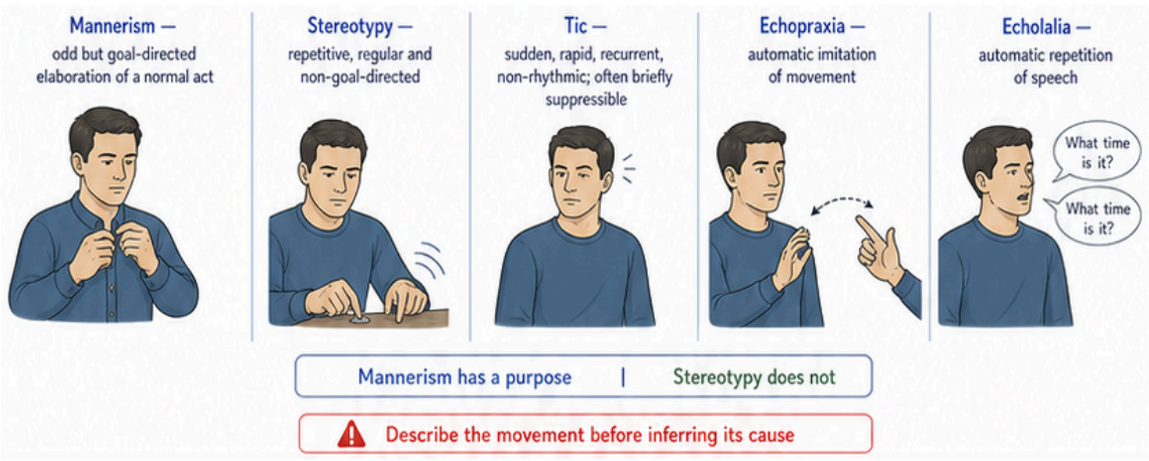


Fig 6.29 Abnormal movements: purpose is the discriminator.

Mannerism

An odd, stilted or bizarre *goal-directed* movement or elaboration of a normal action: a stereotyped, theatrical salute instead of an ordinary greeting. The movement has a purpose, but it is performed strangely.

Stereotypy

A repetitive, regular, *non-goal-directed* movement (rocking, rubbing, repeatedly touching an object) carried out uniformly and without evident purpose. Purposelessness is the discriminator from mannerism.

Tic

A sudden, involuntary, rapid, recurrent, non-rhythmic movement or vocalisation, often briefly suppressible, distinct from the catatonic and stereotyped signs.

Echopraxia

The automatic, senseless imitation of another person's *movements*, even when asked not to; its verbal counterpart is echolalia, the imitation of another's speech.

CLINICAL ANCHOR

Catatonic signs are a medical alert, not just a description. Recognising waxy flexibility, mutism, negativism or posturing should prompt a lorazepam challenge and a search for an organic cause, because untreated catatonia risks dehydration, venous thromboembolism and malignant catatonia. The phenomenology is the entry point to a treatable emergency; the acute management is developed on the emergency psychiatry notes.

RULE **Purpose separates mannerism from stereotypy.** A mannerism is an odd but goal-directed movement; a stereotypy is repetitive and non-goal-directed.

19.4 Attention and concentration

Definition. **Attention** is the active direction of consciousness onto a target, and concentration is its sustained maintenance over time (Sims 2015). Disorders include distractibility (attention captured by every passing stimulus, as in mania and delirium), impersistence (inability to sustain

attention), and the narrowed, poorly shifted attention of clouded consciousness. At the bedside attention and concentration are tested by digit span (registration and immediate attention), serial sevens or serial threes, and reciting the months of the year in reverse.

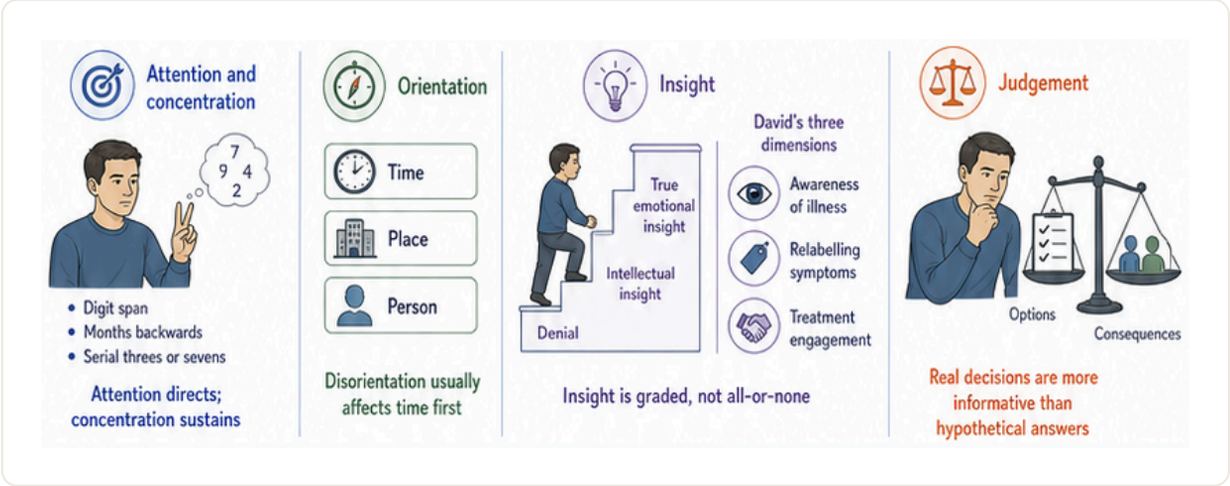


Fig 6.30 The cognitive close of the mental state examination.

19.5 Orientation

Definition. Orientation is the awareness of oneself in relation to time, place and person: knowing the date and time, the current location, and the identity of oneself and others. Disorientation, a loss of this awareness, is a cardinal sign of an *organic* disturbance of consciousness (delirium and dementia) and characteristically affects time first, then place, then person. Orientation is closely tied to attention and memory: a patient cannot be oriented if attention or new learning is grossly impaired. Preserved orientation in a floridly psychotic patient points away from an organic confusional state.

19.6 Insight

Definition, and why it is graded. Insight is the patient's awareness of their own mental state: that they are ill, that the illness is psychiatric, and that treatment is needed. The examinable point is that insight is not all-or-none but a graded, multidimensional phenomenon. A useful clinical grading runs: complete denial of illness; slight awareness of being ill and needing help but denying it at the same time; awareness of being ill but attributing it to external or physical causes; awareness that the illness is due to something unknown in the self; intellectual insight (admitting illness and that symptoms are due to a disturbance in the self, but without applying this knowledge); and true (emotional) insight, in which the awareness leads to changed behaviour and adherence. David's three-dimensional model (awareness of illness, correct relabelling of symptoms as pathological, and treatment adherence) is the modern operationalisation. Insight has prognostic and medico-legal weight (it bears on capacity and on adherence).

HOLD THIS

Catatonia is a transdiagnostic dysregulation of the will (commoner in mood disorders than schizophrenia), and insight is graded, not all-or-none. David's three dimensions: awareness of illness, relabelling of symptoms, and treatment adherence.

19.7 Judgement

Definition. **Judgement** (also spelt **judgment**) is the capacity to assess a situation correctly and to act sensibly within it, weighing options and consequences. It is assessed by observing the patient's real decisions (social, financial, personal) and, historically, by hypothetical test questions, though these are unreliable. Impaired judgement is prominent in mania (reckless spending, disinhibition), in the frontal-executive syndromes, in intoxication, and in psychosis when actions follow delusional beliefs. Judgement and insight together complete the cognitive close of the mental state examination.

PEARL

Orientation is the fastest bedside organic screen. A patient who is disoriented to time and place, especially with fluctuation and inattention, has an organic disturbance of consciousness (delirium) until proven otherwise, whatever the psychiatric content; a patient with florid hallucinations and delusions but intact orientation and attention is more likely in a primary psychotic state. Test orientation early.

MNEMONIC

Movement: "MSET" and "WooNBAG"

The abnormal movements to separate: **M**annerism (goal-directed, odd), **S**tereotypy (non-goal-directed, repetitive), **E**chopraxia (imitates movement), **T**ic. The catatonic core: **W**axy flexibility, autOmatic obedience, **N**egativism, **B**izarre posturing/catalepsy, **A**mbitendency, **G**egenhalten (with mutism and stupor).

GOOD TO REMEMBER

- **Disorders of motor function:** abnormalities of movement and behaviour of psychological origin; the catatonic syndrome is the core.
- **Catatonia:** a transdiagnostic syndrome of psychomotor and volitional dysregulation (Kahlbaum); commoner in mood disorders than schizophrenia; retarded/stuporous and excited forms.
- **Waxy flexibility (cerea flexibilitas):** limbs hold an imposed posture with smooth plastic resistance.
- **Negativism:** motiveless resistance to instruction, doing the opposite; a disorder of will.
- **Automatic obedience and ambitendency:** automaton-like compliance; alternation between opposite movements from conflict of will.
- **Catalepsy / posturing, gegenhalten, mitgehen:** maintained abnormal posture; force-proportional opposition; exaggerated go-along movement.
- **Mannerism:** odd but *goal-directed* movement; **stereotypy:** repetitive *non-goal-directed* movement (the discriminator is purpose).
- **Echopraxia:** automatic imitation of movement (echolalia = imitation of speech).
- **Attention:** active direction of consciousness (concentration = its maintenance); tested by digit span and serial sevens; distractibility in mania and delirium.
- **Orientation:** awareness of time, place and person; lost first for time; disorientation is a cardinal organic sign.
- **Insight:** awareness of being ill, of the illness being psychiatric, and of needing treatment; graded, not all-or-none (David's three dimensions: awareness, relabelling, adherence).
- **Judgement (judgment):** capacity to assess a situation and act sensibly; impaired in mania, frontal syndromes, intoxication and psychosis.

Discriminate

20 · Discriminate: pick the test, name the sign

The two bands of these notes are examined most often as acts of discrimination: choosing the right instrument for a clinical problem, and separating one phenomenological sign from its near-neighbour. This is the step that turns a catalogue into a usable skill. Read the tables below as decision aids, not lists.

20.1 Band A: choosing the right test for the clinical question

Match the tool to the referral question. A psychological test is chosen to answer a specific **clinical question**, and good **test selection** starts from the **referral question** rather than from a favourite instrument. The table pairs the common questions with the answer to "**which test**", so you can **choose the test** that the problem actually needs.

THE CLINICAL / REFERRAL QUESTION	CHOOSE THE TEST	WHY
Is there intellectual disability, and how severe?	**A Wechsler (WAIS/WISC) or Binet-Kamat intelligence test plus a Vineland/VSMS adaptive measure**	ID needs both a low IQ and a deficit in adaptive behaviour; measure both
Which cognitive domains are impaired after this head injury or in this dementia?	**A neuropsychological battery (Halstead-Reitan or a flexible battery; NIMHANS battery in India)**	Profiles attention, memory, language, visuospatial and executive function domain by domain
What is the personality structure, or is the presentation being feigned?	**The MMPI (with its validity scales) and, for depth, the projectives**	The MMPI validity scales detect faking; the projectives add a dynamic picture
Is this child developing normally?	**A developmental screen (DST, Denver) or a Bayley / Gesell assessment**	Yields a developmental quotient against age norms
How severe is this depression, and is it responding to treatment?	**A symptom rating scale: HAM-D or MADRS (mania YMRS; psychosis PANSS; OCD Y-BOCS)**	Quantifies severity and tracks change over time

Test selection is driven by the referral question: ability, cognitive profile, personality, development or symptom severity each point to a different family of instrument.

20.2 Band B: illusion versus hallucination versus pseudohallucination

The perception triad. The single most tested discrimination in phenomenology separates the **illusion** (a real stimulus misperceived) from the **hallucination** (a percept with no stimulus, in outer objective space, full reality, insight lost) and from the **pseudohallucination** (a percept in inner subjective space, lacking full substantiality, insight relatively preserved). The two discriminators that do most of the work are the presence of a real stimulus and the locus (outer versus inner space).

DISCRIMINATOR	ILLUSION	HALLUCINATION	PSEUDOHALLUCINATION
Real external stimulus	Yes, misperceived	**No**	**No**
Locus	Outer space	Outer objective space	**Inner subjective space**
Reality status	Real object transformed	Full force of a true percept	**Lacks full substantiality**
Insight	Corrects on attention	Absent	**Relatively preserved**

Real stimulus separates the illusion from the other two; the outer-versus-inner locus separates the hallucination from the pseudohallucination.

20.3 Band B: obsession versus overvalued idea versus delusion

The belief triad. The second high-yield discrimination separates the **obsession** (ego-dystonic, resisted, insight retained) from the **overvalued idea** (a comprehensible belief held strongly but not with absolute fixity) from the **delusion** (ego-syntonic, unresisted, held with absolute

conviction and no insight). Read the row for insight and resistance and the answer usually falls out.

DISCRIMINATOR	OBSESSION	OVERVALUED IDEA	DELUSION
Conviction / fixity	Recognised as senseless, doubted	Strong, not absolute	**Absolute, incorrigible**
Resistance	**Actively resisted**	Not resisted	Not resisted
Ego relation	**Ego-dystonic (intrusive)**	Ego-syntonic	Ego-syntonic
Insight	Preserved	Partial	**Absent**
Typically in	OCD	Anorexia, dysmorphophobia, morbid jealousy	Psychotic disorders

Resistance and retained insight mark the obsession; absolute conviction with no insight marks the delusion; the overvalued idea sits in between.

HOLD THIS

Band A discriminates by the referral question; Band B by two triads. Perception: real stimulus and locus separate illusion, hallucination and pseudohallucination. Belief: resistance and insight separate obsession, overvalued idea and delusion.

20.4 The other near-misses worth pre-empting

Three more pairs the exam rewards. A few further discriminations recur across the phenomenology and are worth rehearsing together.

Blunting versus incongruity of affect

Blunting is a *loss of range and depth* of emotional expression; incongruity is emotion that is present but *does not match* the thought content (laughing while describing a bereavement). Both occur in schizophrenia; one is reduced affect, the other mismatched affect.

Delirium versus dementia

Delirium is acute, fluctuating, with clouded consciousness and impaired attention; dementia is chronic, stable or progressive, with a clear sensorium until late. Consciousness and time-course separate them.

Mannerism versus stereotypy

A mannerism is an odd but *goal-directed* movement; a stereotypy is a repetitive *non-goal-directed* movement. Purpose is the discriminator.

COMMON TRAP

The word "unreal" hides three different phenomena. A patient who feels the world is unreal but knows it is a feeling has *derealisation* (insight kept); a patient who has a vivid inner-space voice they know is not a true perception has a *pseudohallucination*; a patient convinced as fact that they do not exist has a *nihilistic delusion*. The content overlaps; the discriminators are the locus and the insight, never the word itself. Likewise "hears voices" is not automatically a hallucination: an illusion elaborates a real sound, a functional hallucination is triggered by one, and a pseudohallucination sits in inner space.

GOOD TO REMEMBER

- **Test selection:** start from the referral question (ability, cognitive profile, personality, development, symptom severity), then choose the test family that answers it.
- **ID work-up:** intelligence test plus an adaptive-behaviour measure (both are required for the diagnosis).
- **Illusion vs hallucination vs pseudohallucination:** real stimulus (illusion only); outer vs inner space (hallucination vs pseudohallucination); insight lost only in the true hallucination.
- **Obsession vs overvalued idea vs delusion:** resisted and ego-dystonic with insight (obsession); absolute conviction, unresisted, no insight (delusion); the overvalued idea is in between.
- **Blunting vs incongruity:** reduced range vs mismatched affect. **Delirium vs dementia:** clouded fluctuating vs clear chronic. **Mannerism vs stereotypy:** goal-directed vs purposeless.

Apply and consolidate

21 · Apply: four assessment and phenomenology vignettes

Four short worked vignettes put these notes to use. Each gives a brief presentation, then the reasoning, then the read. Cover the reasoning and try to reach the answer first; the discriminator that carries each case is the point to internalise.

21.1 A test-selection case

VIGNETTE 1

Case: A 7-year-old boy is referred because he is failing at school and is slow to acquire self-care skills. The paediatrician asks whether he has intellectual disability and, if so, how severe.

Reasoning. Intellectual disability is defined by two things together, significantly subaverage intellectual functioning *and* a deficit in adaptive behaviour, both with childhood onset. A single test cannot answer the referral. The intellectual limb needs an individually administered intelligence test normed for Indian children (the Binet-Kamat test or Malin's MISIC; a Wechsler where appropriate). The adaptive limb needs an adaptive-behaviour measure (the Vineland scales or the VSMS, yielding a social quotient). Severity is then graded, and in current systems (DSM-5, ICD-11) the grading rests on adaptive functioning rather than the IQ score alone.

The read. Choose an intelligence test *and* an adaptive-behaviour scale, not an IQ test alone. Grade severity primarily on adaptive functioning. Add a developmental and sensory work-up to exclude a treatable cause.

21.2 An IQ interpretation

VIGNETTE 2

Case: An adult completes the WAIS. The Verbal Comprehension index is 108, the Perceptual Reasoning index is 71, and the Full-Scale IQ is 88. What does the profile mean?

Reasoning. The deviation IQ has a mean of 100 and a standard deviation of 15, so a Full-Scale IQ of 88 falls in the low-average band (80 to 89), which alone might be read as mild global underfunctioning. But the Full-Scale IQ conceals a large split: verbal comprehension is average (108) while perceptual reasoning is borderline (71), a 37-point gap. When the index scores diverge this much, the Full-Scale IQ is not a meaningful summary of a single ability; the split itself is the finding and points to a selective, often perceptual-organisational or nonverbal, deficit that warrants a neuropsychological work-up.

The read. Do not report the Full-Scale IQ of 88 as "low average and homogeneous". Report the significant verbal-performance split and investigate the low perceptual reasoning index; the pattern, not the single number, carries the clinical meaning.

RULE **When the index scores scatter widely, the Full-Scale IQ stops being a meaningful summary.** The split itself is the finding; report and investigate it rather than the single global number.

21.3 A phenomenology spotter

VIGNETTE 3

Case: A man reports that whenever, and only when, the ceiling fan is running, he hears a clear voice speaking to him through the room; he still hears the fan as a fan at the same time. Name the sign.

Reasoning. There is a real external stimulus (the fan) that is *necessary* to provoke the experience, the stimulus is still perceived normally as itself, and the added percept, the voice, occurs together with it and in the same (auditory) modality. That is not an illusion (the voice is not an elaboration of the fan noise), not an ordinary hallucination (a real stimulus is required), and not a reflex hallucination (which would cross to a different modality). It is a functional hallucination.

The read. Functional hallucination: same modality as the triggering stimulus, both perceived together. Contrast the reflex hallucination, where a stimulus in one modality triggers a hallucination in another.

21.4 A first-rank-symptom read

VIGNETTE 4

Case: A young woman says that when she saw a red car pass in the street, she knew instantly and with total certainty that she had been chosen by God to save the country. She is sure the two are connected. What is the sign, and does it clinch a diagnosis?

Reasoning. A real, normally perceived object (the red car) is given an immediate, self-referential delusional meaning that is not understandable from the perception itself. This is a delusional perception, one of Jaspers' primary delusional experiences and a Schneiderian first-rank symptom. The important second half of the question is diagnostic weight: first-rank symptoms are suggestive of schizophrenia but are *not* pathognomonic (they occur in mania and organic psychoses, and a fifth of people with schizophrenia never show them; Carpenter and colleagues 1973), and DSM-5 and ICD-11 have removed their special diagnostic status.

The read. Delusional perception (a primary delusion and a first-rank symptom). It raises the probability of schizophrenia but does not clinch it; the diagnosis rests on the whole picture and course, not one sign.

HOLD THIS

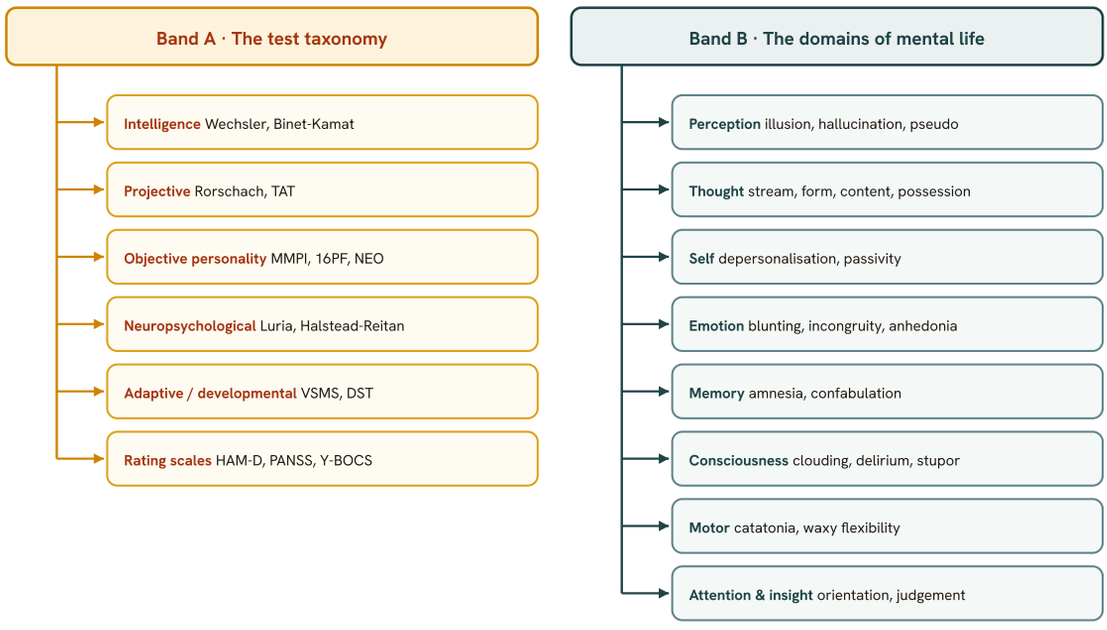
Two meta-lessons carry every vignette: on the test side, the pattern beats the single number; on the phenomenology side, the named form beats the content, and no single sign clinches a diagnosis.

GOOD TO REMEMBER

- **Vignette 1 (test selection):** intellectual disability needs an intelligence test *and* an adaptive-behaviour scale; grade severity by adaptive function.
- **Vignette 2 (IQ interpretation):** a large verbal-performance / index split makes the Full-Scale IQ misleading; report and investigate the split.
- **Vignette 3 (spotter):** a real stimulus needed, perceived alongside the hallucination in the same modality, is a functional hallucination.
- **Vignette 4 (first-rank):** a real percept given sudden delusional meaning is a delusional perception; first-rank symptoms are suggestive of, but not pathognomonic for, schizophrenia.

22 · Consolidate: mnemonic, retrieval and synthesis

Three devices close these notes: a master mnemonic that chains both bands, a set of answer-free retrieval prompts to test yourself against, and the whole-day synthesis map to redraw from memory. Use them in that order.



Cover one band and redraw it from memory, then check against the other.

Fig 6.31 The synthesis map. It has two halves that mirror each other: a taxonomy of the tests we use to measure the mind, and a taxonomy of the domains in which the mind can go wrong. Redraw each band from memory, filling in the example instruments on the left and the example signs on the right, then check one against the other. If both columns come back, the material is consolidated.

22.1 The master mnemonic

MNEMONIC

Tests "I-PONAR"; Mind "PT-SEM-CMA"

This **mnemonic** chains the whole set of notes. The **test taxonomy (I-PONAR)**: **I**ntelligence, **P**rojective, **O**bjective personality, **N**europsychological, **A**daptive/developmental, **R**ating scales. The **domains of mental life (PT-SEM-CMA)**: **P**erception, **T**hought (with delusions and the first-rank symptoms), **S**elf, **E**motion, **M**emory, **C**onsciousness, **M**otor, and **A**ttention/orientation/insight. Recall the two strings and every topic in these notes hangs off a letter: the tests you would reach for, and the domains in which the mind can go wrong.

22.2 Retrieval practice

RETRIEVAL PRACTICE, COVER THE ANSWERS

Use these as **active recall** prompts: cover the answers, answer each aloud or on paper, then check. Spaced **retrieval practice** across the two bands is what fixes this material.

1. Name the six families of psychological test and one instrument in each.
2. Distinguish the types of reliability from the types of validity; which one caps the other?
3. What is the mean and standard deviation of the deviation IQ, and where does the intellectual-disability range begin?
4. Describe the two-phase administration of the Rorschach and name the five Exner coding axes.
5. List the MMPI validity scales and say what each detects.
6. Name three frontal or executive neuropsychological tests and what each measures.
7. Which Indian tests adapt the Stanford-Binet and the WISC?
8. Define an illusion, a hallucination and a pseudohallucination on the discriminators of stimulus, locus and insight.
9. Give the four Jaspersian ways thought can be disordered (stream, form, content, possession) with one example of each.
10. Separate a primary from a secondary delusion, and name three primary delusional experiences.
11. List the Schneiderian first-rank symptoms, and state whether they are pathognomonic of schizophrenia.
12. Define depersonalisation and derealisation, and give the feature that keeps them from being delusions.
13. Distinguish blunting from incongruity of affect, and anhedonia from alexithymia.
14. Separate the four types of stupor, and name the two changes in the field of consciousness.
15. Define waxy flexibility, negativism, mannerism and stereotypy, and give the discriminator between mannerism and stereotypy.

22.3 The synthesis map

Redraw these notes in two halves. The final device is the **synthesis map** for these notes, which you should **redraw from memory** on a blank page. It has two mirror halves. On the left, the test-taxonomy band: the six families, each with what it measures and one anchoring instrument. On the right, the domains-of-mental-life band: the eight domains, each with its characteristic signs. Cover one half and reconstruct it from the other, filling in the example instruments on the left and the example signs on the right. If both columns come back cleanly, the material is consolidated; if a branch is blank, that is the topic to revisit. The figure accompanying this section is the map to copy first, then reproduce unaided.

Previously asked

Real long-essay (25-mark) and short-note (10-mark) questions from the psychometry, assessment and descriptive-psychopathology territory, most-recent year shown.

LONG ESSAYS (25 MARKS)

- Define emotions. Discuss the various theories and the psychopathological presentations related to emotion. MUHS 2017
- Describe the perceptual processes and the various disorders of perception. MUHS 2014

SHORT NOTES (10 MARKS)

- Rorschach test. MUHS 2020
- Indications of various psychiatric disorders on the Rorschach test. MUHS 2020
- Minnesota Multiphasic Personality Inventory (MMPI). MUHS 2016
- Rating scales in psychiatry. MUHS 2020
- Clinical features of catatonia. MUHS 2020

This territory is examined in two recurring ways. On the psychometry side the projectives (the Rorschach above all), the MMPI and the rating scales come as short notes, so prepare each test to give its author, what it measures and how it is scored in a few lines. On the phenomenology side the disorders of perception and the psychopathology of emotion recur as full essays, and catatonia as a short note, so be able to define each sign precisely and place it in its domain. The theories of intelligence, emotion and personality are covered in the General and Developmental Psychology and Social, Learning & Personality Psychology notes; the acute assessment and management of the catatonic or violent patient is developed in the emergency psychiatry chapter; and the fuller catalogue of clinician rating scales is taken up again in the Paper III psychotherapy and assessment notes.

Quick review

The whole picture in one table	
THE TEST TAXONOMY	Every test belongs to a family defined by what it measures: intelligence (ability), projective and objective (personality), neuropsychological (brain-behaviour), adaptive and developmental (functioning), and rating scales (symptom severity). Read a referral by deciding which family it points to.
TEST CONSTRUCTION	A good test is standardised (uniform administration, norms), reliable and valid. Reliability (consistency): test-retest, inter-rater, internal consistency (Cronbach's alpha, split-half). Validity (measures what it claims): face, content, criterion (concurrent, predictive), construct (convergent, discriminant). Item analysis and test bias. Validity requires reliability, not the reverse. Distinct from research reliability and validity, covered in the Research Methods, Biostatistics & Epidemiology notes.
INTELLIGENCE TESTS	Ratio IQ (mental age over chronological age) gave way to the deviation IQ (mean 100, SD 15). The Stanford-Binet lineage and the Wechsler scales (WAIS, WISC): verbal and performance subtests to four index scores to a Full-Scale IQ. Classification bands: average 90 to 109, intellectual disability below 70 (mild, moderate, severe, profound). DSM-5 grades ID by adaptive function. Theories are covered in the General and Developmental Psychology notes.
RORSCHACH	Ten inkblot cards (Hermann Rorschach, 1921); a projective test of personality and thought organisation. Free-association then inquiry; scored on the Exner system by location, determinants, form quality, content and popular responses (R-PAS is the successor). Reliability and validity are contested.
PROJECTIVE TESTS	The projective hypothesis: ambiguous stimuli reveal unconscious material. TAT (Murray, story-telling to ambiguous cards; CAT for children), sentence completion (Rotter), Draw-a-Person (Machover). Rich but low-reliability; the counterpart to the objective inventories.
OBJECTIVE PERSONALITY	Structured self-report with fixed scoring. MMPI (Hathaway and McKinley, empirically keyed; validity scales L, F, K and ten clinical scales), 16PF (Cattell), NEO-PI-R (the Big Five), EPQ (Eysenck's PEN). Operationalise the personality theories from the Social, Learning & Personality Psychology notes; validity scales guard against faking.
NEUROPSYCHOLOGICAL TESTING	Fixed batteries (Halstead-Reitan) versus the flexible, hypothesis-driven approach; the Luria-Nebraska battery. Frontal and executive tests: Wisconsin Card Sorting (set-shifting), Stroop (inhibition), Trail Making A and B, verbal fluency. Bender Gestalt for visuoconstruction. Used in dementia, TBI and localisation.
ADAPTIVE AND DEVELOPMENTAL SCALES	Adaptive behaviour (now central to the ID diagnosis): the Vineland scales and the VSMS (social

The whole picture in one table	
	quotient). Developmental screening and assessment: DST (Bharat Raj), Denver, Bayley, Gesell schedules, Seguin form board. Yield developmental and social quotients.
RATING SCALES	Clinician-rated symptom scales by domain: depression (HAM-D, MADRS), mania (YMRS), psychosis (BPRS, PANSS), OCD (Y-BOCS); global (CGI), movement (AIMS), cognition (MMSE, MoCA). Used to quantify severity and track response in trials and in the clinic.
INDIAN TEST ADAPTATIONS	Intelligence: Binet-Kamat (BKT), Malin's MISIC (Indian WISC), Bhatia's performance battery. Adaptive and developmental: VSMS Indian norms (Malin), DST (Bharat Raj). Neuropsychology: the NIMHANS neuropsychological battery, PGI Battery of Brain Dysfunction and PGI Memory Scale. Adaptation matters for norms, language and literacy.
DESCRIPTIVE PSYCHOPATHOLOGY	The theory-free description of abnormal experience (Jaspers, Fish, Sims). Form (the kind of experience) before content (what it is about). The domains of mental life: perception, thought, self, emotion, memory, consciousness, motor, and attention, orientation and insight. A precise sign is the unit of the mental state examination.
DISORDERS OF PERCEPTION	Sensory distortions (a real percept altered); illusions (a real stimulus misperceived); hallucinations (a percept without a stimulus, in outer space, full reality, no insight) by modality and the special types (functional, reflex, extracampine, autoscopy, hypnagogic); pseudohallucination (inner subjective space, lacks full substantiality, insight retained). Mechanism theories are covered in the General and Developmental Psychology notes.
DISORDERS OF THOUGHT	Stream (flight of ideas, thought block, circumstantiality), form (formal thought disorder: loosening, derailment, knight's-move, neologisms), content (overvalued ideas, obsessions, delusions), and possession (thought insertion, withdrawal, broadcast, the first-rank alienations).
DELUSIONS	A fixed false belief, absolute conviction, incorrigible, out of cultural keeping. Jaspers' primary (autochthonous: delusional mood, delusional perception, sudden idea, delusional memory) versus secondary. Types by content: persecutory, grandiose, referential, nihilistic (Cotard), erotomanic, jealousy, misidentification (Capgras). Delusion versus overvalued idea versus obsession.
FIRST-RANK SYMPTOMS	Schneider's pragmatic markers: thought echo and third-person voices with running commentary; thought insertion, withdrawal, broadcast; passivity of act, impulse, affect and somatic passivity; delusional perception. Not pathognomonic (seen in mania and organic states; absent in some schizophrenia) and stripped of special status in DSM-5 and ICD-11.
DISORDERS OF SELF AND EMOTION	Self: loss of the ego boundary, depersonalisation (the self feels unreal) and derealisation (the world does),

The whole picture in one table	
	the "as if" quality preserving insight; the dimensions of ego-consciousness; passivity. Emotion: distinguish affect, mood and emotion; blunting and flattening, incongruity of affect, lability, anhedonia and alexithymia.
MEMORY AND CONSCIOUSNESS	Memory: anterograde and retrograde amnesia, Korsakoff, transient global amnesia, and the paramnesias (confabulation, deja vu, jamais vu). Consciousness: the level (clouding, delirium, stupor, coma) and the field (twilight and oneiroid states). Cross-refers to the delirium and organic psychiatry chapters.
MOTOR, ATTENTION AND INSIGHT	Motor: catatonia (waxy flexibility, negativism, posturing, stupor, automatic obedience) and abnormal movements (mannerism, stereotypy, echopraxia). Then the cognitive close of the mental state: attention and concentration, orientation, insight (graded, not all-or-none) and judgement.
DISCRIMINATE AND APPLY	Band A: match the clinical or referral question to the right test (query ID to a Wechsler plus a Vineland; cognitive decline to a neuropsychological battery; depression severity to HAM-D or MADRS). Band B: tell illusion from hallucination from pseudohallucination, and obsession from overvalued idea from delusion. Then read four vignettes: a test choice, an IQ interpretation, a phenomenology spotter and a first-rank read.

References

These notes are grounded in the standard texts (the source hierarchy below); each test is attributed to its author and each sign to its describing text, and landmark, contested or numeric claims carry a PubMed-verified journal citation. Every PMID here was checked against PubMed this build, and any citation whose identifier did not resolve to the cited paper was corrected or dropped.

SOURCE HIERARCHY (TEXTBOOKS)

1. Boland R, Verduin ML, eds. Kaplan & Sadock's Comprehensive Textbook of Psychiatry. 11th ed. Philadelphia: Wolters Kluwer; 2024. [primary depth bar; psychological assessment, psychometrics, the phenomenology of the mental state]
2. Oyebode F. Sims' Symptoms in the Mind: Textbook of Descriptive Psychopathology. 6th ed. Edinburgh: Elsevier; 2018. [the descriptive-psychopathology bar for Band B]
3. Casey P, Kelly B. Fish's Clinical Psychopathology: Signs and Symptoms in Psychiatry. 4th ed. Cambridge: Cambridge University Press; 2019. [the sign-by-sign taxonomy of the phenomenology]
4. Jaspers K. General Psychopathology. Hoenig J, Hamilton MW, trans. Baltimore: Johns Hopkins University Press; 1997 (original 1913). [the method of descriptive psychopathology; form versus content]
5. Geddes JR, Andreasen NC, Goodwin GM, eds. New Oxford Textbook of Psychiatry. 3rd ed. Oxford: Oxford University Press; 2020. [assessment and psychopathology chapters]
6. Anastasi A, Urbina S. Psychological Testing. 7th ed. Upper Saddle River, NJ: Prentice Hall; 1997. [the psychometrics reference: reliability, validity, test construction]
7. Clinical Methods in Psychiatry. 1st ed. New Delhi: AIIMS Publication / CBS; 2024. [the mental state examination and the clinical use of the scales]

FOUNDATIONAL PRIMARY SOURCES

1. Rorschach H. Psychodiagnostik. Bern: Bircher; 1921. And Exner JE. The Rorschach: A Comprehensive System. New York: Wiley; 1974. [the inkblot test and its modern scoring]
2. Binet A, Simon T. Methodes nouvelles pour le diagnostic du niveau intellectuel des anormaux. Annee Psychologique. 1905;11:191-244. And Wechsler D. The Measurement of Adult Intelligence. Baltimore: Williams & Wilkins; 1939. [the intelligence scales and the deviation IQ]
3. Morgan CD, Murray HA. A method for investigating fantasies: the Thematic Apperception Test. Arch Neurol Psychiatry. 1935;34:289-306. [the TAT]
4. Hathaway SR, McKinley JC. The Minnesota Multiphasic Personality Inventory. Minneapolis: University of Minnesota Press; 1943. [the MMPI]
5. Cattell RB, Eber HW, Tatsuoka MM. Handbook for the Sixteen Personality Factor Questionnaire (16PF). Champaign, IL: IPAT; 1970. And Costa PT, McCrae RR. Revised NEO Personality Inventory (NEO-PI-R) Manual. Odessa, FL: PAR; 1992. [the trait inventories]
6. Doll EA. Vineland Social Maturity Scale: Condensed Manual of Directions. Circle Pines, MN: American Guidance Service; 1965. [adaptive behaviour and the social quotient]
7. Schneider K. Clinical Psychopathology. Hamilton MW, trans. New York: Grune & Stratton; 1959. [the first-rank symptoms]
8. Kahlbaum KL. Die Katatonie oder das Spannungsirresein. Berlin: Hirschwald; 1874. [the catatonic syndrome]
9. Kamat VV. Measuring Intelligence of Indian Children. Bombay: Oxford University Press; 1934. And Malin AJ. Malin's Intelligence Scale for Indian Children (MISIC). Lucknow: Indian Psychological Corporation; 1969. [the Indian intelligence-test adaptations]

JOURNAL REFERENCES (PUBMED-VERIFIED, 11)

1. Hamilton M. A rating scale for depression. J Neurol Neurosurg Psychiatry. 1960;23(1):56-62. PMID: 14399272.
2. Montgomery SA, Asberg M. A new depression scale designed to be sensitive to change. Br J Psychiatry. 1979;134:382-9. PMID: 444788.
3. Young RC, Biggs JT, Ziegler VE, Meyer DA. A rating scale for mania: reliability, validity and sensitivity. Br J Psychiatry. 1978;133:429-35. PMID: 728692.
4. Kay SR, Fiszbein A, Opler LA. The positive and negative syndrome scale (PANSS) for schizophrenia. Schizophr Bull. 1987;13(2):261-76. PMID: 3616518.
5. Goodman WK, Price LH, Rasmussen SA, et al. The Yale-Brown Obsessive Compulsive Scale. I. Development, use, and reliability. Arch Gen Psychiatry. 1989;46(11):1006-11. PMID: 2684084.
6. Nelson HE. A modified card sorting test sensitive to frontal lobe defects. Cortex. 1976;12(4):313-24. PMID: 1009768.
7. Andreasen NC. Thought, language, and communication disorders. I. Clinical assessment, definition of terms, and evaluation of their reliability. Arch Gen Psychiatry. 1979;36(12):1315-21. PMID: 496551.
8. Carpenter WT, Strauss JS, Muleh S. Are there pathognomonic symptoms in schizophrenia? An empiric investigation of Schneider's first-rank symptoms. Arch Gen Psychiatry. 1973;28(6):847-52. PMID: 4707991.
9. Sierra M, Berrios GE. Depersonalization: neurobiological perspectives. Biol Psychiatry. 1998;44(9):898-908. PMID: 9807645.
10. Bush G, Fink M, Petrides G, Dowling F, Francis A. Catatonia. I. Rating scale and standardized examination. Acta Psychiatr Scand. 1996;93(2):129-36. PMID: 8686483.
11. Folstein MF, Folstein SE, McHugh PR. "Mini-mental state". A practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res. 1975;12(3):189-98. PMID: 1202204.

PART VII

VII

Research Methods, Biostatistics & Epidemiology

Reading a study and reasoning about its numbers: designs, statistics and the epidemiology of disorder.

01 Research methodology

02 Biostatistics

03 Epidemiology

04 Discriminate

05 Apply and consolidate

PAPER I

Research Methods, Biostatistics & Epidemiology

Five sections · 21 topics · one complete notes document

This material gathers the whole research and statistics toolkit of the first paper into one place, because the examiner tests it as one skill: the ability to read a study and reason about numbers. The first band is **research methodology**, the architecture of clinical evidence: the hierarchy that ranks a case report below a cohort below a randomised trial below a systematic review, the three observational designs and their measures of association, the randomised controlled trial with its machinery of randomisation, allocation concealment, blinding and intention-to-treat analysis, the taxonomy of validity and bias and confounding, the sampling methods, the qualitative approaches, the systematic review and meta-analysis with its forest plot, and the reporting guidelines and research ethics that govern the whole enterprise. The second band is **biostatistics**, the grammar of the numbers: the scales of measurement that decide which test is legal, the description of data and the normal curve, the parametric and non-parametric significance tests, correlation and regression, the diagnostic test statistics built on the 2x2 table, statistical inference with its p-values and confidence intervals and errors, and survival and ROC analysis. The third band is **epidemiology**: incidence and prevalence, relative risk and the odds ratio, the National Mental Health Survey numbers that anchor the Indian picture, and the principles of screening. The examinable skill is judgement on both fronts: choosing the right design for a question, choosing the right test for the data, and interpreting the result correctly.

HOW TO USE THESE NOTES

Read the three bands as one argument. In **methodology**, every named design ends with a **Good to remember** box giving what it is, when to use it, and the one number or feature to know, so the reference facts sit apart from the reasoning. In **biostatistics**, the same box holds each test's one-line rule: what data it takes and what it answers. The **Discriminate** section is the payoff, two selector tables, one to choose a study design from the question and one to choose a statistical test from the data, and the **Apply** section works four short appraisal cases end to end. Figures declare one axis and code it in colour and shape: **petrol** marks structure and the neutral case, **coral** marks the trap or the point of discrimination, and **marigold** highlights the number to hold. Two boundaries are worth stating up front: the twin, family and adoption designs as genetic methods live in the Psychiatric Genetics notes, and the epidemiology of specific disorders lives in the clinical chapters of Paper II, while this material owns the general research toolkit and the national survey numbers; and the reliability and validity of a study here are a different idea from the psychometric reliability and validity of a test in the Psychometry, Assessment and Descriptive Psychopathology notes. This is doctor-facing revision, so the full technical vocabulary is used.

Research methodology

1 · Orientation: evidence-based medicine and the hierarchy of evidence

These notes cover one examinable skill wearing two hats: reading a study and reasoning about its numbers. That is all research methods and statistics really are for the psychiatrist. A paper arrives; you must place its **design**, judge whether its **data** were handled correctly, and decide whether its estimate of a **disease frequency** or a treatment effect can be trusted enough to change what you do on Monday. Methods and statistics are not two subjects but the front and back of the same coin, and the examiner tests them together: a viva that hands you an abstract wants to hear you name the design, state what it can and cannot answer, spot the trap, and read the confidence interval.

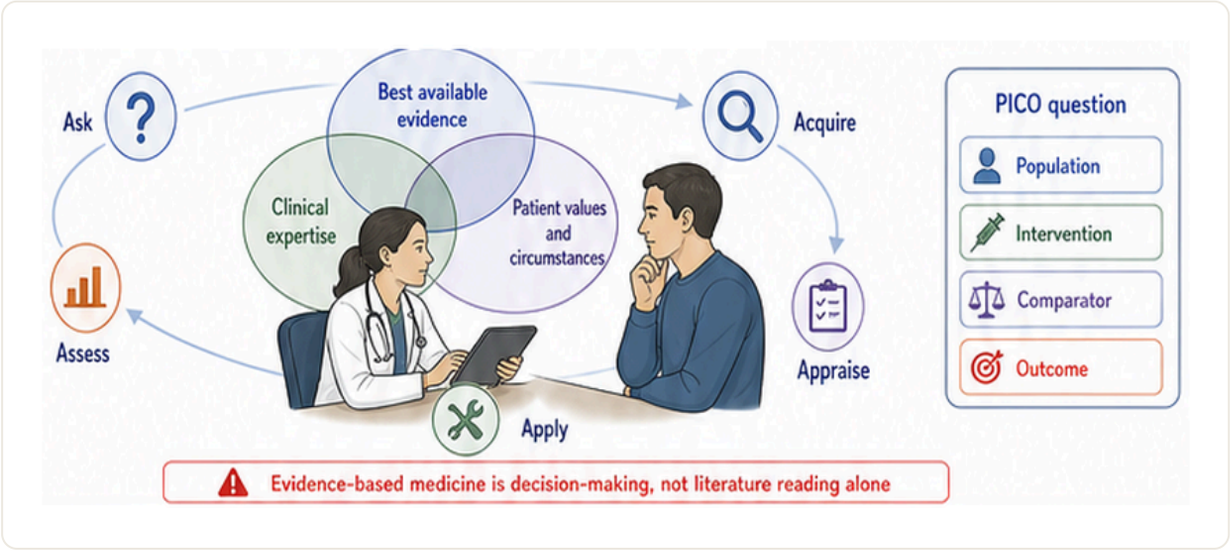


Fig 7.1 Evidence-based medicine: three sources, five actions.

Why this opens the notes. Before any single test or design can be defended, you need the map that ranks them, because the whole discipline is organised around one idea: some kinds of evidence are more trustworthy than others, and the trustworthiness follows from the design, not from the conclusion. That map is the **study-design hierarchy**, the evidence pyramid of evidence-based medicine. Learn the pyramid first and every later topic, from the randomised controlled trial to the odds ratio to the funnel plot, hangs on a rung you already recognise (Cipriani, Leucht & Geddes 2020).

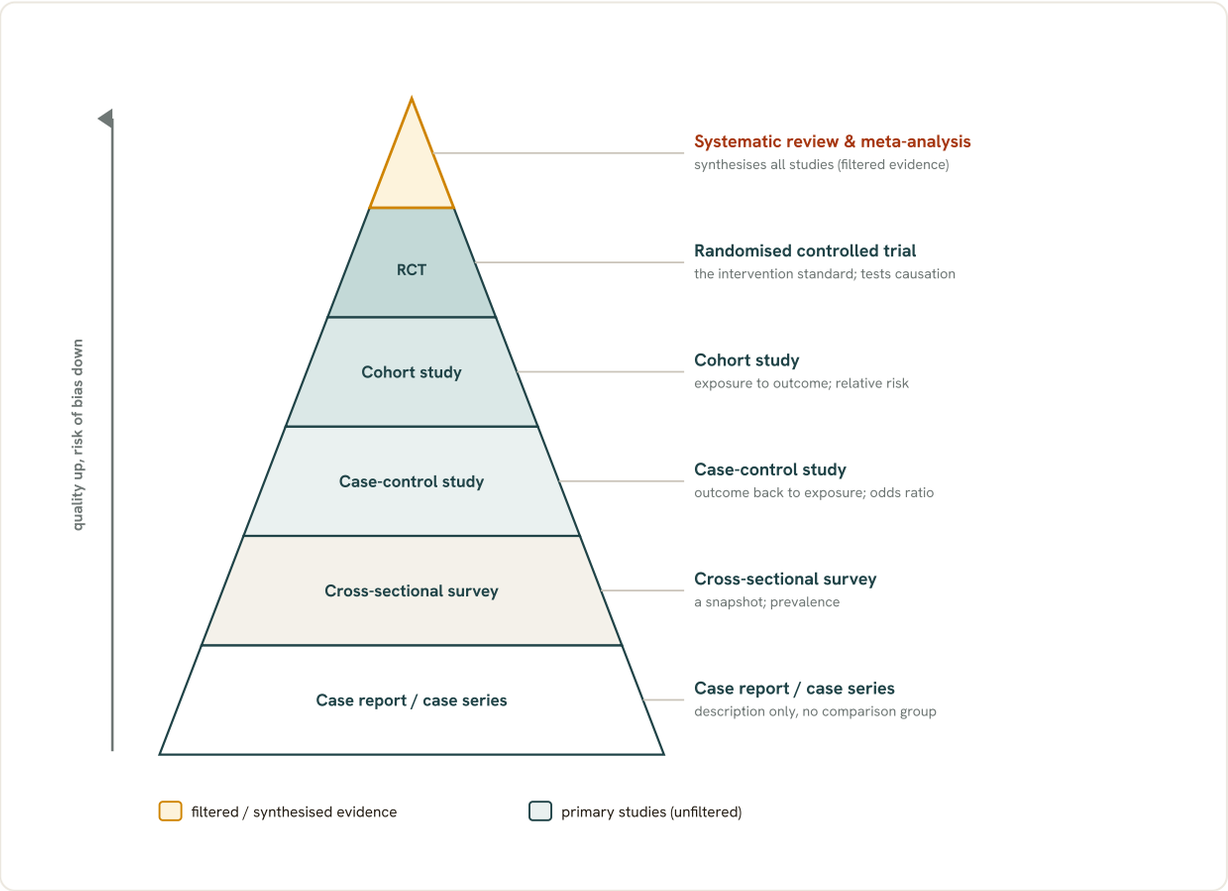


Fig 7.2 The hierarchy of evidence. Study designs rank by how well they control bias: the case report at the base offers only description, the observational designs add comparison groups, the randomised controlled trial adds the control of randomisation, and the systematic review with meta-analysis at the apex synthesises the whole body of filtered evidence. Reading a study begins with placing it on this pyramid, because the design sets the ceiling on the strength of the conclusion.

1.1 Evidence-based medicine: the definition and why it is more than "reading trials"

Definition. Evidence-based medicine is the conscientious integration of best research evidence with clinical expertise and patient values (Sackett et al. 1996). Three legs, not one: the *best current research evidence* from methodologically sound studies, the *clinician's own expertise* built from cumulative experience and skill, and the *patient's preferences, concerns and values*. Drop any leg and the stool falls: pure evidence with no clinical judgement is cookbook medicine, pure expertise with no evidence is eminence-based medicine, and neither is defensible. The term **ebm** in an exam answer should always cue all three components, because a common one-mark question is simply "define evidence-based medicine and name its components".

RULE **Three legs or none.** If your answer names research evidence but omits clinical expertise and patient values, it is not a definition of ebm.

1.2 The five steps of practising ebm (the 5 A's)

The cycle. Practising evidence-based medicine is a repeatable loop. The classic account gives four steps of doing plus a fifth of self-audit, usually taught as the **five steps** or 5 A's. These are the **5 steps** the examiner wants listed in order:

1. Ask

Convert the clinical problem into a focused, answerable question (the PICO question, 1.3).

2. Acquire
Search efficiently for the best available evidence, choosing the right resource (systematic reviews before single trials).

3. Appraise
Critically appraise the evidence for validity (closeness to the truth), size of effect, and applicability.

4. Apply
Integrate the appraised evidence with clinical expertise and the patient's values, and act.

5. Assess
Evaluate your own performance in steps 1 to 4 and the outcome in this patient, then refine.

Steps 1.2 through the rest of these notes are really an expansion of the middle three A's: how to frame the question, how to rank and read the designs you acquire, and how to appraise the numbers.

WORKED EXAMPLE

A patient with treatment-resistant depression asks about adding lithium. **Ask:** in adults with unipolar depression not responding to an antidepressant (P), does adjunctive lithium (I) versus continued antidepressant alone (C) improve response (O)? **Acquire:** a systematic review of augmentation trials sits above any single trial. **Appraise:** check randomisation, blinding, effect size and confidence interval. **Apply:** weigh the benefit against monitoring burden and the patient's willingness to have blood levels checked. **Assess:** review response and tolerability at follow-up. That is one full turn of the five steps.

1.3 The focused question: PICO

Framing. A vague question ("does lithium help?") cannot be searched or answered; a focused one can. The standard scaffold for a therapy or aetiology research question is PICO: **P**opulation (which patients), **I**ntervention (or exposure), **C**omparison (against what, often placebo or usual care), and **O**utcome (measured how). The word **pico** is the examiner's cue for "how do you build an answerable clinical question", and a well-built PICO also tells you which design and which search terms you need. For a harm or prognosis question the I becomes an exposure and the C its absence, but the four slots hold.

TRAP **The missing comparator.** A "study" with no C slot ("patients on drug X improved") is a case series, not a comparative study, and cannot establish that X did the improving.

1.4 The hypothesis: why we test the null

Setting up the test. Once the PICO question is framed, the statistics needs it as a testable hypothesis. Two hypotheses are stated as a pair. The null hypothesis (H0) is the hypothesis of *no difference* between the groups, or *no association* between exposure and outcome; the alternative hypothesis (H1) is that a difference or association does exist. The logic of frequentist testing is deliberately conservative: we never "prove" the alternative, we assume the null is true, compute how surprising the observed data would be under it, and reject the null only if that probability (the p-value) is small enough (Bland 2015).

Why the null, and not the alternative. The null is a single, precise statement ("the two means are equal") whose sampling behaviour we can calculate exactly; the alternative is a whole family of possibilities ("they differ, by some unknown amount") that we cannot. So we test the tractable one. Rejecting H0 lets us *infer* H1; failing to reject H0 is not proof of H0, only absence of evidence against it. This asymmetry, assume no difference and demand the data disprove it, is the spine of null hypothesis significance testing (Altman 1991).

HOLD THIS

We test the null hypothesis of "no difference" because it is the one precise, calculable statement; a small p-value lets us reject it and infer the alternative, but failing to reject it never proves the null.

1.5 The study-design hierarchy: the evidence pyramid

The map. Studies are ranked into a hierarchy of evidence, drawn as a pyramid, because design determines how well a study controls bias and confounding, and therefore how close its estimate sits to the truth. From the broad, weak base up to the narrow, strong apex the study-design hierarchy runs: the case report and case series, then the cross-sectional survey, then the case-control study, then the cohort study, then the randomised controlled trial, and at the top the systematic review and meta-analysis. The base is wide because weak evidence is abundant and easy to generate; the apex is narrow because strong evidence is scarce and expensive. Only the designs near the top are called "best evidence" for a question of treatment efficacy, where the randomised controlled trial reigns because randomisation is the only known way to balance both known and unknown confounders (Cipriani, Leucht & Geddes 2020).

Filtered versus unfiltered. A second cut runs across the pyramid. The single studies (case report through randomised controlled trial) are *unfiltered* or primary evidence: you must appraise each one yourself. The top band, systematic reviews and meta-analyses, is *filtered* or secondary evidence: someone has already gathered, appraised and (in a meta-analysis) statistically pooled the primary studies for you. This is why an efficient search in step 2 starts at the top and works down only if nothing filtered exists.

RULE **Match the design to the question.** The pyramid ranks designs for *treatment efficacy*; for a rare adverse effect a case-control study, not a randomised controlled trial, may be the best feasible design.

1.6 Levels of evidence: Oxford CEBM and GRADE

From pyramid to grade. The pyramid is a picture; formal schemes turn it into ranked levels of evidence. The Oxford Centre for Evidence-Based Medicine (Oxford CEBM) scheme assigns levels by design and question type, with systematic reviews of randomised trials at the top level and expert opinion at the bottom. GRADE takes a different route: rather than ranking by design alone, it starts each body of evidence high (for trials) or low (for observational studies) and then rates it up or down across four certainty grades, high, moderate, low and very low, according to risk of bias, inconsistency, indirectness, imprecision and publication bias. The examinable contrast is that Oxford CEBM ranks *studies* by design, whereas GRADE rates the *certainty of the*

whole body of evidence for a specific outcome, which is why the same trial can support a high or a low GRADE depending on the other studies and the outcome asked about.

INDIA

Indian clinical practice guidelines, including those of the Indian Psychiatric Society, adopt GRADE-style certainty ratings and the same evidence hierarchy, so the language of levels of evidence is not academic decoration: it is how a national guideline tells you the strength behind each recommendation you are expected to follow in a viva on management.

1.7 Systematic review and meta-analysis: the apex, and why it is not automatic

The top band. A systematic review answers a narrow, pre-specified question by an explicit, reproducible method: define the question, search comprehensively, apply stated inclusion criteria, appraise every included study, and synthesise. A *meta-analysis* is the optional statistical step inside a systematic review that pools the primary studies into one summary effect size with greater power than any single trial (Cipriani, Leucht & Geddes 2020). Not every systematic review contains a meta-analysis, and pooling is only justified when the studies are similar enough to combine.

The apex can still mislead. A meta-analysis inherits the flaws of its inputs. Its most serious threat is publication bias: if positive trials are published and negative ones buried, pooling the survivors overstates the effect. This is why the apex is a rung on a ladder, not a guarantee, and appraisal never stops just because a paper says "systematic review" in the title.

TRAP **Apex worship.** "It is a meta-analysis, so it is true" ignores publication bias and heterogeneity; a meta-analysis of biased trials produces a precise, confident, wrong answer.

1.8 The master mnemonic: design, then data, then disease-frequency

The spine of everything that follows. These notes move through three bands in order, and one chained mnemonic holds the whole arc: read the **design** first, then check the **data** handling, then interpret the **disease-frequency** (or effect) it estimates. Every topic is a D in that chain. The pyramid you have just learned is the design band; the statistics topics are the data band; the epidemiology topics are the disease-frequency band.

MNEMONIC

THREE D's: Design, Data, Disease-frequency

The three bands of these notes, in reading order. **Design**: place the study on the evidence pyramid and know what its rung can answer. **Data**: was the null tested correctly, the right statistical test chosen, the p-value and confidence interval read honestly. **Disease-frequency**: what does it say about incidence, prevalence, risk or effect, and can that be generalised. Name the design, trust the data, then quote the frequency: any exam abstract can be attacked in that order.

1.9 What each design answers and where it sits

The catalogue in one view. Before the detailed topics, fix each rung with the question it answers and the one measure it yields. This table is the reference you will return to whenever an abstract lands.

DESIGN (BASE TO APEX)	WHAT IT ANSWERS	SIGNATURE MEASURE	DIRECTION / TIME
Case report / series	Does this phenomenon exist at all; hypothesis generation	**Description only, no rate**	None (no comparator)
Cross-sectional	How common is it now; is there an association at one time point	**Prevalence**	Snapshot (one moment)
Case-control	What exposures differ between those with and without the disease (good for rare disease)	**Odds ratio**	Backward (outcome to exposure)
Cohort	Do the exposed develop the outcome more than the unexposed (good for rare exposure)	**Relative risk, incidence**	Forward (exposure to outcome)
Randomised controlled trial	Does the intervention cause the outcome (efficacy)	**Relative risk, NNT**	Forward, allocation randomised
Systematic review / meta-analysis	What does all the evidence together say	**Pooled effect size**	Synthesis of the above

The evidence pyramid as a table: each rung with the one question it answers and the one measure it yields. The odds ratio, relative risk and NNT are unpacked in their own topics; here, hold which design gives which.

GOOD TO REMEMBER

- **Case report / series:** a description of one or a few patients with no comparison group; use to flag a novel presentation or generate a hypothesis; gives no rate at all.
- **Cross-sectional study:** measures exposure and outcome at a single time point in a defined population; use to estimate how common something is; the one number is prevalence = cases / total population at that time.
- **Case-control study:** starts from the outcome and looks back at past exposures, comparing cases with controls; best for rare diseases; the one measure is the odds ratio = odds of exposure in cases / odds of exposure in controls.
- **Cohort study:** follows exposed versus unexposed forward in time to see who develops the outcome; best for rare exposures and gives incidence; relative risk = incidence in exposed / incidence in unexposed.
- **Randomised controlled trial:** allocates the intervention by a concealed random method to balance confounders; the top design for efficacy; yields relative risk and number needed to treat, NNT = 1 / absolute risk reduction.
- **Systematic review and meta-analysis:** an explicit, reproducible synthesis of primary studies, optionally pooling them statistically; the apex and filtered evidence; the meta-analysis gives one pooled effect size with tighter confidence intervals than any single study.
- **Oxford CEBM levels:** a scheme that ranks individual studies into levels by design and question type; use to grade a single paper; systematic reviews of trials sit at the top level.
- **GRADE:** rates the certainty of a whole body of evidence per outcome into four grades; use in guideline-making; starts high for trials, low for observational studies, then moves up or down.

PEARL

Two axes cross the pyramid and both are examinable. The *vertical* axis is trustworthiness by design (case report at the base, meta-analysis at the apex). The *horizontal* axis is filtered versus unfiltered: everything from case report to randomised controlled trial is unfiltered primary evidence you must appraise yourself, while systematic reviews and meta-analyses are filtered secondary evidence pre-appraised for you. Say both axes and you have shown you understand the pyramid, not just memorised its order.

For genetic study designs such as twin, family and adoption studies, which sit alongside the cohort and case-control designs but answer questions of heritability, see the Psychiatric Genetics notes. For the epidemiology of specific disorders and the national prevalence figures, see the clinical chapters of Paper II. The reliability and validity discussed here belong to *study* quality; the distinct psychometric reliability and validity of a *test* are covered in the Psychometry, Assessment and Descriptive Psychopathology notes.

2 · Observational study designs

This is the map every research question is read against. In an **observational** study the investigator watches nature take its course and measures exposures and outcomes, but does not allocate the exposure. That single fact separates the whole family from the interventional trial (where the investigator assigns the exposure) and is the first fork in any study-design question. The examiner wants you to name a design from a one-line vignette, state its **direction of enquiry** (does it start from exposure or from outcome?), and give the correct measure of

association it yields. Get those three right and almost every stem falls open (Bland 2015; Altman 1991).

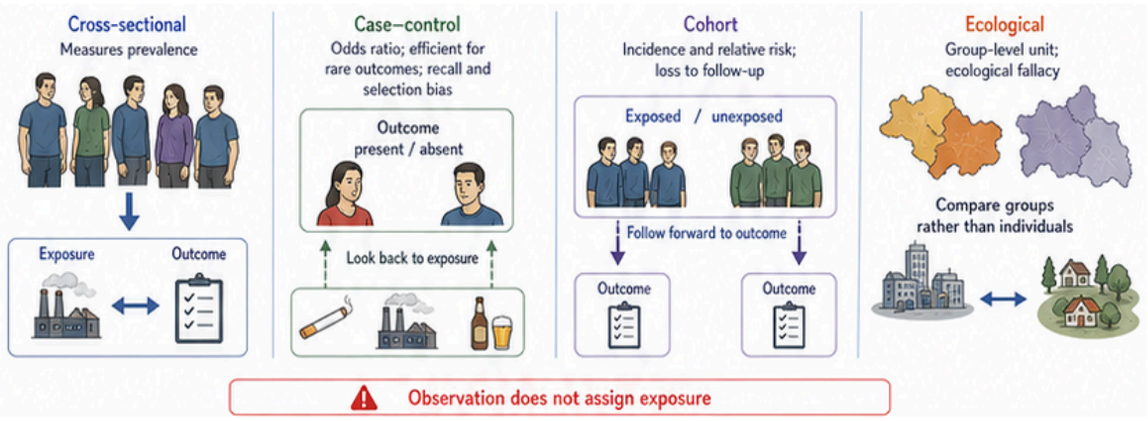


Fig 7.3 Observational designs: direction determines the measure.

The three analytic **observational** designs (cross-sectional, case-control, cohort) differ chiefly in their **time axis**: the cross-sectional design takes a single snapshot, the case-control design looks backward from outcome to exposure, and the cohort design follows forward from exposure to outcome. The ecological study sits apart because its unit of analysis is the group, not the individual. Hold the time axis and the rest is bookkeeping.

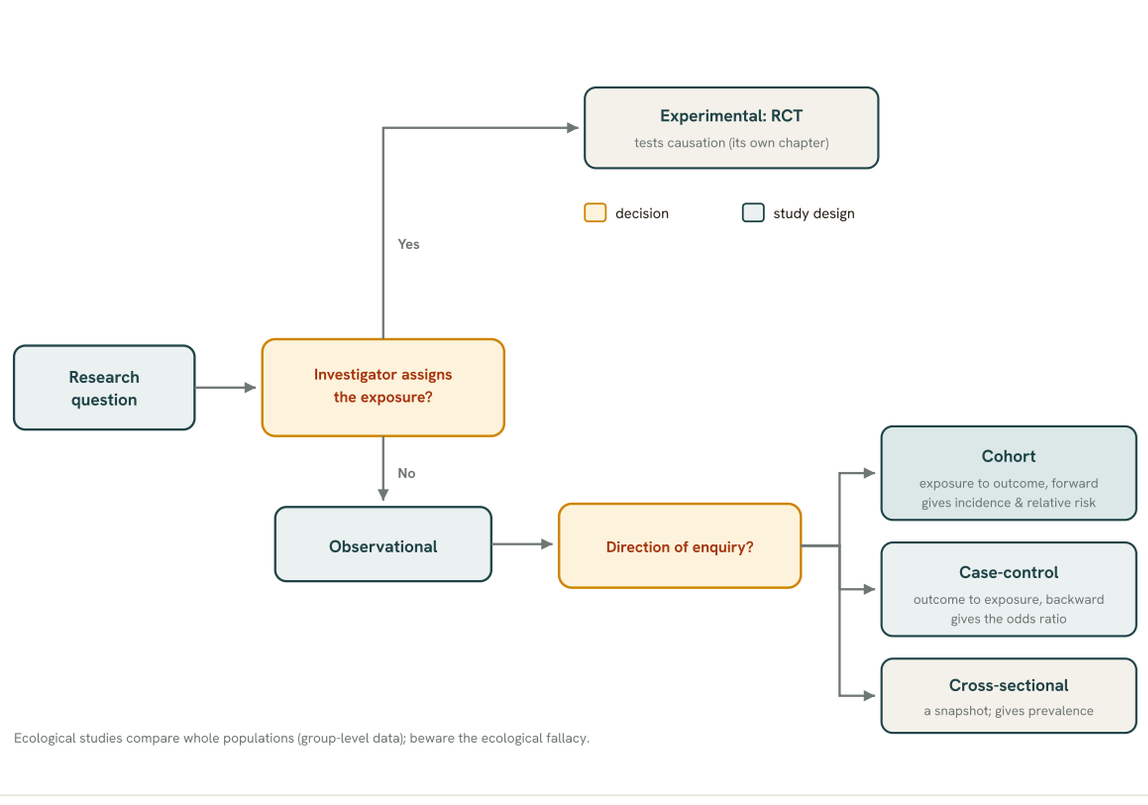


Fig 7.4 Choosing a study design. The first question is whether the investigator assigns the exposure: if so the design is experimental (the randomised controlled trial, developed in its own chapter). If not, the study is observational, and the direction of enquiry then decides the design: following exposure forward to outcome is a cohort (yielding incidence and the relative risk), tracing outcome back to exposure is a case-control (yielding the odds ratio), and measuring both at once is cross-sectional (yielding prevalence).

2.1 Observation versus intervention: the first fork

Who assigns the exposure? The primary division of all analytic study designs is **intervention vs observation**. In an interventional (experimental) study, most rigorously the randomised controlled trial, the investigator allocates the exposure or treatment; randomisation balances known and unknown confounders and gives the design its causal strength (Bland 2015). In an **observational** study the investigator only measures what is already happening, so confounding must be handled at analysis (matching, stratification, regression) rather than by design. Everything in this topic lives on the observational side of that fork; the trial designs are treated with the experimental methods. Descriptive observational work (case reports, case series, correlational surveys) describes without a comparison group; the analytic designs below all carry a comparison group and can test associations.

RULE **Name the fork first.** If the investigator assigned the exposure it is a trial; if they only observed it, it is one of the observational designs, and you then read the direction of enquiry.

2.2 The cross-sectional study: the snapshot

Exposure and outcome measured together, at one point in time. A **cross-sectional** study measures exposure and outcome simultaneously in a defined population at a single point, a snapshot (Bland 2015; Altman 1991). Because everyone is examined once, it is the natural design for a prevalence survey: it counts how many people currently have the condition. It is quick, comparatively cheap, needs no follow-up, and can study several exposures and several outcomes at once, which is why community psychiatric morbidity surveys use it. Its defining weakness is that it *cannot establish temporality*: measuring cause and effect at the same instant, you usually cannot tell which came first, so it is weak for causal inference and prone to reverse causation. It also over-represents long-lasting (prevalent) cases and under-represents brief or rapidly fatal ones.

Direction of enquiry

None: exposure and outcome are read simultaneously.

Measure yielded

Prevalence; and the prevalence ratio as a crude measure of association.

Best for

Prevalence surveys, health-service planning, generating hypotheses.

INDIA

The National Mental Health Survey of India (2015 to 2016) is the flagship cross-sectional example: a snapshot across 12 states giving a current weighted prevalence of any mental disorder of about **10.6** percent (lifetime near **13.7** percent) and a treatment gap for common mental disorders exceeding **80** percent. A snapshot design is exactly right for asking "how many people have the disorder now and how many are in treatment", but it cannot tell you what caused the disorder.

2.3 Prevalence and incidence: what each design can count

Existing cases versus new cases. The measure a design can produce depends on its time axis, so the two frequency measures anchor the whole topic. **Prevalence** is the proportion of a population that *has* the condition at a given time (existing cases); it is what a cross-sectional snapshot yields. **Incidence** is the rate of *new* cases arising over a period in a population initially free of the disease; it requires follow-up over time and so is the property of the cohort design. The link is that prevalence rises with both incidence and duration (roughly, prevalence is proportional to incidence multiplied by average duration in a steady state), which is why chronic disorders look common on a snapshot even when few new cases arise.

TRAP **Prevalence is not incidence.** A cross-sectional survey gives prevalence (existing cases); only a cohort followed over time gives incidence (new cases). Do not report a snapshot as an incidence.

2.4 The case-control study: from outcome, looking back

Start with the outcome, look back for the exposure. A **case-control** study assembles a group who already have the outcome (cases) and a comparable group who do not (controls), then looks back to compare how often each group was exposed. It is therefore **retrospective** in direction: the enquiry runs from outcome to exposure (Bland 2015; Altman 1991). Because you start from the diseased, you can study a rare outcome efficiently with a modest sample, examine several exposures at once, and get an answer quickly and cheaply. Its measure of association is the odds ratio (the odds of exposure among cases divided by the odds among controls). The design's weaknesses are its Achilles heel in exams: it is vulnerable to recall bias (cases search their memories harder for exposures than controls do) and to selection bias (through the way controls are chosen), and it cannot directly measure incidence or, ordinarily, relative risk.

Direction of enquiry

Outcome to exposure (backward): starts from cases and controls.

Measure yielded

The odds ratio (OR); OR approximates the relative risk only when the outcome is rare.

Best for

Rare outcomes, diseases with long latency, quick and cheap enquiry, multiple exposures.

Main weakness

Recall bias and selection bias; cannot give incidence.

TRAP **Case-control gives an odds ratio, not a relative risk.** Because there is no denominator population followed over time, a case-control study cannot yield incidence or a true relative risk; the OR only approximates the RR when the disease is rare.

2.5 The nested case-control study

A case-control study built inside a cohort. The **nested case-control** design draws its cases and controls from within an already-assembled cohort: as outcomes accrue in the cohort, each case is matched to one or more members of the same cohort who were at risk at that time but had not (yet) developed the outcome. Because exposure was recorded at cohort entry, before any outcome,

the nested design largely defeats recall bias, and because controls come from the same source population as the cases it reduces selection bias. It is efficient when a costly exposure measurement (an assay on stored blood, an expensive biomarker) need only be run on the sampled cases and controls rather than the whole cohort. It keeps the temporal clarity of the cohort while retaining the economy of the case-control approach.

2.6 The cohort study: from exposure, following forward

Start with the exposure, follow forward to the outcome. A **cohort** study starts by defining a group free of the outcome, classifies them by exposure status, and follows them forward in time to see who develops the outcome (Bland 2015; Altman 1991). Its direction of enquiry runs from exposure to outcome, and when the following is done in real time it is **prospective**. Because it observes new cases arising in a defined population over time, the cohort design is the one that measures **incidence** directly and yields the relative risk (incidence in the exposed divided by incidence in the unexposed). It is the design of choice for a rare exposure (you deliberately oversample the exposed at entry), it can examine multiple outcomes of a single exposure, and its temporal sequence (exposure demonstrably precedes outcome) makes it the strongest observational design for causal inference. Its costs are the mirror image: large samples, long follow-up, expense, and loss to follow-up, and it is inefficient for a rare outcome.

Direction of enquiry

Exposure to outcome (forward): starts from exposed and unexposed.

Measure yielded

Incidence and the relative risk (RR); the incidence rate ratio.

Best for

Rare exposures, multiple outcomes, establishing temporality.

Main weakness

Slow, costly, loss to follow-up; inefficient for rare outcomes.

2.7 Prospective versus retrospective cohorts

Same design, different position of the observer in time. A **cohort** can be run two ways. In a **prospective** cohort the investigator defines the cohort now, measures exposure now, and waits for outcomes to accrue: exposure data are collected before any outcome, which maximises data quality but demands time and money. In a retrospective (historical) cohort the whole exposure-to-outcome sequence has already happened by the time the study begins; the investigator reconstructs it from existing records (occupational registers, case notes, linked health databases), classifying past exposure and then counting outcomes that have already occurred. Both are true cohort studies (they follow exposure forward to outcome and yield incidence and relative risk); the retrospective form is faster and cheaper but is limited by the completeness and quality of the pre-existing records. Do not confuse the retrospective *cohort* (still exposure to outcome) with the retrospective *direction* of a case-control study (outcome to exposure).

WORKED EXAMPLE

Read a 2x2 table by design. In a **cohort** of 200 exposed and 200 unexposed, followed forward, 40 exposed and 10 unexposed develop the disorder. Incidence in exposed = 40/200 = **0.20**; incidence in unexposed = 10/200 = **0.05**; relative risk = 0.20 / 0.05 = **4.0** (the exposed are four times as likely to develop the outcome). Now take the same table as a **case-control** study (50 cases, 350 controls): odds of exposure in cases = 40/10 = 4; odds of exposure in controls = 160/190; odds ratio = (40 x 190) / (10 x 160) = 7600 / 1600 = **4.75**. Same underlying data, read in two directions: the cohort reads *across* the exposure rows (incidence in the exposed is a / (a + b)) to give an RR, the case-control reads *down* the disease columns (fixing the outcome, then comparing exposure odds) to give an OR, and the OR overshoots the RR because the outcome here is not rare.

2.8 The ecological study and the ecological fallacy

The unit of analysis is the group, not the person. An **ecological** study correlates an exposure and an outcome measured at the level of populations (countries, states, districts, time periods) rather than individuals: for example, plotting national fish consumption against national depression rates, or state lithium-in-water levels against state suicide rates. It is cheap and fast because it reuses routinely collected aggregate data, and it is a useful hypothesis-generating and hypothesis-testing tool at the population level. Its signature hazard is the ecological fallacy: an association that holds between group averages need not hold for the individuals within those groups, so a group-level correlation cannot be assumed to describe individual-level risk. It is the price of losing the individual as the unit of analysis, and it is the single most examined criticism of the ecological design.

TRAP **The ecological fallacy.** Inferring an individual-level relationship from a group-level (aggregate) correlation is invalid; a correlation between country averages tells you nothing certain about any individual.

2.9 The design decision logic: intervention, direction, time axis

Three questions choose the design. Faced with a vignette, decide in order. First, *intervention vs observation*: did the investigator assign the exposure (trial) or merely observe it (one of these designs)? Second, the *direction of enquiry*: does the study start from the outcome and look back (case-control) or start from the exposure and look forward (cohort), or read both at once (cross-sectional)? Third, the *time axis*: a single snapshot (cross-sectional, gives prevalence), backward from outcome (case-control, gives an odds ratio), or forward from exposure (cohort, gives incidence and relative risk). Layered onto that is the frequency logic that decides between the two comparative designs: match the rarity of the exposure or the outcome to the design that captures it efficiently.

DESIGN	DIRECTION OF ENQUIRY	MEASURE OF ASSOCIATION	BEST FOR	MAIN WEAKNESS
Cross-sectional	Simultaneous (snapshot)	**Prevalence (prevalence ratio)**	Prevalence surveys; service planning; several exposures and outcomes at once	No temporality; reverse causation; favours long-duration cases
Case-control	Outcome to exposure (backward, retrospective)	**Odds ratio (OR)**	Rare outcomes; long-latency disease; quick and cheap; multiple exposures	Recall bias; selection bias; no incidence, no true RR
Nested case-control	Outcome to exposure, within a cohort	**Odds ratio (OR)**	Costly exposure assays; when exposure was recorded before outcome	Still smaller than the full cohort; needs the parent cohort
Cohort	Exposure to outcome (forward, prospective)	**Relative risk (RR); incidence**	Rare exposures; multiple outcomes; establishing temporality	Slow, costly, loss to follow-up; poor for rare outcomes
Ecological	Group-level correlation	**Correlation of population rates**	Cheap hypothesis generation from aggregate data	Ecological fallacy: group data cannot be read to the individual

The observational family read across design, direction, measure, best use and main weakness; the hit column is the measure of association each design yields.

2.10 The two selectors: choosing a design, choosing a test

Match the question to the design; match the data to the test. Two interleaved selectors carry a research vignette from question to result. The first turns a clinical question into a design; the second turns the resulting data into the correct statistical test. The design chooser turns on the rarity of what is being studied and on whether temporality matters; the test chooser turns on the type of data, the number of groups, and whether the groups are paired or unpaired.

QUESTION / SITUATION	DESIGN TO PICK
How common is the condition now?	**Cross-sectional (prevalence)**
Rare outcome, or a long-latency disease?	**Case-control**
Rare exposure, or many outcomes of one exposure?	**Cohort**
Need to prove exposure preceded outcome (temporality)?	**Prospective cohort**
Costly exposure assay, cases arising inside a cohort?	**Nested case-control**
Only aggregate, population-level data available?	**Ecological (mind the fallacy)**

Study-design chooser: read the clinical question, pick the design (question to design).

DATA TYPE	GROUPS / PAIRING	CORRECT TEST
Continuous, normal	2 unpaired groups	**Unpaired (independent) t-test**
Continuous, normal	2 paired groups	**Paired t-test**
Continuous, normal	3+ groups	**One-way ANOVA**
Continuous, non-normal	2 unpaired groups	**Mann-Whitney U**
Continuous, non-normal	2 paired groups	**Wilcoxon signed-rank**
Continuous, non-normal	3+ groups	**Kruskal-Wallis**
Categorical (counts)	2+ unpaired groups	**Chi-squared test**
Categorical, paired	2 paired groups	**McNemar test**

Statistical-test chooser: data type by number of groups by pairing, giving the correct parametric or non-parametric test.

HOLD THIS

Case-control starts from the outcome and gives an odds ratio; cohort starts from the exposure and gives incidence and a relative risk; cross-sectional is a snapshot and gives prevalence.

COMMON TRAP

Two mirror-image errors examiners set. First, reporting a **relative risk** from a case-control study, which cannot yield one (it gives an odds ratio; the OR approximates RR only when the outcome is rare). Second, choosing a case-control design for a rare *exposure* (it is efficient for a rare *outcome*): a rare exposure calls for a cohort that oversamples the exposed. Keep the pairing straight: rare outcome to case-control, rare exposure to cohort.

PEARL

The odds ratio and the relative risk converge when the outcome is rare (the "rare disease assumption"): with a low outcome frequency the OR from a case-control study is a good approximation of the RR you would have got from a cohort. As the outcome becomes common the OR increasingly exaggerates the RR, which is exactly why a case-control OR should never be quoted as a relative risk when the outcome is frequent.

MNEMONIC

"Cohort = Cause first; Case-control = Consequence first."

The cohort begins with the putative cause (the exposure) and follows forward; the case-control begins with the consequence (the outcome) and looks back. Both start with C, so anchor on the second word: cause-first is forward-looking cohort (relative risk), consequence-first is backward-looking case-control (odds ratio).

GOOD TO REMEMBER

- **Cross-sectional:** measures exposure and outcome at once, a snapshot; use it for a prevalence survey; gives prevalence and the prevalence ratio, and cannot establish temporality.
- **Case-control:** starts from the outcome and looks back for exposure (retrospective); use it for a rare outcome or long-latency disease; gives the odds ratio, OR = (odds of exposure in cases) / (odds of exposure in controls).
- **Nested case-control:** a case-control study drawn from within a cohort; use it when a costly exposure assay makes measuring the whole cohort wasteful; gives an odds ratio while defeating recall bias.
- **Cohort:** starts from exposure and follows forward to outcome (prospective); use it for a rare exposure or multiple outcomes; gives incidence and the relative risk, RR = (incidence in exposed) / (incidence in unexposed).
- **Ecological:** correlates exposure and outcome at the group level; use it for cheap hypothesis generation from aggregate data; the number to fear is the ecological fallacy, that group correlations do not transfer to individuals.

3 · The randomised controlled trial and drug-trial phases

The randomised controlled trial (RCT) is the only truly **experimental design** in clinical research and the acknowledged gold standard for testing whether a treatment actually works (Companion to Psychiatric Studies 2010). Everything else in this chapter observes; the RCT intervenes, allocates by chance, and so earns the right to speak about cause. The examiner rarely asks "what is an RCT"; the marks sit in the machinery that protects it: randomisation, allocation concealment, blinding, and an analysis that keeps the randomised groups intact.

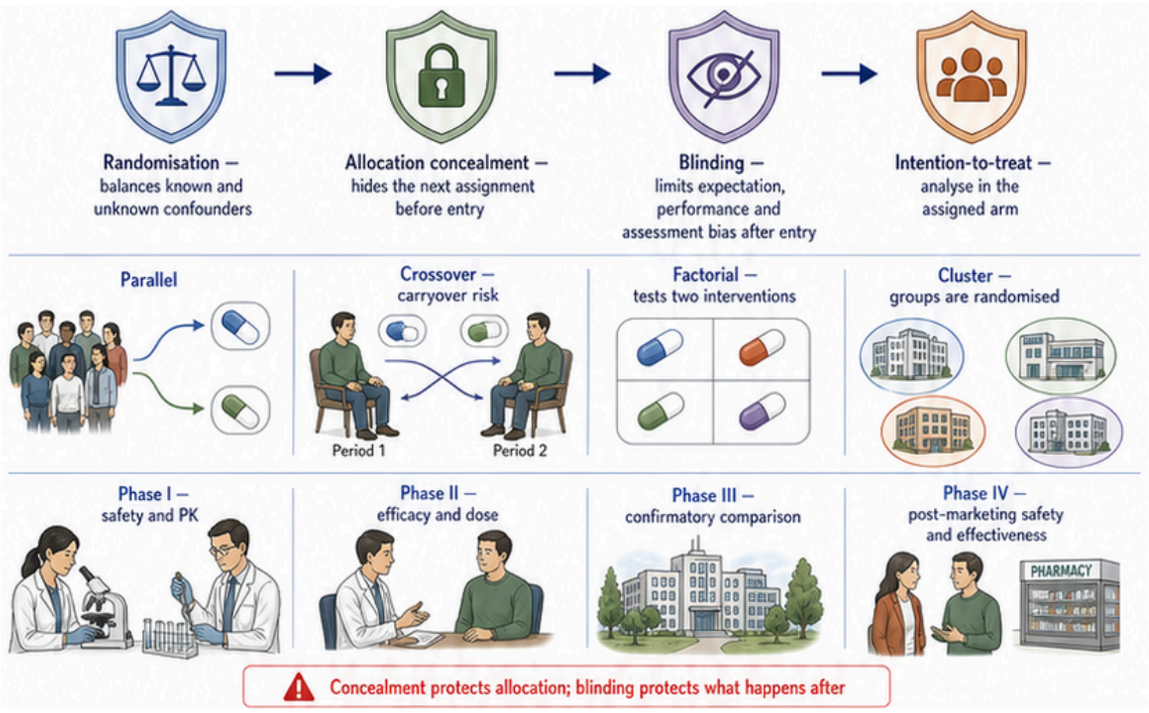


Fig 7.5 Randomised trials: safeguards do different jobs.

Read this topic as a chain in which each link defends the last. Randomisation creates comparable groups; allocation concealment stops the recruiter from breaking the randomisation at entry;

blinding stops patients, clinicians and assessors from biasing what happens afterwards; intention-to-treat keeps the analysis faithful to the randomisation. Break any link and the design degrades toward a mere observational comparison.

3.1 The randomised controlled trial as the experimental design

Only a controlled trial with random allocation is truly experimental. In a **randomised controlled** trial the investigator assigns each consenting participant to an intervention or a comparator by a chance mechanism, follows both arms forward, and compares a pre-specified outcome. Because the investigator controls the exposure and allocates it at random, the RCT (equivalently the **randomized controlled** trial in American spelling) is the design least vulnerable to selection bias and confounding, which is why it anchors the top of the evidence hierarchy for questions of therapy. Non-randomised comparisons tend to over-estimate benefit because the newer treatment is quietly given to less severe or better-prognosis patients, the problem of confounding by indication. Even within randomised trials, inadequate or unclear allocation concealment inflates the estimated treatment effect, by around **30% to 41%** on average (Schulz et al 1995).

3.2 Randomisation: why it works, and its varieties

Randomisation balances known and, crucially, unknown confounders. Randomisation (American: randomization) has two purposes. First and foremost it distributes both the measured prognostic factors (age, sex, severity) and, more importantly, the *unmeasured and unknown* ones evenly between arms, so that any difference in outcome can be attributed to the treatment. Second, it removes the recruiter's ability to steer sicker or healthier patients into a preferred arm. No other design can balance confounders you have not even thought to measure, and that is the single reason the RCT out-ranks the cohort study for causal claims about therapy (Bland 2015).

Simple randomisation

Each patient allocated independently by chance (the equivalent of a fair coin or a random-number list); can leave unequal group sizes in small trials, but self-corrects as the trial grows large.

Block randomisation

Patients randomised in small blocks (of four, six, and so on) so that the numbers in each arm stay equal throughout recruitment; the cost is that the last allocation in a block can become guessable.

Stratified randomisation

A separate randomisation schedule is run within each level of a key prognostic variable (for example centre, or baseline severity) so that variable is balanced by design rather than left to chance; usually combined with blocks.

RULE **Random allocation is the only tool that balances unknown confounders.** That is why the RCT, not the cohort study, is the design for a causal therapy question.

3.3 Allocation concealment: guarding the moment of assignment

Concealment hides the *upcoming* assignment from whoever recruits. Allocation concealment means that the person enrolling a patient cannot know, at the moment of consent, which arm that patient is about to enter. If the schedule is predictable (allocation by date of birth, alternate

days, an unsealed list), a recruiter can consciously or unconsciously include or exclude patients to load one arm, and the randomisation is subverted before it happens. The robust solution is a remote or independent system: the recruiter phones or logs in, enters the patient's baseline data, and only then is the allocation released. Trials in which allocation was easy to subvert were far more likely to report a significant effect (trials with subvertible allocation were consistently more likely to report a significant effect; Chalmers et al 1983).

TRAP **Allocation concealment and blinding are not the same thing.** Concealment protects the assignment *before* and *at* entry; blinding protects perception *after* entry. A trial can be double-blind yet have had broken concealment, and vice versa.

3.4 Blinding: what single, double and triple each conceal

Blinding stops knowledge of the assigned arm from biasing what comes next. Where allocation concealment guards entry, blinding (masking) guards everything after it by keeping the arm hidden from one or more parties. A **placebo** is the instrument that makes patient-level blinding possible, an inert comparator indistinguishable from the active drug. The purpose of single, double and **double-blind** or triple blinding is to reduce observer, performance and ascertainment bias.

LEVEL	WHO IS KEPT UNAWARE OF THE ARM	BIAS IT CHIEFLY PREVENTS
Open (unblinded)	Nobody; all parties know the arm	None; maximally biased
Single-blind	**The patient only**	Patient expectation / placebo response
Double-blind	**Patient and treating clinician**	Patient expectation plus clinician performance bias
Triple-blind	**Patient, clinician and the outcome assessor / analyst**	Adds ascertainment (assessment) bias

Each blinding level conceals the arm from one more party; the extra party in triple-blind is the outcome assessor or data analyst (Companion to Psychiatric Studies 2010).

Blinding is often imperfect. Distinctive side-effects (anticholinergic dryness, extrapyramidal signs) can unmask the active drug; a useful check is to ask patients and raters to guess their arm at the end, where successful blinding gives no-better-than-chance guessing (Even et al 2000).

3.5 Intention-to-treat versus per-protocol analysis

Analyse everyone in the arm to which they were randomised. Intention-to-treat (ITT) analysis includes every randomised participant in their originally allocated arm, regardless of whether they completed, complied with, or even received the treatment. Per-protocol (completer) analysis restricts to those who finished per protocol. ITT is preferred because the moment you exclude dropouts or non-compliers you re-introduce exactly the selection you randomised away: dropout is rarely random (people leave because a drug failed or was not tolerated), so a completer analysis flatters the treatment. ITT preserves the randomisation and therefore keeps the groups

comparable and the comparison unbiased (Altman 1991). It gives the more conservative, more clinically realistic estimate of effectiveness; per-protocol answers the narrower "did it work in those who took it" question and is more prone to bias.

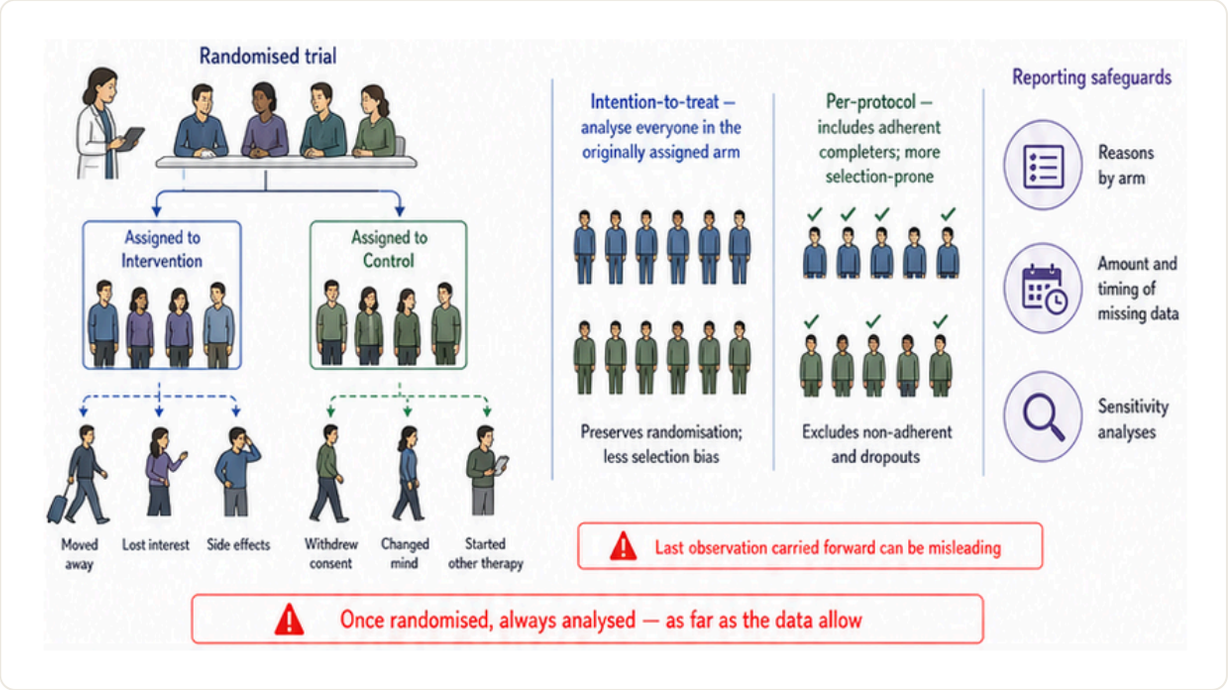


Fig 7.6 Attrition, adherence and missing outcomes.

RULE **ITT analyses everyone as randomised.** "Once randomised, always analysed" preserves the balance that randomisation bought and gives the honest, conservative effect estimate.

For missing outcomes, last-observation-carried-forward (LOCF) has been used to complete ITT datasets but is statistically dubious, since trial dropout in psychiatry is often substantial and treatment-related, so the missing values are rarely missing at random (Companion to Psychiatric Studies 2010).

3.6 Trial architectures: parallel-group, crossover, factorial, cluster randomised

The architecture is chosen to fit the disease and the question. The default is the parallel-group trial, in which independent arms run side by side. Three important variants exist. A crossover trial gives every participant each treatment in turn in random order, so each patient serves as their own control. A factorial design tests two or more interventions simultaneously in one trial (a two-by-two factorial gives A, B, both, or neither), efficiently answering more than one question and probing interaction. A cluster randomised (American: cluster randomized) trial randomises whole groups, such as clinics or general-practice teams, rather than individuals.

ARCHITECTURE	STRUCTURE	WHEN TO USE IT	THE CATCH
Parallel-group	Separate arms compared concurrently	**The default for most therapeutic questions**	Needs the full sample size in each arm

ARCHITECTURE	STRUCTURE	WHEN TO USE IT	THE CATCH
Crossover	**Each patient receives both treatments in random sequence, own control**	Rare or stable chronic disease where recruiting large parallel groups is impractical	Carryover and order effects; needs a washout; not for curable or unstable conditions
Factorial	**Two-plus interventions crossed in one trial (2x2: A, B, both, neither)**	Two questions, or a suspected interaction, tested economically at once	Assumes no strong interaction between the factors; complex to interpret if there is one
Cluster randomised	**Groups (clinics, practices, wards) randomised, not individuals**	Service-level or educational interventions that cannot be given to one person without contaminating neighbours	The cluster is the unit of analysis; needs many clusters for power

Crossover buys power in rare stable diseases at the price of carryover; factorial buys a second answer if the factors do not interact; cluster is forced whenever the intervention cannot be confined to an individual (Companion to Psychiatric Studies 2010).

3.7 Superiority, non-inferiority and equivalence framing

The objective determines the hypothesis and the sample size. A **superiority** trial asks whether the new treatment beats the comparator (or placebo). A **non-inferiority** trial asks whether the new treatment is not worse than an established one by more than a pre-specified acceptable margin, used when a new agent offers a side benefit (cheaper, safer, easier) and a placebo arm would be unethical. An **equivalence** trial asks whether two treatments differ by no more than a margin in either direction, typical for bioequivalence. The direction of the question changes the null hypothesis, so a non-inferiority or equivalence claim cannot simply be read off a failed superiority test.

> **COMMON TRAP**
>
> "No significant difference" in a superiority trial does not prove the treatments are equivalent; absence of evidence is not evidence of absence. Equivalence and non-inferiority must be designed and powered as such from the outset, with a pre-stated margin. A small, underpowered superiority trial that finds nothing is uninformative, not reassuring.

3.8 The drug trial phases

A new drug is licensed by climbing four phases. Before an RCT can even ask about efficacy, a compound must pass through the staged programme of a drug trial. Each phase has a distinct question and a characteristically larger sample. **Phase i** is first-in-human: small groups of usually healthy volunteers receive the drug to establish safety, tolerability and pharmacokinetics (in oncology and some psychiatry contexts, patients rather than healthy volunteers are used). **Phase ii** moves to patients with the target disorder to look for preliminary efficacy and to find the dose. **Phase iii** is the large, comparative, confirmatory RCT that supplies the pivotal evidence for licensing. Phase IV is post-marketing surveillance after approval, detecting rare or long-term adverse effects in real-world use (Goodman & Gilman 2018).

20 to 100	100 to 300	300 to 3000	thousands
PHASE I (HEALTHY VOLUNTEERS)	PHASE II (PATIENTS)	PHASE III (PATIENTS)	PHASE IV (POST-MARKETING)

PHASE	CORE QUESTION	WHO / TYPICAL N	DESIGN
Phase I	**Is it safe in humans, and how is it handled (PK)?**	Small healthy volunteer groups, roughly **20 to 100**	First-in-human, dose-escalation, often open
Phase II	**Does it show efficacy, and at what dose?**	Patients with the disorder, roughly **100 to 300**	Dose-finding; often placebo-controlled, randomised
Phase III	**Does it work better than the comparator, confirmed?**	Larger patient samples, roughly **300 to 3000**	Large double-blind confirmatory RCT for licensing
Phase IV	**What happens in real-world, long-term use?**	Post-marketing, thousands of users	Surveillance / pharmacovigilance after approval

The question, the sample and the setting all scale up phase by phase: safety in a handful, efficacy and dose in hundreds, confirmation in thousands, then lifelong surveillance (typical Ns per standard pharmacology texts; Goodman & Gilman 2018).

WORKED EXAMPLE

A novel antidepressant: phase I gives single ascending oral doses to about 40 healthy volunteers, measuring half-life and tolerability with no efficacy claim. Phase II randomises roughly 200 patients with major depression across three doses versus placebo to identify the effective dose. Phase III runs two double-blind placebo-controlled RCTs of around 500 patients each, the pivotal evidence submitted for licensing. After approval, phase IV registries pick up a rare hepatic signal invisible in the pre-licensing sample. The same drug thus needs every phase; a positive phase III cannot substitute for phase IV vigilance.

3.9 How the pieces defend one another

The safeguards form one continuous chain. Randomisation manufactures comparable groups; allocation concealment stops that randomisation being unpicked at recruitment; blinding stops it being eroded during treatment and assessment; intention-to-treat stops it being discarded at analysis. Assess any published RCT by walking this chain, and you will locate its weakest link. A trial that is randomised but with poor concealment, or double-blind but analysed per-protocol, has a leak precisely where the exam wants you to point.

HOLD THIS

Allocation concealment guards the assignment at entry; blinding guards perception after entry; intention-to-treat keeps everyone analysed in the arm they were randomised to; and the drug phases climb safety (20 to 100 healthy volunteers) to efficacy/dose (100 to 300 patients) to confirmatory licensing (300 to 3000) to post-marketing surveillance.

GOOD TO REMEMBER

- **Randomised controlled trial:** the only experimental therapeutic design; allocates treatment by chance and compares arms forward; the one design that balances unknown confounders, which is why it is the therapy gold standard.
- **Randomisation:** distributes known and unknown confounders evenly; simple (self-corrects when large), block (keeps arms equal), stratified (balances a key prognostic factor by design).
- **Allocation concealment:** hides the upcoming assignment from the recruiter at the moment of entry; best achieved by remote / independent randomisation; distinct from blinding, which acts after entry.
- **Blinding:** conceals the arm to cut bias; single (patient), double (patient plus clinician), triple (plus outcome assessor); a placebo makes patient blinding possible.
- **Intention-to-treat vs per-protocol:** ITT analyses all as randomised and preserves comparability (conservative); per-protocol counts only completers and is prone to bias; "once randomised, always analysed".
- **Parallel-group:** concurrent independent arms; the default; needs full N per arm.
- **Crossover:** each patient is own control receiving both treatments in sequence; use in rare, stable, chronic disease; watch carryover and order effects, use a washout.
- **Factorial:** two-plus interventions crossed (2x2 = A, B, both, neither); answers two questions and tests interaction in one trial; valid if the factors do not strongly interact.
- **Cluster randomised:** randomises groups (clinics, practices) not individuals; use for service or educational interventions that cannot be confined to one person; needs many clusters because the cluster is the analysis unit.
- **Superiority / non-inferiority / equivalence:** beats comparator / not worse than by a set margin / differs by no more than a margin either way; a non-significant superiority result does not prove equivalence.
- **Drug trial phases:** phase I safety and PK in 20 to 100 healthy volunteers; phase II efficacy and dose-finding in 100 to 300 patients; phase III large confirmatory comparative RCT of 300 to 3000 for licensing; phase IV post-marketing surveillance in thousands.

4 · Validity, bias and confounding

This is the quality-control chapter of research. Every study makes a claim; validity, bias and confounding are the three questions the examiner uses to decide whether the claim is believable. **Validity** asks whether the finding is true (for this sample, and then for the wider world); **bias** is systematic error that pushes the answer consistently in one direction; and **confounding** is a third variable that mimics a real association. The stem usually gives a scenario and asks you to name the flaw, classify it, and say how it should have been controlled. Get the taxonomy clean and almost every methods question falls open (Kaplan & Sadock 2024; Bland 2015; Altman 1991).

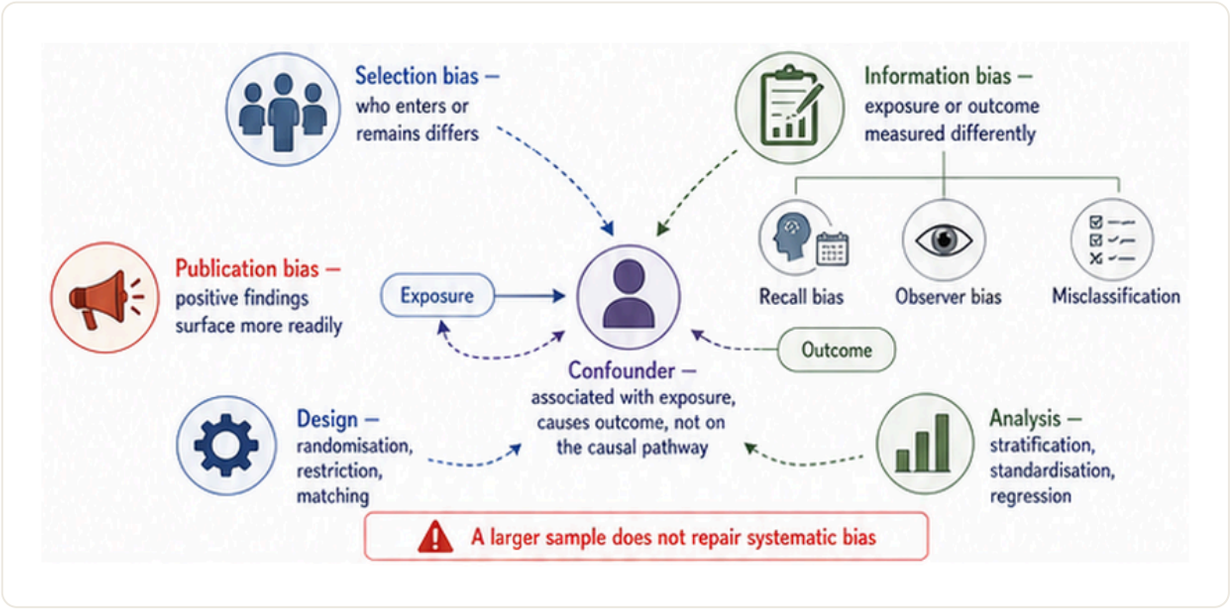


Fig 7.7 Bias and confounding: how results move away from truth.

Two housekeeping distinctions anchor the topic before the detail. First, keep **systematic error** (bias, which no increase in sample size removes) apart from **random error** (chance, which shrinks as the sample grows and is quantified by confidence intervals and the p-value). Second, the study reliability and validity discussed here (are the study's results reproducible and true) are a DIFFERENT idea from the psychometric-test reliability and validity of a scale or instrument (whether a questionnaire measures consistently and measures what it claims); those are taught in the Psychometry, Assessment and Descriptive Psychopathology notes. Keep the two vocabularies separate: here the object is a whole study, there it is a single test; this separation is restated where reliability is defined below.

4.1 Internal validity: are the results true for this sample

Does the study answer its own question correctly, free of bias and confounding? Internal validity is the degree to which the observed association between exposure and outcome is real for the participants actually studied, rather than an artefact of bias, confounding or chance (Kaplan & Sadock 2024). It is the first and non-negotiable property: a study with poor internal validity is simply wrong, and how far it generalises is then irrelevant. Threats to internal validity are exactly the topics of this chapter, selection bias, information bias and confounding, plus chance. The randomised controlled trial earns its rank because randomisation and blinding protect internal validity better than any observational design can. Efficacy trials in tightly selected samples are built to maximise internal validity (Kaplan & Sadock 2024).

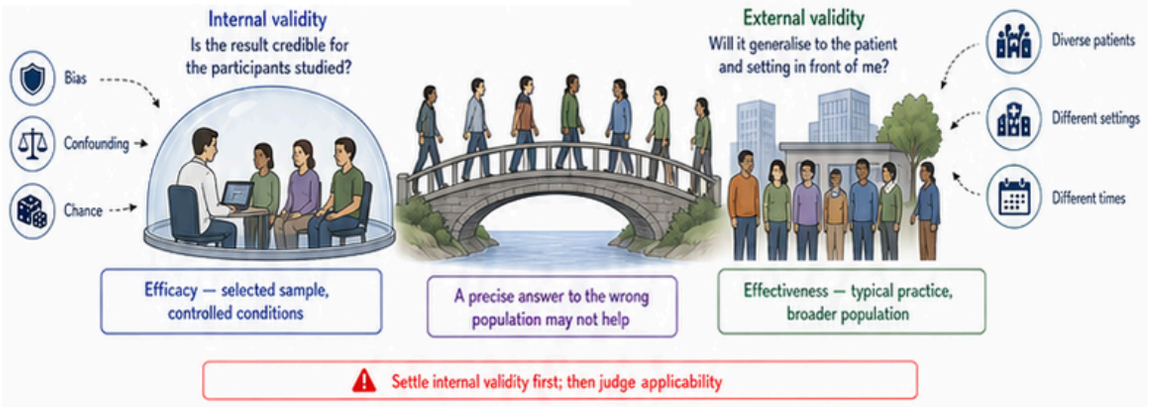


Fig 7.8 Internal and external validity: true here, useful there.

RULE **Internal validity comes first.** If the result is not true for the sample studied, it does not matter whether it generalises: settle internal validity before external validity.

4.2 External validity: do the results generalise

Can the finding be applied beyond the study sample? External validity, also called **generalisability** (spelled generalisability or generalizability), is the extent to which the results hold for patients outside the study, in other settings, populations and times (New Oxford Textbook of Psychiatry). A trial can have flawless internal validity yet poor external validity if its participants were so narrowly selected (single-diagnosis, no comorbidity, no substance use) that the result cannot be read across to the messy patients of ordinary practice. This is the efficacy versus effectiveness trade-off: efficacy studies maximise internal validity in narrow samples, whereas effectiveness (pragmatic) studies use broad, typical, comorbid populations so that external validity carries more weight (Kaplan & Sadock 2024). The two validities pull against each other, and reading which one a design has bought is a favourite question.

WORKED EXAMPLE

An antidepressant RCT enrols only patients aged 18 to 45 with major depression and no comorbidity, randomises and double-blinds cleanly, and reports a clear drug effect. Its internal validity is high: the effect is real for those studied. Its external validity is low: most real depressed patients are older, or have anxiety, alcohol use or physical illness, so the result may not generalise to them. High internal, low external validity is the signature of a tightly controlled efficacy trial.

4.3 Reliability: reproducibility of the measurement

Does the study measure the same thing consistently on repetition? Study reliability, or **repeatability**, is the reproducibility of a measurement: if the same subjects were measured again under the same conditions, would the same values result. Reliability concerns precision (random scatter), whereas validity concerns accuracy (systematic closeness to the truth); the classic image is the target, where a reliable-but-invalid instrument lands its shots tightly clustered but off the bullseye, and a valid-but-unreliable one scatters around the centre. A measurement can be reliable without being valid, but it cannot be valid without first being reliable, so reliability is necessary

but not sufficient for validity. As flagged in the intro, this study-level reliability is distinct from the psychometric-test reliability (internal consistency, test-retest, inter-rater) covered in the Psychometry, Assessment and Descriptive Psychopathology notes.

4.4 Bias: systematic error, and its two great families

Bias is systematic error, built into the study, that no larger sample can fix. Bias is any systematic (non-random) error in the design, conduct or analysis of a study that produces a result consistently distorted away from the truth (Kaplan & Sadock 2024). Because it is systematic, adding participants only makes a biased study more precisely wrong; bias is defeated by better design, not by size. Following the standard epidemiological division, bias falls into two great families plus a literature-level form: selection bias (who gets into the study and who is compared), information bias (how exposure and outcome are measured once in), and publication bias (which studies reach print). Naming the family first, then the specific mechanism, is the reliable way to answer a bias vignette (Tasman Psychiatry).

4.5 Selection bias

A systematic error in how participants enter or are compared. Selection bias arises when the way subjects are selected, or the way exposed and unexposed (or cases and controls) are chosen, is related to both the exposure and the outcome, so the compared groups differ systematically from the start (Tasman Psychiatry). Textbook varieties include the **healthy worker effect** (employed populations are healthier than the general population, so occupational cohorts understate harm), **self-selection (volunteer) bias** (people who respond to advertisements or agree to take part differ from those who do not, and the exposed with symptoms may be keener to enrol), **Berkson's bias** (hospital-based cases and controls carry a spurious association because admission itself depends on having one or more conditions), and **loss to follow-up (attrition) bias** (differential dropout in a cohort or trial, a serious threat when dropout is related to both exposure and outcome). The defence is at the design stage: define the source population, sample and recruit exposed and unexposed identically, and choose controls from the same population that produced the cases.

4.6 Information bias, including recall and observer bias

A systematic error in how exposure or outcome is measured. Information bias (also called observation or measurement bias) occurs when data on exposure or outcome are obtained in a non-comparable manner between the groups, so that one group's information is systematically more or less accurate than the other's (Tasman Psychiatry). Its named forms are high-yield. Recall bias is differential accuracy of memory: people with the outcome (cases, or mothers of an affected child) search their past for exposures more thoroughly than unaffected controls, inflating the apparent association; it is the classic weakness of the retrospective case-control study. **Observer (or interviewer) bias** is systematic error introduced by the assessor: an interviewer who knows a subject's disease status probes exposure harder in cases, or an unblinded rater scores the outcome more favourably in the treated arm; masking (keeping the assessor blind to exposure or allocation) is the remedy (Tasman Psychiatry). **Misclassification** is the mechanism by which information bias operates: non-differential misclassification (errors unrelated to group) usually

dilutes an association toward the null, whereas differential misclassification (errors that differ by group, as in recall bias) can bias in either direction and is the more dangerous. Publication bias is a special, literature-level information problem addressed at 4.7.

TRAP **Recall bias needs two groups remembering differently.** It is not just "poor memory": it is differential recall between cases and controls. Symmetric forgetting in both groups is non-differential misclassification, not recall bias.

4.7 Publication bias

Positive studies are published more, and sooner, than negative ones. Publication bias is the tendency for studies with statistically significant, positive or striking results to be submitted, accepted and published more readily than studies with null or negative findings, so the published literature systematically overstates an effect. It is the reason a meta-analysis restricted to published trials can be misleading, and it is detected within the systematic-review and meta-analysis topic by an asymmetric funnel plot (and formal tests such as Egger's), and mitigated by trial registries and grey-literature searching. It is treated more fully as a threat to the systematic review; here, hold that it is a bias of the literature, not of a single study's internal conduct.

4.8 Confounding: the third variable

A third variable linked to both exposure and outcome, and not on the causal path. Confounding occurs when a third factor is associated with the exposure and is independently a cause of the outcome, so that it distorts (creates, inflates, masks or even reverses) the apparent exposure to outcome association (Tasman Psychiatry). Three conditions define a **confounder**: it must be associated with the exposure, it must be an independent risk factor for (cause of) the outcome, and it must NOT lie on the causal pathway between exposure and outcome (a variable on that pathway is a mediator, not a confounder). The examiner's classic is the unemployment to suicidal behaviour question in which **alcoholism** is related both to unemployment and to suicidal behaviour, and therefore confounds the association unless it is adjusted for (Tasman Psychiatry). Other stock examples are age and sex confounding almost any exposure to disease link, and the coffee to lung-cancer association confounded by smoking (smokers drink more coffee and smoking causes lung cancer). Spot a confounder by testing the three conditions, then decide whether it was handled at design or at analysis.

WORKED EXAMPLE

A study finds unemployed people have more suicidal behaviour, crude relative risk **2.0**. But alcohol dependence is commoner among the unemployed AND independently raises suicide risk, so it satisfies both confounder arms and is not on the causal path. Stratify by alcohol status: within non-drinkers RR is **1.2** and within dependent drinkers RR is **1.3**. The crude 2.0 was inflated by confounding; the adjusted (stratum-specific) estimate near 1.2 to 1.3 is the truer effect of unemployment itself. Adjusting for alcohol is correct here because alcohol is a confounder, not a step on the pathway from unemployment to suicide.

4.9 Controlling confounding at the design stage

Prevent the imbalance before data are collected. Four design tools handle confounding up front. Randomisation allocates subjects to groups by chance so that, on average, both known AND unknown confounders are balanced between arms; it is the only method that controls confounders you have not measured or even thought of, which is the entire reason the RCT is the reference standard. **Restriction** admits only one level of the confounder (for example, study only non-smokers), which removes its confounding effect but narrows external validity. **Matching** selects comparison subjects to have the same distribution of a confounder as the index group (individual or frequency matching), balancing that specific confounder, though matched data then require a matched analysis. Two further design safeguards protect randomisation itself: allocation concealment hides the upcoming assignment from the recruiter so that selection into arms cannot be manipulated, and blinding (masking) keeps participants, clinicians and assessors unaware of allocation; a **double-blind** design masks both the participant and the assessor, defending against performance and observer (ascertainment) bias. Note that allocation concealment governs entry into the arms whereas blinding governs everything after; they are distinct and both matter.

RULE **Only randomisation controls unknown confounders.** Restriction, matching, stratification, standardisation and regression each control confounders you have measured; solely randomisation balances the ones you have not.

4.10 Controlling confounding at the analysis stage

Correct for the measured confounder after data are collected. When a confounder was recorded but not designed out, three analytic tools adjust for it. Stratification divides the data into strata of the confounder, computes the association within each stratum where the confounder is held constant, and pools the stratum-specific estimates (the Mantel-Haenszel method); comparing the crude and adjusted estimates reveals how much confounding was present. **Standardisation** re-weights rates to a common reference population (direct or indirect), the method behind age-standardised and sex-standardised rates, so groups with different age or sex structures can be compared fairly. Multivariable (regression) adjustment models the outcome on the exposure plus the confounders simultaneously (linear, logistic or Cox regression), yielding an exposure effect adjusted for every covariate in the model; it is the workhorse of modern observational analysis. The hard limit of every analytic method is that you can only adjust for confounders you measured, and residual confounding from unmeasured or mismeasured variables always remains, which is precisely why randomisation is unique.

TRAP **Never adjust for a mediator.** Confounding is NOT a type of bias, and a variable on the causal pathway (a mediator) is not a confounder: adjusting for it wrongly blocks part of the real effect (overadjustment).

CLASS	NAMED FORM	WHAT GOES WRONG	TYPICAL DESIGN	DEFENCE
Selection bias	**Healthy worker effect**	Employed sample healthier than general population, harm understated	Occupational cohort	Suitable external comparison group
Selection bias	**Self-selection (volunteer)**	Participants differ systematically from non-participants	Advertised recruitment, surveys	Define and sample a source population
Selection bias	**Berkson's bias**	Hospital cases and controls carry a spurious association via admission	Hospital case-control	Community (population) controls
Selection bias	**Loss to follow-up (attrition)**	Differential dropout related to exposure and outcome	Cohort, RCT	Minimise and analyse dropout; intention-to-treat
Information bias	**Recall bias**	Cases remember exposures more thoroughly than controls	Retrospective case-control	Records-based exposure; nested design
Information bias	**Observer / interviewer bias**	Unblinded assessor probes or scores groups differently	Any assessed study	Masking (blinding) of assessors
Information bias	**Misclassification**	Errors in exposure/outcome labelling (non-differential dilutes; differential either way)	Any measurement	Validated, objective, blinded measures
Publication bias	**Positive-result bias**	Significant studies published more, literature overstates effect	Meta-analysis of published trials	Trial registries; funnel plot; grey literature
Confounding	**Third-variable distortion**	Factor linked to exposure and outcome, not on causal path, mimics association	Any observational study	Randomisation, restriction, matching; stratification, standardisation, regression

Bias taxonomy: the family, a named form, the mechanism, the design it typically strikes, and the defence; the hit column is the named entity to recognise in a stem.

HOLD THIS

Bias is systematic error split into selection bias and information bias; confounding is a third variable tied to both exposure and outcome but not on the causal path; and randomisation is the only tool that balances UNKNOWN confounders.

COMMON TRAP

Two errors examiners love. First, calling confounding a type of bias: it is a separate category (a real third-variable effect, controllable at analysis), not a systematic measurement or selection error. Second, treating internal and external validity as the same axis: a narrow efficacy trial can have excellent internal validity and poor external validity at once, and confusing the two mis-reads the whole study.

PEARL

To separate confounding from mediation, ask where the third variable sits. If it independently causes the outcome and merely travels with the exposure, it is a confounder, so adjust for it. If the exposure causes it and it in turn causes the outcome, it is a mediator on the causal path, so adjusting for it (overadjustment) erases part of the true effect. The same statistical adjustment is right for the first and wrong for the second.

MNEMONIC

Confounder = "linked to Exposure, causes Outcome, off the Path" (E-O-P).

A confounder must be associated with the Exposure, be an independent cause of the Outcome, and lie off the causal Path. Fail any one of the three and it is not a confounder: variables on the path are mediators, and variables unrelated to the exposure are simply other risk factors.

GOOD TO REMEMBER

- **Internal validity:** are the results true for this sample; threatened by selection bias, information bias, confounding and chance; the one to secure first, before generalisability.
- **External validity (generalisability):** do the results apply beyond the sample; use it to judge whether an efficacy-trial finding transfers to ordinary patients; bought by broad, typical, pragmatic samples.
- **Reliability (repeatability):** reproducibility of a measurement on repetition; the target-clustering property; necessary but not sufficient for validity (reliable can be invalid, but valid needs reliable).
- **Bias:** systematic (not random) error; use design, not sample size, to defeat it; the number to fear is that no increase in n reduces bias.
- **Selection bias:** non-comparable entry or comparison groups; suspect it in volunteer, hospital-based or high-dropout studies; defended by sampling a defined source population identically.
- **Information bias:** non-comparable measurement of exposure or outcome; includes recall bias and observer bias; defended by blinding (masking) and validated measures.
- **Recall bias:** differential memory between cases and controls; the classic flaw of the retrospective case-control study; defended by records-based or nested designs.
- **Confounding:** a third variable associated with exposure and causing the outcome, off the causal path; controlled at design (randomisation, restriction, matching, allocation concealment, blinding) or at analysis (stratification, standardisation, regression); only randomisation handles unknown confounders.

5 · Sampling methods

Whether a study can speak beyond its own participants is decided the moment the sample is drawn. **Sampling** is the process of drawing a subset of units (the sample) from the whole population of interest so that a study can be run on the few while conclusions are made about the many. The examiner wants three things from a one-line vignette: name the sampling method, say whether it is **probability** or **non-probability**, and state whether the resulting sample can support a generalisation to the population (Bland 2015; Altman 1991; Kaplan & Sadock 2024). The single hinge is that only probability sampling, where every unit has a known non-zero chance of selection, permits valid statistical inference from sample to population.

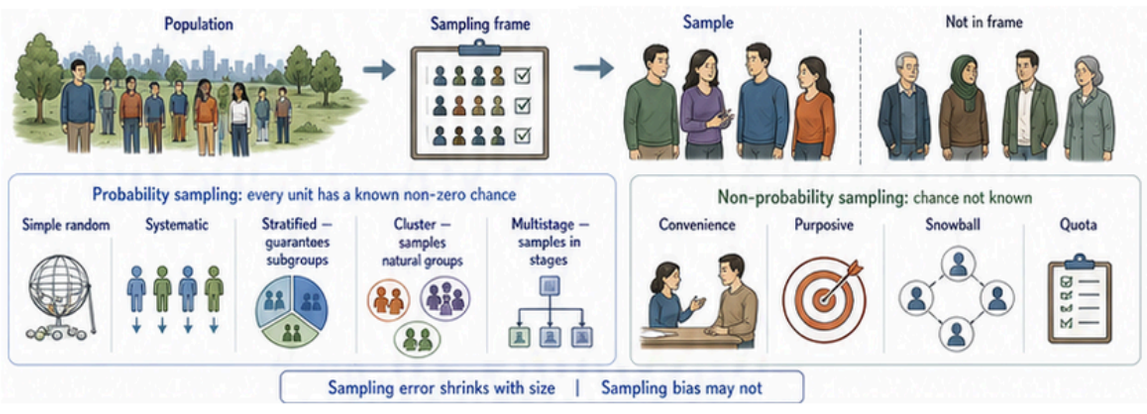


Fig 7.9 Sampling: who gets a chance to enter?.

The family splits cleanly. **Probability sampling** methods (simple random, systematic sampling, stratified, cluster sampling, multistage) use a chance mechanism and allow generalisable

inference; **non-probability sampling** methods (convenience, purposive, snowball, quota) select by accessibility or judgement and cannot support a population estimate. Hold the two-column split and the rest is detail.

5.1 The population, the sampling frame, and the sample

Three nested objects define every sampling problem. The **target population** is the whole group about whom conclusions are wanted (for example, all adults in a district). The sampling frame is the actual list or operational device from which units are drawn (an electoral roll, a household census list, a clinic register): it is the population as it is really available to be sampled. The **sample** is the subset finally selected and measured (Bland 2015). Most real-world error enters at the frame: if the frame omits part of the target population (the homeless are missing from an electoral roll, the untreated are missing from a clinic register) the sample is biased before a single unit is drawn, however elegant the selection method. Naming the frame is the first discipline of good sampling.

Target population

The whole group about whom inference is wanted.

Sampling frame

The actual list or device from which the sample is drawn; the practical stand-in for the population.

Sampling unit

The element selected at each draw (a person, a household, a school, a village).

5.2 Probability sampling: the licence to generalise

Every unit has a known, non-zero chance of selection. In **probability sampling** selection is by a chance mechanism, so the probability that any given unit enters the sample is known and greater than zero (Kaplan & Sadock 2024; Bland 2015). This is what makes a sample *representative in expectation* and lets sampling theory attach a standard error and a confidence interval to an estimate: only under a known selection probability can the mathematics of inference from sample to population be justified. Community psychiatric morbidity surveys therefore use probability sampling (the National Comorbidity Survey and the WHO World Mental Health surveys used multistage household probability sampling precisely to license generalisation to their target populations). If you cannot state each unit's chance of selection, you are not doing probability sampling and you forfeit the right to a population estimate.

RULE **Known chance, valid inference.** Only when every unit has a known non-zero probability of selection can a sample estimate be generalised to the population with a valid standard error and confidence interval.

5.3 Simple random and systematic sampling

The two basic single-stage probability methods. In **simple random** sampling every unit in the frame has an equal and independent chance of selection, drawn by lottery or random-number generator; it is the reference standard against which all other methods are judged and needs a complete numbered frame (Bland 2015; Altman 1991). **Systematic sampling** selects every kth unit from the ordered frame after a random start, where the sampling interval k equals the

population size divided by the desired sample size (draw a random start between 1 and k, then take that unit, then every kth thereafter). Systematic sampling is quicker and needs no full random draw, and it spreads the sample evenly through the list; its one hazard is a hidden periodicity in the frame that coincides with the interval k (for example, if every kth bed is a corner bed), which can bias the sample. Both are probability methods and both give generalisable estimates.

Simple random
Equal, independent chance for every unit; the gold-standard probability method.

Systematic sampling
Every kth unit after a random start, k = population size / sample size; fast but vulnerable to periodicity in the frame.

WORKED EXAMPLE

You have a numbered frame of 1,000 clinic attenders and want a sample of 100. The sampling interval is k = 1000 / 100 = **10**. Pick a random start between 1 and 10, say 7, then take attender 7, 17, 27, 37 and so on to 997: that is **systematic sampling**. Every attender had a 1 in 10 (0.1) chance of selection, so it is a probability sample and the estimate generalises, provided the list carries no repeating pattern that lands on the interval of 10.

5.4 Stratified sampling: guaranteeing the subgroup

Divide first into strata, then sample within each. Stratified sampling first partitions the population into mutually exclusive strata defined by a characteristic that matters (age band, sex, urban versus rural, diagnosis), then draws a random sample *within each stratum* (Bland 2015; Altman 1991). Its whole purpose is to guarantee representation: because each stratum is sampled deliberately, even a small subgroup that a simple random draw might miss by chance is certain to appear, and stratifying on a variable related to the outcome also reduces the standard error of the overall estimate (it removes between-stratum variability). In proportionate stratification each stratum contributes in proportion to its share of the population; in disproportionate stratification a small but important stratum is deliberately over-sampled and the estimates are re-weighted at analysis (this is the oversampling of older adults or minority groups seen in the large epidemiological surveys). Stratified sampling never increases the standard error relative to simple random sampling and usually lowers it.

RULE **Stratify to represent a small subgroup.** When you must be certain a small but important subgroup appears in adequate numbers, stratify on that characteristic and sample within the stratum, oversampling it if needed and re-weighting at analysis.

5.5 Cluster sampling and the design effect

Sample whole groups, not individuals. Cluster sampling divides the population into naturally occurring groups (clusters) such as villages, schools, wards or households, takes a random sample of whole clusters, and then studies every unit within the selected clusters (or a further sample of them). It is chosen for logistics and cost: when the population is geographically dispersed and no

complete individual-level frame exists, it is far cheaper to enumerate and travel to a handful of clusters than to chase a random scatter of individuals across the whole area (Bland 2015). The price is statistical. People within a cluster tend to resemble one another (they share environment, culture, exposures), so each additional person in a cluster adds less new information than an independent draw would. This within-cluster correlation inflates the variance, quantified by the design effect (Deff): the ratio of the variance under the cluster design to the variance a simple random sample of the same size would have given. A design effect greater than 1 means the cluster sample carries less information than its nominal size, so the sample size must be multiplied by the design effect to recover the intended precision. Stratified sampling reduces variance; cluster sampling increases it. That contrast is examined constantly.

Cluster sampling
Randomly sample whole clusters (villages, schools), then study units within them; cheaper for dispersed populations, no individual frame needed.

Design effect (Deff)
Variance under the cluster design divided by the variance a simple random sample would give; Deff > 1 means precision is lost and the sample must be enlarged.

TRAP **Ignoring the design effect.** Analysing a cluster sample as if it were a simple random sample understates the true standard error and gives falsely narrow confidence intervals and falsely small p-values; the sample size must be inflated by the design effect.

5.6 Multistage sampling: the real-world survey design

Sampling in successive nested stages. **Multistage** sampling combines the above into a sequence: sample large primary units first, then sub-sample within them, over two or more stages. A national survey might first randomly select districts (stage 1), then villages within selected districts (stage 2), then households within selected villages (stage 3), then one adult within each selected household (stage 4). It is the practical backbone of almost every large community survey because it needs a frame only for the units selected at the next stage down, never a complete individual-level frame for the whole country. Each clustering stage carries its own design effect, so multistage designs are analysed with survey-weighted methods that account for the stratification, the clustering and the unequal selection probabilities. The National Mental Health Survey type of design and the WHO World Mental Health surveys are multistage household probability samples of exactly this kind (Kaplan & Sadock 2024).

5.7 Non-probability sampling: convenience, purposive, snowball, quota

Selection by access or judgement, not chance. In **non-probability sampling** the chance of any unit being selected is unknown, so no valid generalisation to a population can be made; these methods belong to qualitative research, pilot studies and situations where a frame is impossible (Kaplan & Sadock 2024). **Convenience** sampling takes the units easiest to reach until the target size is met (a volunteer sample, the patients who happen to be in clinic today); it is fast and cheap but its self-selection makes it the least representative method. **Purposive** (judgemental) sampling selects units the researcher judges most informative for the question, the standard approach in

qualitative work where the aim is depth and typicality, not statistical representativeness. **Snowball** sampling starts from a few index participants who then recruit further participants from their own networks, and is the method of choice for hidden or hard-to-reach populations (people who inject drugs, sex workers, the homeless) where no frame exists (Kaplan & Sadock 2024). Quota sampling fills preset quotas for chosen characteristics (so many men, so many women) by non-random selection until each quota is met: it superficially resembles stratified sampling but the crucial difference is that within each quota the units are chosen non-randomly, so it remains a non-probability method. Consecutive sampling, a near neighbour, recruits every accessible unit over a defined period.

TRAP **Convenience sampling cannot support a prevalence estimate.** A prevalence figure is a population statement; a convenience sample has an unknown selection probability, so it can describe only the sample and can never be quoted as the prevalence in the population.

5.8 Sampling error versus sampling bias

Two different failures, only one of which shrinks with size. Sampling error is the random difference between a sample estimate and the true population value that arises simply because a sample, not the whole population, was measured; it is unavoidable, it has no direction, and it is exactly what the standard error and the confidence interval quantify. Crucially, sampling error *decreases as the sample size increases* and disappears if the whole population is measured (Bland 2015). Sampling bias (selection bias) is a systematic error in the way units are selected, so that the sample is unrepresentative in a consistent direction (the classic example is drawing a sample of the mentally ill only from treated populations, which over-represents the more severe and help-seeking). Bias has a direction, it distorts the estimate the same way every time, and, fatally, *a larger sample does not fix it*: it merely produces a more precise wrong answer. Distinguishing the two, and knowing that size cures error but not bias, is one of the most reliably examined points in the whole topic.

Random	Systematic
SAMPLING ERROR: NO DIRECTION, SHRINKS WITH N	SAMPLING BIAS: HAS DIRECTION, NOT FIXED BY N

TRAP **A bigger sample does not cure bias.** Increasing the sample size narrows sampling error but a biased sampling method just gives a more precise wrong estimate; only a better sampling design removes bias.

5.9 The selector: matching the situation to the method

Read the constraint, pick the method. A sampling vignette turns on three things: is a complete frame available, must a particular subgroup be guaranteed, and is generalisation required at all? A complete individual frame invites simple random or systematic sampling; the need to represent a small subgroup calls for stratification; a dispersed population with no individual frame forces cluster or multistage sampling and its design effect; and a hidden population or a purely exploratory aim drops you into the non-probability methods, which forfeit generalisation.

METHOD	PROBABILITY?	HOW IT SELECTS	WHEN TO USE IT
Simple random	**Yes**	Equal, independent chance for every unit (lottery / random numbers)	Complete numbered frame available; the reference standard
Systematic sampling	**Yes**	Every kth unit after a random start, k = N / n	Ordered frame; quicker than a full random draw; no periodicity in the list
Stratified	**Yes**	Split into strata, random-sample within each	Must guarantee a small subgroup; improves precision
Cluster sampling	**Yes**	Randomly sample whole clusters, study units within	Dispersed population, no individual frame; cheaper, but larger design effect
Multistage	**Yes**	Successive nested sampling stages	Large national surveys; needs a frame only stage by stage
Convenience	**No**	Whoever is easiest to reach	Quick pilots only; cannot generalise
Purposive	**No**	Researcher's judgement of who is informative	Qualitative work seeking depth and typicality
Snowball	**No**	Index participants recruit further participants	Hidden or hard-to-reach populations with no frame
Quota	**No**	Fill preset quotas by non-random selection	Fast field surveys; looks stratified but is not random

The sampling family read across method, whether it is probability-based, how it selects, and when to use it; the hit column is the probability-versus-non-probability discriminator that decides generalisability.

HOLD THIS

Probability sampling (known non-zero selection chance) is the only route to a generalisable population estimate; stratify to guarantee a small subgroup, cluster to save cost at the price of a design effect greater than 1, and never quote a prevalence from a convenience sample.

INDIA

The National Mental Health Survey of India (2015 to 2016) is the flagship probability-sampled example: a **multistage** stratified random household survey across 12 states that could therefore report a generalisable current weighted prevalence of any mental disorder of about **10.6** percent and a treatment gap for common mental disorders exceeding **80** percent. Because selection probabilities were known and the design was multistage with clustering, the estimates carry valid confidence intervals: a convenience sample of clinic attenders could never have produced a national prevalence figure.

PEARL

Stratification and clustering pull the standard error in opposite directions. Stratified sampling *reduces* variance because it removes between-stratum variability, so its design effect is typically below 1; cluster sampling *increases* variance because units within a cluster are correlated, so its design effect is above 1. A survey that stratifies and then clusters, as most national surveys do, trades the two off, which is why the net design effect must be computed and the sample size inflated accordingly.

MNEMONIC

Probability sampling: "Some Systems Sort Clusters Multiply."

Simple random, Systematic, Stratified, Cluster, Multistage: the five probability methods. Everything else (convenience, purposive, snowball, quota) is non-probability and cannot generalise.

GOOD TO REMEMBER

- **Simple random:** every unit has an equal, independent chance of selection; use it whenever a complete numbered frame exists; it is the gold-standard probability method against which the design effect of others is judged (Deff = 1).
- **Systematic sampling:** take every kth unit after a random start; use it for a quick draw from an ordered list; the one number is the interval k = population size / sample size.
- **Stratified:** split into strata and random-sample within each; use it to guarantee a small subgroup is represented and to improve precision; its design effect is usually below 1 (it lowers the standard error).
- **Cluster sampling:** randomly sample whole clusters and study units within them; use it for a dispersed population with no individual frame to save cost; the number to know is the design effect (Deff greater than 1), by which the sample size must be multiplied.
- **Multistage:** sample in successive nested stages (districts, then villages, then households, then individuals); use it for large national surveys; it needs a frame only for the units at the next stage, and each clustering stage adds its own design effect.
- **Convenience:** take whoever is easiest to reach; use it only for quick pilots; the fact to hold is that its unknown selection probability means it cannot support a prevalence estimate.
- **Purposive:** select by the researcher's judgement of who is most informative; use it in qualitative research for depth and typicality; it makes no claim to statistical generalisation.
- **Snowball:** index participants recruit further participants through their networks; use it for hidden or hard-to-reach populations with no sampling frame; it is non-probability, so no population estimate follows.

6 · Qualitative research methods

Some questions cannot be answered by counting. **Qualitative** research is the systematic study of meaning and experience: it asks how people understand their illness, why an intervention fails in practice, what a service feels like from the inside. It collects and analyses data that cannot be reduced to numbers (interview transcripts, field notes, documents) and reads them for categories and themes rather than for means and p-values (Bowling 2014; Greenhalgh 2014). The exam wants three things from you: the named approaches with a one-line signature each, the concepts that give a qualitative study its **rigour** (the qualitative counterpart of reliability and validity), and a clear sense of when a qualitative design beats a quantitative one. Get those and any stem, whether it names grounded theory or asks "what is saturation", falls open.

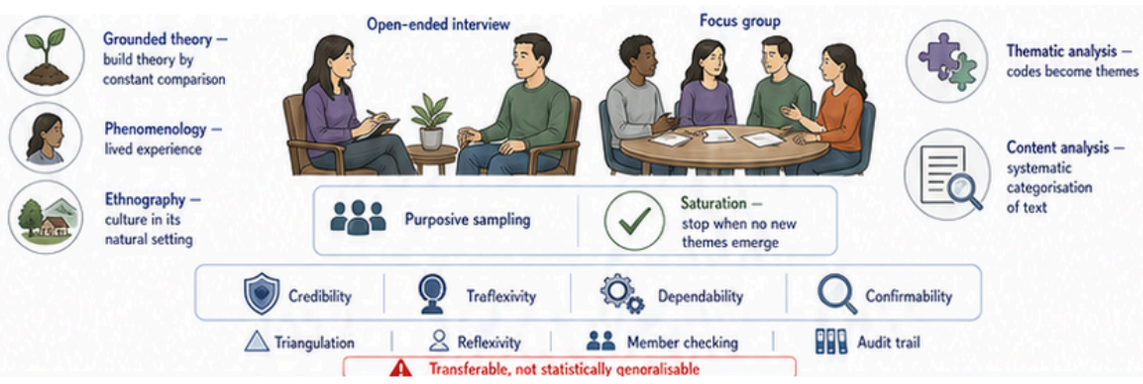


Fig 7.10 Qualitative research: depth, meaning and context.

The mental model is a mirror image of the quantitative one. Where a trial samples randomly to represent a population and tests a pre-set hypothesis, a qualitative study samples **purposively** to capture informative cases and lets categories emerge from the data, with analysis running iteratively alongside collection (Bowling 2014). It generalises to theory and concepts, never to population statistics. Hold that contrast and the rest is detail.

6.1 What qualitative research is, and how it differs from quantitative

Understanding meaning, not measuring frequency. A **qualitative** study seeks to understand a phenomenon through personal encounter, observation and text, rather than to quantify it (Bowling 2014; Greenhalgh 2014). The defining differences from quantitative work are consistent and examinable: sampling is purposive (subjects chosen deliberately for characteristics relevant to the question, not random selection); data collection is open-ended and often interactive (interviews, observation, documents, not standardised scales); analysis is **iterative** and interpretive, generating categories and themes as the data accumulate rather than testing a fixed hypothesis at the end; and the aim is depth of understanding and **transferability** of concepts, not statistical generalisation to a population. The two are complementary, not rivals: a mixed-methods study can nest a qualitative arm inside a randomised trial to explain why participants responded as they did.

RULE **Purpose sets the design.** If the question is "how many / how much / does it work", think quantitative; if it is "why / how / what is it like", think qualitative.

6.2 The five main approaches

Each approach has a distinct object and analytic move. The examiner most often wants the named approach matched to its one-line signature (Bowling 2014; Greenhalgh 2014). Grounded theory builds a theory upward from the data themselves through **constant comparison**, comparing each new piece of data with the emerging categories until a theory grounded in the material is generated (Glaser and Strauss 1967). Thematic analysis is the workhorse method that codes the data and organises those codes into themes, without committing to a deeper theoretical framework (Braun and Clarke 2006). The phenomenological approach studies the **lived experience** of a phenomenon, the essence of what it is like to undergo it, from the first-person perspective. Ethnography studies a culture or group in its natural setting, classically through prolonged participant observation and immersion. Content analysis systematically categorises the content of text or documents, and can be handled qualitatively (interpreting meaning) or quantitatively (counting the frequency of coded categories).

Grounded theory

Theory built from the data by constant comparison; sampling continues until the theory is saturated.

Thematic analysis

Codes distilled into themes; flexible, framework-light, the commonest entry-level method.

Phenomenological

The lived experience and essence of a phenomenon, from the person's own perspective.

Ethnography

A culture or group studied in its natural setting through immersion and participant observation.

Content analysis

Systematic categorisation of textual or documentary content; qualitative or quantitative in flavour.

6.3 Data collection: focus groups and in-depth interviews

Two workhorses, chosen by what you want to hear. The two staple methods of qualitative data collection are the **focus group** and the in-depth interview (Bowling 2014). A focus group brings a small group of participants (typically six to ten) together with a moderator so that the *interaction between members* generates data: people react to and build on each other's views, which surfaces shared meanings, norms and disagreements that a one-to-one setting would miss. Its weakness is that dominant voices can silence others and it is poor for private or sensitive material. In-depth (semi-structured or unstructured) interviews put one participant with one interviewer using an open topic guide, giving privacy and depth on an individual's experience, and are the natural choice for sensitive subjects or for reconstructing a personal account. Both are complemented by participant observation (watching behaviour in its natural context) and by documentary sources. Sampling for all of these is purposive, and often theoretical (sampling driven by the emerging analysis) in grounded theory.

6.4 Saturation and when to stop sampling

Stop when new data stop teaching you anything. Because a qualitative sample is chosen for information richness rather than for a pre-calculated power, its size is not fixed in advance; it is governed by saturation. **Saturation** is the point at which no new codes, categories or themes emerge from further data collection, so additional interviews or observations add nothing to the analysis (Bowling 2014). Reaching it is the qualitative analogue of an adequate sample size: you keep sampling and analysing in parallel and stop once the emerging framework has stabilised. This is why qualitative sample sizes are small and cannot be stated at the outset the way a trial's n can.

RULE **Stop sampling at saturation.** Continue collecting and analysing data until no new codes or themes emerge; that point, not a pre-set number, sets the qualitative sample size.

6.5 Rigour: the trustworthiness criteria and the techniques that build them

The qualitative counterpart of reliability and validity. A good qualitative study is judged not by statistical validity but by its **trustworthiness**, the framework of Lincoln and Guba (1985) with four criteria: credibility (are the findings a believable account of the participants' reality, the qualitative parallel of internal validity), transferability (can the findings be applied to other settings, the parallel of external validity, supported by rich "thick description" so a reader can judge fit), dependability (would the process be consistent and traceable if repeated, the parallel of reliability), and confirmability (are the findings grounded in the data rather than the researcher's bias, the parallel of objectivity). Several techniques operationalise these criteria (Bowling 2014; Greenhalgh 2014):

Triangulation

Triangulation: using multiple sources, methods, investigators or theories and checking that they converge on the same finding; convergence strengthens credibility.

Reflexivity

Reflexivity: the practice of reflexivity means the researcher explicitly examines and discloses how their own values, assumptions and presence shape the data and its interpretation.

Member checking

Member checking (respondent validation): taking the findings back to participants to confirm they recognise their experience in the account; supports credibility.

Audit trail

An audit trail: a transparent, documented record of every decision from raw data to conclusion, so an outsider can follow the reasoning; supports dependability and confirmability.

TRAP **Qualitative findings are not statistically generalisable.** They transfer to theory and to similar settings (transferability), but they cannot be projected onto a population the way a representative quantitative estimate can.

6.6 When qualitative beats quantitative

Reach for qualitative when the number is not the answer. Qualitative methods are the right tool, not a weaker substitute, in defined situations (Bowling 2014; Greenhalgh 2014). Use them for **hypothesis generation**, where little is known and the aim is to discover the relevant concepts and variables before any quantitative study can be designed. Use them to understand **barriers** and facilitators, why an evidence-based intervention is not adopted in practice, why patients disengage, what obstructs help-seeking, since these turn on meaning and context that a scale cannot capture. Use them for **service evaluation**, to gather the attitudes, beliefs and experiences of service users, carers and professionals, and to see how a service actually works on the ground. They also excel at exploring sensitive or stigmatised experiences and at explaining the "why" behind a surprising quantitative result.

6.7 The selector: matching approach and method to the question

Read the aim, then pick the approach. Faced with a stem, first confirm the question is qualitative (a why/how/what-is-it-like question), then match the specific aim to the approach and the setting to the collection method. If the aim is to build an explanatory theory, choose grounded theory; if it is simply to identify patterns of meaning, thematic analysis; if it is the essence of an experience, phenomenology; if it is a group's culture in situ, ethnography; if it is the systematic content of documents, content analysis. The collection method then follows from whether you need group interaction or private depth.

AIM OF THE STUDY	APPROACH / METHOD TO PICK
Build a new explanatory theory from the ground up	Grounded theory (constant comparison)
Identify patterns of meaning across the data	Thematic analysis (codes to themes)
Capture the essence of a lived experience	Phenomenological approach
Understand a group's culture in its natural setting	Ethnography (participant observation)
Systematically categorise documents or text	Content analysis
Surface shared norms and group interaction	Focus group
Explore a private or sensitive individual account	In-depth interview

Qualitative selector: read the study aim, pick the approach or data-collection method (aim to method).

WORKED EXAMPLE

A team wants to know why patients started on a depot antipsychotic stop attending the clinic. A trial can measure the drop-out rate, but not the reasons. They run in-depth interviews with disengaged patients and two focus groups with community nurses, code the transcripts, and organise the codes into themes (a thematic analysis). They keep interviewing until no new theme appears (saturation), take the draft themes back to a few patients to check they ring true (member checking), and confirm that the interview, the nurse focus groups and the case notes all point to the same barriers (triangulation). The output is not a percentage but a set of transferable themes ("feeling talked down to", "side effects not discussed") that a service can act on, and that can seed a later quantitative study.

HOLD THIS

Qualitative research studies meaning and experience, samples purposively, and stops at saturation; its four trustworthiness criteria are credibility, transferability, dependability and confirmability, and its findings transfer to theory, not to population statistics.

COMMON TRAP

Two errors examiners set. First, treating qualitative findings as if they were statistically generalisable to a population: they are not, they offer transferability to similar contexts, judged from thick description, not statistical generalisation. Second, criticising a qualitative study for a "small, unrepresentative sample": small purposive samples are correct by design, and adequacy is judged by saturation, not by size or representativeness. Judge a qualitative study by its trustworthiness, not by the yardstick of a trial.

MNEMONIC

Trustworthiness = "Can These Data Convince" (Credibility, Transferability, Dependability, Confirmability).

The four Lincoln and Guba criteria map onto the quantitative quartet: Credibility to internal validity, Transferability to external validity, Dependability to reliability, Confirmability to objectivity. "Can These Data Convince" fixes the order and reminds you the whole point is a believable, traceable account.

GOOD TO REMEMBER

- **Grounded theory:** builds theory upward from data; use it to generate a new explanatory model; the one move to know is constant comparison, sampling to theoretical saturation.
- **Thematic analysis:** the flexible framework-light method; use it to find patterns of meaning; the one move is coding the data then grouping codes into themes.
- **Phenomenological:** studies the lived experience and essence of a phenomenon; use it to capture what an experience is like from the inside.
- **Ethnography:** studies a culture or group in its natural setting; use it for immersion and participant observation over time.
- **Content analysis:** systematically categorises text or documents; use it on documentary data; it can be qualitative (meaning) or quantitative (frequency counts).
- **Focus group:** six to ten participants with a moderator; use it when the interaction between members is the data; weak for private or sensitive material.
- **Saturation:** the stopping rule; the point at which no new codes or themes emerge; it, not a pre-set n, sets qualitative sample size.
- **Triangulation:** convergence of multiple sources, methods or investigators on one finding; the number to state is at least two independent sources agreeing.
- **Trustworthiness (Lincoln and Guba):** credibility, transferability, dependability, confirmability; the qualitative parallels of internal validity, external validity, reliability and objectivity.

7 · Systematic review and meta-analysis

This is the apex of the evidence pyramid. A single trial answers one question in one sample; the **systematic review** gathers every trial that ever asked that question and reads them together, and the **meta-analysis** is the statistics that pool their numbers into one estimate. The examiner treats these as the most senior methods in the paper, and the marks sit in two places: the distinction between the reproducible **systematic review** method and the older narrative review, and the interpretation of the forest plot with its diamond, its heterogeneity and its funnel-plot check for publication bias (Shorter Oxford Textbook of Psychiatry; Companion to Psychiatric Studies 2010).

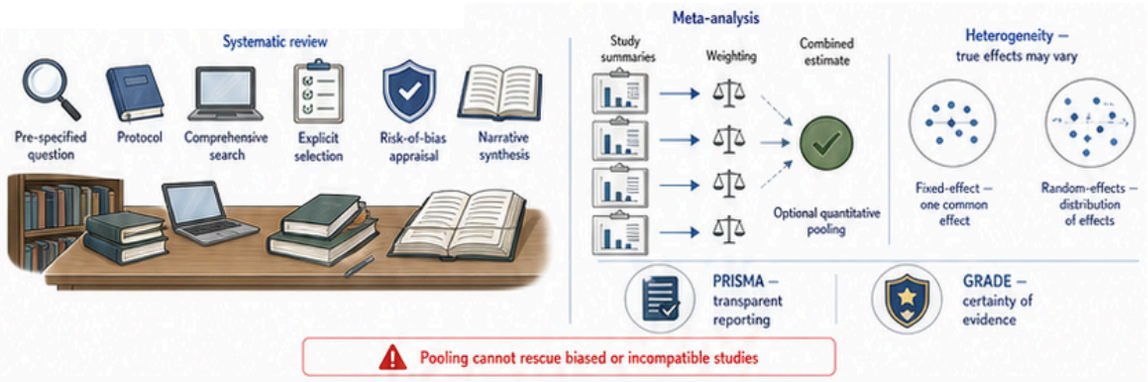


Fig 7.11 Systematic review and meta-analysis.

Read the topic as one pipeline. A protocol fixes the question and the search; a comprehensive search finds the trials; explicit criteria include or exclude them; risk-of-bias appraisal weighs each; and only then, optionally, a meta-analysis pools the results, tests them for heterogeneity, checks the literature for publication bias, and grades the certainty of the whole. The systematic review is the method; the meta-analysis is the arithmetic that may or may not sit inside it (Bland 2015).

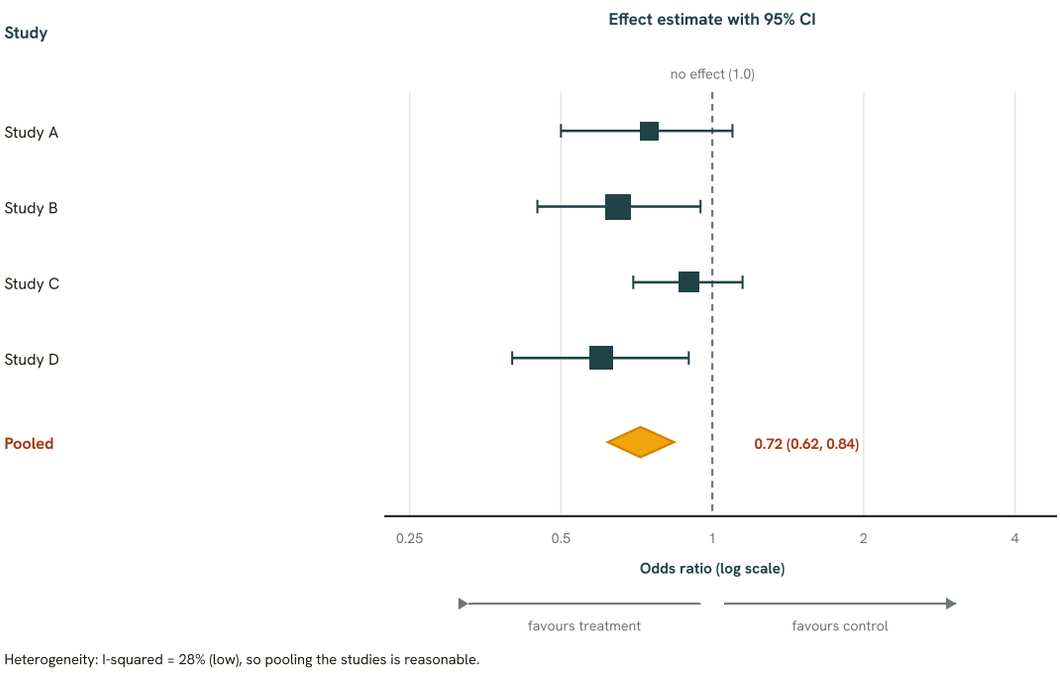


Fig 7.12 Reading a forest plot. Each study is a horizontal line (its 95% confidence interval) with a box marking the point estimate, the box sized by the study's weight in the pool. The vertical dashed line is the value of no effect. A confidence interval that crosses it is non-significant for that study. The diamond at the foot is the pooled estimate, its width the pooled confidence interval. Before trusting the pool, check heterogeneity: a high I-squared means the studies disagree and a single summary may mislead.

7.1 The systematic review: a protocol-driven synthesis, not a narrative review

A systematic review is a reproducible, protocol-driven summary of all the evidence on one question. A systematic review starts from a clearly formulated question, then follows a pre-

registered protocol: a comprehensive and documented search, explicit and pre-stated inclusion and exclusion criteria, formal risk-of-bias (quality) appraisal of each included study, and a transparent synthesis. Because every step is specified in advance and reported, a second team following the same protocol should reach the same set of studies and the same conclusion, which is precisely what makes it reproducible (Shorter Oxford Textbook of Psychiatry). This is the opposite of the traditional **narrative review**, in which an expert selects and weights the literature by personal judgement, so the reader cannot tell what was searched, what was left out, or why. The two-investigator rule, that study selection and quality scoring be done independently by at least two reviewers, exists to keep even the systematic review honest.

RULE **Systematic review = method; meta-analysis = optional statistics.** A systematic review is defined by its protocol (question, search, criteria, appraisal); the pooling is a separate step that it may or may not include.

TRAP **Not every systematic review contains a meta-analysis, and not every pooled analysis is systematic.** If the trials are too clinically diverse to combine, a good systematic review synthesises them narratively and refuses to pool; pooling them anyway is the error.

7.2 The Cochrane Collaboration and the evidence hierarchy

The systematic review of randomised trials sits at the top of the therapy hierarchy. The movement grew from Archibald Cochrane, whose 1972 book argued that treatment provision should rest on randomised controlled trials because they are more reliable than any other evidence (Cochrane 1972). His call for a critical, periodically updated summary of all relevant trials was realised by the Cochrane Collaboration, formed in 1993 and now the largest organisation in the world producing and maintaining systematic reviews (Shorter Oxford Textbook of Psychiatry). In the conventional hierarchy of evidence for questions of therapy, level Ia is a systematic review of randomised controlled trials and level Ib is at least one randomised controlled trial, both above non-randomised controlled studies (II), descriptive studies (III), and expert opinion (IV). The **cochrane** review, with its rigorous protocol and standard software, is the archetype of the method.

INDIA

Cochrane reviews are freely accessible in India through the Cochrane Library and are the standard evidence base cited by Indian clinical practice guidelines. When guidance from a Western meta-analysis is read across to Indian practice, the external-validity question of whether the pooled sample resembles Indian patients still applies, because most pooled trials are from high-income settings.

7.3 Meta-analysis: the statistical pooling, distinguished from the systematic review

Meta-analysis is the quantitative pooling of results across studies into one weighted estimate. A meta-analysis takes the effect estimate from each included study (an odds ratio or relative risk for a binary outcome, a mean difference or standardised effect size for a continuous one) and combines them into a single pooled estimate of greater precision than any one trial

(Shorter Oxford Textbook of Psychiatry). Crucially, it does not weight studies equally: each contributes in proportion to its information, so larger and more precise studies pull the pooled estimate more (the inverse-variance principle). A meta-analysis is therefore a step that lives inside a systematic review, never a substitute for it: pooling numbers gathered without a systematic, unbiased search simply averages a biased sample of the literature. The systematic review supplies the trustworthy set of studies; the meta-analysis does the arithmetic on them.

WORKED EXAMPLE

Three trials of a drug versus placebo report relative risks of relapse of 0.80, 0.70 and 0.75, each with its own confidence interval. A meta-analysis does not average these as (0.80 + 0.70 + 0.75) / 3; it weights each by the inverse of its variance, so the largest, most precise trial dominates, yielding a pooled relative risk near **0.75**, its exact value set by the study weights, with a confidence interval narrower than any single trial. The narrowing of the confidence interval, not a new point estimate, is the gain from pooling.

7.4 Reading the forest plot

The forest plot displays every study, its weight, and the pooled result on one axis. The forest plot is the signature graphic of a meta-analysis, and reading it is a standard viva task (Shorter Oxford Textbook of Psychiatry). Each horizontal line is one study: the central box is its point estimate, and the size of the box marks its weight in the pool (larger box = more precise study, more influence), while the horizontal whiskers are its 95% confidence interval. A vertical **null line** runs down the plot at the value of no effect: **1.0** for a ratio measure (odds ratio, relative risk) and **0** for a difference measure (mean difference). A study whose confidence interval crosses the null line is individually non-significant. At the foot of the plot sits the pooled result as a diamond: its centre is the pooled point estimate and its width is the pooled confidence interval, so if the diamond touches or crosses the null line the overall result is non-significant.

Box (point estimate)

Each study's own effect; the box centre is the estimate, the box size is that study's weight in the pool.

Whiskers

The 95% confidence interval of that single study; if they cross the null line, that study alone is not significant.

Null line

The vertical line of no effect: 1.0 for odds ratio or relative risk, 0 for a mean difference.

Diamond

The pooled estimate at the bottom: centre is the combined effect, width is its confidence interval; crossing the null line means the pooled result is non-significant.

7.5 Fixed-effect versus random-effects models

The choice of model depends on whether the studies share one true effect. The pooling can assume one of two things. A fixed-effect model assumes every study is estimating the same single underlying treatment effect, and all variation between them is sampling error; it weights purely by study size and gives the narrowest, most confident pooled confidence interval. A random-

effects model assumes the true effect genuinely differs from study to study (because of differing doses, patients or settings), so it adds this between-study variance to the weighting; it gives a wider, more conservative confidence interval and lets small studies count for relatively more (Shorter Oxford Textbook of Psychiatry). The decision follows the heterogeneity: when studies are consistent, a fixed-effect model is appropriate and efficient; when significant heterogeneity is present, the random-effects model is the more appropriate and honest choice, and its wider interval reflects the real uncertainty.

RULE **Significant heterogeneity means use a random-effects model.** Fixed-effect assumes one shared true effect; when effects genuinely vary, random-effects widens the interval to tell the truth about that variation.

7.6 Heterogeneity: the chi-square test and the I-squared statistic

Heterogeneity is real variation in the true effect between studies, beyond chance. When some trials show a large effect and others none at all, the studies show heterogeneity, meaning their results are more different than sampling error alone can explain (Shorter Oxford Textbook of Psychiatry). It is assessed two ways. The **chi-square test for heterogeneity** (Cochran's Q, a modification of the chi-square test) formally tests the null hypothesis that all studies share one true effect; because it has low power when trials are few, a lenient threshold such as p less than **0.10** is conventionally used, and a significant result signals heterogeneity. The **I-squared** statistic (written **i-squared** or **i2**) is the more useful companion: it is the percentage of total variation across studies that is due to real heterogeneity rather than chance, running from 0% to 100% and, unlike the chi-square, not dependent on the number of studies. The rough Cochrane bands are worth holding.

0 to 40%	30 to 60%	50 to 90%	75 to 100%
I2: MIGHT NOT BE IMPORTANT	I2: MODERATE	I2: SUBSTANTIAL	I2: CONSIDERABLE

Overlapping I-squared bands from the Cochrane Handbook (Higgins et al 2023); the overlap is deliberate, since the importance of a given value also depends on the direction and strength of the evidence.

TRAP **A tidy pooled diamond over high heterogeneity is misleading.** High i-squared means the studies are answering different questions; the neat pooled estimate then averages apples and oranges and should not be reported as if it were one clean effect.

7.7 Publication bias: the funnel plot and Egger's test

Positive studies are published more, so the pooled literature can overstate the effect. Publication bias is the tendency for statistically significant, positive trials to be submitted and published more readily than null or negative ones, so a meta-analysis restricted to published studies systematically exaggerates the benefit (Shorter Oxford Textbook of Psychiatry). The classic detection tool is the funnel plot: each study's effect estimate is plotted against its precision (or standard error), so small imprecise studies scatter widely at the bottom and large precise ones cluster tightly at the top. With no bias the cloud is a symmetric inverted funnel about the pooled estimate; **funnel plot** asymmetry, a corner of small negative studies missing, suggests that such

studies were run but never published. Because eyeballing asymmetry is subjective, Egger's test provides a formal statistical test of funnel-plot asymmetry (Egger et al 1997). The remedies are structural: trial registries, and searching grey literature and unpublished data.

COMMON TRAP

Funnel-plot asymmetry is not proof of publication bias. Genuine heterogeneity, poorer methodology in smaller trials, or chance can all bend the funnel. Asymmetry is a prompt to investigate, not a verdict; and a funnel plot needs roughly ten or more studies before its shape can be judged at all.

7.8 PRISMA reporting and GRADE certainty

PRISMA standardises the reporting; GRADE rates how much to trust the result. Two frameworks bracket the modern systematic review. The **PRISMA** statement (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) standardises what must be reported about how the review was searched, conducted, analysed and presented, including the flow diagram of records screened, excluded and included (Moher et al 2009). Separately, GRADE (Grading of Recommendations Assessment, Development and Evaluation) rates the certainty of the pooled evidence for each outcome as high, moderate, low or very low. It starts a body of randomised evidence at high certainty and rates it down for five problems, risk of bias, inconsistency (heterogeneity), indirectness, imprecision and publication bias, and can rate observational evidence up for a large effect or dose-response. GRADE is thus the summary judgement on top of the whole pipeline: a meta-analysis can produce a precise diamond and still be graded low certainty if the trials were biased or heterogeneous.

PEARL

Keep the three frameworks in their lanes. PRISMA governs reporting (how the review is written up), the risk-of-bias tool (such as the Cochrane RoB tool) appraises each included trial, and GRADE rates the certainty of the pooled evidence per outcome. A review can be PRISMA-compliant in its reporting yet GRADE-low in its certainty; good reporting of weak evidence is still weak evidence.

7.9 How the pipeline holds together

Each stage protects the next, and the meta-analysis is only as good as the review beneath it. The protocol fixes an unbiased question and search; the comprehensive search and explicit criteria assemble a fair set of studies; risk-of-bias appraisal weighs their quality; the meta-analysis pools them, but only after heterogeneity is checked so that pooling is legitimate; the funnel plot and Egger's test interrogate the literature for missing negative trials; and GRADE delivers the final verdict on certainty. Assess any published meta-analysis by walking this chain: an impressive pooled diamond built on a narrow search, unappraised trials, or ignored heterogeneity is exactly where the exam wants you to point.

HOLD THIS

The systematic review is the reproducible protocol-driven method (question, search, criteria, appraisal); the meta-analysis is the optional inverse-variance pooling inside it, read off a forest plot whose box is each study's weight, whisker is its CI, null line is 1.0 (or 0), and bottom diamond is the pooled estimate; high i-squared means use a random-effects model and distrust a tidy diamond, and a lopsided funnel plot (tested by Egger's) flags publication bias.

GOOD TO REMEMBER

- **Systematic review:** a reproducible synthesis of all evidence on one question; use it whenever a therapy question needs the whole literature, not one trial; defined by its protocol (question, comprehensive search, explicit inclusion, risk-of-bias appraisal), which separates it from a narrative review.
- **Cochrane Collaboration:** the world's largest producer of systematic reviews (formed 1993, from Archibald Cochrane's 1972 argument); the archetype of the method; its review of randomised trials is level Ia, the top of the therapy evidence hierarchy.
- **Meta-analysis:** the statistical pooling of study results into one weighted estimate; use it inside a systematic review when studies are similar enough to combine; weights each study by the inverse of its variance, so the gain is a narrower pooled confidence interval, not a new average.
- **Forest plot:** the display of a meta-analysis; read it to see each study and the pooled result; box = point estimate and weight, whisker = 95% CI, null line = 1.0 for a ratio (0 for a difference), bottom diamond = pooled estimate and its CI.
- **Fixed-effect vs random-effects model:** fixed assumes one shared true effect (narrower CI); random assumes effects vary between studies (wider, more conservative CI); use random-effects when heterogeneity is significant.
- **Heterogeneity (i-squared, chi-square):** real between-study variation beyond chance; the chi-square (Cochran's Q) test flags it at p less than 0.10, and i-squared gives the percentage of variation due to heterogeneity (bands: 0 to 40% low, 30 to 60% moderate, 50 to 90% substantial, 75 to 100% considerable).
- **Publication bias (funnel plot, Egger's test):** positive studies published preferentially, so the pool overstates effect; plot effect against precision, look for an asymmetric funnel (missing small negative studies), and test asymmetry formally with Egger's test; needs roughly ten or more studies.
- **GRADE:** rates the certainty of the pooled evidence per outcome as high, moderate, low or very low; starts randomised evidence high and rates down for risk of bias, inconsistency, indirectness, imprecision and publication bias; the final judgement on top of the whole review.

8 · Reporting guidelines and research ethics

Two examiner favourites sit back to back here: how a finished study should be *reported* so a reader can appraise it, and how a study is allowed to be *done* to human beings in the first place. The first half is a matching game, match the **reporting guideline** to the study design, and it underpins every structured critical appraisal. The second half is the ethical spine, from Nuremberg to the four principles to the consent process to the committee that signs off. Vivas love the crisp discriminators: which checklist for a trial versus a review, which document introduced "respect for persons", why consent is a process and not a signature.

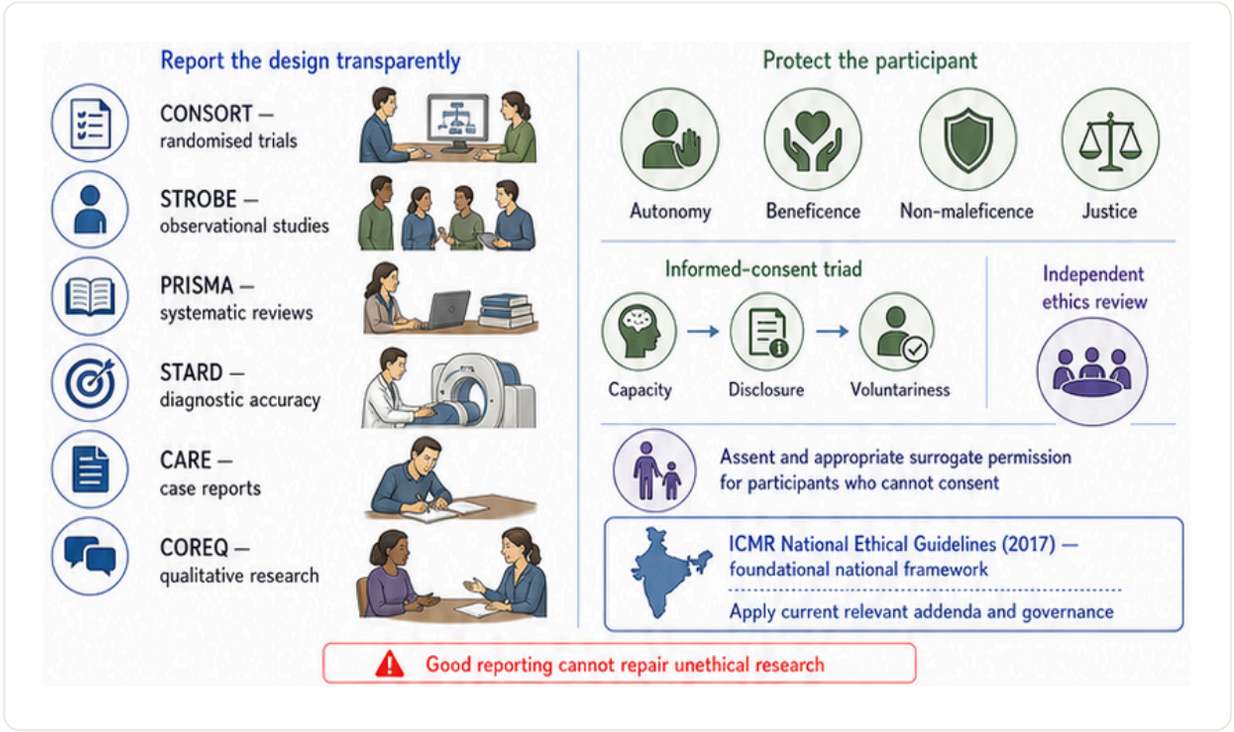


Fig 7.13 Reporting and ethics: transparency does not replace protection.

Frame it as two questions. "Is this paper reported well enough to trust?" is answered by the **reporting guideline** matched to the design. "Was this study ethical to run?" is answered by the historical codes, the four principles, valid **informed consent**, and clearance by an **ethics committee**. Keep them separate and the topic collapses into two short tables.

8.1 Critical appraisal: reading a paper against a checklist

What it is. Critical appraisal is the structured process of judging whether a study's results are valid, what they mean, and whether they apply to your patients (Greenhalgh 2019). It weighs internal validity (was the study done well) against external validity (does it generalise). Reporting guidelines exist so that authors give you every item you need to make that judgement, so a well-reported paper is not automatically a good study, but a badly reported one cannot be appraised at all. This is why a critical appraisal always begins by asking which reporting guideline the design demanded, then checking the paper against it.



Fig 7.14 Critical appraisal: six questions a paper must survive.

RULE **Appraise before you apply.** Judge validity, size of effect, and applicability in that order; a statistically significant result from a biased study still does not change practice.

8.2 Reporting guidelines matched to design

The matching game. Each major design has its own EQUATOR-network checklist. The three you must never confuse: CONSORT for randomised trials, PRISMA for systematic reviews and meta-analyses, and STROBE for observational studies (Schulz et al. 2010; Moher et al. 2009; von Elm et al. 2007). Two more are named constantly alongside them: GRADE is not a reporting checklist but a system for rating the *certainty* of a body of evidence and the strength of a recommendation (Guyatt et al. 2008), and MOOSE is the reporting guideline for meta-analyses of *observational* studies where PRISMA (built for reviews of trials) is a poor fit (Stroup et al. 2000).

GUIDELINE	DESIGN IT REPORTS	ONE-LINE DISCRIMINATOR
consort (CONSORT)	Randomised controlled trials	**Has the flow diagram: enrolled → randomised → analysed**
prisma (PRISMA)	Systematic reviews and meta-analyses (of trials)	**Has the four-phase flow: identified, screened, eligible, included**
strobe (STROBE)	Observational: cohort, case-control, cross-sectional	**Default for any study with no randomisation by the investigator**
moose (MOOSE)	Meta-analyses of observational studies	**Observational meta-analysis, where PRISMA does not fit**
grade (GRADE)	Certainty of a body of evidence + strength of recommendation	**Rates evidence high/moderate/low/very-low, not a reporting checklist**

Match the guideline to the design; CONSORT, PRISMA, STROBE are the three that get swapped in vivas.

RULE **CONSORT for trials, PRISMA for reviews, STROBE for observational.** Name the design first, then the checklist follows automatically.

TRAP **PRISMA is for the review, not the trials in it.** A meta-analysis of RCTs is reported with PRISMA; each included RCT was reported with CONSORT. GRADE then rates the certainty of the pooled evidence. Do not answer "CONSORT" for a meta-analysis.

GOOD TO REMEMBER

- **CONSORT:** reporting guideline for randomised trials; use when appraising an RCT; the signature item is the participant flow diagram (enrolled, randomised, lost, analysed).
- **PRISMA:** reporting guideline for systematic reviews and meta-analyses; use for a pooled synthesis of studies; the signature item is the four-phase study-selection flow diagram.
- **STROBE:** reporting guideline for observational studies; use for cohort, case-control, or cross-sectional designs; the rule is "no investigator-controlled randomisation, use STROBE".
- **MOOSE:** reporting guideline for meta-analyses of observational studies; use when the pooled studies are observational, not trials; distinguishes itself from PRISMA by the observational input.
- **GRADE:** system to rate certainty of evidence and strength of recommendation; use when grading a body of evidence for a guideline; the four certainty levels are high, moderate, low, very low.

8.3 Research ethics: the historical spine

Why the codes exist. Research ethics is a reaction to abuse. Every modern safeguard traces to a documented harm, and examiners want the three landmark documents in order with what each one introduced (Emanuel et al. 2000; Kaplan & Sadock 2024).

Nuremberg Code (1947)

Written after the Nazi doctors' trial; the first international code; its cardinal rule is that voluntary consent of the human subject is absolutely essential.

Declaration of Helsinki (1964, WMA, revised repeatedly)

The physician-authored standard for medical research; introduced the research ethics committee, the written protocol, and the primacy of the participant's interests over those of science and society.

Belmont Report (1979, USA)

Followed the Tuskegee syphilis study; named three foundational principles, respect for persons, beneficence, and justice, and mapped them to consent, risk-benefit assessment, and fair subject selection.

MNEMONIC

Never Have Bad Research

Nuremberg → Helsinki → Belmont, the chronological spine. Nuremberg gave consent, Helsinki gave the ethics committee and protocol, Belmont gave the principles that became the four-principle framework.

8.4 The four principles

The Beauchamp and Childress framework. Biomedical ethics is standardly taught as four principles that must be balanced against each other (Beauchamp & Childress 2019; Kaplan &

Sadock 2024). Belmont named three; the fourth, non-maleficence, is usually split out from beneficence in the clinical version.

Autonomy
Respect for the person's right to make their own informed, voluntary decision; the ground of informed consent and confidentiality.

Beneficence
Act in the participant's or patient's best interest; maximise benefit.

Non-maleficence
"First, do no harm"; minimise and justify risk; favourable risk-benefit ratio.

Justice
Fair distribution of the burdens and benefits of research; do not exploit vulnerable groups nor exclude those who could benefit.

PEARL

The Belmont-to-four-principles map is a classic one-mark question: respect for persons becomes autonomy, beneficence stays beneficence (and yields non-maleficence), and justice stays justice. Belmont's three "applications" (informed consent, risk-benefit assessment, selection of subjects) map onto the same principles.

8.5 Informed consent: capacity, disclosure, voluntariness

The three elements. Valid informed consent requires three things together (Kaplan & Sadock 2024; Appelbaum 2007). **Capacity** (the clinical term; "competence" is the legal, court-determined term) is the ability to communicate a choice, understand the relevant information, appreciate its application to oneself, and reason with it. **Disclosure** means giving the information a reasonable patient (patient-based standard) or reasonable physician (professional standard) would need: nature, purpose, risks, benefits, and alternatives including no treatment. **Voluntariness** means the decision is free of coercion, manipulation, or undue influence.

TRAP **Consent is a process, not a signature.** A signed form with no genuine understanding is not valid consent; consent is ongoing and can be withdrawn at any time. The form documents the process, it does not constitute it.

GOOD TO REMEMBER

- **Capacity:** the clinical ability to decide; four abilities, communicate a choice, understand, appreciate, reason; "capacity" is clinical, "competence" is the legal adjudication.
- **Disclosure:** the information given; must cover nature, risks, benefits, and alternatives including the option of no treatment; standard is reasonable-patient or reasonable-physician.
- **Voluntariness:** a free choice; invalidated by coercion, manipulation, or undue influence.

8.6 Vulnerable groups, surrogate consent, and assent

When the participant cannot consent alone. Some groups cannot give fully autonomous consent and need extra protection: children, those with impaired capacity (intellectual disability, dementia, acute psychosis), prisoners, and the economically or educationally disadvantaged

where consent may not be truly voluntary. Two mechanisms cover them. **Surrogate consent** is proxy consent by a legally authorised representative (a legally acceptable representative or LAR) who decides in the person's best interest when the participant lacks capacity. **Assent** is the affirmative agreement of a minor or a person lacking full capacity who cannot legally consent; the child's assent is sought *in addition to* the parent's or guardian's consent, and a clear refusal (dissent) is generally respected in non-therapeutic research.

RULE **Consent from the guardian, assent from the minor.** A minor cannot legally consent; obtain the guardian's consent and the child's assent, and honour the child's dissent where the research offers no direct benefit.

GOOD TO REMEMBER

- **Surrogate (proxy) consent:** consent given by a legally authorised representative for a participant who lacks capacity; use for adults unable to decide; the standard is the participant's best interest.
- **Assent:** a minor's affirmative agreement alongside guardian consent; use for children and others lacking legal capacity; a documented refusal (dissent) is respected in non-beneficial research.

8.7 Governance: the ethics committee and institutional review board

The gatekeeper. No human research proceeds without prior clearance by an independent committee, called an ethics committee (EC, the common term in the UK, India, and Europe) or an institutional review board (IRB, the US term). Its job is to protect the rights, safety, and wellbeing of participants: it reviews the protocol, the risk-benefit balance, the consent process and documents, compensation, and provisions for vulnerable groups, and it continues to monitor the study through progress reports and adverse-event reporting. A conflicted or poorly reported study is stopped here before a single participant is enrolled.

GOOD TO REMEMBER

- **Ethics committee / institutional review board:** the independent body that reviews and monitors human research; use before enrolment and throughout; its remit is participant rights, safety, and wellbeing (protocol, risk-benefit, consent, vulnerable-group safeguards).

8.8 The Indian frame: ICMR National Ethical Guidelines 2017

The national standard. In India, biomedical and health research on human participants is governed by the ICMR National Ethical Guidelines for Biomedical and Health Research Involving Human Participants, 2017, which replaced the 2006 edition. They mandate prior review by a registered ethics committee, informed consent, additional safeguards for vulnerable participants, and are the reference standard for Indian institutional ethics committees. New Drugs and Clinical Trials Rules 2019 (under CDSCO) add the statutory regulatory layer for drug trials.

INDIA

Cite the ICMR National Ethical Guidelines 2017 for any Indian human-research question; they are the exam's expected answer for ethics-committee review and consent standards in India, superseding the 2006 guidelines. For a drug trial, add the New Drugs and Clinical Trials Rules 2019 (CDSCO). Registration of the ethics committee is mandatory.

8.9 Putting it together in an appraisal

The examiner's synthesis. A complete critical appraisal reads on two axes at once. On the reporting axis: name the design, pick the reporting guideline (CONSORT / PRISMA / STROBE), check the paper against it, and if it is a body of evidence for a recommendation, apply GRADE. On the ethics axis: confirm ethics committee approval, valid informed consent, and appropriate handling of any vulnerable participants. A paper that is beautifully reported but ethically unsound, or ethically clean but unappraisable, fails.

COMMON TRAP

Confusing GRADE with a reporting checklist. GRADE rates the *certainty* of evidence (high, moderate, low, very low) and the strength of a recommendation; it is not the checklist you use to report a single study. Report a single RCT with CONSORT; grade the pooled evidence with GRADE.

HOLD THIS

CONSORT reports randomised trials, PRISMA reports systematic reviews and meta-analyses, STROBE reports observational studies; GRADE rates certainty of evidence, MOOSE reports observational meta-analyses; and informed consent is capacity plus disclosure plus voluntariness, a continuing process and never merely a signature.

Biostatistics

9 · Data types and scales of measurement

Before any statistic is computed, one question decides which analysis is legal: what kind of number is this? The **scales of measurement** are the classification that governs which summary statistic and which statistical test may lawfully be applied to a variable. Every stem that hands you a variable ("tumour stage", "temperature in Celsius", "number of admissions", "diagnosis") is really asking you to name its scale, because the scale, not the researcher's preference, fixes whether you may quote a mean or only a median, and whether a parametric or a non-parametric test is permitted (Bland 2015; Altman 1991; Kaplan & Sadock 2024). Get the scale right and the correct description and test follow almost automatically.

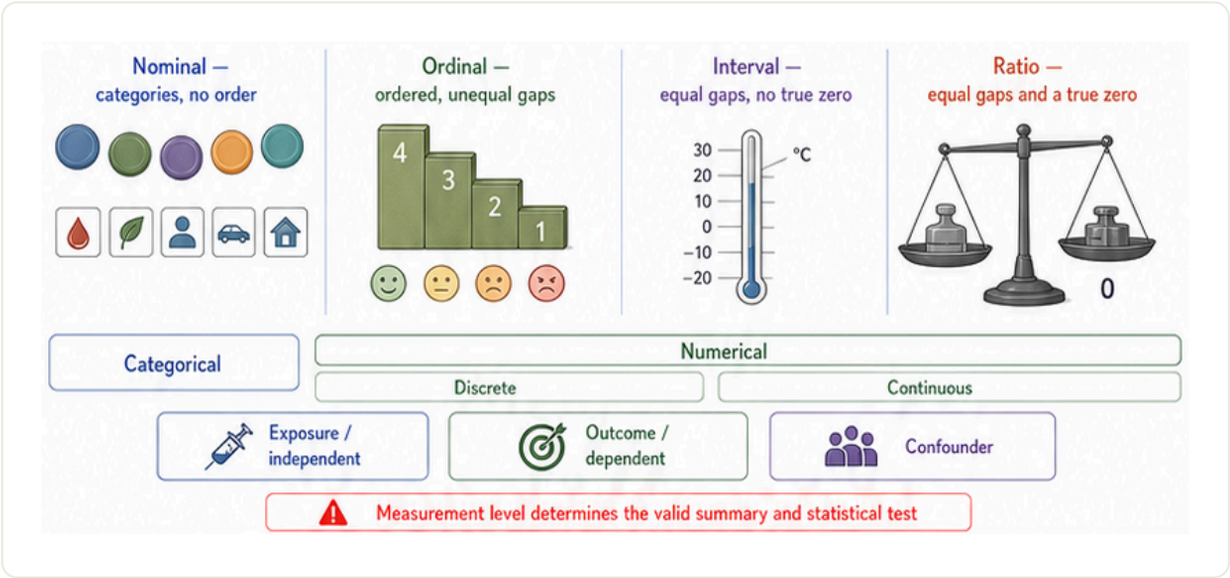


Fig 7.15 Levels of measurement: what can the numbers mean?.

The classification runs on two axes. First, Stevens' four **scales of measurement** in ascending order of information: **nominal**, **ordinal**, **interval**, **ratio** (Stevens 1946). Second, the broader **categorical** versus numerical split, with numerical data further divided into **discrete** and **continuous**. Nominal and ordinal are the two categorical scales; interval and ratio are the two numerical scales. Hold that mapping and the whole topic is scaffolded.

9.1 Why the scale is the first decision, not an abstraction

The scale determines the permitted arithmetic. Distinguishing the **data types** may look like a needless abstraction, but it is essential in fixing the correct method of both data description and analysis (Companion to Psychiatric Studies; Bland 2015). What operations are meaningful climbs with the scale: on a nominal scale you may only count and compare for equality; on an ordinal scale you may additionally rank; on an interval scale you may add and subtract differences because the gaps are equal; on a ratio scale you may also multiply and form ratios because a true zero exists. A mean requires that differences be equal, so it is meaningful only from interval level upward; below that, the median or the mode is the honest centre. This single dependency, arithmetic permitted rises with scale, is the engine behind every choice of statistic and test that follows.

RULE **The measurement scale decides the legal test.** Identify the scale first: it fixes the permissible summary statistic and the permissible test before you look at anything else in the analysis.

9.2 Nominal: named categories with no order

Labels that differ in kind, not in amount. **Nominal** (categorical) data have values that are qualitatively different from one another but carry no inherent order (Companion to Psychiatric Studies). The textbook example is diagnosis: schizophrenia, bipolar disorder and depression are distinct labels, but none is "more" than another, and no arithmetic on the labels is meaningful. Blood group, marital status, sex and the presence or absence of a symptom are all nominal. When a nominal variable has exactly two categories (alive or dead, case or non-case) it is called **dichotomous** or binary. The only legitimate summary is a count or a **proportion**; the only centre

is the mode; and comparisons between groups use the chi-square test (or Fisher's exact test when expected cell counts are small).

9.3 Ordinal: ordered categories with unequal gaps

Rank is real, but the spacing is not. **Ordinal** data have a logical order in their values, but the differences between adjacent values are not necessarily equal and there is no true zero point (Companion to Psychiatric Studies). A single Likert item (strongly disagree, disagree, neutral, agree, strongly agree), tumour stage (I to IV), social class I to V, and a ward observation level all rank the units without promising that the step from stage I to stage II equals the step from stage III to stage IV. Because the gaps are not guaranteed equal, the mean is not strictly meaningful; the correct centre is the **median** with the interquartile range for spread, and group comparisons use **non-parametric** (distribution-free) tests such as the Mann-Whitney U test or the Wilcoxon signed-rank test.

TRAP **Treating an ordinal Likert score as interval without care.** Averaging a single Likert item assumes the psychological distance from "agree" to "strongly agree" equals that from "neutral" to "agree", which is not established; report the median, or justify interval treatment only for a summed multi-item scale.

9.4 Interval: equal gaps, no true zero

Differences are meaningful; ratios are not. **Interval** data have equal differences between values, so the distance between 1 and 2 is the same as the distance between 33 and 34, but the zero is arbitrary rather than absolute (Companion to Psychiatric Studies). The canonical example is temperature in Celsius: 20 degrees is 10 degrees warmer than 10 degrees, yet 20 degrees is not "twice as hot" as 10 degrees, and 0 degrees Celsius does not mean an absence of temperature. Because the zero is not true, sums and differences are valid but ratios are not. Interval data support the **mean** and standard deviation and, given the distributional assumptions, **parametric** tests. Calendar year and IQ score are usually treated as interval.

9.5 Ratio: equal gaps and a true zero

The full arithmetic, ratios included. **Ratio** data share the equal-interval property of interval data and add a true, non-arbitrary zero that marks a complete absence of the quantity (Companion to Psychiatric Studies). Weight, height, age, and number of admissions are ratio: zero admissions genuinely means none, and four admissions are meaningfully twice two. This true zero is what licenses ratios, so "twice as heavy" is a valid statement whereas "twice as hot in Celsius" is not. In practice the interval versus ratio distinction rarely changes the analysis: both are numerical, both are summarised by the mean and standard deviation, and both are analysed with the same parametric tests. That is why many texts pool them as "interval or ratio".

9.6 The categorical versus numerical split, and discrete versus continuous

The higher-level partition that maps onto the four scales. The four scales collapse into two families. **Categorical** data (nominal and ordinal) place units into classes; numerical data (interval and ratio) attach a genuine number. Within numerical data a second cut matters: **discrete** data can take only separate, usually whole-number, values (number of admissions, number of previous

episodes, family size, a count of anything), whereas **continuous** data can in principle take any value on a continuum, limited only by measurement precision (weight, height, serum lithium concentration, reaction time). A count is discrete even though it is ratio-scaled; a measured quantity is continuous. The practical payoff is that discrete-count and continuous data, being numerical, are described by the mean and analysed by parametric methods, while categorical data are described by proportions and analysed by chi-square and its relatives (Companion to Psychiatric Studies).

Categorical
Data sorted into classes; comprises the nominal and ordinal scales; summarised by counts and proportions.

Numerical
Data carrying a true number; comprises the interval and ratio scales; summarised by the mean (or median if skewed).

Discrete
Numerical data limited to separate values, typically whole-number counts (number of admissions).

Continuous
Numerical data able to take any value on a continuum to the limit of measurement precision (weight, concentration).

9.7 Variable roles: independent and dependent, exposure and outcome, confounder

The scale says what a variable is; its role says what it does. A single **variable** can occupy different roles in a study, and naming the role is separate from naming the scale. The independent variable is the presumed cause, the one manipulated or classified on (treatment arm, drug dose); the dependent variable is the measured response that is expected to change with it (symptom score, remission). In epidemiology the same idea is renamed: the exposure is the putative causal factor (a risk factor, a treatment) and the **outcome** is the event or state under study (relapse, mortality). A confounder is a third variable independently associated with both the exposure and the outcome, and not on the causal pathway between them, which distorts the crude exposure-outcome association if left unadjusted (age confounding an association between comorbidity and mortality is the standard example). Roles and scales are orthogonal: an outcome can be nominal (relapsed or not), ordinal (much improved to much worse), or ratio (days to relapse), and the outcome's scale, not its role, still dictates the test.

WORKED EXAMPLE

A trial randomises patients to drug A or drug B and, at 12 weeks, records (i) remission, yes or no, and (ii) the Hamilton depression score. Here the treatment arm is the **independent variable** (nominal, two categories); remission is a **nominal** dependent variable, so groups are compared with a chi-square test on proportions; the Hamilton score is treated as an **interval** dependent variable, so groups are compared with an independent-samples t-test on means. One study, two outcomes, two different tests, chosen entirely by the scale of each outcome. Had the outcome instead been an **ordinal** global-improvement rating, the correct test would switch to the non-parametric Mann-Whitney U test.

9.8 How the scale drives the summary statistic and the test

One scale, one centre, one test family. The chain is direct (Companion to Psychiatric Studies; Bland 2015). **Nominal** data are summarised by proportions and compared with the chi-square test; there is no meaningful measure of central tendency beyond the mode, and no measure of spread. **Ordinal** data are summarised by the median and interquartile range and compared with non-parametric tests. **Interval** and **ratio** data, if reasonably normally distributed, are summarised by the mean and standard deviation and compared with parametric tests (t-test, ANOVA). The scale you can always safely drop *down* to a lower one (treat interval data as ordinal by ranking, or as nominal by categorising) at the cost of throwing away information; you may never lawfully move a variable *up* a scale it does not possess. This is the reason ordinal-treated-as-interval is a genuine error while interval-treated-as-ordinal is merely wasteful.

SCALE	EXAMPLE	CENTRE STATISTIC	TEST FAMILY
Nominal	Diagnosis; blood group; alive or dead	Mode; report as a proportion	**Chi-square (Fisher's exact if cells small)**
Ordinal	Likert item; tumour stage I to IV; social class	Median, interquartile range	**Non-parametric (Mann-Whitney, Wilcoxon)**
Interval	Temperature in Celsius; IQ; calendar year	Mean, standard deviation	**Parametric (t-test, ANOVA)**
Ratio	Weight; age; number of admissions	Mean, standard deviation	**Parametric (t-test, ANOVA)**

The four scales of measurement read across a worked example, the honest measure of central tendency, and the family of tests each scale permits; the hit column is the test-family discriminator that the scale forces on you.

9.9 The statistical-test selector: data type by groups by pairing

Once the scale is fixed, three further questions name the test. Beyond the scale, the correct test for a difference depends on how many groups are compared and whether the observations are paired (the same subjects measured twice, or matched) or unpaired (independent groups). The selector below is the version examined most heavily: read the data type down and the group structure across (Companion to Psychiatric Studies; Altman 1991). Note the clean parallelism, every parametric test on the right has a non-parametric partner on the left, and every categorical row uses a chi-square-family test.

COMPARISON	CATEGORICAL (NOMINAL)	ORDINAL / NON-NORMAL	INTERVAL OR RATIO (NORMAL)
Two groups, unpaired	**Chi-square test**	Mann-Whitney U test	Independent-samples t-test
Two groups, paired	**McNemar test**	Wilcoxon signed-rank test	Paired t-test
Three or more, unpaired	**Chi-square test**	Kruskal-Wallis test	One-way ANOVA
Three or more, paired	**Cochran's Q test**	Friedman test	Repeated-measures ANOVA

COMPARISON	CATEGORICAL (NOMINAL)	ORDINAL / NON-NORMAL	INTERVAL OR RATIO (NORMAL)
Association of two variables	**Chi-square / phi**	Spearman rank correlation	Pearson correlation

The statistical-test selector: data type down, number of groups and pairing across; the hit column is the categorical-data route, and each non-parametric test in the ordinal column is the distribution-free partner of the parametric test to its right.

HOLD THIS

The measurement scale is the first and decisive choice: nominal gives proportions and chi-square, ordinal gives the median and non-parametric tests, interval and ratio give the mean and parametric tests, and you may drop a variable down a scale but never push it up one it does not have.

COMMON TRAP

The commonest measurement error in psychiatry research is running a t-test or reporting a mean on data that are only **ordinal**, above all on single **Likert** items and clinician rating scales, as if the equal-interval property held. A count that looks numerical can also mislead: "number of admissions" is **discrete** and often heavily skewed, so its mean can be a poor centre and a non-parametric test or a count model is safer than a naive t-test.

PEARL

Interval and ratio almost never need to be distinguished in practice: both are numerical, both take the mean and standard deviation, and both are analysed by the same parametric tests, which is why textbooks routinely pool them as "interval or ratio" in their test tables (Companion to Psychiatric Studies). The examinable distinction is one rung lower, categorical (nominal, ordinal) versus numerical (interval, ratio), because that boundary is exactly where parametric analysis becomes permissible.

MNEMONIC

NOIR, in rising order of information: Nominal, Ordinal, Interval, Ratio.

Read left to right you climb from named categories (Nominal) to ranked categories (Ordinal) to equal gaps without a true zero (Interval) to equal gaps with a true zero (Ratio). Each step adds one permitted operation: equality, then order, then addition, then ratios. The categorical break sits between Ordinal and Interval, which is the parametric threshold.

GOOD TO REMEMBER

- **Nominal:** named categories with no order (diagnosis, blood group); use it for qualitative labels; the summary is a proportion and the test is the chi-square test (Fisher's exact when expected cell counts are small).
- **Ordinal:** ordered categories with unequal, unguaranteed gaps and no true zero (Likert item, tumour stage); use it for ranked data; the centre is the median with the interquartile range and the tests are non-parametric (Mann-Whitney, Wilcoxon).
- **Interval:** equal gaps but an arbitrary zero (temperature in Celsius, IQ); use it for numerical data with no true zero; the summary is the mean and standard deviation and the tests are parametric (t-test, ANOVA); differences are valid but ratios are not.
- **Ratio:** equal gaps and a true zero marking absence of the quantity (weight, age, number of admissions); use it for numerical data where "twice as much" is meaningful; summary and tests are the same as interval (mean, SD, parametric), with ratios now also valid.
- **Discrete versus continuous:** discrete numerical data take separate whole-number values (counts of admissions); continuous numerical data take any value on a continuum (weight, concentration); both are numerical, but a skewed count may need a non-parametric test despite being ratio-scaled.
- **Confounder:** a variable independently linked to both exposure and outcome and not on the causal pathway; recognise it whenever a crude association may be distorted by a third factor; it is controlled by stratification, matching, restriction or multivariable adjustment.

10 · Describing data: central tendency, dispersion and the normal curve

Every dataset must first be described before it can be tested. Descriptive statistics answer three questions about a set of numbers: where is its centre, how spread out is it, and what shape does it take. **Central tendency** gives the typical value, **dispersion** gives the scatter around that value, and **shape** (symmetry and peakedness) decides which summary is honest and which inferential test is legitimate. The examiner tests this as a chain: read the data type and shape, pick the correct measure of centre and spread, and only then reach for a test (Companion to Psychiatric Studies; Bland 2015; Altman 1991).

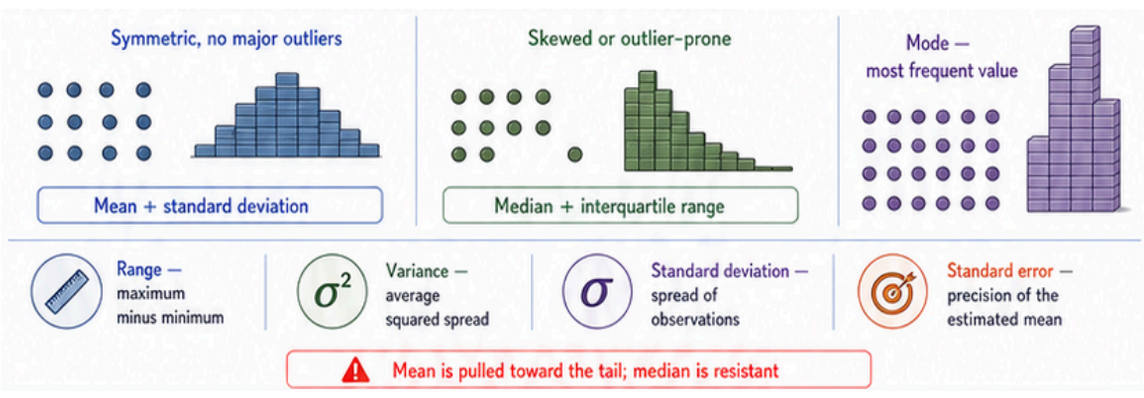


Fig 7.16 Describing data: choose the centre and spread together.

The keystone is the **normal distribution**, the bell-shaped curve on which most parametric statistics rest. Two properties make it examinable: it is fully specified by just its mean and standard deviation, and a fixed, memorisable fraction of the data falls within one, two and three standard deviations of the mean. Master that and a large block of biostatistics becomes

arithmetic (Kaplan & Sadock 2024). The rest of this topic builds the vocabulary of centre, spread and shape, then closes with the probability ideas that feed formal inference.

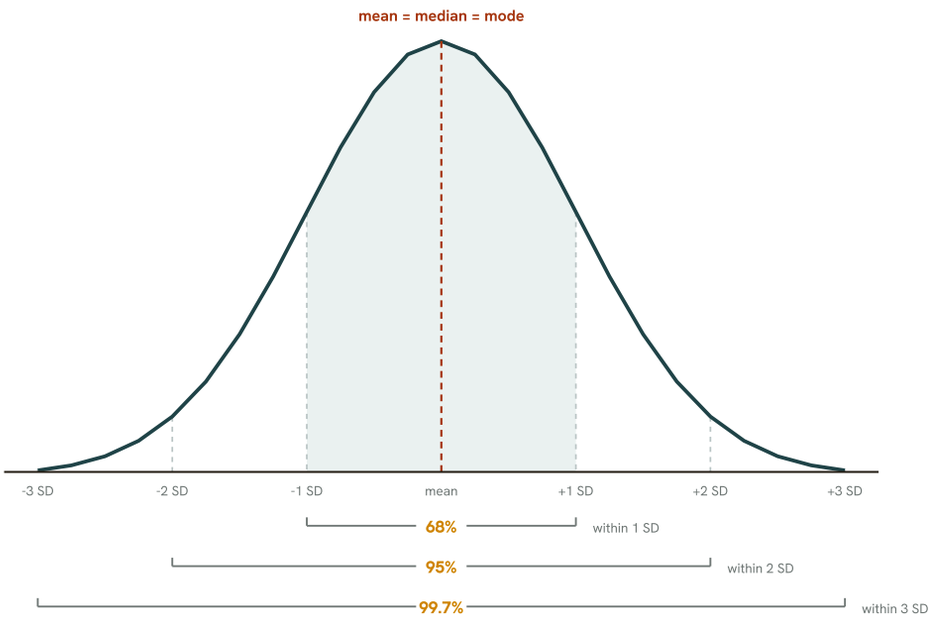


Fig 7.17 The normal curve and the empirical rule. In a normal distribution the mean, median and mode coincide at the peak and the spread is measured in standard deviations. The empirical rule fixes the proportions worth memorising: about 68 percent of observations lie within one SD of the mean, about 95 percent within two, and about 99.7 percent within three. The 95 percent band (more precisely 1.96 SD) is the basis of most confidence intervals and reference ranges.

10.1 Central tendency: the three averages

Central tendency is the single value that best represents a distribution. There are three measures, and the correct one depends on the data type and the shape (Companion to Psychiatric Studies). The mean (the arithmetic average, sum of values divided by n) uses every observation and is the natural summary for symmetric, interval or ratio data. The median (the middle value when the data are ranked, the 50th percentile) splits the data into two equal halves and is the summary of choice for skewed data and for ordinal data. The mode (the most frequently occurring value) is the only measure that can be used for nominal (categorical) data, and it is the one that can be read off a bar chart. A distribution can have one mode (unimodal), two (bimodal), or none.

RULE **Report the median for skewed data.** When a distribution is skewed or the data are ordinal, the median is the honest centre; the mean is right only for symmetric interval or ratio data, and the mode is for nominal data.

10.2 Choosing the centre: mean vs median vs mode

Match the measure to the data. The mean is efficient but not robust: a single extreme value (an outlier) or a long tail pulls it away from the bulk of the data. The median ignores the actual magnitude of extreme values and cares only about rank, which is exactly why it is robust and why

it is preferred for incomes, lengths of stay, symptom counts and any right-skewed clinical variable. The mode is crude for continuous data but indispensable for nominal categories such as diagnosis or blood group, where "average" is otherwise meaningless. The selector table at 10.10 formalises this against data type.

WORKED EXAMPLE

Length of stay for seven patients: 3, 4, 4, 5, 6, 8, 40 days. The mean is (3+4+4+5+6+8+40)/7 = 70/7 = **10** days, which no typical patient experienced. The ranked middle value, the median, is **5** days and describes the group far better. The mode is **4** days (the commonest value). The single long-stay outlier at 40 days has dragged the mean upward, the classic reason to report the median for a right-skewed variable.

10.3 Dispersion: range and interquartile range

Dispersion measures how widely the observations scatter around the centre. The simplest is the range (the difference between the largest and smallest value, or the pair itself). It is quick but fragile, because it depends entirely on the two most extreme observations and grows with sample size. Its robust cousin is the interquartile range, the IQR, which is the distance between the 25th percentile (first quartile, Q1) and the 75th percentile (third quartile, Q3), so it spans the central 50 percent of the data. Because the IQR discards the extreme quarters at each end, it is the natural partner of the median for skewed data, and it is what the box of a box-and-whisker plot draws.

10.4 Dispersion: variance and standard deviation

The variance and standard deviation are the dispersion measures that pair with the mean. The variance is the average of the squared deviations of each value from the mean (Companion to Psychiatric Studies). Squaring removes the sign so that deviations do not cancel, but it leaves the answer in squared units (for example, days squared), which is hard to interpret. Taking the square root of the variance returns to the original units and gives the standard deviation (SD), the single most used measure of spread for symmetric data. A large SD means the data are widely scattered about the mean; a small SD means they cluster tightly. For sample data the variance divides the sum of squared deviations by n minus 1 (Bessel's correction) rather than n, which corrects the tendency of a sample to underestimate the population spread.

HOLD THIS

The mean pairs with the standard deviation (symmetric data); the median pairs with the interquartile range (skewed data). Never a mean with an IQR, never a median with an SD.

10.5 Standard deviation vs standard error of the mean

SD describes the data; the standard error of the mean describes the estimate. This is the single most confused pair in the examination (Companion to Psychiatric Studies). The standard deviation quantifies the spread of the individual observations in your sample and does not shrink as you collect more data (a bigger sample just estimates the true SD more precisely). The standard error of the mean (SEM) is the standard deviation of the sample mean itself, that is, how much

the sample mean would vary from study to study; it equals the SD divided by the square root of n. Because n is in the denominator, the SEM falls as the sample grows, so it is always smaller than the SD. Use the SD to describe how variable the patients are; use the SEM (and the confidence interval built from it) to say how precisely you have pinned down the mean.

TRAP **SEM is not a measure of data spread.** Authors quote the small SEM instead of the SD to make data look tidy; the SEM only reflects precision of the mean and always shrinks with n. To describe the scatter of patients, report the SD.

10.6 The normal distribution and the empirical rule

The normal distribution is the symmetric, bell-shaped curve where mean = median = mode. Also called the gaussian distribution, it is unimodal and perfectly symmetric about its centre, so the three measures of central tendency coincide at the single peak (Companion to Psychiatric Studies; Kaplan & Sadock 2024). It is completely defined by two parameters, the mean (which locates the peak) and the standard deviation (which sets the width). Its key examinable property is the empirical rule (the 68 to 95 to 99.7 rule): for any normal distribution, about **68** percent of observations lie within one SD of the mean, about **95** percent within two SDs, and about **99.7** percent within three SDs. These bands, **68/95/99.7**, are the most reusable numbers in descriptive statistics.

68/95/99.7	**1.96**
PERCENT WITHIN 1, 2, 3 SD	SD FOR CENTRAL 95 PERCENT

10.7 The standard normal and the z-score

Standardising turns any normal value into a position on one universal curve. The standard normal distribution is the special normal distribution with mean 0 and SD 1. Any raw value x from a normal distribution is converted to it by the z-score, z = (x minus mean) / SD, which expresses how many standard deviations the value sits above (positive z) or below (negative z) the mean. A z-score lets you compare values from different scales and read off the proportion of the distribution beyond any point from a single z-table. The empirical rule is just the z-table at round numbers: z = 1, 2 and 3 cut off the 68, 95 and 99.7 percent bands. More precisely, the central 95 percent of a normal distribution lies within **1.96** SDs of the mean (not exactly 2), and 99 percent within 2.58 SDs; the 1.96 value is the one to memorise because it reappears as the multiplier in the 95 percent confidence interval.

WORKED EXAMPLE

IQ is scaled to a normal distribution with mean **100** and SD **15**. A score of 130 has z = (130 minus 100)/15 = 30/15 = **2**, so it sits exactly two SDs above the mean. By the empirical rule 95 percent of people lie within two SDs (between 70 and 130), leaving 5 percent outside; because the curve is symmetric, about 2.5 percent score above 130. Intellectual disability is conventionally set near two SDs below the mean, an IQ around 70, which is the same 2.5 percent tail at the lower end (DSM-5-TR 2022).

10.8 Shape: skew

Skew is asymmetry, and it tells you where the mean has gone. A skewed distribution has one tail longer than the other, and the direction is named for the tail, not the hump. In a right (positive) skew the long tail points to the high values (as in length of stay, income, or reaction times), and the mean is dragged toward that tail so the order becomes mode less than median less than mean. In a left (negative) skew the long tail points to the low values and the order reverses to mean less than median less than mode. This is the mechanism behind the rule to report the median for skewed data: the mean chases the tail while the median stays near the bulk. A quick sign of right skew is a mean noticeably larger than the median.

> **PEARL**
>
> The mean is pulled toward the longer tail; the median resists it. So in right (positive) skew the mean exceeds the median, and in left (negative) skew the mean falls below the median. Comparing mean and median is a one-line skew detector.

10.9 Shape: kurtosis

Kurtosis describes the peakedness and tail-weight of a distribution. Where skew is about asymmetry, kurtosis is about how heavy the tails are and how sharp the peak is relative to a normal curve. A distribution with positive excess kurtosis (leptokurtic) is more sharply peaked with fatter tails, meaning more extreme outliers than a normal distribution predicts; one with negative excess kurtosis (platykurtic) is flatter with thinner tails. The normal distribution is the reference (mesokurtic, excess kurtosis 0). Kurtosis matters because fat tails break the assumptions of tests that presume normality, and heavy-tailed data are better summarised by the median and IQR.

10.10 Selector: which centre and spread for which data

The measure of centre and its matched spread follow directly from data type and shape. This is the practical payoff of the whole topic and a favourite one-mark question. Read the row, name the pair.

DATA / SHAPE	BEST CENTRE	MATCHED SPREAD	WHY
Symmetric interval/ratio	**Mean**	Standard deviation	Uses all data; SD and mean fully describe a normal curve
Skewed continuous	**Median**	Interquartile range	Median and IQR ignore the outlying tail; robust
Ordinal (ranked)	**Median**	Interquartile range / range	Ranks have order but no equal intervals, so no true mean
Nominal (categorical)	**Mode**	(Counts / proportions)	Only the commonest category is meaningful; no arithmetic

Descriptive selector: data type and shape fix the centre, which fixes the matched spread; the hit column is the answer the stem wants.

10.11 Probability: the bridge to inference

Probability turns a described distribution into a basis for inference. A probability is the long-run relative frequency of an outcome, a number from 0 (impossible) to 1 (certain), and the probabilities of all mutually exclusive outcomes sum to 1 (Companion to Psychiatric Studies). Two ideas carry forward. First, for a continuous variable, area under the curve equals probability, which is exactly what the empirical rule computes: the 95 percent within 1.96 SDs is the area under the central slab of the normal curve. Second, two events are independent when the occurrence of one does not change the probability of the other, the assumption behind treating patients as independent observations. These feed the sampling distribution of the mean, the standard error, confidence intervals and hypothesis tests, where descriptive statistics hand over to formal inference.

MNEMONIC

Mean Moves, Median Middles

In a skewed distribution the MEAN MOVES toward the long tail while the MEDIAN holds the MIDDLE. So report the median (and IQR) whenever the data are skewed, and reserve the mean (and SD) for symmetric data.

GOOD TO REMEMBER

- **Mean:** arithmetic average using all values; for symmetric interval/ratio data; = sum of values / n; pairs with the SD.
- **Median:** the middle (50th percentile) ranked value; for skewed or ordinal data; robust to outliers; pairs with the IQR.
- **Mode:** the most frequent value; the only centre valid for nominal data; read off a bar chart.
- **Range:** largest minus smallest value; crude, driven by the two extremes and grows with n.
- **Interquartile range:** Q3 minus Q1 (75th minus 25th percentile); spans the central 50 percent; robust partner of the median.
- **Variance:** mean of squared deviations from the mean; in squared units; sample version divides by n minus 1.
- **Standard deviation:** square root of the variance, back in original units; the spread that pairs with the mean.
- **Standard error of the mean:** SD of the sample mean = SD / square root of n; a precision measure that shrinks with n, always smaller than the SD.
- **Normal distribution (gaussian):** symmetric bell curve, mean = median = mode; fixed by mean and SD; empirical rule 68/95/99.7 percent within 1, 2, 3 SDs.
- **Standard normal / z-score:** normal with mean 0, SD 1; z = (x minus mean) / SD = number of SDs from the mean; central 95 percent lies within 1.96 SDs.
- **Skew:** asymmetry named for the long tail; right/positive tail drags the mean above the median, left/negative below it.
- **Kurtosis:** peakedness and tail-weight vs normal; leptokurtic = peaked, fat tails; platykurtic = flat, thin tails.

11 · Tests of significance: parametric and non-parametric

A significance test asks a single question: is the difference or association we observed likely to be real, or is it plausibly the play of chance? Every test we cover here re-frames the clinical question

as a **null hypothesis** of no difference, computes a test statistic, and returns a p-value, the probability of a result this extreme (or more) if the null were true (Johnstone et al 2010, Bland 2015). The examiner rarely asks you to compute anything by hand. What they test is whether you can **choose the right test** for the data in front of you, and whether you know why the wrong choice is wrong. That choice turns on three things: the scale of your outcome (nominal, ordinal, interval or ratio), how many groups you are comparing, and whether the observations are paired.

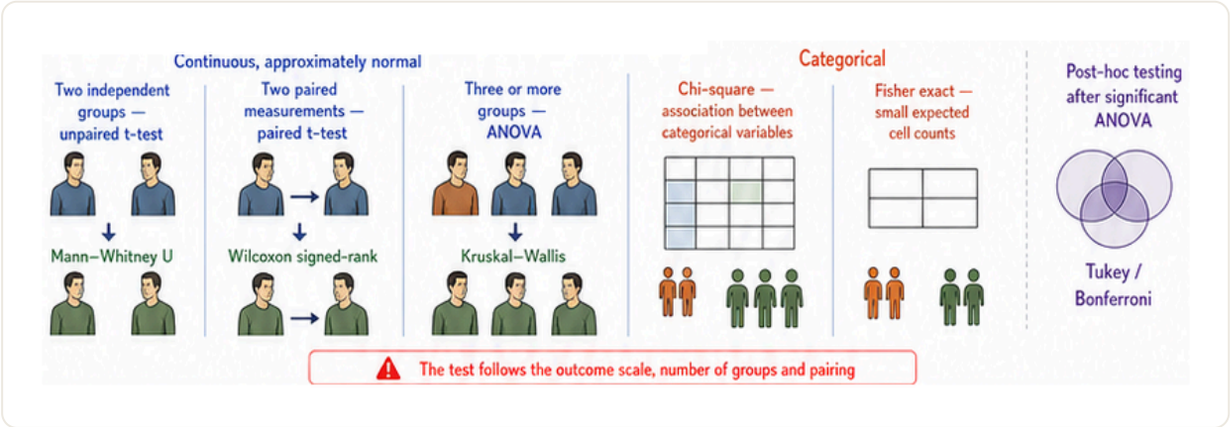


Fig 7.18 Significance tests: match the question, data and pairing.

The great divide is between parametric tests, which assume the data follow a known distribution (usually normal) and estimate population parameters like the mean and variance, and non-parametric tests (also called distribution-free tests), which make no such assumption and work on ranks (Altman 1991, Kaplan & Sadock 2024). Parametric tests are more powerful when their assumptions hold; the non-parametric twins are the safety net for ordinal or non-normal data.

11.1 The three parametric assumptions

What must be true for a parametric test. The correct choice of a parametric test rests on a short list of assumptions (Johnstone et al 2010): the outcome is **interval or ratio** data (continuous, with meaningful equal intervals); each observation is **independent** of the others; the data are approximately **normally distributed**; and the **variance of each group is approximately equal** (homoscedasticity). Meet these and you may use the powerful mean-based tests that use every data value. Fail them badly and you switch to a non-parametric rank-based test, or transform the data (e.g. by taking the log or square root) to restore normality first.

Robustness. A useful caveat: parametric tests are relatively robust to minor departures from normality, contrary to the common misperception that perfect normality is the overriding requirement (Johnstone et al 2010). With a large sample the central limit theorem means sample means tend to normality even when the raw data do not, so a large-sample t-test remains safe even when some assumptions are mildly violated.

RULE **Scale plus groups plus pairing.** Match the test to the data scale, the number of groups, and whether the observations are paired: those three facts pick the test.

11.2 The student t-test: one-sample, two-sample, and paired

The parametric workhorse for one or two groups. The student t-test (use the t-distribution) is one of the most quoted statistics in medicine (Johnstone et al 2010). It comes in three flavours matched to the design:

One-sample t-test

Compares a single sample mean against a known or hypothesised population mean (e.g. mean age of a dementia cohort vs mean age of all elderly inpatients).

Independent two-sample t-test

Compares the means of two independent groups; needs continuous or interval data, approximate normality, and roughly equal variances.

Paired t (paired t-test)

For matched data: the same subjects measured twice (before/after) or individually matched pairs. It analyses the within-pair differences, not the raw group means.

The statistic. In essence, t = observed difference in means / standard error of that difference, with **n1 + n2 – 2** degrees of freedom for the two-sample test. t grows (and significance becomes more likely) as the difference in means widens or as the standard error shrinks with larger samples (Johnstone et al 2010). Where the equal-variance assumption fails there is a modified two-sample t-test (Welch). Always state t and its degrees of freedom.

11.3 ANOVA and the F-test: three or more groups

Why you cannot just run more t-tests. When there are three or more groups you test the null hypothesis that all group means are equal using the analysis of variance (ANOVA). Counter-intuitively, ANOVA answers a question about means by comparing variances (Johnstone et al 2010). It partitions the total variation into the between-groups variance (how far the group means sit from the grand mean) and the within-groups (residual) variance (scatter inside each group), and takes their ratio as the test statistic F-test (the F ratio):

F = variation between groups / variation within groups = MS(between) / MS(residual). If the groups share one population mean, F is near 1; if the group means truly differ, the between-groups term swells and F rises. A significant F says at least one group differs, but not which one.

TRAP **Do not t-test three groups.** Running a t-test on every pair of three groups inflates the type I error (each test carries its own 5% false-positive risk). Use ANOVA for three or more groups.

Extensions. One-way ANOVA handles a single grouping factor; factorial ANOVA handles several factors at once and reveals interactions; repeated-measures ANOVA is the paired extension for the same subjects measured on several occasions; ANCOVA adjusts the group comparison for a confounding covariate such as age or premorbid IQ (Johnstone et al 2010).

11.4 Post-hoc comparisons: Tukey and Bonferroni

Finding which groups differ, safely. A significant ANOVA licenses post-hoc pairwise comparisons to locate the difference, but every extra comparison is another chance for a false positive, so these tests build in a correction (Johnstone et al 2010). The Tukey Honestly

Significant Difference and the Bonferroni adjustment are the two most-quoted. Bonferroni is the simplest and most conservative: to keep the overall false-positive rate at 0.05 across n tests, each individual test must clear a threshold of p = **0.05** / number of tests. For two comparisons that is roughly 0.025; for many comparisons it becomes very stringent, so critics note it can over-correct and inflate the type II error (Perneger 1998).

11.5 Mann-Whitney U: the non-parametric two independent groups test

The rank-based twin of the independent t-test. When the outcome is ordinal, or interval data that badly break normality, and you are comparing two independent groups, use the mann-whitney U test (Johnstone et al 2010). It ranks all observations across both groups combined and compares the rank sums, so it makes no distributional assumption. It assumes only that the two groups are independent, and because it can detect differences in spread as well as in location, report each group's median alongside a note on skew (e.g. a box plot). Always state U.

11.6 Wilcoxon signed-rank: the non-parametric paired test

The rank-based twin of the paired t. Where measurements are paired (two measurements from the same individual, or matched pairs) and the data are ordinal or non-normal, the wilcoxon signed-rank (matched-pairs) test replaces the paired t-test (Johnstone et al 2010). It ranks the within-pair differences by magnitude, attaches the sign, and tests whether the signed ranks balance around zero. Pair Mann-Whitney with independent groups and Wilcoxon with paired data: swapping them is the classic slip.

11.7 Kruskal-Wallis: the non-parametric ANOVA

The rank-based twin of one-way ANOVA. For three or more independent groups on ordinal or non-normal data, the kruskal-wallis test is the non-parametric equivalent of one-way ANOVA (Johnstone et al 2010). It ranks all observations and tests whether the mean ranks differ across groups; a significant result, like a significant F, says at least one group differs and is followed by post-hoc rank comparisons. Its paired counterpart (repeated measures on the same subjects) is the Friedman test.

11.8 Chi-square: association for categorical data

Comparing proportions, not means. For categorical (nominal) outcomes the chi-square (chi square) test of association is the key test (Johnstone et al 2010). It is distribution-free, and it asks whether the proportion with a characteristic differs across two or more independent groups by comparing observed cell counts with the counts expected under the null. The statistic is chi-square = sum of (O − E)squared / E, where O and E are observed and expected frequencies. Two rules the examiner loves: the cells must contain absolute counts, never percentages (percentages falsely inflate the sample size); and you must state chi-square with its degrees of freedom. The paired analogue for a 2x2 table (same subjects, before/after) is the McNemar test.

11.9 Fisher exact: the small-cell rescue

When the chi-square approximation breaks. The chi-square distribution is an approximation that fails when expected cell counts are small. The classic rule: when any expected frequency falls below **5**, use the fisher exact test (or Yates's continuity correction) instead of the ordinary chi-

square (Johnstone et al 2010). Fisher's exact computes the exact probability of the observed table and is more conservative, so it is less likely to reject the null wrongly when numbers are tiny.

0.05	< 5	n−1
ALPHA THRESHOLD	EXPECTED CELL TO FISHER	DEGREES OF FREEDOM IDEA

11.10 The test-map: pairing each parametric test with its non-parametric twin

The one table to hold. Read across data type, number of groups, and pairing to land on the correct test. This is the selector the examiner is really testing (Johnstone et al 2010, Altman 1991).

GROUPS & PAIRING	CATEGORICAL (NOMINAL)	ORDINAL / NON-NORMAL	INTERVAL OR RATIO (PARAMETRIC)
One sample vs known value	Chi-square goodness-of-fit	Sign test	**One-sample t-test**
Two groups, independent (unpaired)	Chi-square test of association	Mann-Whitney U	**Independent two-sample t-test**
Two groups, paired / matched	McNemar test	Wilcoxon signed-rank	**Paired t-test**
Three or more groups, independent	Chi-square test	Kruskal-Wallis	**ANOVA (one-way), F-test**
Three or more groups, repeated / paired	Cochran's Q	Friedman test	**Repeated-measures ANOVA**
Association between two variables	Chi-square / phi coefficient	Spearman rank correlation	**Pearson correlation**

Every parametric test in the right-hand column has a rank-based non-parametric twin to its left; small-cell categorical data drop to the Fisher exact test.

11.11 Worked reads: a chi-square 2x2 and an ANOVA F

WORKED EXAMPLE

Chi-square, 2x2. Sex distribution is compared between 60 people with schizophrenia and 60 healthy controls (Johnstone et al 2010). Expected counts under no difference are 30 per cell. Summing (O − E)squared / E across the four cells gives chi-square = 1.2 on 1 degree of freedom, p = 0.27. Because 0.27 far exceeds alpha 0.05, the null of no difference in the population proportion cannot be rejected: the sexes are not shown to differ.

ANOVA F. Ages of three diagnostic groups of four patients are compared (Johnstone et al 2010). The between-groups sum of squares is 1711.5 (2 degrees of freedom) and the within-groups (residual) sum of squares is 648.5 (9 degrees of freedom). Dividing each by its degrees of freedom gives mean squares of 855.75 and 72.1, so F = 855.75 / 72.1 = 11.9, p = 0.003. Since 0.003 is below 0.05 we reject the null: at least one group mean differs, and post-hoc tests would then locate which.

COMMON TRAP

Feeding percentages into a chi-square test instead of raw counts falsely inflates the sample size and manufactures spurious significance; chi-square needs absolute frequencies. Equally, treating an ordinal scale (e.g. a 1-to-6 social-class rank) as interval data and running a t-test is a scale error: use Mann-Whitney or Kruskal-Wallis instead.

MNEMONIC

MWWK guards the non-parametric gate: Mann, Wilcoxon, Whitney's cousin, Kruskal.

Two independent groups to **Mann**-Whitney; two paired to **Wilcoxon**; three or more independent to **Kruskal**-Wallis. Their parametric partners are the independent t-test, the paired t-test, and ANOVA in the same order.

HOLD THIS

Match the test to data scale x number of groups x paired?: t-test twin is Mann-Whitney (2 independent) or Wilcoxon (paired), ANOVA twin is Kruskal-Wallis (3+), and categorical goes to chi-square, dropping to Fisher exact when any expected cell is under 5.

GOOD TO REMEMBER

- **Student t-test:** parametric test of means for one or two groups; use when data are interval/ratio, roughly normal, with equal variance; two-sample t has n1 + n2 − 2 degrees of freedom.
- **Paired t-test:** parametric test for matched/before-after data; use when the same subjects are measured twice; it tests the mean of the within-pair differences.
- **ANOVA (analysis of variance):** parametric test for three or more group means; use to avoid multiple t-tests; its statistic is F = MS(between) / MS(residual).
- **F-test:** the ratio of between-groups to within-groups variance inside ANOVA; use to test whether group means are equal; F near 1 supports the null.
- **Post-hoc (Tukey, Bonferroni):** follow a significant ANOVA to find which groups differ; use to control the family-wise error; Bonferroni threshold = 0.05 / number of tests.
- **Mann-Whitney U:** non-parametric test for two independent groups on ordinal/non-normal data; use as the t-test's rank-based twin; report the group medians.
- **Wilcoxon signed-rank:** non-parametric test for paired ordinal/non-normal data; use as the paired-t twin; it ranks the signed within-pair differences.
- **Kruskal-Wallis:** non-parametric test for three or more independent groups; use as the one-way ANOVA twin; the paired counterpart is the Friedman test.
- **Chi-square (chi square) test of association:** non-parametric test comparing proportions across categorical groups; use on absolute counts, never percentages; statistic = sum of (O − E)squared / E, stated with its degrees of freedom.
- **Fisher exact test:** the small-cell rescue for a 2x2 table; use when any expected cell frequency is under 5; it gives the exact, more conservative probability.

For test-selection detail and worked derivations see Altman (1991) and Bland (2015). Reliability and validity of a psychometric instrument itself (as distinct from the reliability of a study) are covered in the Psychometry, Assessment and Descriptive Psychopathology notes; the disorder-specific applications of these tests appear in the clinical chapters of Paper II.

12 · Correlation, regression and factor analysis

Once two or more variables are in play, three questions dominate the stems: do they move together, can one predict another, and do many items collapse into a few? This topic covers the three workhorse techniques of association and prediction: **correlation** quantifies how strongly two variables move together, **regression** models and predicts one variable from others (and adjusts for confounders), and **factor analysis** reduces many correlated items to a few underlying latent dimensions (Companion to Psychiatric Studies; Bland 2015; Altman 1991). The examinable spine is simple: correlation measures, regression predicts and adjusts, factor analysis simplifies, and none of the three, on its own, proves cause.

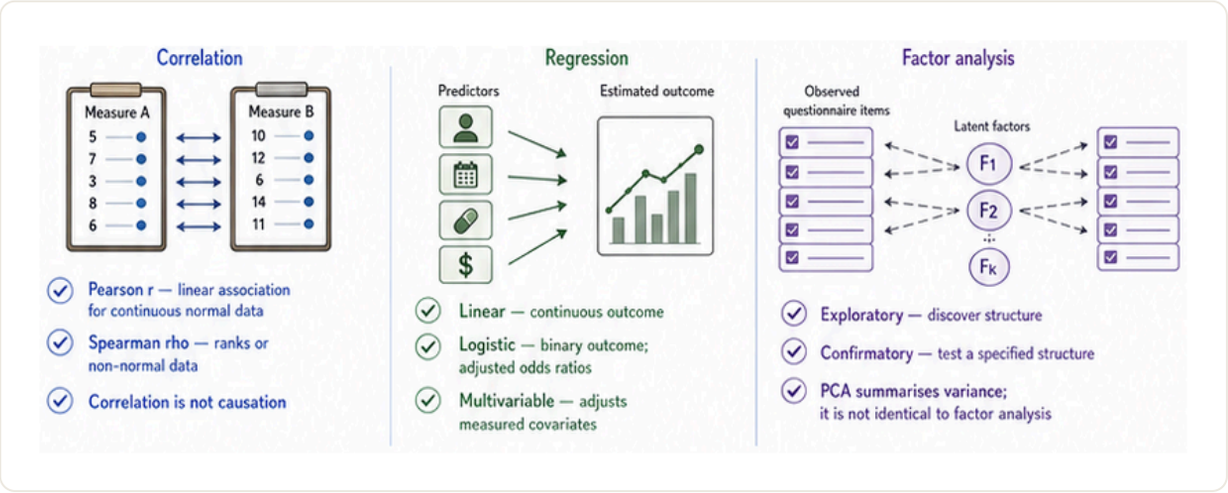


Fig 7.19 Association, prediction and latent structure.

The single most tested idea across the whole cluster is the divorce of statistical association from causation: a strong correlation, a steep regression slope, or a high coefficient of determination each describes the data at hand but licenses no causal claim without a design that supports one (Tasman 2015; Kaplan & Sadock 2024). Hold that caveat and the traps in this area mostly disarm themselves.

12.1 Correlation: measuring linear association between two variables

A single number for how tightly two variables track. A correlation coefficient summarises the strength and direction of the linear relationship between two variables in one number that runs from **-1** through **0** to **+1** (Bland 2015; Altman 1991). A coefficient of **+1** is a perfect positive linear relationship (as one rises the other rises exactly in step), **-1** is a perfect negative linear relationship, and **0** means no linear relationship at all. The sign gives the direction and the magnitude gives the strength; a value near zero does not exclude a strong non-linear (for example U-shaped) relationship, because the coefficient only captures the linear component. Correlation is symmetric: the correlation of x with y equals the correlation of y with x, which is exactly why it can never, by itself, say which variable is cause and which is effect.

12.2 Pearson r: the coefficient for two continuous, normal variables

The parametric correlation, for continuous normally distributed data. The pearson product-moment correlation coefficient, written r, is the correlation used when both variables are

continuous and approximately normally distributed and their relationship is linear (Bland 2015; Altman 1991). It is the default numerical-versus-numerical association measure and the parametric member of the correlation family, the natural partner of the t-test and ANOVA. Being parametric, it is sensitive to outliers and assumes a straight-line relationship; a single extreme point can inflate or collapse it, and a curved relationship can hide behind a near-zero r. Its square, r-squared, carries a direct interpretation covered below.

RULE **Match the correlation to the data.** Two continuous normal variables in a linear relationship take pearson r; ordinal or non-normal (or outlier-ridden) data take the spearman rank correlation.

12.3 Spearman rank correlation: for ordinal or non-normal data

The non-parametric correlation, computed on ranks. The spearman rank correlation coefficient (rho) is the distribution-free counterpart of pearson r: it is applied when the data are ordinal, when a continuous variable is markedly non-normal or skewed, or when outliers make a rank-based measure safer (Bland 2015; Companion to Psychiatric Studies). It is simply the pearson correlation computed on the ranks rather than the raw values, so it captures any monotonic (consistently increasing or decreasing) relationship, not only a strictly linear one, and it shares the same -1 to +1 range and the same sign convention. Because it works on ranks it is robust to outliers and makes no normality assumption, at the modest cost of discarding the exact spacing of the values.

WORKED EXAMPLE

A study reports that clinician-rated illness severity (a five-point ordinal scale) and number of prior admissions (a skewed count) rise together, with a coefficient of **0.62**. Because one variable is ordinal and the other is a skewed count, pearson r is inappropriate and the **spearman** rank correlation is the correct choice; rho of 0.62 says the ranks move together moderately and positively. Note what it does not say: it does not establish that more admissions cause greater severity, nor the reverse, because a third factor (chronicity, poor adherence) could drive both. The coefficient describes the monotonic association and nothing more.

12.4 Correlation is not causation

The caveat that governs the entire topic. A correlation, however strong, cannot by itself be logically read as evidence that one variable causes the other; the non-equivalence of correlation and causation applies with full force (Tasman 2015). Two variables can correlate because one causes the other, because the other causes the one, because a third variable (a **confounder**) causes both, or by chance. The classic pattern is confounding: ice-cream sales correlate with drowning deaths, but summer heat drives both. Reverse causation and selection effects produce the same trap. Establishing causation needs a design that controls these alternatives (a randomised trial, or at minimum multivariable adjustment plus the Bradford Hill considerations), never a correlation coefficient on its own.

TRAP **Reading a strong r, slope, or r-squared as proof of cause.** Association is not causation; a confounder, reverse causation, or chance can generate any of them without a causal link between the two variables.

12.5 Linear regression: predicting a continuous outcome from a line

From describing association to predicting a value. Where correlation gives a symmetric strength-of-association number, linear regression fits an asymmetric predictive model: it estimates the straight line that best predicts a continuous outcome (the dependent variable, y) from one or more predictors (independent variables, x), by minimising the squared vertical distances of the points from the line (least squares) (Bland 2015; Altman 1991). The fitted line has the form y equals intercept plus slope times x. The **slope** (regression coefficient, b) is the heart of the output: it states how many units the outcome changes for each one-unit rise in the predictor, and its sign gives the direction. Unlike correlation, regression is directional, it names a predictor and an outcome, which is why the stem's phrasing ("predict", "for each unit increase") signals regression rather than correlation.

12.6 The coefficient of determination: r-squared as variance explained

The proportion of the outcome's variability the model explains. The coefficient of determination, written **r-squared**, is the square of the correlation coefficient and gives the proportion of the total variability in the outcome that is predictable from, or accounted for by, the predictor(s) (Tasman 2015; Bland 2015). It runs from **0** to **1** (often quoted as a percentage): an r of **0.5** gives an r-squared of **0.25**, meaning **25%** of the variance in the outcome is explained by the predictor and the remaining 75% is unexplained. It is the standard effect-size and goodness-of-fit measure for a linear model. The critical exam point is that a high r-squared measures only how much variance the model captures; it says nothing about whether the relationship is causal.

-1 to +1	0 to 1	0.25
CORRELATION RANGE (R, RHO)	R-SQUARED RANGE	R-SQUARED WHEN R = 0.5

12.7 Logistic regression: predicting a binary outcome, yielding adjusted odds ratios

The regression for yes/no outcomes. When the outcome is binary (relapsed or not, dead or alive, case or non-case) a straight line is inappropriate, and logistic regression is used instead: it models the log-odds of the outcome as a linear function of the predictors (Bland 2015; Kaplan & Sadock 2024). Its coefficients, once exponentiated, yield odds ratios, and because several predictors sit in one model, each odds ratio is *adjusted* for the others in the model. An adjusted odds ratio therefore estimates the effect of one predictor on the odds of the outcome while holding the remaining predictors constant, which is precisely how confounding is handled in observational analyses. An odds ratio of **1** means no association; above 1 means increased odds; below 1 means decreased odds.

12.8 Multivariable regression: adjusting for confounders

Putting several predictors in one model to isolate an effect. The shared power of the regression family is that entering more than one predictor at once gives a **multivariable** model

in which each coefficient is estimated with the others held constant, statistically adjusting the exposure-outcome association for measured confounders (Bland 2015; Altman 1991). This is the analytic answer to confounding introduced in the data-types and validity material: rather than adjusting by design (randomisation, matching, restriction), multivariable regression adjusts in the analysis. Linear regression does this for a continuous outcome, giving adjusted slopes; logistic regression does it for a binary outcome, giving adjusted odds ratios; and the parallel Cox model does it for time-to-event outcomes, giving adjusted hazard ratios. The unadjusted (crude) estimate becomes the adjusted estimate once confounders enter the model.

PEARL

The word that tells you which member of the regression family is meant is the *outcome*. A continuous outcome (symptom score, blood level) means **linear regression** and a slope; a binary outcome (relapse yes/no) means **logistic regression** and an adjusted odds ratio; a time-to-event outcome (time to relapse) means Cox regression and an adjusted hazard ratio. Same logic of adjustment, three outcome types, three models.

12.9 Factor analysis: reducing many correlated items to a few latent factors

Data reduction for scale construction. Factor analysis is a multivariate technique that identifies a small set of underlying, unobserved dimensions (latent factors) that explain the pattern of intercorrelations among a larger set of measured items (Tasman 2015; Kaplan & Sadock 2024). Its starting point is the correlation matrix of all item pairs; from that matrix it extracts factors, each a weighted combination of items that hang together. The paradigm use in psychiatry is scale construction and validation: nine anxiety items might collapse to two factors (say, somatic and cognitive anxiety), giving a more parsimonious measure. The technique underpins the trait structure of personality questionnaires, where extraversion, neuroticism and psychoticism emerge as factors reproducible across cultures (Companion to Psychiatric Studies). Test-construction reliability and validity concepts sit alongside this in the Psychometry, Assessment and Descriptive Psychopathology notes.

12.10 Exploratory versus confirmatory factor analysis

Discovering structure versus testing a hypothesised one. Exploratory factor analysis is used when there is no firm prior model: it lets the data reveal how many factors exist and which items load on which, and is the tool for the first pass at a new scale (Tasman 2015). Confirmatory factor analysis is used later, when a specific factor structure is already hypothesised (from theory or from prior exploratory work): it tests how well a pre-specified model fits new data, reporting goodness-of-fit indices, and is a form of structural equation modelling. The examinable contrast is direction of reasoning: exploratory factor analysis generates the structure, confirmatory factor analysis tests a structure that is already specified.

12.11 Eigenvalues, the Kaiser criterion, loadings and the scree plot

The machinery for deciding how many factors to keep and what they mean. An eigenvalue is the amount of total variance in the item set that a given factor accounts for; larger eigenvalues mark more important factors. The commonest retention rule is the **Kaiser criterion**: retain only

factors with an eigenvalue greater than **1**, on the logic that a retained factor should explain more variance than a single original item (Tasman 2015). The scree plot graphs eigenvalues in descending order; the factors above the "elbow" (the point where the curve levels into scree) are retained. A factor loading is the correlation between an item and a factor, and is how factors are interpreted: an item with a high loading defines that factor, and rotation (commonly varimax) is applied to sharpen the pattern so that each item loads highly on one factor and negligibly on the rest. Determining the number of factors is a matter of judgement, not an automatic output.

12.12 Principal component analysis and its relation to factor analysis

The close cousin, and how it differs. Principal component analysis is the related and often-conflated data-reduction method that repackages a set of correlated variables into a smaller set of uncorrelated components ordered by the variance each explains (Tasman 2015). In the factor-analysis workflow, using a communality estimate of 100% (retaining 1.0 on the diagonal of the correlation matrix) makes the extraction principal components. The conceptual difference is what each seeks: principal component analysis is purely descriptive and maximises variance explained, treating components as summaries of the observed variables, whereas true factor analysis (for example principal-factors extraction) posits latent factors that *cause* the observed correlations and separates common variance from item-specific and error variance. In practice they often give similar solutions, and principal component analysis is frequently the default first step.

RULE **Correlation quantifies association; regression predicts and adjusts.** Reach for a correlation coefficient to measure how two variables move together, and for regression to predict an outcome and to adjust for confounders.

TECHNIQUE	WHAT IT DOES	OUTCOME / DATA	KEY OUTPUT NUMBER
Pearson r	Linear association, two variables	Both continuous, normal	**r, from -1 to +1**
Spearman rho	Monotonic association on ranks	Ordinal or non-normal	**rho, from -1 to +1**
Linear regression	Predict a continuous outcome; adjust	Continuous outcome	**Slope (b); r-squared**
Logistic regression	Predict a binary outcome; adjust	Binary outcome	**Adjusted odds ratio**
Factor analysis	Reduce items to latent factors	Many correlated items	**Eigenvalue; factor loading**

The association-and-prediction selector: read the technique down, and let the data type and the question (measure, predict, or reduce) fix the choice; the hit column is the signature output number each technique returns.

HOLD THIS

Correlation measures association (pearson r for continuous normal data, spearman rho for ordinal or non-normal, both running -1 to +1); regression predicts and adjusts (linear regression for a continuous outcome with a slope and r-squared as variance explained, logistic regression for a binary outcome with adjusted odds ratios); factor analysis reduces many correlated items to a few latent factors (eigenvalue greater than or equal to 1 by the Kaiser criterion, interpreted through factor loadings); and none of them, alone, proves causation.

COMMON TRAP

The flagship error is reading a high r-squared (or a strong r, or a steep slope) as proof of causation: the coefficient of determination only states how much of the outcome's variance the model explains, not why, and a confounder or reverse causation can produce a large r-squared with no causal link. A second, quieter trap is applying pearson r to ordinal or skewed data where the spearman rank correlation is required, or reading a near-zero pearson r as "no relationship" when the true relationship is strong but curved.

MNEMONIC

Correlation Counts, Regression Reaches, Factor analysis Folds.

Correlation *counts* the association between two variables (one number, symmetric, no direction of cause). Regression *reaches* forward to predict an outcome and to adjust for confounders (directional, predictor to outcome). Factor analysis *folds* many correlated items into a few latent factors (data reduction for scale building).

GOOD TO REMEMBER

- **Pearson r:** the correlation coefficient for two continuous, approximately normal variables in a linear relationship; use it as the default numerical-versus-numerical association measure; r runs from -1 to +1 and its square is r-squared.
- **Spearman rank correlation (rho):** the non-parametric correlation for ordinal or non-normal data, computed on ranks; use it when data are ordinal, skewed, or outlier-prone; captures any monotonic relationship and also runs from -1 to +1.
- **Linear regression:** predicts a continuous outcome from predictors by a best-fit least-squares line; use it to predict a value or to adjust for confounders; the slope (b) gives the change in outcome per one-unit rise in the predictor, and r-squared gives the proportion of outcome variance explained.
- **Coefficient of determination (r-squared):** the square of the correlation, the fraction of outcome variance the model explains; use it as the goodness-of-fit and effect-size measure; runs 0 to 1, so r of 0.5 gives r-squared of 0.25, that is 25% of variance explained.
- **Logistic regression:** predicts a binary outcome by modelling its log-odds; use it when the outcome is yes/no; its exponentiated coefficients are adjusted odds ratios, an odds ratio of 1 meaning no association.
- **Multivariable regression:** any regression with several predictors entered together; use it to adjust the exposure-outcome estimate for measured confounders in the analysis; each coefficient is estimated with the others held constant, turning a crude estimate into an adjusted one.
- **Factor analysis:** reduces many correlated items to a few latent factors from their correlation matrix; use it for scale construction and validation; retain factors with an eigenvalue greater than or equal to 1 (Kaiser criterion) and interpret each factor through its factor loadings (the item-factor correlations).
- **Exploratory factor analysis versus confirmatory factor analysis:** exploratory factor analysis lets the data reveal the number and structure of factors (new scale, no prior model); confirmatory factor analysis tests a pre-specified factor structure against new data (a form of structural equation modelling) and reports fit.
- **Principal component analysis:** a data-reduction method that repackages correlated variables into uncorrelated components ranked by variance explained; use it as a descriptive first pass or when maximising variance summarised is the goal; it differs from true factor analysis in seeking components that summarise, not latent factors that cause, the observed correlations.

13 · Diagnostic test statistics

Every screening or diagnostic test makes two kinds of promise and two kinds of mistake, and the whole apparatus of test statistics is a way of pinning numbers to those promises and mistakes. It is built on one small object: the **2x2 table** that cross-tabulates the true state of the world (disease present or absent, fixed by a reference or gold standard) against what the test said (positive or negative). The examiner reliably asks you to read that table, to compute an index from it, and above all to know which indices are intrinsic to the test and which shift with the population you apply it to (Kaplan & Sadock 2024, Bland 2015). Get that last distinction wrong and you will over-trust a positive screen in a low-risk clinic, the single most common error in this whole area.

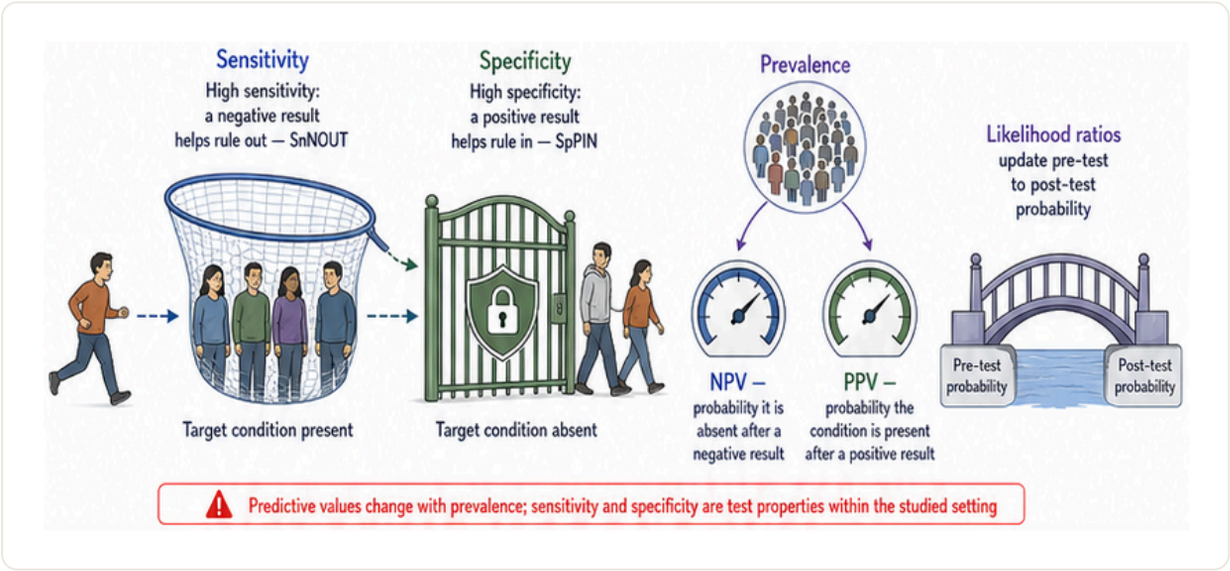


Fig 7.20 Diagnostic tests: from detection to post-test probability.

The four cells carry standard labels (Kaplan & Sadock 2024). The test correctly flags a diseased person as a **true positive** (cell a) and correctly clears a healthy person as a **true negative** (cell d). It errs two ways: a **false positive** (cell b) labels a healthy person diseased, and a **false negative** (cell c) misses a diseased person. Read the table two ways: down the columns (given the true state, how did the test perform?) gives sensitivity and specificity; across the rows (given the test result, what is the true state?) gives the predictive values. That change of direction is the heart of the topic.

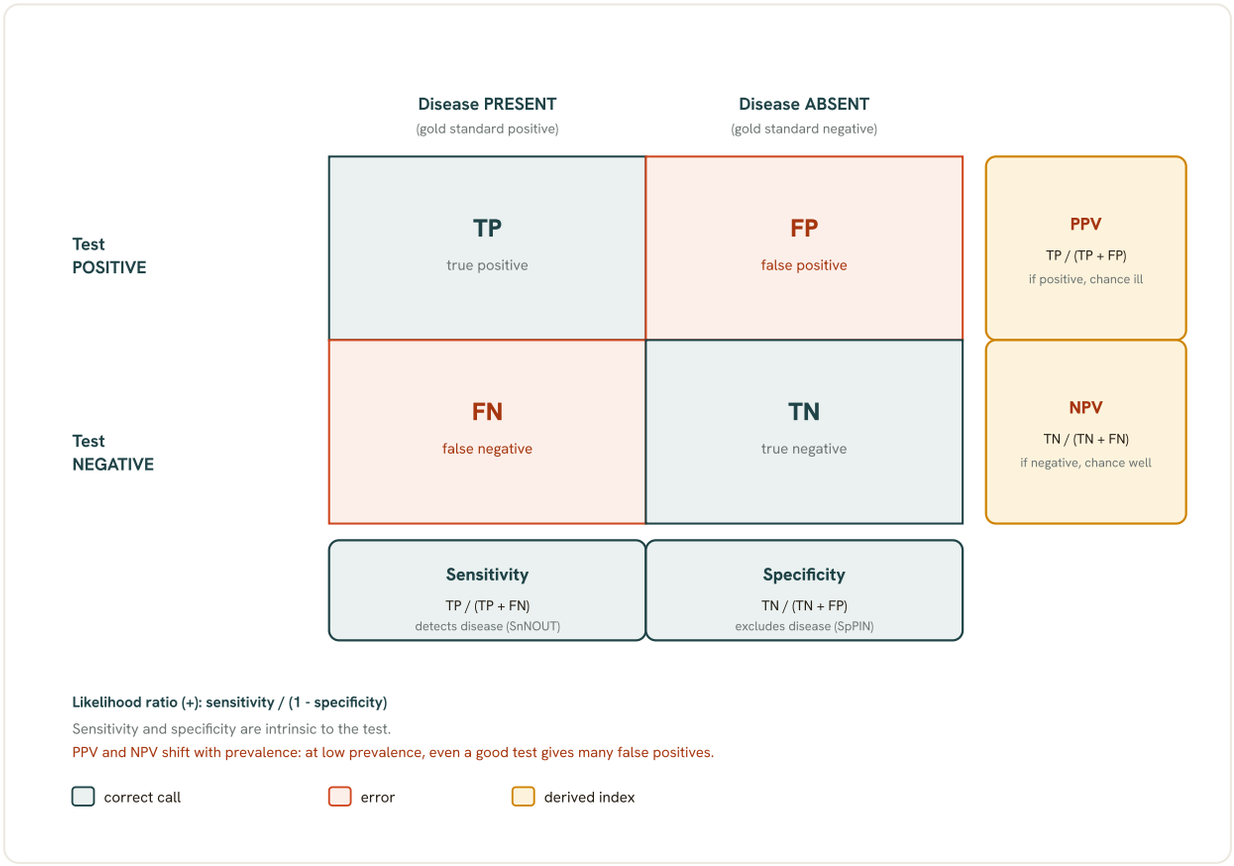


Fig 7.21 The diagnostic 2x2 and its indices. Reading DOWN a column gives the intrinsic properties: sensitivity is the detection rate among the diseased, specificity the correct-negative rate among the well. Reading ACROSS a row gives the predictive values the clinician actually wants: the positive predictive value of a positive test and the negative predictive value of a negative one. The crucial caveat is that sensitivity and specificity stay fixed while the predictive values move with prevalence, which is why a positive screening test in a low-prevalence population is so often a false alarm.

13.1 The 2x2 table: the object everything is read from

Fix the layout before you compute anything. Disease status runs across the top (the gold-standard truth), test result down the side. Populate the four cells with absolute counts, never percentages, and always check that the marginal totals add up before you divide (Bland 2015). Every index below is just one ratio taken from this grid.

TEST RESULT	DISEASE PRESENT	DISEASE ABSENT	ROW TOTAL
Test positive	True positive (a)	False positive (b)	**a + b**
Test negative	False negative (c)	True negative (d)	**c + d**
Column total	a + c (all diseased)	b + d (all non-diseased)	**N**

Columns fix the true state and yield the intrinsic indices (down); rows fix the test result and yield the predictive values (across).

13.2 Sensitivity: detecting the diseased (SnNOUT)

The test's ability to detect disease. Sensitivity (use "sensitivity") is the proportion of truly diseased people the test correctly calls positive: true positives divided by all diseased (Kaplan & Sadock 2024). It is read down the disease-present column, so it is a property of the test in diseased people and does not depend on how common the disease is.

Formula

Sensitivity = a / (a + c) = true positives / all diseased.

Complement

The false-negative rate = c / (a + c) = 1 − sensitivity: the fraction of diseased people the test misses.

Use

A sensitive test misses few cases, so it is what you want for a screening test or for ruling out a dangerous diagnosis.

SnNOUT. A highly **sensitive** test with a **Negative** result rules the disease **OUT**: if a test rarely misses disease, a negative from it makes disease unlikely. Hold the mnemonic SnNOUT (Sackett et al 2000).

RULE **SnNOUT rules out.** A negative result on a highly sensitive test rules the diagnosis out.

13.3 Specificity: clearing the healthy (SpPIN)

The test's ability to exclude disease. Specificity (use "specificity") is the proportion of truly non-diseased people the test correctly calls negative: true negatives divided by all non-diseased (Kaplan & Sadock 2024). It is read down the disease-absent column, so like sensitivity it is intrinsic to the test and independent of prevalence.

Formula

Specificity = d / (b + d) = true negatives / all non-diseased.

Complement

The false-positive rate = b / (b + d) = 1 − specificity: the fraction of healthy people wrongly flagged.

Use

A specific test rarely raises a false alarm, so it is what you want as a confirmatory test after a positive screen.

SpPIN. A highly **specific** test with a **Positive** result rules the disease **IN**: if a test rarely fires falsely, a positive from it makes disease likely. Hold the mnemonic SpPIN (Sackett et al 2000). The two indices trade off against each other along a threshold, which is exactly what an ROC curve plots (covered in the sampling and measurement notes).

13.4 Positive predictive value: trusting a positive result

Of those who test positive, how many are truly diseased. The positive predictive value (use "ppv", "positive predictive") reads across the test-positive row: of everyone the test flagged, what fraction actually has the disease (Kaplan & Sadock 2024). This is the number the clinician and the patient in front of you actually care about, and, crucially, it depends on prevalence.

Formula

PPV = a / (a + b) = true positives / all who test positive.

Reads

Across the top (test-positive) row, so it mixes the test's behaviour with how common the disease is in this population.

Moves with

Prevalence: the rarer the disease, the more the positive column is diluted by false positives, and the lower the PPV.

13.5 Negative predictive value: trusting a negative result

Of those who test negative, how many are truly healthy. The negative predictive value (use "npv", "negative predictive") reads across the test-negative row: of everyone the test cleared, what fraction really is disease-free (Kaplan & Sadock 2024). Like PPV it moves with prevalence, but in the opposite direction: in a low-prevalence setting almost everyone is truly healthy, so a negative result is nearly always right and NPV is high.

Formula

NPV = d / (c + d) = true negatives / all who test negative.

Reads

Across the bottom (test-negative) row.

Moves with

Prevalence, inversely: NPV rises as the disease becomes rarer.

13.6 The worked case: the same test, two prevalences

WORKED EXAMPLE

Setup. Take a test with sensitivity 90% and specificity 90%, and apply it first in a clinic where disease prevalence is 10%. Among 1000 people, 100 are diseased and 900 are not. Sensitivity 90% gives true positives a = 90 and false negatives c = 10. Specificity 90% gives true negatives d = 810 and false positives b = 90.

Verify the intrinsic indices. Sensitivity = a / (a + c) = 90 / 100 = 0.90; specificity = d / (b + d) = 810 / 900 = 0.90. Both reproduce the inputs, as they must.

Predictive values. PPV = a / (a + b) = 90 / (90 + 90) = 90 / 180 = 0.50, so a positive result carries only a 50% chance of true disease even with this good test. NPV = d / (c + d) = 810 / (810 + 10) = 810 / 820 = 0.988, about 99%.

Now drop the prevalence to 1%. Among 10000 people, 100 are diseased and 9900 are not. Sensitivity 90% still gives a = 90 and c = 10; specificity 90% now gives false positives b = 990 and true negatives d = 8910. Sensitivity and specificity are unchanged (90 / 100 and 8910 / 9900 = 0.90). But PPV = 90 / (90 + 990) = 90 / 1080 = 0.083, only about 8%. The very same test, in a low-prevalence population, turns nine out of ten positives into false alarms while its sensitivity and specificity never budge.

50%	8%	90/90
PPV AT 10% PREVALENCE	PPV AT 1% PREVALENCE	SENS/SPEC, UNCHANGED

13.7 Prevalence dependence: what is fixed and what moves

The one distinction the examiner is really testing. Sensitivity and specificity are **intrinsic** properties of the test: they are computed within the diseased and non-diseased columns respectively, so changing the mix of diseased to healthy people leaves them alone (Kaplan &

Sadock 2024, Altman 1991). Predictive values are computed across the test-result rows, which are populated by both true and false cases, so they are hostages to **prevalence** (the pre-test probability). As prevalence falls, PPV falls (a good test still yields many false positives in a low-prevalence screen) and NPV rises; as prevalence climbs toward that of a specialist referral clinic, PPV rises and NPV falls. This is why a screening programme that looks superb in a high-risk clinic can generate a flood of false positives when rolled out to the general population (Wilson & Jungner 1968).

RULE **PPV rises with prevalence; sensitivity and specificity do not.** Predictive values move with the population; intrinsic indices are fixed to the test.

TRAP **A positive is not a confirmation.** A highly sensitive test with a positive result does not confirm disease in a low-prevalence population, where most positives are false; only a specific test's positive rules in.

13.8 The likelihood ratio and Fagan's nomogram

The prevalence-proof way to update probability. The likelihood ratio (use "likelihood ratio") folds sensitivity and specificity into a single number that says how much a given result shifts the odds of disease, and unlike the predictive values it is itself independent of prevalence (Sackett et al 2000, Bland 2015). The positive likelihood ratio, LR+ = sensitivity / (1 − specificity), is how much more likely a positive result is in a diseased than a non-diseased person; the negative, LR− = (1 − sensitivity) / specificity, does the same for a negative result.

Pre-test to post-test. The clinical value of an LR is that it converts a **pre-test** probability (use "pre-test", essentially the prevalence or your clinical suspicion before testing) into a **post-test** probability (use "post-test") after the result is known: post-test odds = pre-test odds x likelihood ratio. Fagan's nomogram (use Fagan for the eponym) draws this as three parallel scales; a ruled line from the pre-test probability through the likelihood ratio reads off the post-test probability without any arithmetic (Fagan 1975). For the worked test above, LR+ = 0.90 / (1 − 0.90) = 0.90 / 0.10 = 9, and LR− = (1 − 0.90) / 0.90 = 0.10 / 0.90 = 0.11. A rough reading: an LR+ above 10 or an LR− below 0.1 shifts probability decisively, while an LR near 1 barely moves it.

13.9 Hold the picture together

Down the columns for the test, across the rows for the patient. Sensitivity and specificity answer "how good is this test?" and are read down the true-state columns; predictive values answer "what does this result mean for this patient?" and are read across the result rows, dragging prevalence in with them; the likelihood ratio is the bridge that carries a pre-test probability to a post-test probability regardless of prevalence (Kaplan & Sadock 2024, Sackett et al 2000). If you can place a formula on each margin of the 2x2 and say in one breath which side prevalence touches, you have the topic.

HOLD THIS

Sensitivity and specificity are intrinsic to the test and do not move with prevalence; PPV and NPV do, so a positive result on even a good test means little in a low-prevalence population (SnNOUT rules out, SpPIN rules in).

MNEMONIC

SnNOUT and SpPIN

A **Sn**sitive test, when **N**egative, rules the disease **OUT** (few misses, so a clear negative is trustworthy). A **Sp**ecific test, when **P**ositive, rules the disease **IN** (few false alarms, so a positive is trustworthy).

PEARL

Predictive values are what the patient hears, but they are borrowed from the population, not the test. Quote sensitivity and specificity to describe a test in the literature; compute PPV and NPV only for the specific prevalence you are working in; quote the likelihood ratio when you want a prevalence-proof measure to carry across settings.

GOOD TO REMEMBER

- **Sensitivity:** the proportion of diseased people the test calls positive; use it when you must not miss cases (screening, ruling out); sensitivity = a / (a + c) = true positives / all diseased; a Negative on a sensitive test rules OUT (SnNOUT); intrinsic, does not move with prevalence.
- **Specificity:** the proportion of healthy people the test calls negative; use it to confirm disease and avoid false alarms; specificity = d / (b + d) = true negatives / all non-diseased; a Positive on a specific test rules IN (SpPIN); intrinsic, does not move with prevalence.
- **Positive predictive value (PPV):** of those testing positive, the fraction truly diseased; use it to interpret a positive result in a given population; PPV = a / (a + b); rises with prevalence.
- **Negative predictive value (NPV):** of those testing negative, the fraction truly healthy; use it to interpret a negative result; NPV = d / (c + d); falls as prevalence rises.
- **Likelihood ratio (LR):** how much a result shifts the odds of disease, independent of prevalence; use it with Fagan's nomogram to move pre-test to post-test probability; LR+ = sensitivity / (1 − specificity), LR− = (1 − sensitivity) / specificity.
- **Prevalence (pre-test probability):** the proportion diseased before testing; recognise it as the lever that moves PPV and NPV while leaving sensitivity and specificity untouched; a low-prevalence screen yields many false positives even with a good test.

The prevalence figures used in a real screening decision come from population studies, so a test that performs well in a specialist clinic must be re-appraised at general-population prevalence before it is offered as a screen (Wilson & Jungner 1968); the prevalence of specific disorders is set out in the clinical chapters of Paper II, and the reliability and validity of a psychometric instrument itself, as distinct from these diagnostic indices, are covered in the Psychometry, Assessment and Descriptive Psychopathology notes.

14 · Statistical inference: p-values, confidence intervals, errors and power

Every significance test hands back two numbers, and the whole of applied statistical inference is the discipline of reading them without over-reading them. The first is the **p-value**, a verdict on chance; the second, ideally beside it, is the **confidence interval**, a statement of where the truth plausibly lies. The examiner is far less interested in whether you can compute either than in whether you know exactly what each does and does not mean, because the classic errors here are errors of interpretation, not arithmetic (Johnstone et al 2010, Bland 2015). Master four ideas and the topic is yours: what a p value actually measures, why the confidence interval is the more honest companion, the two ways an inference can be wrong, and the arithmetic of designing a study large enough to give a true effect a fair chance of being seen.

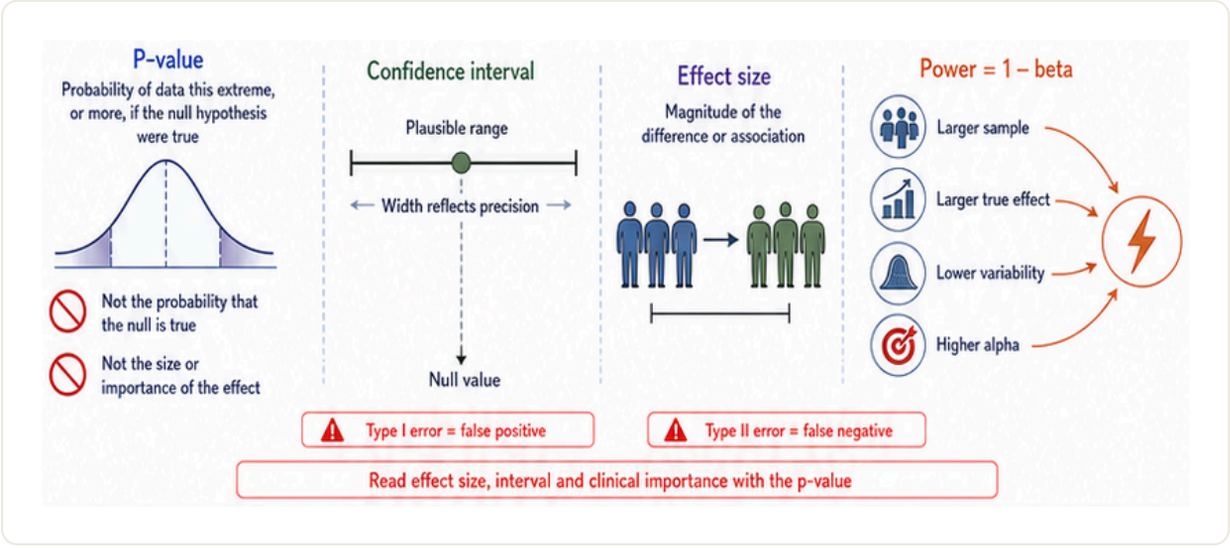


Fig 7.22 Inference: p-value, confidence interval, effect and power.

The **throughline** is the null hypothesis. A test assumes there is no difference (or no association) in the population, then asks how surprising the observed data would be under that assumption. The p value quantifies the surprise; the confidence interval turns the same information into a range of plausible effects. Errors and power then describe what can go wrong and how to guard against it before a single patient is recruited.

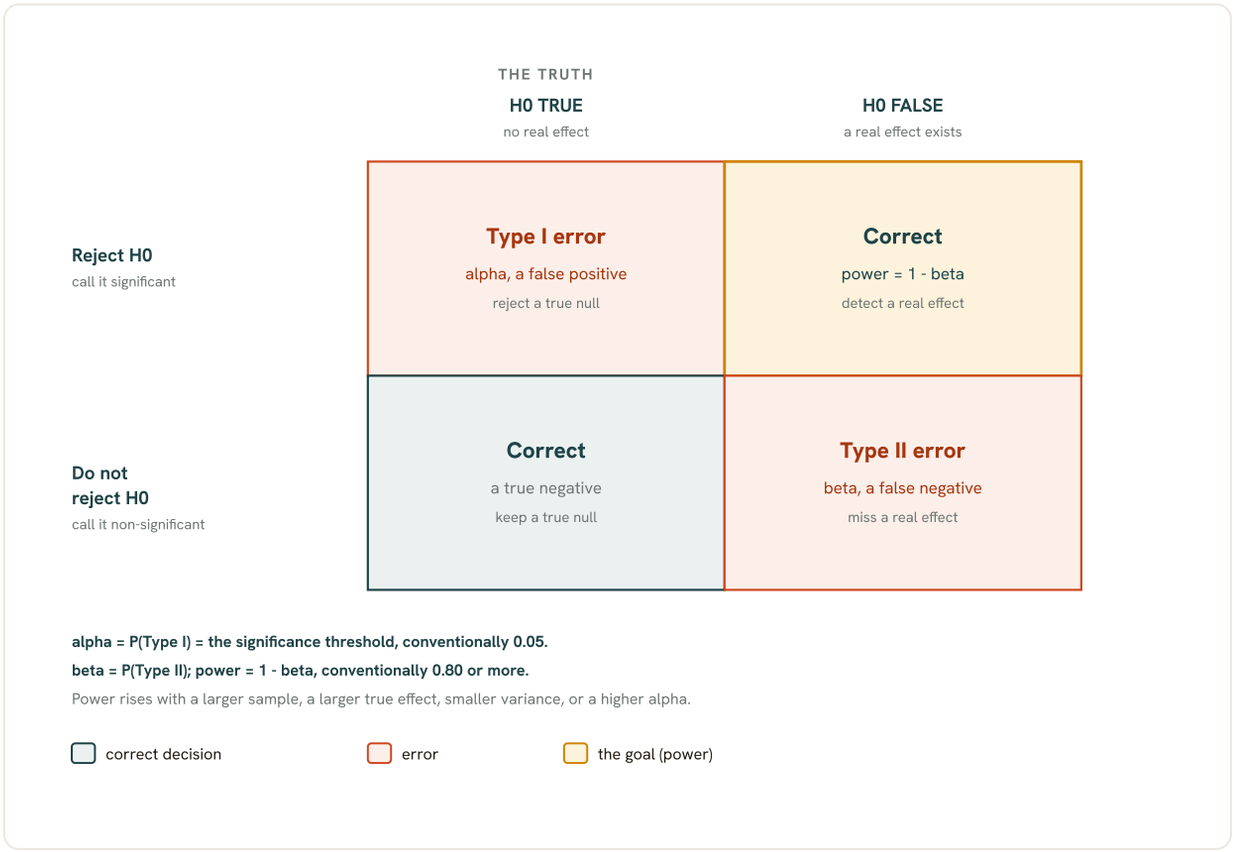


Fig 7.23 Type I and type II error. A test can go wrong two ways. Rejecting a null that is actually true is a type I error, a false positive, and its probability is alpha, the significance level we set. Failing to reject a null that is actually false is a type II error, a false negative, and its probability is beta. Power, the ability to detect a real effect, is one minus beta, and an underpowered study is the commonest reason a true effect is missed.

14.1 The p-value: what it is

A probability about the data, given the null. The p-value (also written p value) is the probability of obtaining the observed result, or one more extreme, if the null hypothesis of no true difference were correct (Johnstone et al 2010, Altman 1991). It is a measure of how compatible the data are with the null: a small p value says the data would be a rare fluke under the null, so we doubt the null. It is emphatically a probability about the data assuming the null is true, not a probability about the null itself.

14.2 The alpha threshold and significance

Where the line is drawn. Before the study, an investigator fixes a significance threshold, alpha, the p value at or below which the null will be rejected. By convention alpha = **0.05** (Johnstone et al 2010, Bland 2015). A result with p at or below 0.05 is called statistically significant: under a true null we would see a result this extreme less than 1 time in 20, which we treat as evidence against the null. The 0.05 line is a convention, not a law of nature, and stricter thresholds (0.01, or a Bonferroni-adjusted value across many tests) are used where false positives are costly. Alpha is chosen by the investigator; the p value is computed from the data.

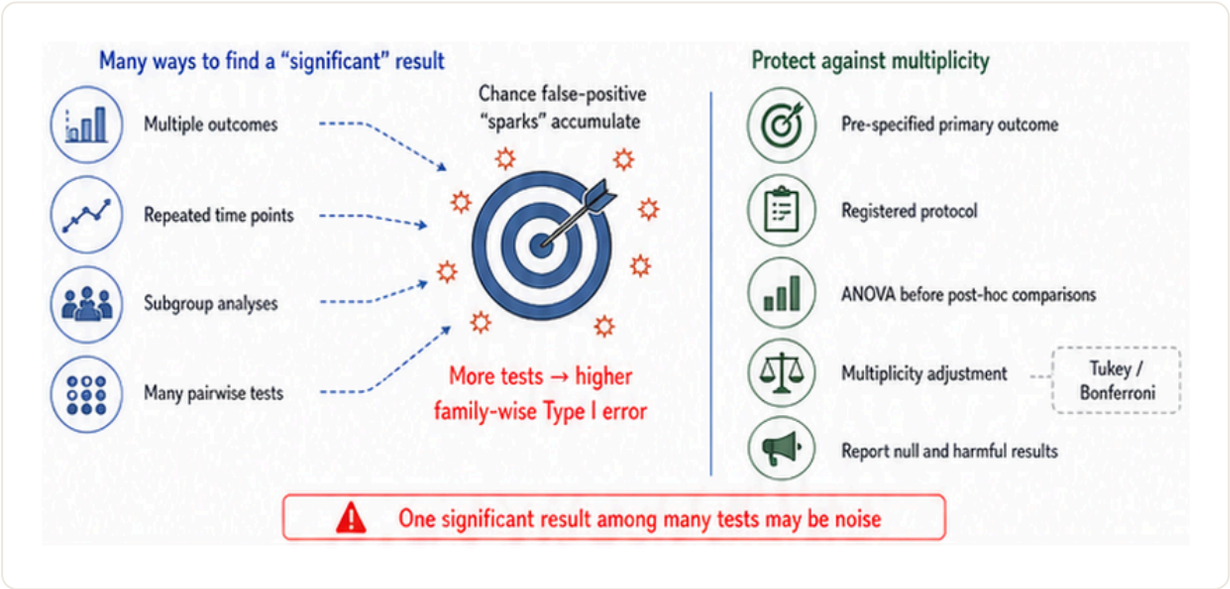


Fig 7.24 Multiplicity and selective reporting.

RULE **Set alpha first, then read p.** Alpha is your pre-declared tolerance for a false positive; the p value is what the data return. Compare the one to the other, never move the goalposts after seeing the data.

14.3 What a p-value is NOT

The three misreadings the examiner hunts for. A p value is not the probability that the null hypothesis is true, and it is not the probability that the result arose by chance in any absolute sense; it is conditional on the null being true (Altman 1991, Sterne & Davey Smith 2001). Nor does a p value measure the size or the importance of an effect: a vanishingly small p value can attach to a trivial difference if the sample is huge, and a non-significant p can hide a large effect measured imprecisely. And a non-significant result (p above 0.05) is not proof that the null is true, absence of evidence is not evidence of absence. Hold these three: p is not the probability the null is true, p is not the effect size, and a large p does not prove no effect.

TRAP **p is not P(null is true).** A p of 0.03 does not mean a 3% chance the null holds; it means data this extreme would occur 3% of the time if the null held. The direction of the conditional is the whole trap.

14.4 The confidence interval: the plausible range

From a yes/no verdict to a range of effects. A confidence interval is the range of plausible values within which the true population value is likely to lie, given the data (Johnstone et al 2010, Altman 1991). A **95 percent CI** is the interval such that, over many repeated samples, 95% of the intervals so constructed would contain the true value; loosely, we can be 95 percent confident the true effect lies inside it. Where the p value gives a bare significant/non-significant verdict, the CI shows both the best estimate (the point estimate at its centre) and its precision (its width). A trial that finds chlorpromazine beats placebo with a relative risk of 2.3 (95% CI 1.2 to 3.5) tells you not only that the effect is real but that the true effect plausibly ranges from modest to large (Johnstone et al 2010).

14.5 Reading significance off a confidence interval

The no-effect value inside the interval. A CI carries the significance test within it, read against the value of no effect. For a **difference** of means (or any subtracted quantity) the no-effect value is **0**: if the 95% CI for the difference crosses 0, the result is non-significant at the 0.05 level. For a **ratio** measure, a relative risk (RR) or odds ratio (OR), the no-effect value is **1** (equal risk, equal odds): if the 95% CI for the ratio crosses 1, the result is non-significant (Johnstone et al 2010, Altman 1991). So an RR of 2.3 with CI 1.2 to 3.5 is significant (the interval sits wholly above 1), whereas an OR of 1.4 with CI 0.9 to 2.1 is not (the interval straddles 1). The CI is more informative than the p value alone because it delivers the significance verdict and the effect size and its precision in a single line.

RULE **Cross 0 for a difference, cross 1 for a ratio.** A 95% CI for a difference that includes 0, or for an RR/OR that includes 1, means the result is not statistically significant.

14.6 Type I and Type II error

The two ways an inference can be wrong. Because inference is a decision under uncertainty, two mistakes are possible against the two states of the truth (Johnstone et al 2010, Bland 2015).

Type I error

A false positive: rejecting a null hypothesis that is actually true, that is, declaring a difference that does not exist. Its probability is alpha, so setting alpha at 0.05 caps the false-positive rate at 5%.

Type II error

A false negative: failing to reject a null hypothesis that is actually false, that is, missing a difference that truly exists. Its probability is termed beta.

The 2x2 of truth against decision. Cross the true state of the world (null true, or null false) with the decision taken (do not reject, or reject) and four cells result: two correct (a true negative, and a true positive), and the two errors above. The type I error sits where a true null is rejected; the type II error where a false null is retained. Tightening alpha to reduce type I error, all else equal, raises beta and so raises type II error: the two trade off against each other.

14.7 Power and its four drivers

The chance of finding a real effect. The power of a test is 1 minus beta, the probability that it will detect a true effect of a given size when one genuinely exists (Tasman et al 2015, Johnstone et al 2010). Colloquially, power is the chance of finding what you are looking for. The convention is to design for power of **80** percent or more, meaning at most a 20% chance of missing a true effect (beta = 0.2). Power is fixed by four interlocking quantities, and moving any one moves power:

Alpha

The significance threshold. A stricter (smaller) alpha lowers power, all else equal.

Sample size

The number of participants. A larger sample sharpens the estimate and raises power; too small a sample is the commonest reason a true effect is missed.

Effect size

The magnitude of the true difference sought. Larger effects are easier to detect, so they need smaller samples.

Variance

The scatter of the data. Less variability sharpens the estimate and raises power.

Fix any four of alpha, power, effect size, variance and sample size and the fifth is determined, which is exactly how a sample-size calculation works.

TRAP **A non-significant result may be an underpowered study.** Failure to reject the null can mean there is no effect, or that the study was too small to see one. The two are not the same, and the p value alone cannot tell them apart.

14.8 Effect size and Cohen's d

Standardising the size of a difference. An effect size expresses the magnitude of a difference in units that are comparable across studies and scales, independent of sample size (New Oxford Textbook 2020). For a difference between two means the standard measure is cohen's d, the difference in means divided by the pooled standard deviation. Cohen's conventional benchmarks are small = **0.2**, medium = **0.5**, and large = **0.8** (New Oxford Textbook 2020). For a correlation, the effect size is r itself (or r-squared, the shared variance). Effect size is the input that most strongly governs the sample a study needs: a large expected effect can be caught with few participants, a small one demands many.

14.9 Sample size, the MCID and the power calculation

Sizing the study before it runs. A sample size calculation is performed at the design stage to find the number of participants needed to give a study adequate power (Johnstone et al 2010, Altman 1991). Its inputs are the other four quantities: the chosen alpha (usually 0.05), the target power (usually 0.8), the smallest effect worth detecting, and an estimate of the variance or event rate (from pilot data or the literature). Feed these in and the power calculation returns the required n; equivalently, given a fixed n, it returns the power to detect a stated effect. The effect fed in should be the minimum clinically important difference, the smallest change that would matter to a patient or alter management, not merely the smallest that could be made statistically significant. Powering a trial for a statistically detectable but clinically trivial difference wastes participants; powering it for the minimum clinically important difference sizes it to answer a question worth asking.

0.05	80%	0.2 / 0.5 / 0.8
ALPHA	POWER	COHEN D SMALL/MED/LARGE

14.10 A worked read: p, CI and the four cells together

WORKED EXAMPLE

Reading a trial result. A relapse-prevention trial reports a relative risk of relapse on active drug versus placebo of RR = 2.3, 95% CI 1.2 to 3.5, p = 0.01 (Johnstone et al 2010). Read it in three moves. First the p value: under a true null of no difference, a result this extreme would occur about 1 time in 100, below alpha 0.05, so we reject the null and call it significant. Second the confidence interval: the 95% CI runs 1.2 to 3.5 and does not cross the ratio no-effect value of 1, which confirms significance, and its width tells us the true effect plausibly ranges from modestly (1.2) to substantially (3.5) raised, an imprecise estimate a larger sample would tighten. Third the error frame: in rejecting the null we risk a type I error, capped at alpha = 0.05; had the CI instead been 0.8 to 4.1 (crossing 1, non-significant) we could not conclude no effect, because a small, underpowered study courts a type II error of missing a real difference.

A difference, not a ratio. Had the outcome been a mean symptom-score difference of 2.0 points with 95% CI −0.5 to 4.5, the no-effect value is 0, the interval crosses it, and the result is non-significant, whatever the point estimate suggests.

COMMON TRAP

Statistical significance is not clinical importance. A trial of 10,000 patients can return $p < 0.001$ for a 1-point difference on a 60-point depression scale, a difference no patient would notice; conversely a genuinely useful effect in a small study may miss significance purely for want of power. Always read the effect size and its confidence interval, not the p value alone, and anchor the judgement to the minimum clinically important difference.

MNEMONIC

Type I is the FALSE ALARM (reject a true null); Type II is the MISS (retain a false null).

A smoke alarm going off with no fire is a type I error (false positive, probability alpha); a fire the alarm sleeps through is a type II error (false negative, probability beta). Power (1 minus beta) is the alarm actually sounding for a real fire.

HOLD THIS

A 95% CI that crosses 0 (for a difference) or 1 (for a ratio) is non-significant; type I error = alpha = 0.05 (false positive), type II error = beta (false negative), power = 1 minus beta, conventionally 80% or more.

GOOD TO REMEMBER

- **p-value:** the probability of the observed data or more extreme if the null were true; use it to judge compatibility with the null; the significance threshold is alpha = 0.05.
- **Confidence interval (95% CI):** the plausible range for the true population value; use it instead of the p value alone because it shows effect size and precision; non-significant if it crosses 0 (difference) or 1 (ratio RR/OR).
- **Type I error:** a false positive, rejecting a true null; guarded by the significance threshold; its probability is alpha (conventionally 0.05).
- **Type II error:** a false negative, retaining a false null; arises chiefly from too small a sample; its probability is beta.
- **Power:** the ability to detect a true effect; design for it before recruiting; power = 1 minus beta, conventionally 80% or more.
- **Effect size (Cohen's d):** the standardised magnitude of a difference; use it to compare across studies and drive sample size; d = 0.2 small, 0.5 medium, 0.8 large.
- **Sample size / power calculation:** the design-stage arithmetic sizing a study; use it to secure adequate power; its inputs are alpha, power, the minimum clinically important effect size, and the variance.

For the derivations and worked confidence-interval formulae see Altman (1991) and Bland (2015); the interpretation traps are set out plainly in Sterne & Davey Smith (2001). Where these inference tools drive the choice of a specific significance test, that selection is treated in the sibling tests-of-significance material; twin, family and adoption designs that generate the effect estimates fed into these calculations are covered in the Psychiatric Genetics notes, and the reliability and validity of the measuring instrument itself (as distinct from a study's statistical validity) belong to the Psychometry, Assessment and Descriptive Psychopathology notes.

15 · Survival analysis, NNT and ROC

Three tools cluster here because each squeezes a clinically useful single number out of a slightly awkward kind of data. Survival analysis handles data where the outcome is **time to an event** and some patients have not had the event when the study ends. The number needed to treat converts a trial's risk difference into a bedside count of patients. The ROC curve measures how well a diagnostic test separates the sick from the well across every possible cut-off. The examiner tests interpretation, not derivation: what the curve, the count and the area under the curve mean, and the one trap that goes with each (Kaplan & Sadock 2024, Bland 2015).

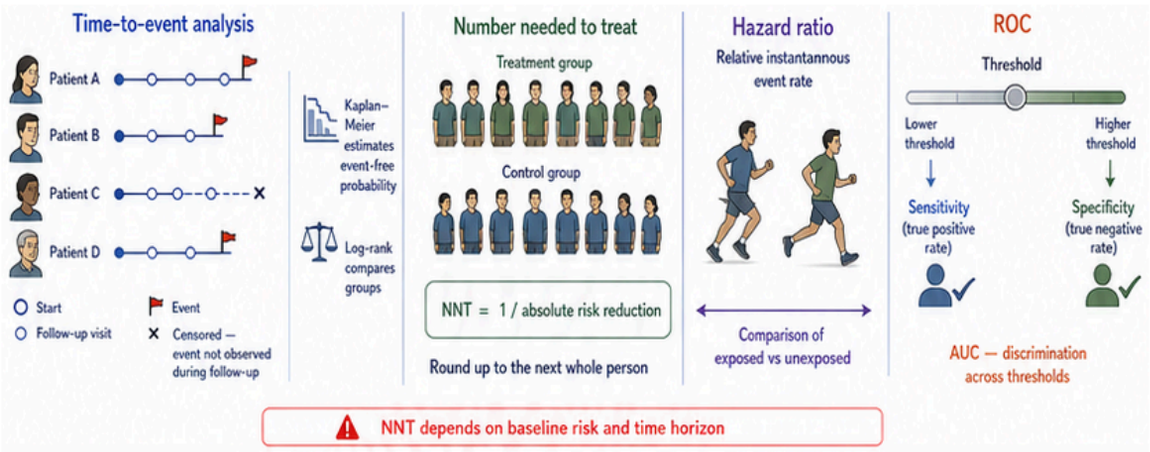


Fig 7.25 Time-to-event, NNT and discrimination.

Why time-to-event needs its own method. If you simply compared the proportion relapsed at two years between two arms, you would throw away everyone who left the study early or who had not relapsed at the cut-off. Survival analysis keeps them by treating their incomplete follow-up as partial information rather than as a missing value to be discarded (Kaplan & Sadock 2024).

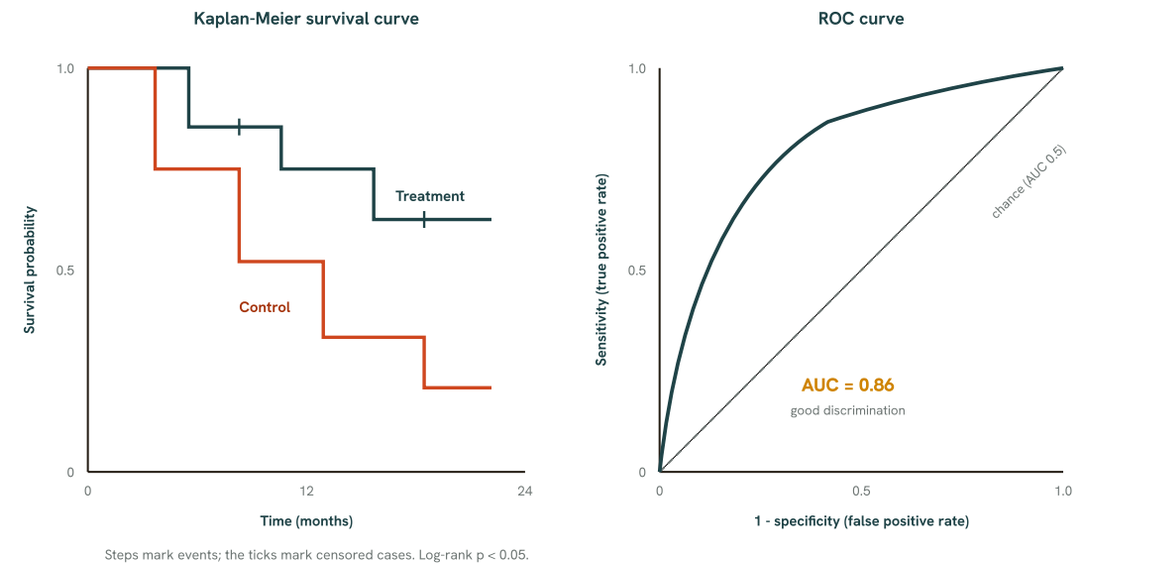


Fig 7.26 Survival and ROC curves. The Kaplan-Meier curve plots the probability of remaining event-free over time, stepping down at each event, with ticks for censored cases; two curves are compared with the log-rank test and summarised by the hazard ratio. The ROC curve plots sensitivity against one minus specificity across every cut-off of a test: the further it bows toward the top-left corner the better, and the area under it (AUC) grades overall discrimination from 0.5 (chance) to 1.0 (perfect).

15.1 Time-to-event data and censoring

What survival data are. Many psychiatric questions live in the time domain: time to onset of drug effect, time in hospital, time to remission or to relapse (Kaplan & Sadock 2024). These are generically **time-to-event** or survival variables; the original event studied was time to death,

which gives the field its name. The two things recorded for each subject are the length of follow-up and whether the event had occurred by the end of it.

Censoring. A subject is censoringed (a censored observation) when the event has not occurred by the time observation stops: the study ends, or the patient is lost to follow-up, or withdraws (Kaplan & Sadock 2024). We know only that their true time-to-event exceeds the time we watched them. This is right-censoring, the common form. Crucially, a censored patient is not the same as a dropout to be deleted: they contribute information for the whole period they were observed, and are only removed from the risk set after the point at which we lose sight of them.

TRAP **Censoring is not ignored loss to follow-up.** Censoring is not the same as loss to follow-up being ignored: Kaplan-Meier accounts for it by using each censored case up to the moment it is censored, then dropping it from the denominator, rather than discarding it or coding it as an event.

15.2 The Kaplan-Meier survival curve

The step curve everyone draws. The kaplan-meier estimator is the nonparametric way to draw the survival function from the data: it makes no assumption about the shape of the underlying distribution (Kaplan & Sadock 2024). It splits time at each point where an event actually occurs and, at each such point, multiplies the running survival probability by the proportion of those still at risk who survived that instant. The probability of surviving beyond time t is therefore the product of the conditional survival probabilities across all preceding intervals, which is why it is a step function that drops only when an event happens, not at fixed calendar intervals.

Reading the curve. The y-axis is the cumulative proportion still event-free; the curve starts at 1.0 and steps down. A small vertical tick on the curve marks a **censored** case (a patient still event-free when last seen). The height where the curve crosses 0.5 gives the **median survival time**, the single summary statistic most often quoted (Kaplan & Sadock 2024). Flatter curve means better survival.

15.3 Comparing two curves: the log-rank test

Is the gap between two curves real? Two survival curves, for example two treatment arms, are compared with the log-rank test, the most commonly used nonparametric comparison (Kaplan & Sadock 2024, Bland 2015). At every event time it compares the number of events observed in each group with the number expected if the two curves shared one underlying survival experience, then sums those observed-minus-expected contributions across all event times into a single chi-square-type statistic. Its null hypothesis is that the two survival distributions are identical. It uses the whole curve, not survival at one arbitrary time-point, and weights every event time equally (the related Gehan and generalized Wilcoxon tests weight early events more).

RULE **Curves to log-rank, effect size to Cox.** Use the log-rank test to ask whether two Kaplan-Meier curves differ; use Cox regression to put a number, the hazard ratio, on how much they differ and to adjust for covariates.

15.4 The hazard ratio from Cox regression

The instantaneous relative risk. The hazard rate is the probability of the event happening at time t given the subject has survived event-free up to t: an instantaneous risk. Cox proportional hazards regression models this time-to-event outcome against covariates without assuming a distributional shape (it is semiparametric), and yields the hazard ratio (Kaplan & Sadock 2024). The hazard ratio is the ratio of the hazard in one group to the hazard in the other: read it like a relative risk that applies at every instant. A hazard ratio of **1.0** means no difference in the rate of events; above 1 the exposure or treatment raises the instantaneous event rate, below 1 it lowers it. The name comes from the **proportionality assumption**: the model assumes the two hazards stay in constant proportion over time (Kaplan & Sadock 2024).

15.5 The number needed to treat

From a risk difference to a headcount. The number needed to treat (nnt) translates a trial result into a clinical unit a resident can quote at the bedside: how many patients must receive the treatment for one extra good outcome, compared with the control (Cooper & Jacoby 2013, Bland 2015). It is the reciprocal of the **absolute risk reduction** (ARR), the difference between the control event rate and the experimental event rate:

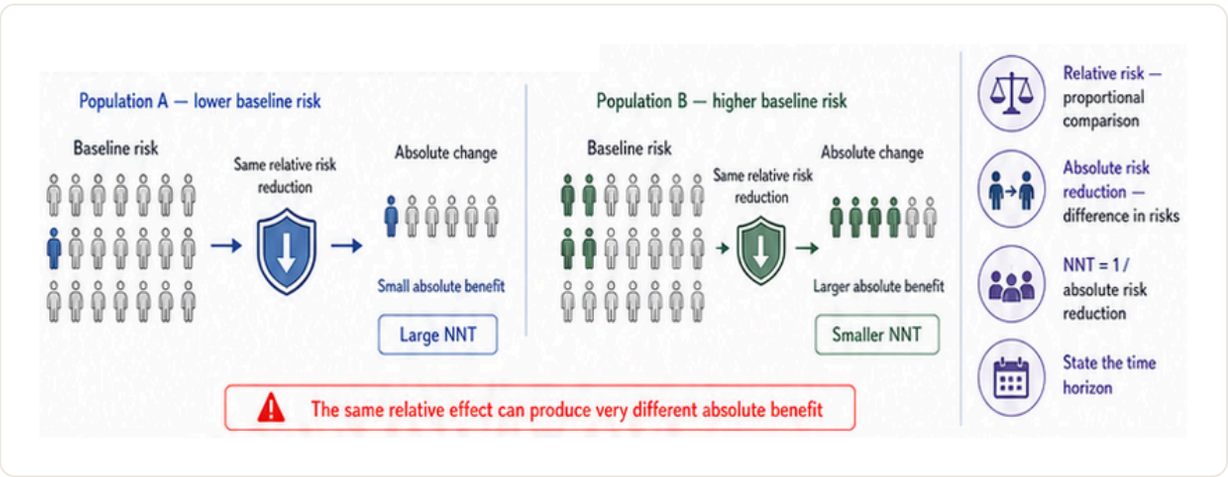


Fig 7.27 Relative effects need absolute context.

Absolute risk reduction (ARR)

Control event rate minus experimental event rate, expressed as a proportion (a difference in the probability of the bad outcome).

Number needed to treat (NNT)

NNT = 1 / ARR, rounded up to the next whole number; the count of patients to treat to prevent one extra bad outcome.

Number needed to harm (NNH)

The mirror image: 1 / absolute risk increase in an adverse outcome; the count of patients to treat before one extra harm occurs.

Direction of merit. A large NNT means many patients must be treated for a single benefit, so the treatment is weak; a small NNT means the benefit comes cheaply, so the treatment is strong. As a rough guide an NNT under **10** denotes a clinically useful treatment effect, and NNTs for

psychiatric interventions are broadly comparable to those in general medicine (Cooper & Jacoby 2013). NNH is best large (harm is rare); NNT is best small.

RULE **Smaller NNT, stronger treatment.** A smaller NNT is a stronger treatment: fewer patients treated per good outcome gained. A small NNT and a large NNH together is the ideal profile.

15.6 A worked NNT

WORKED EXAMPLE

CBT for residual depression (Paykel et al 1999, via Cooper & Jacoby 2013). Patients with residual depressive symptoms were randomised to clinical management alone or clinical management plus cognitive behaviour therapy. Over follow-up the relapse rate was **47%** in the clinical-management (control) arm and **29%** in the CBT arm. The absolute risk reduction is the difference in these relapse rates: ARR = 47% − 29% = **18%** (0.18). The NNT is the reciprocal: NNT = 1 / 0.18 = 5.56, rounded up to **6**. Reading: about six patients with residual depressive symptoms must be treated with CBT to prevent one extra relapse. Since 6 is under 10, this is a clinically worthwhile effect.

15.7 The ROC curve

A test across all its thresholds. A continuous diagnostic test (a rating-scale score, a biomarker level) gives a different sensitivity and specificity at every cut-off you might pick. The roc (receiver operating characteristic) curve plots this whole family of trade-offs on one graph: the **sensitivity** (the true positive rate) on the y-axis against **1 minus specificity** (the false positive rate) on the x-axis, traced across all thresholds (Altman 1991, Bland 2015). Lowering the cut-off catches more true cases (sensitivity rises) at the cost of more false alarms (specificity falls), so the curve sweeps from the bottom-left corner up to the top-right. A test with no discriminating power sits on the diagonal from (0,0) to (1,1); a good test bows up towards the top-left corner, where sensitivity is high and false positives are few.

15.8 The area under the curve (AUC)

One number for overall discrimination. The auc, the area under the ROC curve, summarises the whole curve as a single index of discrimination: the probability that the test will score a randomly chosen affected patient higher than a randomly chosen unaffected one (Altman 1991). Its scale is anchored at two points a resident must hold: an AUC of **0.5** is the diagonal, chance-level discrimination (no better than a coin toss), and an AUC of **1.0** is a perfect test that separates the two groups completely. In between, higher is better, so AUC lets you rank competing tests on one axis.

0.5	1.0	< 10
AUC AT CHANCE	AUC PERFECT TEST	NNT: USEFUL EFFECT

15.9 Choosing a cut-off from the curve

Where to set the threshold. The ROC curve also guides the practical choice of a single operating cut-off (Altman 1991, Bland 2015). The point closest to the top-left corner gives the best overall

balance of sensitivity and specificity, but the clinically right cut-off depends on the costs of the two error types. For a screening test, where missing a case is worse than a false alarm, slide the cut-off to favour **sensitivity** (accept more false positives); for a confirmatory test, where a false positive is costly, favour **specificity**. The curve does not choose for you; it shows the trade-off you are buying at each threshold. The reliability and validity of the underlying instrument itself are covered in the Psychometry, Assessment and Descriptive Psychopathology notes.

TRAP **The diagonal is not a bad test drawn badly.** An ROC curve sitting on the 45-degree line (AUC 0.5) is not an error of plotting: it is a test with zero discriminating ability. Only a curve bowing above the diagonal carries diagnostic information.

HOLD THIS

Kaplan-Meier steps down and keeps censored cases (median survival where it hits 0.5); log-rank compares two curves, Cox gives the hazard ratio (1.0 = no effect); NNT = 1 / ARR and smaller is stronger; ROC plots sensitivity against 1 minus specificity, and AUC runs 0.5 (chance) to 1.0 (perfect).

GOOD TO REMEMBER

- **Survival analysis:** the family of methods for time-to-event data with censoring; use when the outcome is time until an event and some subjects have not had it by study end; the key summary is the median survival time.
- **Censoring:** incomplete follow-up where the event has not yet occurred; use it (do not delete it) whenever a subject is lost, withdraws, or the study ends event-free; it contributes to the risk set only up to the point of censoring.
- **Kaplan-Meier curve:** the nonparametric step estimate of the survival function; use to picture and summarise survival in one or more groups; it drops only at observed event times and marks censored cases with ticks.
- **Log-rank test:** the nonparametric test comparing two or more survival curves; use to ask whether the curves differ overall; null hypothesis is identical survival distributions, and it weights all event times equally.
- **Hazard ratio (Cox regression):** the ratio of instantaneous event rates between groups from a semiparametric model; use to quantify and covariate-adjust the difference between curves; a hazard ratio of 1.0 means no difference.
- **Number needed to treat (NNT):** the count of patients treated per one extra good outcome; use to translate a trial's ARR into clinical terms; NNT = 1 / absolute risk reduction, and under 10 is a useful effect.
- **Number needed to harm (NNH):** the count of patients treated before one extra adverse event; use to size the harm of a treatment; NNH = 1 / absolute risk increase, and larger is safer.
- **ROC curve:** the plot of sensitivity against 1 minus specificity across all thresholds; use to show a continuous test's sensitivity-specificity trade-off and to pick a cut-off; the best-balance point sits nearest the top-left corner.
- **AUC (area under the ROC curve):** a single index of overall discrimination; use to rank or compare diagnostic tests; it runs from 0.5 (chance) to 1.0 (perfect).

For survival methods and Cox regression see Kaplan & Sadock (2024); for ARR, NNT and NNH see Cooper & Jacoby (2013); for ROC and AUC see Altman (1991) and Bland (2015). Genetic

time-to-onset study designs are treated in the Psychiatric Genetics notes, and the disorder-specific efficacy figures these tools summarise appear in the clinical chapters of Paper II.

Epidemiology

16 · Measures of disease frequency and association

Epidemiology begins with two questions and answers each with a number: how common is a disorder, and what raises the chance of getting it. The first question is answered by the **measures of disease frequency**, of which the two workhorses are incidence and prevalence; the second by the **measures of association**, which quantify how much an exposure changes the risk of an outcome (Kaplan & Sadock 2024). The examiner reliably asks you to separate a count of new cases from a count of existing cases, to know which measure of association comes from which study design, and to read an association off a 2x2 table without confusing risk with odds. Get the incidence-versus-prevalence distinction wrong and every downstream inference about causation and burden goes with it.

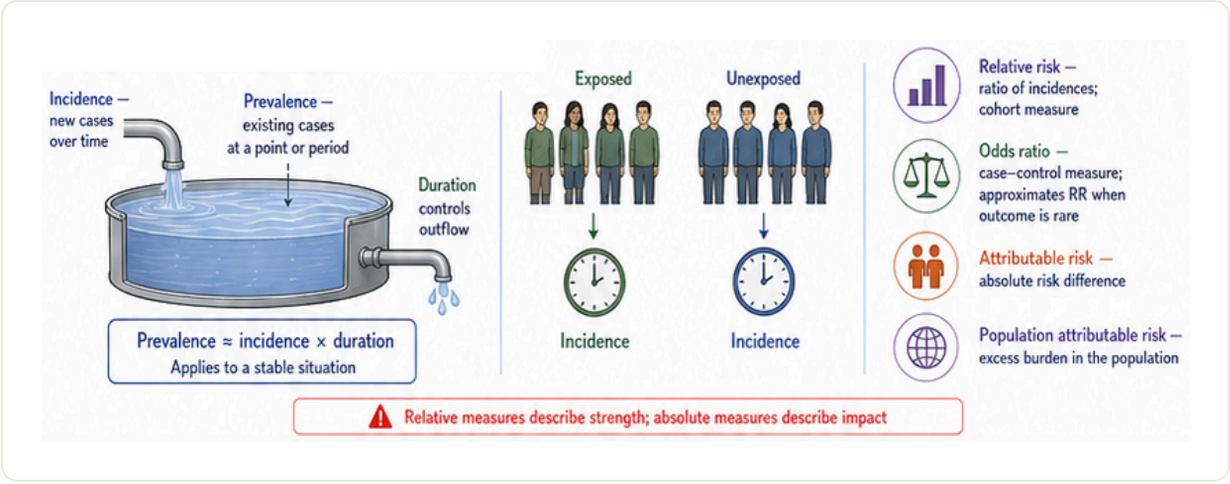


Fig 7.28 Disease frequency and association.

This material owns the measures themselves and the national numbers; the **epidemiology of specific disorders** (the prevalence figures for schizophrenia, mood disorders, and the rest) is developed in the clinical chapters of Paper II. Here we keep the tools, verified formula by formula, and the study design each tool is native to.

16.1 Prevalence: the stock of existing cases

Existing cases in a population at a point or over a period. Prevalence (use "prevalence") is a ratio: the number of people who have the disorder divided by the number in the population, over a designated time window (Kaplan & Sadock 2024). It is a snapshot of the burden of disease, so it counts both old and new cases indiscriminately and depends heavily on how long the disorder lasts. Three elements fix any prevalence estimate: the case definition, the target population, and the time.

Point prevalence

Point prevalence (use "point prevalence"): the proportion of a population who have the disorder at a single specified point in time.

Period prevalence

Period prevalence (use "period prevalence"): the proportion who have the disorder at any time within a stated interval (1-month, 1-year, or lifetime prevalence).

Lifetime prevalence

The proportion who have had the disorder at any point in their lives; useful for risk-factor studies but subject to recall bias.

For mental disorders, point prevalence is hard to pin down because symptoms wax and wane daily, so the **1-month** and **1-year** period prevalences are used in practice; the 1-year figure is what service planners multiply by the population to forecast caseload (Kaplan & Sadock 2024).

16.2 Incidence: the flow of new cases

New cases arising over a period, per population at risk. Incidence (use "incidence") counts only fresh cases: its numerator is the number of new events arising in a specified time period, and its denominator is the population at risk of having that event (Kaplan & Sadock 2024). People who already have the disorder at the start are removed from the denominator because they are, by definition, no longer at risk of developing it. Incidence is the measure of choice for a first-onset question, for example the number of new cases of post-traumatic stress disorder after a natural disaster.

Cumulative incidence

Cumulative incidence (use "cumulative incidence"): new cases divided by the number of disease-free people at risk at the start of the period; a proportion (risk), so it needs a stated time period to mean anything.

Incidence rate

Incidence rate (use "incidence rate"): new cases divided by the total person-time at risk; a true rate with units of per person-year, robust to people entering and leaving the population.

Person-time

The sum, over all individuals, of the time each was actually at risk until they became a case or were lost to follow-up; the denominator of the incidence rate.

Because most mental disorders are rare per year, estimating incidence needs large populations at risk followed for long periods, and most incidence rates are quoted over a 1-year window (Kaplan & Sadock 2024).

RULE **Incidence is a rate of the new, prevalence is a stock of the existing.** Ask "new cases over a period" for incidence, "all cases at a moment" for prevalence.

16.3 The bridge: prevalence is roughly incidence times duration

The equation that ties the two frequency measures together. Prevalence, incidence, and mean duration are mathematically related: the prevalence of a disorder is proportional to its incidence multiplied by its mean duration, written **prevalence is roughly incidence times duration** (P is

proportional to I x d) for a stable, low-prevalence condition (Kaplan & Sadock 2024). The consequence is the single most examined trap in this topic. Prevalence reflects not only how many new cases appear but also how long cases stay ill, so anything that lengthens duration inflates prevalence even when incidence is flat.

TRAP **Prevalence rises with longer illness duration even if incidence is unchanged.** A treatment that prolongs life without curing (the AIDS example) raises prevalence; a cure lowers it. Rising prevalence never by itself proves rising incidence.

COMMON TRAP

Do not read a climbing prevalence as more people falling ill. Because prevalence is roughly incidence times duration, better survival or chronification lifts prevalence with incidence untouched. When you need to tell a genuine rise in new cases apart from improved survival, you must use an incidence measure, not prevalence.

16.4 From frequency to association: the 2x2 table

Every measure of association is one ratio read off this grid. The **measures of association** compare disease frequency between an exposed and an unexposed group, and all of them are computed from a single 2x2 table that cross-tabulates exposure against outcome (Kaplan & Sadock 2024). Fix the layout first, populate the four cells with absolute counts, and check the marginal totals before you divide. In the standard orientation the exposed row holds cells a and b, the unexposed row cells c and d.

GROUP	DISEASE PRESENT	DISEASE ABSENT	ROW TOTAL
Exposed	a	b	**a + b**
Unexposed	c	d	**c + d**

Risk in the exposed reads across the top row, a / (a + b); risk in the unexposed reads across the bottom row, c / (c + d).

16.5 Relative risk: the ratio of two incidences, from a cohort

How many times more likely the exposed are to develop disease. The relative risk (use "relative risk") is the ratio of two incidences: the incidence in the exposed divided by the incidence in the unexposed (Kaplan & Sadock 2024). Because it needs the actual risk (incidence) in each group, it can only be computed when you have followed disease-free people forward and watched new cases arise, that is, from a cohort study. A relative risk greater than 1 marks the exposure as a risk factor; less than 1, a protective factor; exactly 1, no association.

Formula

Relative risk = incidence in exposed / incidence in unexposed = [a / (a + b)] / [c / (c + d)].

Design

Cohort study (follows exposed and unexposed forward), because only a cohort yields incidence directly.

Reads

A risk ratio: "the exposed are RR times as likely to develop the disorder as the unexposed."

RULE **Relative risk comes from a cohort, the odds ratio from a case-control.** If the design gives you incidence you can quote a relative risk; if it starts from cases and controls you are limited to an odds ratio.

16.6 Odds ratio: the case-control measure that approximates RR when disease is rare

The ratio of the odds of exposure, computed when you cannot measure incidence. The odds ratio (use "odds ratio") is used when the study begins with cases and controls rather than exposed and unexposed cohorts, so incidence and therefore relative risk cannot be measured directly (Kaplan & Sadock 2024). It compares the odds of disease in the exposed (a / b) with the odds of disease in the unexposed (c / d), which reduces to the cross-product ad / bc. Like the relative risk it varies around 1: above 1 an association with disease, below 1 an association with not having disease, exactly 1 no association.

Formula

Odds ratio = (a / b) / (c / d) = ad / bc (the cross-product ratio).

Design

Case-control study (starts from diseased cases and disease-free controls, then looks back at exposure).

Key property

The odds ratio approximates the relative risk when the outcome is rare (the rare-disease assumption), so for uncommon disorders a case-control odds ratio stands in for a cohort relative risk.

16.7 Worked read of a 2x2: relative risk and odds ratio side by side

WORKED EXAMPLE

Setup. A cohort of 200 exposed and 200 unexposed people is followed. Among the exposed, 40 develop the disorder and 160 do not, so a = 40, b = 160. Among the unexposed, 10 develop it and 190 do not, so c = 10, d = 190.

Relative risk. Incidence in exposed = a / (a + b) = 40 / 200 = 0.20. Incidence in unexposed = c / (c + d) = 10 / 200 = 0.05. Relative risk = 0.20 / 0.05 = **4.0**, so the exposed are four times as likely to develop the disorder.

Odds ratio. Odds of disease in exposed = a / b = 40 / 160 = 0.25. Odds in unexposed = c / d = 10 / 190 = 0.0526. Odds ratio = ad / bc = (40 x 190) / (160 x 10) = 7600 / 1600 = **4.75**.

The lesson. With a disease this common (20 percent in the exposed) the odds ratio (4.75) overstates the relative risk (4.0): the odds ratio always lies further from 1 than the risk ratio. Make the disease rare (say 5 new cases in the exposed rather than 40) and the two converge, which is exactly the rare-disease approximation in action.

4.0	4.75	1
RELATIVE RISK (COHORT)	ODDS RATIO (CROSS-PRODUCT)	VALUE MEANING NO ASSOCIATION

16.8 Attributable risk and the population attributable risk

The excess risk, not the ratio of risks. Where relative risk and odds ratio are ratios, the attributable risk (use "attributable risk") is a difference: the excess risk of disease in the exposed

that can be attributed to the exposure, calculated as the incidence in the exposed minus the incidence in the unexposed (Kaplan & Sadock 2024). It answers "how much extra disease does this exposure add", and expressed as a fraction of the exposed incidence it becomes the attributable fraction, the share of disease in exposed people that the risk factor explains.

Attributable risk

Excess risk in the exposed = incidence in exposed minus incidence in unexposed.

Population attributable risk

The excess disease in the whole population attributable to the exposure; it depends on how common the exposure is, so a rare exposure yields a small population attributable risk however strong its relative risk.

Public-health use

If the exposure is truly causal, the population attributable risk estimates the disease reduction achievable by removing the exposure.

In the worked cohort, attributable risk = 0.20 minus 0.05 = **0.15**, meaning 15 excess cases per 100 exposed persons are attributable to the exposure; the ratio measures (RR, OR) tell you the strength of association, the attributable measures tell you the burden.

16.9 The measures assembled: formula, design, interpretation

One table to hold the whole toolkit. The examiner wants you to match each measure to its formula, the study design it is native to, and how to read it (Kaplan & Sadock 2024, Bland 2015).

MEASURE	FORMULA	DESIGN IT COMES FROM	INTERPRETATION
Point prevalence	cases at a point / population	**Cross-sectional survey**	Burden now; stock of existing cases
Period prevalence	cases in interval / population	**Cross-sectional / repeated survey**	Burden over an interval (1-month, 1-year, lifetime)
Cumulative incidence	new cases / at-risk at start	**Cohort**	Risk of new disease over the period
Incidence rate	new cases / person-time at risk	**Cohort**	Speed of new-case occurrence, per person-year
Relative risk	incidence exposed / incidence unexposed	**Cohort**	How many times more likely disease is in exposed; RR > 1 harmful
Odds ratio	ad / bc	**Case-control**	Approximates RR when disease is rare; OR > 1 associated with disease
Attributable risk	incidence exposed minus incidence unexposed	**Cohort**	Excess (extra) risk in the exposed due to exposure

MEASURE	FORMULA	DESIGN IT COMES FROM	INTERPRETATION
Population attributable risk	excess disease in population from exposure	**Cohort / population data**	Disease preventable by removing exposure; depends on exposure prevalence

Ratio measures (RR, OR) give strength of association; difference measures (attributable risk) give the excess burden; frequency measures (prevalence, incidence) give how common the disorder is.

16.10 The national numbers this material owns

Where the population figures come from. The flagship Indian frequency estimate is the National Mental Health Survey of India (2015 to 2016), a cross-sectional survey that reported a current weighted prevalence of any mental disorder of about **10.6** percent and a lifetime prevalence near **13.7** percent, with a treatment gap for mental disorders exceeding **70** percent (Gururaj et al 2016). These are prevalence numbers, a stock of existing cases at survey time, not incidence; they belong here as measures of frequency, while the disorder-by-disorder breakdown is carried in the clinical chapters of Paper II.

INDIA

The National Mental Health Survey of India (2015 to 2016) gives a current weighted prevalence of any mental disorder of about **10.6** percent (lifetime near **13.7** percent) and a treatment gap above **70** percent. Read these as point/period prevalence figures: they measure how many people have a disorder now, and because prevalence is roughly incidence times duration they cannot on their own tell you whether new cases are rising.

HOLD THIS

Incidence counts new cases over a period per population at risk; prevalence counts existing cases and equals roughly incidence times duration; relative risk comes from a cohort (incidence exposed / incidence unexposed) and the odds ratio (ad / bc) from a case-control, approximating RR only when the disease is rare.

MNEMONIC

PRIDE: Prevalence Rises with Increased Duration of Existing cases

Prevalence **R**ises with **I**ncreased **D**uration of **E**xisting cases. Because prevalence is roughly incidence times duration, longer illness inflates prevalence with incidence flat; incidence, by contrast, only ever counts the genuinely new.

PEARL

Match the measure to the design before you compute. A cohort hands you incidence, so you may quote a relative risk and an attributable risk. A case-control starts from cases and controls, so incidence is unavailable and you are restricted to the odds ratio, which you trust as a stand-in for relative risk only when the outcome is rare. Never quote a relative risk from a case-control study.

GOOD TO REMEMBER

- **Prevalence:** existing cases in a population over a time window; use it for burden and service planning; prevalence is roughly incidence times duration (P is proportional to I x d). Point prevalence = at one instant; period prevalence = over an interval.
- **Incidence:** new cases over a period per population at risk; use it for first-onset questions and to separate real rises from better survival; cumulative incidence = new cases / at-risk at start, incidence rate = new cases / person-time.
- **Relative risk:** how many times more likely disease is in the exposed; use it from a cohort; RR = incidence in exposed / incidence in unexposed; RR > 1 harmful, < 1 protective, = 1 no association.
- **Odds ratio:** the case-control measure of association; use it when incidence cannot be measured; OR = ad / bc; approximates the relative risk only when the outcome is rare.
- **Attributable risk:** the excess risk in the exposed caused by the exposure; use it to size the burden a risk factor adds; attributable risk = incidence in exposed minus incidence in unexposed; the population attributable risk scales this to the whole population and shrinks when the exposure is rare.

These measures are the shared currency of every observational and experimental design; the prevalence and incidence of individual psychiatric disorders, and the risk factors quantified by these ratios for each condition, are set out in the clinical chapters of Paper II, while the reliability and validity of the instruments used to define a case (as distinct from the frequency measures here) are covered in the Psychometry, Assessment and Descriptive Psychopathology notes.

17 · The National Mental Health Survey and the Indian picture

Every design and test taught earlier exists so that a number can be trusted. This topic is where those methods are spent on a single high-yield application: the National Mental Health Survey (NMHS) of India, 2015 to 2016, led by NIMHANS (Gururaj et al 2016). It is the one dataset an examiner expects an Indian resident to quote by heart, and it is the standing answer to any viva question that begins "what do we know about the burden of mental illness in India". Read it not as a table of prevalences to memorise blindly but as the country-level payoff of the **epidemiology of** mental disorders: a cross-sectional multistage survey applying the sampling logic and cross-sectional design covered earlier in this chapter.

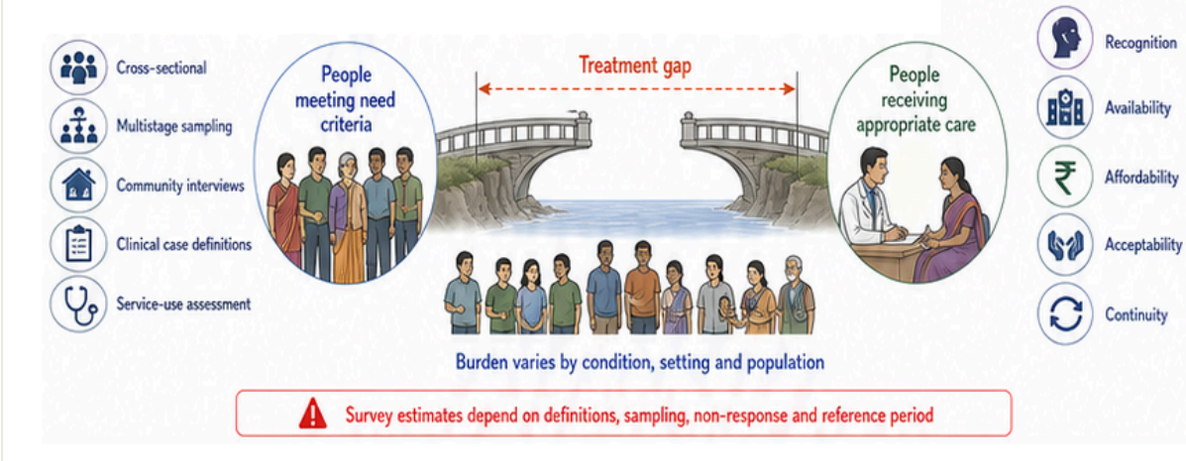


Fig 7.29 India's National Mental Health Survey: burden, care and gap.

The examiner's real interest, and the policy headline, is not how common illness is but how few of the ill are treated. The treatment gap, the proportion of people with a diagnosable disorder who receive no care, is the number that reframes Indian psychiatry from a clinical problem into a public-health one, and it is where the marks sit. Hold the prevalences, but lead with the gap.

17.1 What the NMHS is, and why it is the India device

The NMHS is India's first large multi-state national mental-health epidemiology survey. The **national mental health survey** was conducted across 12 states of India between 2015 and 2016, coordinated by the National Institute of Mental Health and Neuro Sciences (NIMHANS), Bengaluru, and published as NIMHANS Publication No. 129 (Gururaj et al 2016). Its purpose was to estimate the prevalence of mental disorders, describe their patterns and outcomes, and quantify the treatment gap and disability, so that mental-health policy and the District Mental Health Programme could be planned on **indian data** rather than borrowed Western figures. When a question asks for the burden of mental illness in India, this is the citation.

17.2 Methodology: a cross-sectional multistage survey

The NMHS is a prevalence (cross-sectional) survey built on multistage sampling. Methodologically the NMHS is an application of two designs taught earlier: it is a **cross-sectional** design (it measures disorder and non-disorder at one point in time, so it yields prevalence, not incidence, and cannot by itself establish that any exposure came first) delivered through a multistage random sampling frame (state, then district, then taluk or town, then village or ward, then household, then individual) so that a manageable sample can stand in for a vast and stratified population. For the anatomy of the sampling frame see the Sampling methods topic; for why a one-time snapshot gives prevalence and not causal direction see the Observational study designs topic in this chapter. Diagnoses were made with a structured diagnostic instrument (the MINI 6.0.0), the same family of fully structured interviews that anchors modern population psychiatry.

RULE **A cross-sectional survey gives prevalence, never incidence.** The NMHS tells you how many people are currently ill, not how many newly fall ill per year.

17.3 The headline prevalences: current and lifetime

Roughly one Indian in ten currently has a mental disorder; nearly one in seven has had one in their lifetime. The NMHS put the **current** prevalence of mental disorders (excluding tobacco use in most summaries) at **10.6%** (10.56% precisely) and the lifetime prevalence at **13.7%** (13.67%; Gautham et al 2020), drawn from 34,802 adults interviewed across 12 states and translating to an estimated 150 million Indians affected. Read plainly: about one adult in ten is unwell now, and close to one in seven has been unwell at some point. These two figures are the spine of the topic; quote them together and always with their source and survey years.

10.6%	13.7%
CURRENT PREVALENCE, ANY MENTAL DISORDER	LIFETIME PREVALENCE, ANY MENTAL DISORDER

TRAP **Never quote a prevalence bare.** A figure without its year and its source (NMHS 2015 to 2016) is unciteable and marks nothing; pair every number with survey and year.

17.4 The treatment gap: the policy headline

The Indian story is not that illness is common but that care is scarce. The **treatment gap** is the percentage of people who meet criteria for a disorder yet receive no treatment for it. Across mental disorders in India the NMHS found the overall treatment gap to be **84.5%** (Gautham et al 2020), varying widely by disorder. This is the single most examinable NMHS fact, because it is what drives policy: it is the argument for the National Mental Health Programme, task-shifting to primary care, and closing the psychiatrist shortfall (India has fewer than 10,000 psychiatrists against a desirable minimum of about 3 per 100,000 population; Kaplan and Sadock 2024).

RULE **Lead with the gap, not the prevalence.** The treatment gap, not prevalence alone, is the Indian policy headline; it is what an examiner is testing.

17.5 How the gap varies by disorder: the substance-use picture

The gap is widest for the substance-use and tobacco disorders. The treatment gap is not uniform: it is largest where stigma and normalisation are greatest. For substance use disorders overall the NMHS found a treatment gap of about **91.1%**, comprising **86.3%** for alcohol use disorder, **91.8%** for tobacco use disorder, and **72.9%** for other substance use disorders (Clinical Methods in Psychiatry 2024). The lesson to carry into the clinic and the exam is that the people least likely to be in treatment are precisely those with the addictions, which reframes screening and opportunistic case-finding as the front line.

91.1%	86.3%	91.8%	72.9%
TREATMENT GAP, SUBSTANCE USE DISORDERS	TREATMENT GAP, ALCOHOL USE DISORDER	TREATMENT GAP, TOBACCO USE DISORDER	TREATMENT GAP, OTHER SUBSTANCES

NMHS treatment gaps for the substance-use disorders, the group in which the gap is widest (Clinical Methods in Psychiatry 2024).

17.6 Setting India beside the World Mental Health survey frame

The NMHS is India's contribution to a global epidemiological project, not an isolated survey. The World Mental Health Survey Initiative is a suite of surveys conducted throughout the world using comparable sampling and diagnostic instruments (Tasman Psychiatry 2024), carried out in representative community samples in 17 countries across Africa, the Americas, Asia, Europe and the Middle East (Kaplan and Sadock 2024). Its two enduring lessons frame the Indian numbers: mental disorders are common everywhere, and the treatment gap is substantial worldwide and worst in low-income countries. The NMHS is best read as the Indian instance of exactly this pattern, using the same third-generation survey methodology (the ECA, the NCS and NCS-R, and the World Mental Health Survey Initiative, all built on structured instruments such as the CIDI; Tasman Psychiatry 2024). This is why "the treatment gap is global" is a defensible one-line summary and why India's overall gap of 84.5% is not an outlier but an intensification of the global picture.

WORKED EXAMPLE: READING A TREATMENT GAP

A treatment gap is a simple proportion. If a survey identifies 1,000 people who meet criteria for alcohol use disorder and finds that 137 of them have received any treatment for it, the treated proportion is 137 divided by 1000, which is 13.7%, so the treatment gap is 100% minus 13.7%, equals **86.3%**. That is exactly the NMHS alcohol-use-disorder figure. The arithmetic is trivial; the point of the calculation is that the gap is nothing more than one minus the proportion treated, and that a change in either the numerator (who counts as treated) or the denominator (who counts as a case) will move it. Always ask how "treated" and "case" were defined before comparing two gaps.

COMMON TRAP

Do not confuse the NMHS with the separate national drug-use survey. The frequently quoted figure that 14.6% of Indians aged 10 to 75 years use alcohol comes from the national survey on the magnitude of substance use in India, not from the NMHS; it is a use figure, not a disorder prevalence, and not a treatment gap. Mixing the two is a classic viva error. Keep the NMHS for disorder prevalence and treatment gap, and cite the substance-use magnitude survey separately if you quote its use figures.

INDIA

The whole topic is the India device for this chapter. The NMHS 2015 to 2016 (NIMHANS, 12 states) gives the numbers a resident must own: current prevalence **10.6%**, lifetime prevalence **13.7%**, and an overall treatment gap of **84.5%**, widest for the substance-use and tobacco disorders. It is the epidemiological justification for the District Mental Health Programme and for task-shifting mental-health care to primary care, given India's psychiatrist shortfall. When any Paper II clinical chapter needs an Indian prevalence for a specific disorder, the NMHS is the source to cite, but the disorder-specific prevalence must be looked up and quoted with its year, not guessed.

PEARL

Prevalence answers "how common", the treatment gap answers "how neglected". In India the second question carries the marks and the policy weight. A resident who can state the gap and explain why it drives task-shifting has understood the survey; one who only recites prevalences has memorised it.

MNEMONIC

GAP: Gururaj, All-India 12 states, Prevalence 10.6 now.

Gururaj (the lead author, Gururaj et al 2016) fixes the citation; All-India across 12 states fixes the scope; Prevalence 10.6% current (with 13.7% lifetime) fixes the headline number, so the letters of GAP also remind you that the gap itself is the point.

HOLD THIS

NMHS 2015 to 2016: current prevalence of any mental disorder 10.6%, lifetime 13.7%, and an overall treatment gap of 84.5%, which is the Indian policy headline.

GOOD TO REMEMBER

- **National Mental Health Survey (NMHS):** India's cross-sectional multistage prevalence survey (NIMHANS, 12 states, 2015 to 2016); use it for any Indian burden or treatment-gap figure; the two numbers to hold are current prevalence 10.6% and lifetime 13.7%.
- **Treatment gap:** percentage of people with a disorder who receive no care; 84.5% overall in India (widest for substance-use and tobacco); compute it as 100% minus the proportion treated.
- **World Mental Health Survey Initiative:** global suite of surveys using comparable sampling and structured diagnostic instruments (CIDI) across 17 countries; use it as the comparative frame that shows the treatment gap is global and worst in low-income settings.
- **Cross-sectional multistage design:** the NMHS method; use when you need population prevalence at one time point; remember it yields prevalence not incidence, and the one formula to know is prevalence equals cases divided by population sampled.

18 · Principles of screening

Screening is prevention masquerading as a test. **Screening** means testing apparently well people to detect disease early, before symptoms declare themselves, in the belief that earlier detection buys better outcomes (Wilson & Jungner 1968). The examiner rarely wants the mechanics of any one test; the question is whether a screening programme is justified at all, and whether an impressive-looking survival figure is real or an artefact. Two bodies of knowledge answer that: the Wilson and Jungner criteria, which decide when to screen, and the three screening biases, which explain why screening flatters itself. Master both and every screening vignette resolves cleanly (Wilson & Jungner 1968; Kaplan & Sadock 2024).

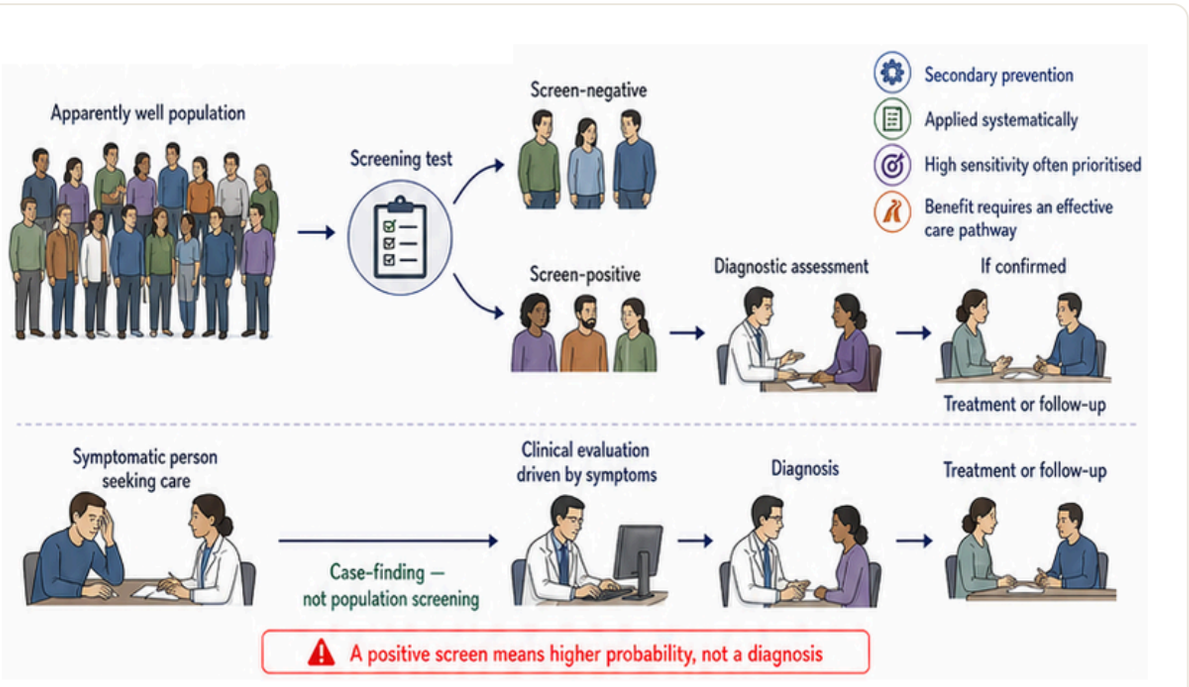


Fig 7.30 Screening is not diagnosis.

One framing distinction anchors the topic before the detail. Screening is applied to people with no complaint, so the base rate of true disease is low and the ethical bar is high: you are intervening on the well, and the harm of a false positive (anxiety, further invasive tests, overtreatment) falls on someone who was never going to be ill. That is the opposite of diagnostic testing, which is applied to the symptomatic, where prevalence is higher and the person has already sought help. This low pre-test probability is exactly why a screening test's positive predictive value collapses in the general population, a point developed in the Diagnostic test statistics topic and restated at 18.5.

18.1 What screening is, and how it differs from diagnosis

Testing apparently well people to detect disease early. Screening is the systematic application of a test to an asymptomatic population to identify those likely to have (or to be at high risk of) a disease, who are then referred for definitive diagnosis and treatment (Wilson & Jungner 1968). Two features define it. First, the target is the well, not the ill, so a screen is never itself a diagnosis: a positive screen selects people for a confirmatory diagnostic test, it does not label them with the disease. Second, the justification is downstream benefit, so a programme is only worth running if earlier detection actually changes the outcome, not merely the timing of the label. The distinction from case-finding (opportunistically testing patients already attending for another reason) and from surveillance is worth holding, but the core contrast the examiner wants is screen (asymptomatic, provisional) versus diagnose (symptomatic, definitive).

18.2 The Wilson and Jungner criteria (1968): the condition

Screen only a disease worth catching early. The classic Wilson and Jungner criteria, published for the World Health Organization in 1968, group the requirements of a sound screening programme, and the first group concerns the condition itself (Wilson & Jungner 1968). The condition must be an **important health problem** (common enough, or serious enough, to justify a population effort); it must have a **recognisable latent or early symptomatic stage** at which the screen can catch it before it declares clinically; and its **natural history must be adequately understood**, including the progression from latent to overt disease, so that we know that intercepting the latent stage genuinely alters the course. A disease with no detectable pre-clinical window, or one whose natural history is a mystery, fails at this first hurdle.

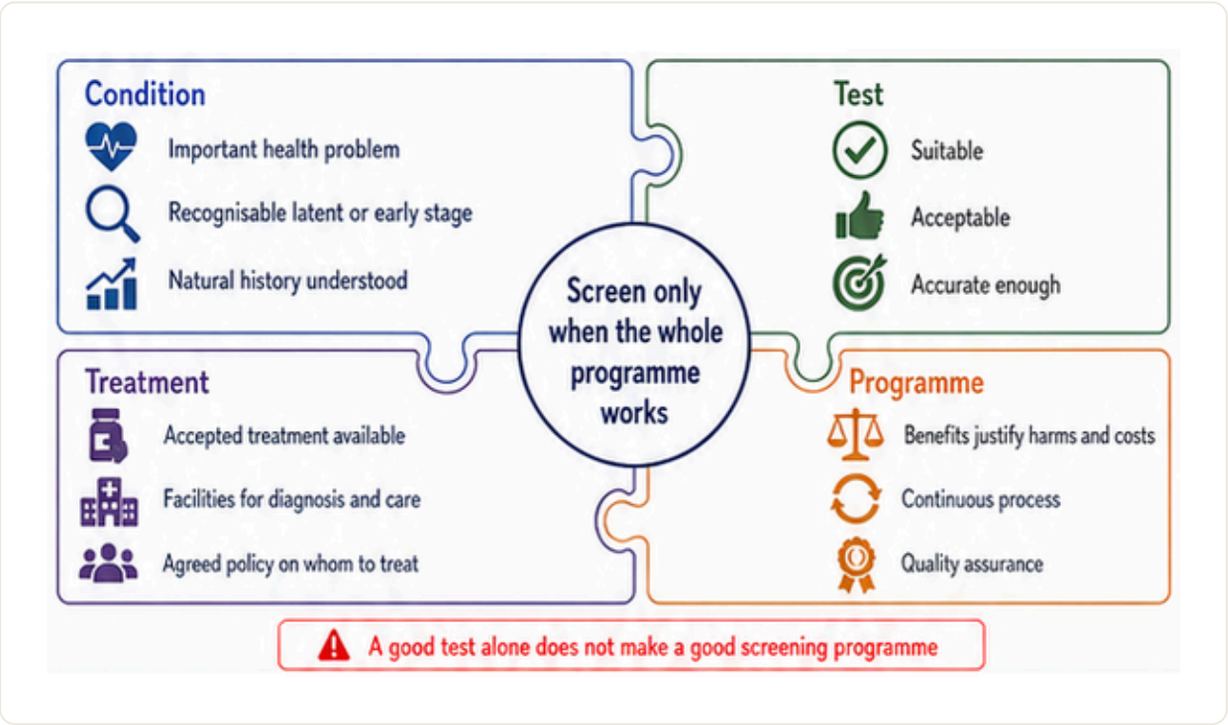


Fig 7.31 Wilson and Jungner: when is screening worth doing?.

18.3 The Wilson and Jungner criteria: the test, the treatment and the programme

A suitable test, an accepted treatment, and an organised, cost-effective programme. The remaining Wilson and Jungner groups concern the test, the treatment and the programme (Wilson & Jungner 1968). The **test** must be suitable (valid, with good sensitivity and specificity), simple, safe and acceptable to the population, since a screen the public will not attend screens no one. The **treatment** must exist and be accepted: there must be an effective, agreed treatment for patients found, together with the facilities to diagnose and treat them, because detecting a disease you cannot help merely inflicts foreknowledge. The **programme** must be organised: an agreed policy on whom to treat (an agreed cut-off and referral pathway), a cost that is balanced against benefit for the whole system (cost-effectiveness), and, critically, screening understood as a continuing process, not a one-off campaign, repeated at an interval matched to the disease's natural history. Later frameworks (the UK National Screening Committee, and a 2008 WHO update) refine these, but the 1968 list remains the examinable canon.

RULE **Screen with a sensitive test, confirm with a specific one.** The first-line screen is tuned for high sensitivity to miss few true cases; every positive is then re-tested with a specific diagnostic test to weed out the false positives.

18.4 The sensitivity-specificity trade-off in a screening test

A screening test is deliberately tuned for high sensitivity. Because the cost of missing a true case (a false negative reassurance) is usually judged worse than the cost of a false alarm, the sensitivity of a screening test is set high, at the price of lower specificity and therefore more false positives (Kaplan & Sadock 2024). Those positives are not treated on the strength of the screen; they pass to a second, more **specific** diagnostic test that confirms or excludes the disease. This two-stage architecture, a sensitive sieve followed by a specific confirmation, is the standard

design of a screening pathway (for example a sensitive immunoassay confirmed by a specific test, or a screening questionnaire confirmed by a structured clinical interview). Moving the cut-off trades the two errors against each other: a more lenient threshold raises sensitivity and lowers specificity; a stricter one does the reverse. The receiver operating characteristic curve, treated in its own topic, is the formal picture of that trade-off.

18.5 Low prevalence wrecks the positive predictive value

In an asymptomatic population, most positives are false. Sensitivity and specificity are intrinsic to the test and do not move with prevalence, but the positive predictive value (the chance a screen-positive person truly has the disease) is a hostage to prevalence: as prevalence falls, PPV falls (Kaplan & Sadock 2024). Screening operates precisely where prevalence is lowest, the general well population, so even an excellent test throws up a flood of false positives, and a positive screen means far less than the same result in a symptomatic clinic. The full 2x2 worked calculation (a good test whose PPV collapses from about **50%** at **10%** prevalence to roughly **8%** at **1%** prevalence) is carried in the Diagnostic test statistics topic; here, hold the consequence: a programme that looks superb in a high-risk clinic can generate mostly false alarms once rolled out to the low-prevalence general population, which is why the confirmatory specific test is not optional.

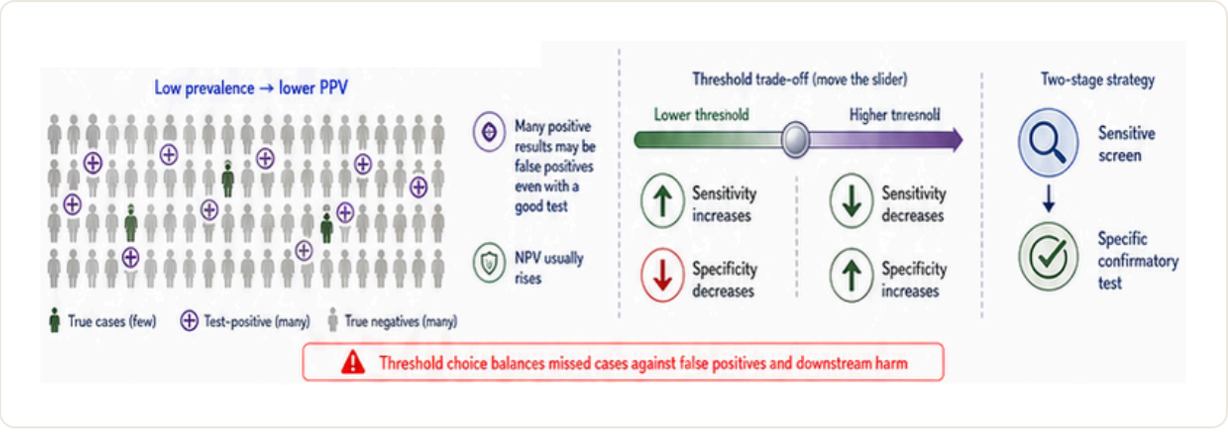


Fig 7.32 Low prevalence and the screening threshold.

18.6 Lead-time bias: earlier does not mean longer

Diagnosing earlier lengthens apparent survival without postponing death. Lead-time bias is the spurious improvement in measured survival that arises simply because screening advances the moment of diagnosis (Kaplan & Sadock 2024). Survival is timed from diagnosis to death; if a screen detects the disease two years before it would have surfaced clinically, but the patient dies at exactly the same moment as they would have unscreened, then the survival clock has been started earlier and reads two years longer, even though no survival time was actually gained. The disease was found sooner, so the patient merely spends longer knowing they have it. Any screening series compared with a clinically-diagnosed series will show longer survival for this reason alone, before any real benefit is considered. The defence is to judge screening by mortality (deaths in the whole screened population versus the unscreened) rather than by survival-from-diagnosis, and to use a randomised trial with mortality as the endpoint.

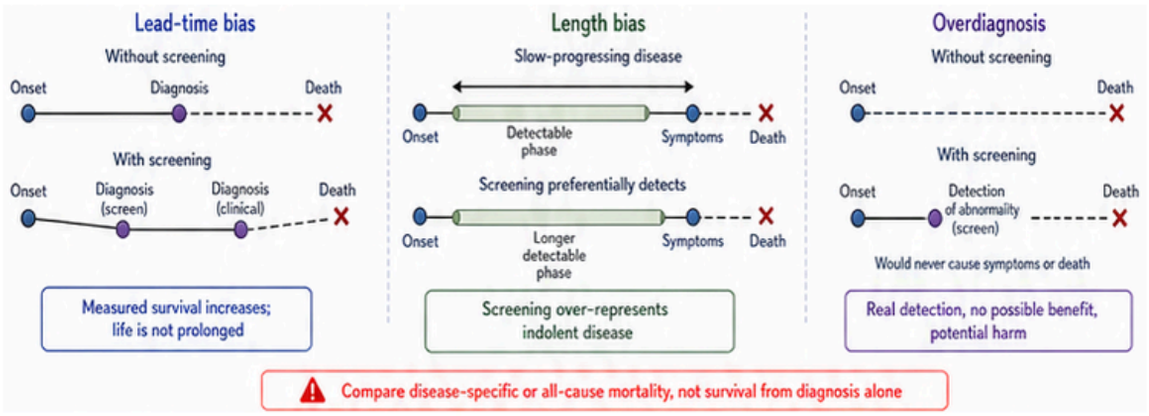


Fig 7.33 Screening biases: apparent benefit without true benefit.

TRAP **Improved survival in a screening series can be pure lead-time bias.** Longer survival-from-diagnosis after screening does not prove lives are saved; only a fall in population mortality does.

18.7 Length bias: the screen catches the indolent

Screening preferentially catches slow, indolent disease. Length bias (also called length-time bias) arises because a periodic screen is more likely to catch diseases that spend a long time in the detectable pre-clinical phase, and those slow-growing cases are, by their nature, the more indolent, better-prognosis ones (Kaplan & Sadock 2024). Fast, aggressive disease passes quickly from undetectable to symptomatic and often surfaces clinically in the gap between screening rounds, so it is under-represented among screen-detected cases. The screened group is therefore enriched for slow, benign disease and looks to do better, but the advantage is a sampling artefact, not a treatment effect. Length bias explains part of the apparent good prognosis of screen-detected cancers and is a cousin of lead-time bias: both make the screened group appear to fare better without any real extension of life.

18.8 Overdiagnosis: the extreme of length bias

Detecting disease that would never have caused harm. Overdiagnosis is the detection, by screening, of a genuine abnormality that met the pathological definition of disease but that would never have progressed to cause symptoms or death in the person's lifetime (Kaplan & Sadock 2024). It is the limiting case of length bias: the disease is so indolent that the patient would have died of something else first. Overdiagnosis is not misdiagnosis, the lesion is real, but the diagnosis is useless or harmful, because the person is then subjected to the anxiety, investigation and treatment of a disease that was never going to trouble them (overtreatment). It inflates apparent survival and incidence figures (more cases detected, all surviving) while doing pure harm to the individual. It is the strongest ethical argument for restraint in screening and the reason every programme must weigh benefit against the harm done to those who could never have benefited.

WORKED EXAMPLE

Two towns, identical disease biology. Town A is screened, Town B is not. Screen-detected patients in Town A are diagnosed on average 3 years earlier and their mean survival-from-diagnosis is 8 years; Town B's clinically-diagnosed patients survive 6 years from diagnosis. It looks like screening added 2 years. But every patient in both towns dies at the same age: the 2-year gap is exactly the lead time (3 years advanced diagnosis, offset by the different case mix), so it is lead-time bias, not benefit. On top of that, Town A's screened cases include several slow, indolent tumours that Town B's clinical presentation would have missed (they surfaced between rounds or never), so Town A's series is further enriched by length bias, and a handful of those lesions would never have harmed anyone at all (overdiagnosis). The only honest comparison is total mortality across each whole town, which shows no difference. Survival-from-diagnosis was the wrong yardstick.

18.9 Judging a screening programme honestly

Mortality in a randomised trial, not survival in a case series. Because lead-time, length and overdiagnosis all inflate survival-from-diagnosis, the correct evaluation of screening compares disease-specific (and ideally all-cause) mortality between a screened and an unscreened population, allocated at random so the two arms are otherwise alike (Kaplan & Sadock 2024). Randomisation defeats length bias (both arms get the same case mix) and the mortality endpoint defeats lead-time bias (the clock is not reset by earlier diagnosis). The benefits of a programme are then weighed against its harms, the false positives and their cascade of tests, the overdiagnosed and overtreated, the cost, and the anxiety inflicted on the well, exactly the balance the Wilson and Jungner programme criteria demand. A screening test that improves survival figures but not population mortality has proved nothing.

HOLD THIS

Screen with a sensitive test and confirm with a specific one; and never trust survival-from-diagnosis in a screened series, because lead-time bias, length bias and overdiagnosis all inflate it, so judge screening only by mortality in a randomised comparison.

BIAS	WHAT HAPPENS	WHY THE SCREENED GROUP LOOKS BETTER	DEFENCE
Lead-time bias	**Diagnosis advanced, death unchanged**	Survival is timed from an earlier diagnosis, so it reads longer without any life gained	Use mortality, not survival-from-diagnosis; randomised trial
Length bias	**Slow disease oversampled**	Periodic screens preferentially catch indolent, long-pre-clinical, good-prognosis cases	Randomised allocation equalises case mix between arms
Overdiagnosis	**Non-progressive disease detected**	Real but harmless lesions are counted as cured cases, inflating incidence and survival	Weigh benefit against overtreatment harm; long-term mortality

The three screening biases: each makes a screened series look better than it is; the hit column is the mechanism to name in a stem.

1968	3	8%
WILSON & JUNGNER YEAR	SCREENING BIASES TO NAME	PPV AT 1% PREVALENCE

COMMON TRAP

Two errors the examiner rewards. First, quoting improved five-year survival as proof that a screen saves lives: that figure is exactly what lead-time and length bias inflate, and only a fall in population mortality is proof. Second, treating a positive screen as a diagnosis: a screen is a sensitive sieve, not a specific verdict, and in a low-prevalence population most of its positives are false until a specific confirmatory test says otherwise.

MNEMONIC

The three biases spell L-L-O: Lead-time, Length, Overdiagnosis.

Lead-time advances the diagnosis clock; Length over-samples slow disease; Overdiagnosis catches disease that never mattered. All three inflate the screened group's apparent survival; none of them extends a single life; only population mortality tells the truth.

INDIA

In a low-resource, high-population setting the Wilson and Jungner programme criteria bite hardest: a screen is only defensible if the confirmatory diagnostic test and the accepted treatment are actually available and affordable at scale, and if the programme can be sustained as a continuing process rather than a one-off camp. Screening for a condition whose treatment or follow-up cannot be delivered breaches the treatment and programme criteria and merely generates anxiety and cost.

GOOD TO REMEMBER

- **Screening:** testing apparently well (asymptomatic) people to detect disease early; a sensitive sieve that selects people for a confirmatory diagnostic test, never a diagnosis itself; justified only if earlier detection improves the outcome, not just its timing.
- **Wilson & Jungner criteria (1968):** when to screen; four groups, the condition (important, recognisable latent stage, understood natural history), the test (suitable, acceptable), the treatment (accepted treatment plus facilities), and the programme (agreed policy on whom to treat, cost-effective, a continuing process); the WHO canon for a justified programme.
- **Screening test trade-off:** tuned for high sensitivity, accepting more false positives, then confirmed by a specific diagnostic test; the one number to hold is that PPV falls as prevalence falls, so most positives in the general population are false.
- **Lead-time bias:** earlier diagnosis lengthens apparent survival without postponing death; suspect it whenever survival-from-diagnosis improves; defeated by using mortality as the endpoint in a randomised trial.
- **Length bias:** screening preferentially catches slow, indolent, good-prognosis disease; suspect it when screen-detected cases look benign; defeated by randomised allocation equalising case mix.
- **Overdiagnosis:** detecting disease that would never have caused harm; the extreme of length bias; the strongest argument for restraint; weighed against benefit through long-term population mortality.

Discriminate

19 · Discriminate: pick the design, pick the test

This is the payoff. Every research vignette in the examination reduces to two acts of discrimination: **which design** answers the question that was asked, and **which test** reads the data that came back. The first act is **choosing a** study design from a clinical question; the second is **test selection** from the shape of the data. The examiner rarely asks you to compute anything by hand. What is tested is whether you can move cleanly from a one-line stem to the right design, and from a described dataset to the correct statistical test, and whether you know why the wrong pick is wrong (Bland 2015; Altman 1991; Johnstone et al 2010). This topic sets out the two selectors side by side, because in practice a question flows through both in turn: the design fixes what data you will have, and that data fixes which test is legitimate.

Think of it as two interleaved lookup tables. PART A, the study-design chooser, turns a **research question** into a design, and its axis is the *nature of the question*: prevalence, a rare outcome, a rare exposure, an intervention, the totality of evidence, or meaning and experience. PART B, the statistical-test chooser, turns the data into a test, and its axes are the *data type* (categorical versus continuous), the *number of groups* (one, two, three or more), and whether the observations are *paired or unpaired*. Hold both grids and almost every methods stem falls open.

data type \ comparison	Two groups unpaired	Two groups paired	Three or more groups	Association of two variables
Continuous, normal (parametric)	Independent t-test	Paired t-test	ANOVA (then post-hoc)	Pearson r linear regression
Ordinal or non-normal (non-parametric)	Mann-Whitney U	Wilcoxon signed-rank	Kruskal-Wallis	Spearman rho
Categorical counts (proportions)	Chi-square (Fisher if small)	McNemar	Chi-square	Chi-square logistic regression

Fig 7.34 Choosing the right statistical test. Read down to the type of your outcome data and across to the comparison you are making, and the cell names the test. Continuous normal data take the parametric tests (t-test, ANOVA, Pearson); ordinal or non-normal data take their non-parametric twins (Mann-Whitney, Wilcoxon, Kruskal-Wallis, Spearman); categorical counts take the chi-square family. This one grid, read with the study-design chooser, is the practical heart of the whole subject.

19.1 The design-choice logic: read the question, not the data

The question dictates the design. Before any data exist, the **design-choice** is settled by what the investigator wants to know and by what is rare or costly to observe (Bland 2015). Four sub-questions run in order. First, is this an *intervention* question (does a treatment work?) or an *observation* question (is this exposure associated with this outcome?): intervention licenses a trial, observation sends you to the observational family. Second, for observation, what is the *direction of enquiry*: from outcome back to exposure, or from exposure forward to outcome? Third, what is *rare*, the outcome or the exposure, since that decides between the two comparative designs. Fourth, does the question ask about the *current burden*, the *totality of the evidence*, or the lived *meaning* of an experience, each of which has its own home. The exact-string task here is naming **which design** fits the vignette.

RULE **Match the question to the design.** A prevalence question to cross-sectional, a rare outcome to case-control, a rare exposure or incidence question to cohort, an efficacy question to a randomised controlled trial, an evidence-synthesis question to systematic review and meta-analysis, a meaning question to qualitative.

19.2 PART A: the study-design chooser

Question in, design out. This is the first selector: read the clinical question in the left column and land on the design in the hit column (question to design). Rarity and temporality do most of the sorting; the last three rows sit apart because they answer questions of totality of evidence and of meaning rather than of individual-level association (Bland 2015; Altman 1991; Greenhalgh 2019).

RESEARCH QUESTION (THE VIGNETTE)	DIRECTION / UNIT	DESIGN TO PICK	MEASURE IT YIELDS
How common is the condition right now?	Snapshot, one time point	**Cross-sectional**	Prevalence, prevalence ratio
What are the risk factors for a rare outcome (or a long-latency disease)?	Outcome to exposure (backward)	**Case-control**	Odds ratio (OR)
What are the effects of a rare exposure, or the incidence of an outcome?	Exposure to outcome (forward)	**Cohort**	Incidence; relative risk (RR)
Must I prove the exposure preceded the outcome (temporality)?	Exposure to outcome, real time	**Prospective cohort**	Incidence; relative risk (RR)
Does this intervention actually work (efficacy)?	Investigator allocates exposure	**Randomised controlled trial**	Risk ratio, absolute risk reduction, NNT
What does the totality of the evidence say?	Studies as the unit	**Systematic review and meta-analysis**	Pooled effect size (95% CI)

RESEARCH QUESTION (THE VIGNETTE)	DIRECTION / UNIT	DESIGN TO PICK	MEASURE IT YIELDS
What is the meaning or lived experience of this?	Words, not numbers	**Qualitative**	Themes; no effect measure
Only aggregate, population-level data are available?	Group is the unit	**Ecological (mind the fallacy)**	Correlation of population rates

Study-design chooser: read the research question, pick the design (question to design); rarity and temporality do the sorting.

TRAP **Rare exposure is not rare outcome.** A case-control study is efficient for a rare *outcome*, a cohort for a rare *exposure*; swapping them is the classic design-choice error.

19.3 The intervention branch: the randomised controlled trial

When the question is efficacy. If the vignette asks whether a treatment *works*, and the investigator can *allocate* the exposure, the answer is the randomised controlled trial (Bland 2015). Randomisation is what separates it from every observational design: allocating treatment by chance balances known and unknown confounders across arms, so a difference in outcome can be attributed to the intervention. The other pillars, allocation concealment, blinding, and intention-to-treat analysis, protect that internal validity downstream. It is the strongest single design for a causal claim about an intervention, which is why the efficacy question routes here and nowhere else.

19.4 The evidence-synthesis branch: systematic review and meta-analysis

When the question is the totality of evidence. If the vignette asks what *all* the studies together show, the design is a systematic review, and where the studies are combined numerically, a meta-analysis (Greenhalgh 2019). A systematic review answers a focused question with a pre-specified, reproducible search and appraisal of every eligible study; the meta-analysis then pools their effect sizes into a single weighted estimate with a narrower confidence interval than any one study could give. It sits at the apex of the evidence hierarchy because it is the design whose unit of analysis is the study itself. Its validity depends wholly on the quality of the studies it pools and on whether publication bias has hidden the negative ones.

19.5 The meaning branch: qualitative designs

When the question is meaning, not magnitude. If the vignette asks about *why*, about experience, or about the meaning people attach to an illness or a service, no numerical design fits: the answer is a qualitative design (Greenhalgh 2019). Qualitative research works in words, gathered by in-depth interview, focus group or observation, and analysed by approaches such as thematic analysis, grounded theory or interpretative phenomenological analysis. It yields themes and understanding, not an effect size or a p-value, and it is judged by trustworthiness criteria (credibility, transferability, dependability, confirmability) rather than by statistical power. It is the correct pick whenever the stem asks about lived experience or meaning, and the wrong pick whenever the stem asks how many or how much.

19.6 The test-selection logic: read the data

The data dictate the test. Once a design has produced data, **test selection** turns on three facts and three facts only (Johnstone et al 2010; Altman 1991). First, the *data type*: is the outcome categorical (counts in categories) or continuous (measured on a scale), and if continuous, is it approximately normal? Second, the *number of groups* being compared: one, two, or three or more? Third, are the observations *paired* (the same subjects measured twice, or matched pairs) or *unpaired* (independent groups)? Read those three off the stem and the **statistical test** is fixed. The parametric tests (which assume an underlying normal distribution and compare means) each have a non-parametric twin (which works on ranks and assumes no distribution) for ordinal or non-normal data.

RULE **Data type x groups x pairing.** Those three coordinates alone pick the statistical test; continuous-normal goes parametric, ordinal or non-normal goes to the rank-based twin, categorical goes to chi-square.

19.7 PART B: the statistical-test chooser

Data in, test out. This is the second selector. Read across the data type, the number of groups, and the pairing to reach the correct **statistical test** in the hit column. Two independent continuous-normal groups give the independent t-test; two paired give the paired t-test; three or more give ANOVA; their non-parametric twins are Mann-Whitney, Wilcoxon and Kruskal-Wallis; two categorical variables give chi-square; an association of two continuous variables gives Pearson (or Spearman if ordinal or non-normal) (Johnstone et al 2010; Altman 1991).

DATA TYPE	NUMBER OF GROUPS	PAIRED OR UNPAIRED	CORRECT STATISTICAL TEST
Continuous, normal	One sample vs known value	n/a (single sample or a pairwise association)	**One-sample t-test**
Continuous, normal	Two	Unpaired (independent)	**Independent t-test**
Continuous, normal	Two	Paired / matched	**Paired t-test**
Continuous, normal	Three or more	Unpaired (independent)	**ANOVA (one-way)**
Continuous, normal	Three or more	Paired / repeated	**Repeated-measures ANOVA**
Continuous, non-normal / ordinal	Two	Unpaired (independent)	**Mann-Whitney U**
Continuous, non-normal / ordinal	Two	Paired / matched	**Wilcoxon signed-rank**
Continuous, non-normal / ordinal	Three or more	Unpaired (independent)	**Kruskal-Wallis**
Continuous, non-normal / ordinal	Three or more	Paired / repeated	**Friedman test**

DATA TYPE	NUMBER OF GROUPS	PAIRED OR UNPAIRED	CORRECT STATISTICAL TEST
Categorical (counts)	Two variables, two or more groups	Unpaired	**Chi-square test**
Categorical (counts), small cells	Two variables, 2x2	Unpaired	**Fisher exact test**
Categorical (counts)	Two variables, 2x2	Paired / before-after	**McNemar test**
Two continuous variables	Association (normal)	n/a (single sample or a pairwise association)	**Pearson correlation**
Two continuous / ordinal variables	Association (non-normal)	n/a (single sample or a pairwise association)	**Spearman rank correlation**

Statistical-test chooser: data type by number of groups by paired/unpaired, resolving to the correct parametric test or its non-parametric twin (categorical drops to Fisher exact when any expected cell is under 5).

TRAP **Three groups is not a job for the t-test.** Running a t-test on every pair of three or more groups inflates the type I error; use ANOVA (or Kruskal-Wallis) once, then post-hoc tests.

19.8 The two-selector worked walk-through

WORKED EXAMPLE

Design, then test, in one vignette. A team asks whether a new brief psychotherapy lowers depression scores more than treatment as usual. The question is *efficacy* and the investigator can *allocate* the therapy, so PART A resolves to a randomised controlled trial. The trial randomises 120 patients to two arms and records the continuous HAM-D score at 12 weeks. Now PART B: the outcome is *continuous*, roughly *normal*, there are *two* groups, and they are *independent* (different patients in each arm), which lands on the independent t-test. Say instead each patient's HAM-D is compared before and after within the same arm: that is *two paired* measurements, so the test becomes the paired t-test. Add a third arm and compare all three endpoints at once and it becomes a one-way ANOVA. Read a 2x2 of responders vs non-responders across the two arms instead, and the counts route to a chi-square test. One vignette, both selectors, four different tests depending only on data type, group number and pairing.

19.9 The classic mix-ups the examiner sets

Four errors sit at the seam of the two selectors. Each is a discrimination the stem deliberately blurs (Bland 2015; Altman 1991).

Odds ratio vs relative risk

A case-control study yields only an odds ratio; the relative risk needs a cohort with a denominator followed over time. The OR approximates the RR only when the outcome is rare.

Sensitivity vs positive predictive value

Sensitivity is a property of the test (proportion of the diseased it catches) and is prevalence-independent; PPV is a property of the setting (proportion of positives who are truly diseased) and falls as prevalence falls.

t-test where ANOVA is needed

Multiple pairwise t-tests across three or more groups inflate the false-positive rate; one ANOVA with post-hoc correction is the legitimate route.

Correlation claimed as causation

A significant Pearson or Spearman correlation quantifies association, not cause; only a design that controls confounding and establishes temporality (ideally a trial) supports a causal claim.

COMMON TRAP

These four mix-ups recur every year. Reporting a *relative risk* from a case-control study (it gives an odds ratio); reading a high *sensitivity* as a high *positive predictive value* (they diverge as prevalence changes); running a *t-test* across three or more groups where *ANOVA* is required (multiplicity inflates type I error); and reading a *correlation* as *causation* (association is not cause). Each is a failure to discriminate at exactly the point the two selectors meet.

19.10 The rare-disease bridge between the two selectors

PEARL

The two selectors are linked by the rare-disease assumption. When the outcome is rare, the odds ratio from a case-control design (PART A) closely approximates the relative risk you would have obtained from a cohort, and the chi-square that tests that 2x2 table (PART B) behaves well because expected cell counts stay large enough. As the outcome becomes common, both approximations fray: the OR exaggerates the RR, and small or lopsided cells push you toward the Fisher exact test. Rarity, the same axis that steers the design choice, quietly governs which test is trustworthy on the data that design returns.

MNEMONIC

QUESTION picks the DESIGN; DATA picks the TEST.

Two selectors, two inputs. Read the **question** (prevalence, rare outcome, rare exposure, efficacy, totality of evidence, meaning) to pick the design; read the **data** (type x number of groups x paired?) to pick the test. Never read the data to pick the design or the question to pick the test.

HOLD THIS

The question picks the design (prevalence to cross-sectional, rare outcome to case-control, rare exposure or incidence to cohort, efficacy to RCT, totality of evidence to systematic review and meta-analysis, meaning to qualitative); the data pick the test (data type x number of groups x paired/unpaired: two continuous-normal independent to independent t-test, paired to paired t-test, three or more to ANOVA, their twins Mann-Whitney / Wilcoxon / Kruskal-Wallis, two categorical to chi-square, two continuous associated to Pearson or Spearman).

GOOD TO REMEMBER

- **Cross-sectional:** the design for a prevalence question; use it to count current cases in a snapshot; yields prevalence = existing cases / population at that time.
- **Case-control:** the design for a rare outcome's risk factors; use it to look back from cases and controls to exposure; yields the odds ratio, OR = (odds of exposure in cases) / (odds of exposure in controls).
- **Cohort:** the design for a rare exposure's effects or for incidence; use it to follow exposed vs unexposed forward; yields incidence and relative risk, RR = (incidence in exposed) / (incidence in unexposed).
- **Randomised controlled trial:** the design for an intervention's efficacy; use it when the investigator can allocate the treatment; randomisation balances confounders and yields a risk ratio, absolute risk reduction and NNT = 1 / absolute risk reduction.
- **Systematic review and meta-analysis:** the design for the totality of evidence; use it to pool a focused question across all eligible studies; yields a single weighted pooled effect size with a 95% confidence interval.
- **Qualitative:** the design for meaning and experience; use it when the stem asks why or about lived experience; yields themes, judged by trustworthiness, with no effect measure or p-value.
- **Independent t-test:** the test for two independent continuous-normal groups; use it to compare two group means; two-sample t has n1 + n2 − 2 degrees of freedom.
- **Paired t-test:** the test for two paired continuous-normal measurements; use it for before-after or matched pairs; it tests the mean of the within-pair differences.
- **ANOVA:** the test for three or more continuous-normal group means; use it to avoid multiple t-tests; its statistic is F = MS(between) / MS(within).
- **Mann-Whitney / Wilcoxon / Kruskal-Wallis:** the non-parametric twins of the independent t-test, paired t-test and ANOVA respectively; use them on ordinal or non-normal data; they work on ranks, not means.
- **Chi-square:** the test for the association of two categorical variables; use it on absolute counts, never percentages; statistic = sum of (O − E)squared / E, dropping to the Fisher exact test when any expected cell is under **5**.
- **Pearson / Spearman:** the test for the association of two continuous variables; use Pearson when both are normal, Spearman (rank-based) when ordinal or non-normal; each returns a correlation coefficient r between −1 and +1, which is association and not causation.

For design selection and worked derivations see Bland (2015), Altman (1991) and Johnstone et al (2010); for the qualitative and evidence-synthesis branches see Greenhalgh (2019). Twin, family and adoption designs used to partition genetic and environmental variance are treated separately in the Psychiatric Genetics notes; the reliability and validity of a psychometric instrument, as distinct from the validity of a study design, are covered in the Psychometry, Assessment and Descriptive Psychopathology notes; the disorder-specific applications of these designs and tests appear in the clinical chapters of Paper II.

Apply and consolidate

20 · Apply: four worked appraisal vignettes

Everything in the research-methods and biostatistics material converges on a single skill the examiner can actually test in an OSCE station or a short-answer stem: put a piece of evidence in

front of the candidate and ask them to read it correctly. This topic drills that skill through four short worked appraisal vignettes, each a compact scenario followed by the reasoning and then the read. The four moves recur across every viva: read a **forest plot**, choose the right statistical test for a described dataset, compute an index from a diagnostic **2x2 table**, and interpret a **p-value** and a **confidence interval** without over-claiming (Bland 2015, Altman 1991).

How to use these. Treat each vignette as a self-contained worked example: cover the read, work the reasoning yourself, then check. The arithmetic in vignette three is fully worked and verified so you can reproduce it under pressure; the other three are conceptual reads where the trap is almost always the same, over-trusting a result the data cannot support (Sackett et al 2000). Every number quoted below has been checked by hand.

20.1 Vignette one: read a forest plot

WORKED EXAMPLE

Case: A meta-analysis pools eight randomised trials of an add-on agent for depression. Each trial is a horizontal line (its point estimate and 95% confidence interval) stacked down the plot; a vertical line marks the no-effect value, and a diamond at the foot marks the pooled estimate. The pooled diamond sits to the left of the no-effect line but its right tip just crosses it. The reported heterogeneity is I-squared = **72%**.

Reasoning. Each study line's width shows that study's precision; a wider line is a smaller or noisier trial. The forest plot diamond is the fixed or random-effects pooled estimate, and its width is the 95% confidence interval of that pooled estimate. Whether the diamond crosses the vertical no-effect line is the whole verdict: for a ratio measure (odds ratio, relative risk) the no-effect value is **1**; for a difference measure (mean difference) it is **0**. Here the diamond crosses the null line, so the pooled effect is not statistically significant at the 0.05 level even though the central estimate favours the drug.

The read. A diamond that crosses the null line means the meta-analysis has not demonstrated a significant pooled effect. Then heterogeneity changes your confidence in that pooled number: I-squared of 72% is high (a common ladder reads under 25% as low, around 50% as moderate, over 75% as high), meaning most of the variation across trials is real between-study difference, not chance. High heterogeneity means the single diamond hides genuinely different underlying effects, so the pooled estimate is less trustworthy and a random-effects model with wider intervals is the honest summary (Higgins et al 2003, Bland 2015).

RULE **Cross the null, lose significance.** A forest-plot diamond touching the no-effect line (1 for ratios, 0 for differences) means a non-significant pooled result.

TRAP **Pooling despite high heterogeneity.** A tidy diamond over an I-squared of 72% is a false comfort: the trials disagree, so the single number oversimplifies.

20.2 Vignette two: choose the test

WORKED EXAMPLE

Case: A trial randomises patients to three treatment arms (drug A, drug B, placebo) and measures a continuous symptom score, the change on a depression rating scale, at eight weeks. The question is whether mean symptom change differs across the three arms. In this vignette the score is roughly normally distributed within each arm and variances are comparable.

Reasoning. Walk the chooser: the outcome is continuous, the groups are independent (different patients per arm), and there are three groups, not two. Three independent groups on a normal continuous outcome is the textbook indication for one-way ANOVA (analysis of variance), whose F statistic is the ratio of between-group to within-group variance (Johnstone et al 2010). A significant ANOVA then licenses post-hoc pairwise comparisons (Tukey or Bonferroni) to locate which arms differ.

Why a t-test would be wrong. A t-test compares only two means. To cover three arms you would run three separate t-tests (A vs B, A vs placebo, B vs placebo), and each carries its own **0.05** chance of a false positive, so the family-wise error inflates well above 5%. ANOVA tests all three at once at a single controlled alpha, which is exactly why it exists.

The non-parametric twin. If the same score were skewed or ordinal (assumptions of normality failing), swap ANOVA for its rank-based twin, the Kruskal-Wallis test, which compares three or more independent groups on medians rather than means. The two-group non-parametric equivalent (had there been only two arms) would have been Mann-Whitney U in place of the t-test.

TRAP **Serial t-tests for three arms.** Three pairwise t-tests inflate the false-positive rate; use ANOVA (or Kruskal-Wallis if non-normal) for three or more groups.

20.3 Vignette three: compute from a 2x2

WORKED EXAMPLE

Case: A brief screening questionnaire for a disorder is validated against a structured interview (the reference standard) in a clinic of 1000 attenders where the disorder prevalence is 10%. The 2x2 table comes out as below. Work out the sensitivity and the positive predictive value, then say what happens to the PPV if the same test is used where the disorder is rarer.

SCREEN RESULT	DISORDER PRESENT	DISORDER ABSENT	ROW TOTAL
Screen positive	80 (a)	90 (b)	**170**
Screen negative	20 (c)	810 (d)	**830**
Column total	100	900	**1000**

Prevalence 10%: 100 diseased, 900 non-diseased among 1000 attenders.

Sensitivity. Read down the disorder-present column: sensitivity = a / (a + c) = 80 / (80 + 20) = 80 / 100 = **0.80**, that is 80%. The screen detects 80% of true cases; it misses 20% (the false-negative rate).

Positive predictive value. Read across the screen-positive row: PPV = a / (a + b) = 80 / (80 + 90) = 80 / 170 = 0.4706, about **47%**. So even with 80% sensitivity and (from d / (b + d) = 810 / 900) 90% specificity, fewer than half of those who screen positive truly have the disorder in this clinic.

If prevalence falls. Sensitivity and specificity are intrinsic to the test and do not move. But re-run the same 80% sensitive, 90% specific test where prevalence is only 1%: among 10000 people, 100 are diseased and 9900 are not. True positives = 80, but false positives now = 9900 x 0.10 = 990, so PPV = 80 / (80 + 990) = 80 / 1070 = 0.0748, about **7.5%**. The very same test turns roughly nine of every ten positives into false alarms once the disorder is rare. That is why a screen that looks good in a high-risk clinic floods the general population with false positives (Wilson & Jungner 1968, Altman 1991).

80%	47%	7.5%
SENSITIVITY	PPV AT 10% PREVALENCE	PPV AT 1% PREVALENCE

RULE **PPV chases prevalence.** Sensitivity and specificity stay fixed; PPV falls as the disorder becomes rarer because false positives swamp the positive row.

20.4 Vignette four: interpret a p-value and a CI

WORKED EXAMPLE

Case: A cohort study reports that an exposure is associated with a disorder at an odds ratio of 1.8 with a 95% confidence interval of 1.2 to 2.7 (p = 0.01). A parallel trial reports a mean difference in symptom score of 1.5 points (95% CI 0.2 to 2.8, p = 0.03) on a scale where clinicians regard 5 points as the smallest change a patient would notice.

Reasoning: the odds ratio. The point estimate 1.8 means the odds of the disorder are 1.8 times higher in the exposed. The 95% confidence interval 1.2 to 2.7 is the range of values compatible with the data; the interpretation is that if the study were repeated many times, 95% of such intervals would contain the true value. Because the interval does not include the no-effect value of **1**, the association is statistically significant, which agrees with the reported p of 0.01 (below the 0.05 threshold). For a ratio measure, crossing 1 and having p above 0.05 are the same statement.

Reasoning: the mean difference. Its no-effect value is **0**. The CI 0.2 to 2.8 excludes 0, so the 1.5-point difference is statistically significant (p = 0.03). But the whole interval lies below the 5-point threshold that patients can actually feel. So the result is statistically significant yet clinically trivial: a real but tiny effect, made detectable only by a large sample. Statistical significance answers "is this likely a chance finding?"; clinical significance answers "is the effect big enough to matter to a patient?", and the two can diverge in either direction (Bland 2015, Altman 1991).

The read. Correct the p-value too: p = 0.01 is the probability of data this extreme if the null were true, not the probability that the null is true and not the size of the effect. A narrow CI that excludes the null and clears the clinical threshold is the strongest result; a CI that excludes the null but sits below it, as here, is significant but not meaningful.

TRAP **Significant equals important.** A p under 0.05 with a CI excluding the null can still be clinically trivial if the effect sits below the minimal important difference.

20.5 The four appraisal moves as one habit

One question runs through all four vignettes. Does this evidence support the claim being made of it, or is someone over-reading it? In the forest plot the check is whether the diamond clears the null and whether heterogeneity undermines the pool; in the test-choice vignette it is whether the analysis matched the data structure or inflated error with serial comparisons; in the 2x2 it is whether a positive result means much given the prevalence; in the p-value and CI it is whether statistical significance was mistaken for clinical importance (Sackett et al 2000, Bland 2015). Learn to ask that one question and every appraisal station collapses to a short, safe read. In each worked example the vignette stem (marked case:) is deliberately terse, because the examiner wants the read, not a recital of the scenario.

HOLD THIS

Diamond crossing the null equals non-significant; three groups need ANOVA (Kruskal-Wallis if non-normal), never serial t-tests; PPV falls with prevalence while sensitivity and specificity hold; and a CI excluding the null is statistically significant but only clinically significant if the effect clears the minimal important difference.

MNEMONIC

READ: Reach for the null line, Elect the right test, Anchor to prevalence, Distinguish significances.

Reach: does the forest-plot diamond (or the CI) cross the no-effect value? **E**lect: match the test to data type by group count by paired status. **A**nchor: read predictive values against the population's prevalence. **D**istinguish: statistical significance is not clinical significance.

GOOD TO REMEMBER

- **Forest plot:** the graphic summary of a meta-analysis; use it to read pooled effect and heterogeneity at a glance; the diamond crossing the null line (1 for ratios, 0 for differences) means non-significant, and I-squared over 75% is high heterogeneity.
- **I-squared (heterogeneity):** the percentage of total variation across trials due to real between-study difference rather than chance; use to judge how much to trust a pooled estimate; roughly under 25% low, around 50% moderate, over 75% high.
- **ANOVA:** the parametric test for three or more independent group means on a normal continuous outcome; use instead of multiple t-tests to control error; statistic is F = between-group variance / within-group variance.
- **Kruskal-Wallis:** the non-parametric twin of one-way ANOVA; use for three or more independent groups when normality fails; it compares ranks, so report medians.
- **Sensitivity:** the proportion of true cases the test correctly flags; use to rule out disease when negative (SnNOUT); formula = a / (a + c), read down the disease-present column and independent of prevalence.
- **Positive predictive value (PPV):** the proportion of test-positives who truly have the disorder; use to judge how much a positive result means in this population; formula = a / (a + b), and it falls as prevalence falls.
- **Odds ratio with 95% CI:** a ratio measure of association read together with its interval; use to test significance and precision at once; significant when the CI excludes the no-effect value of 1.
- **Confidence interval:** the range of values compatible with the data at 95%; use to test significance (does it cross the null?) and to gauge precision; a narrow interval excluding the null that also clears the clinical threshold is the strongest result.
- **p-value:** the probability of data at least this extreme if the null hypothesis were true; use against a pre-set threshold (conventionally 0.05); it is not the probability the null is true and not a measure of effect size.

21 · Consolidate: mnemonic, retrieval and synthesis

Everything in these notes reduces to one reflex the examiner is testing: an abstract lands, and you must place its **design**, judge how its **data** were analysed, and read its **disease frequency** or effect. That is the whole "THREE D's" seed planted at the start, Design then Data then Disease-frequency, and this closing topic ties the three bands into a single memorisable spine so that naming a study, choosing its test, and quoting its measure become one continuous move rather than three separate recalls (Kaplan & Sadock 2024).

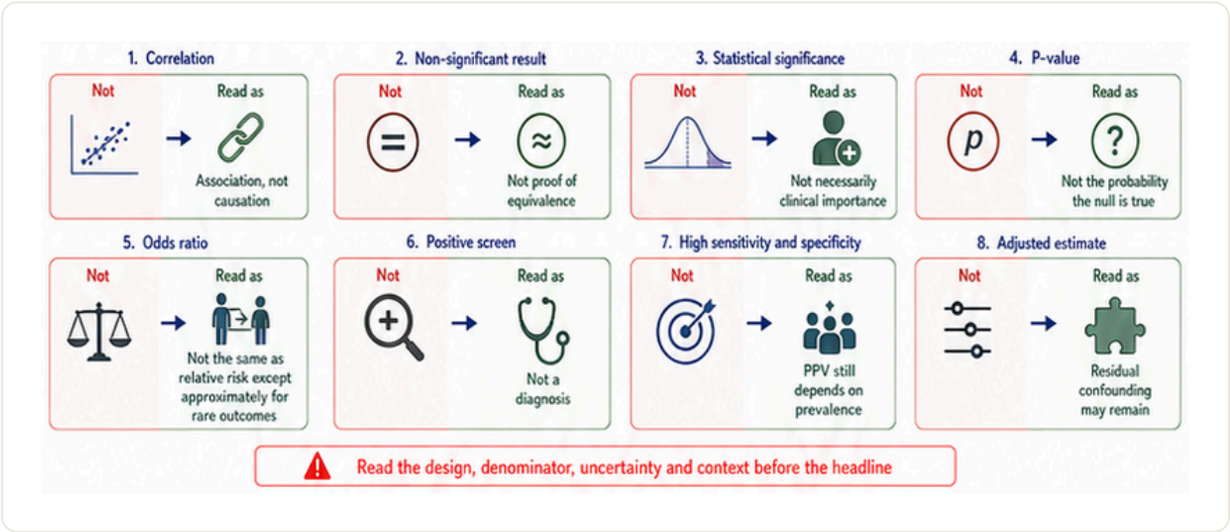


Fig 7.35 Research claims: eight common misreads.

How to use this page. Read the master mnemonic once to fix the skeleton, then close the book and work the recall prompts before you look anything up, then rebuild the whole picture as a branching map from memory. A note read passively fades in days; a note recalled and redrawn under mild difficulty is the version that survives to the viva (Bland 2015). Consolidation is not extra content, it is the retrieval scaffold that makes the preceding content stick.

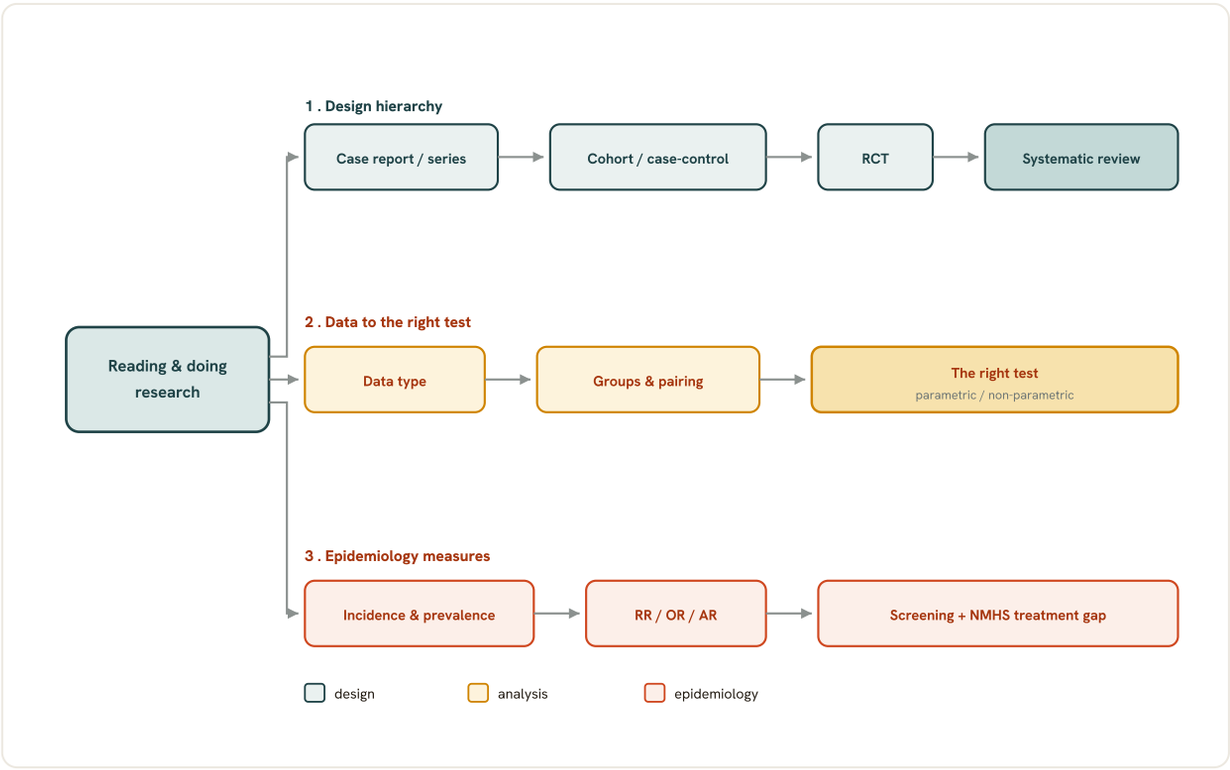


Fig 7.36 The whole-chapter synthesis map. Three branches run out from one hub: the design hierarchy that ranks the strength of a study, the analysis path that takes a dataset from its measurement scale through the number of groups to the correct test, and the epidemiology measures that quantify how common a disorder is and how strongly an exposure raises its risk. Cover the boxes and redraw the three branches from memory as the final retrieval check.

21.1 The master mnemonic: one chain from design to test to measure

The whole document on a single hook. The three bands each get a memory anchor, and the three anchors nest under the "THREE D's" seed so that one cue unpacks the entire note. The design hierarchy runs from the weakest, most abundant evidence at the base up to the strongest, scarcest evidence at the apex; the analysis path runs from the type of data you hold to the one correct test; the epidemiology measures split into the two frequency counts and the two ratio measures of association. Learn the box below and you can reconstruct every earlier topic from its first letter.

MNEMONIC

THREE D's -> "REACH the top, DANCE to the test, FOUR frequencies." (Design climb: Reports, Enquiry cross-sectional, Analyse case-control, Cohort, RCT, Higher-order review. Data path: Discrete-or-continuous, count of groups, Are-they-paired, Normal-or-not, then Choose. Epidemiology: Incidence, Prevalence, Relative risk, Odds ratio.)

Unpack it band by band. **REACH** is the design climb up the evidence pyramid: **R**eports (case report and case series, the base), **E**nquiry-at-a-point (cross-sectional survey, gives prevalence), **A**nalyse-backwards (case-control, gives an odds ratio), **C**ohort (follows forward, gives incidence and relative risk), then the RCT, then the **H**igher-order synthesis (systematic review and meta-analysis, the apex): case report to cohort to RCT to systematic review, base to apex. **DANCE** is the data-to-test path: is the outcome **D**iscrete (categorical) or continuous; how many groups; **A**re they paired or unpaired; is the distribution **N**ormal (parametric) or not; then **C**hoose the cell of the test grid and **E**xecute. **FOUR frequencies** is the epidemiology quartet in reading order: **I**ncidence (new cases, from a cohort), **P**revalence (existing cases, from a cross-sectional survey), **R**elative risk (ratio of two incidences, from a cohort), **O**dds ratio (cross-product, from a case-control). One seed, three chains, the whole note: that is the payoff of the "THREE D's" you were handed at the start.

RULE **Name, test, measure, in that order.** For any abstract, say the design first, then the statistical test its data demanded, then the frequency or effect it reports; the three D's force the sequence so nothing is skipped.

21.2 The three chains laid side by side

What each letter is standing in for. A mnemonic is only worth its unpacking, so the table sets the three chains against each other: the design climb, the analysis path, and the epidemiology quartet, each with the one fact that letter must trigger. Read down any column to rehearse a band; read across to see how a single vignette moves design to test to measure.

BAND (THE D IT SERVES)	THE CHAIN, IN ORDER	THE ONE FACT EACH LINK MUST FIRE
Design climb (Design)	Case report -> cross-sectional -> case-control -> cohort -> RCT -> systematic review	**Trustworthiness rises with control of bias; base is weak and abundant, apex is strong and scarce**
Analysis path (Data)	Data type -> number of groups -> paired or unpaired -> normal or not -> the test	**Categorical vs numerical is the top fork; parametric only when numerical and roughly normal**
Epidemiology quartet (Disease-frequency)	Incidence -> prevalence -> relative risk -> odds ratio	**Incidence and RR need a cohort; prevalence needs a cross-sectional survey; OR is the case-control stand-in**

The three chains under the "THREE D's" seed: one column per band, read across to trace a vignette from design to test to measure.

TRAP **Quoting the wrong measure for the design.** A case-control study cannot yield a relative risk and a cross-sectional survey cannot yield an incidence; if you misname the design you will misname the measure, which is why the chain fixes them together.

21.3 The analysis path in one glance: data type to the right test

The middle chain, filled in. The DANCE letters resolve to a grid whose rows are the group-and-pairing situation and whose columns are the data type. The parametric column (interval or ratio, roughly normal) is the discriminator: pick it only when the data earn it, otherwise drop one column left to the non-parametric equivalent (Bland 2015; Altman 1991). This is the single most examined "choose the test" table, so hold it whole.

GROUPS & PAIRING	CATEGORICAL (NOMINAL)	ORDINAL / NON-NORMAL	INTERVAL OR RATIO (PARAMETRIC)
One sample vs known value	Chi-square goodness-of-fit	Sign test	**One-sample t-test**
Two groups, independent (unpaired)	Chi-square test of association	Mann-Whitney U	**Independent two-sample t-test**
Two groups, paired / matched	McNemar test	Wilcoxon signed-rank	**Paired t-test**
Three or more groups, independent	Chi-square test	Kruskal-Wallis	**ANOVA (one-way), F-test**
Three or more groups, repeated / paired	Cochran's Q	Friedman test	**Repeated-measures ANOVA**
Association between two variables	Chi-square / phi coefficient	Spearman rank correlation	**Pearson correlation**

The DANCE grid: data type across the top, group and pairing down the side, the correct test in the cell; parametric column highlighted.

21.4 The epidemiology quartet, worked once so the numbers stick

The disease-frequency chain, with its arithmetic. The two frequency counts and the two association ratios are best fixed by one 2x2 read that produces both a relative risk and an odds ratio, because seeing the two diverge is what stops you quoting one for the other. Take a cohort of 100 exposed and 100 unexposed people followed forward.

WORKED EXAMPLE

Of 100 exposed, 20 develop the disorder (incidence in exposed = 20/100 = 0.20); of 100 unexposed, 5 develop it (incidence in unexposed = 5/100 = 0.05). The relative risk = incidence in exposed / incidence in unexposed = 0.20 / 0.05 = **4.0**. The odds ratio = ad/bc = (20 x 95) / (80 x 5) = 1900/400 = **4.75**. The attributable risk = incidence in exposed minus incidence in unexposed = 0.20 minus 0.05 = **0.15**, that is 15 excess cases per 100 exposed. Note the odds ratio (4.75) sits further from 1 than the relative risk (4.0) because the disorder is common here (20 percent); make it rare and the two converge, which is the rare-disease approximation. Prevalence would come from a different design entirely, a cross-sectional snapshot, and rises with duration since prevalence is roughly incidence times duration (Kaplan & Sadock 2024).

TRAP **The odds ratio always lies further from 1 than the relative risk.** With a common outcome the OR exaggerates the RR; only when the outcome is rare do they converge, so never read a case-control OR as a relative risk when the disorder is frequent.

21.5 Retrieval practice: close the book and answer these first

Test yourself before you re-read. The evidence for durable memory is unambiguous: active recall beats re-reading, and spaced retrieval practice beats massed review (Bland 2015). So the instruction is literal, cover the answers, attempt each prompt aloud or on paper, and only then check against the earlier topics. The prompts below span all three bands, design then data then disease-frequency, and none of them prints its answer.

RETRIEVAL PRACTICE, COVER THE ANSWERS

1. Name the six rungs of the study-design hierarchy from base to apex, and state what makes the apex more trustworthy than the base.
2. Which single design feature separates the whole observational family from a randomised controlled trial?
3. A vignette gives you a rare, long-latency outcome and asks for the most efficient design: which do you pick, and which measure of association does it yield?
4. State the direction of enquiry for a cohort study versus a case-control study, and the measure each produces.
5. What is the top fork of the data-to-test path, and why does it decide whether a parametric test is even permissible?
6. Two independent groups, a continuous roughly-normal outcome: name the test. Now make the two groups paired: name the test.
7. Three or more independent groups with a numerical outcome: name the parametric test and its non-parametric equivalent.
8. Which test compares paired categorical (before-after yes/no) data in a single group?
9. Define incidence and prevalence, and give the equation that links them through duration.
10. Which study design is needed to measure incidence directly, and why can a cross-sectional survey never give it?
11. Write the formula for the relative risk and the formula for the odds ratio from a 2x2 table.
12. Under what condition does the odds ratio approximate the relative risk, and in which direction does the odds ratio err when that condition fails?
13. Distinguish attributable risk from relative risk in one line: which is a difference and which is a ratio, and what question does each answer?
14. State the "THREE D's" seed and name the memory chain that unpacks each of the three bands.
15. For any abstract, what is the fixed order in which you name the design, the test, and the measure?

21.6 The synthesis map: redraw the whole note from memory

Rebuild the three branches on a blank page. The final consolidation move is to draw a synthesis map, a single branching diagram you redraw from memory with the "THREE D's" seed at the centre and three branches radiating out: the *design hierarchy* climbing case report to cross-sectional to case-control to cohort to RCT to systematic review; the *data-to-test analysis path* forking on data type, group number, pairing and normality down to the named test; and the *epidemiology measures* splitting into the two frequency counts (incidence, prevalence) and the two association ratios (relative risk, odds ratio). If you can redraw the synthesis map with every branch labelled and every leaf carrying its one fact, without the notes open, the note has consolidated; a gap on the map is exactly the topic to revisit.

GOOD TO REMEMBER

- **Design hierarchy:** ranks study designs by control of bias; use it to place any abstract and know what its rung can answer; order (base to apex) = case report -> cross-sectional -> case-control -> cohort -> RCT -> systematic review.
- **Data-to-test path:** selects the correct statistical test; use it once the data are in hand; the deciding chain = data type, number of groups, paired vs unpaired, normal vs not, then the test cell.
- **Incidence:** new cases over a period per population at risk; use it for a first-onset question; needs a cohort, and it is the numerator of the relative risk.
- **Prevalence:** existing cases at a point or over an interval; use it for burden and service planning; from a cross-sectional survey, and prevalence is roughly incidence times duration.
- **Relative risk:** how many times more likely disease is in the exposed; use it from a cohort; RR = incidence in exposed / incidence in unexposed (worked value 4.0).
- **Odds ratio:** the case-control measure when incidence cannot be measured; use it from a case-control study; OR = ad/bc (worked value 4.75), approximating RR only when the outcome is rare.

HOLD THIS

The "THREE D's" seed unpacks the whole note: name the Design on the hierarchy, choose the Data's test by type-groups-pairing-normality, then quote the Disease-frequency measure that design can legitimately give, in that order, every time.

Previously asked

Research methodology, biostatistics and epidemiology sit in the first paper's basic-sciences territory. In the theory catalogue sampled here they have not appeared as standalone long essays or short notes: the subject is examined **applied** rather than recited, folded into clinical questions (the epidemiology and risk factors of a named disorder, the interpretation of a trial in a management answer, the ethics of consent in a forensic scenario) and, above all, in the critical-appraisal and thesis-protocol components. Genuine standalone items will be added here as further question catalogues are sourced, never invented.

? HOW THIS TERRITORY IS ACTUALLY TESTED

Q As an **appraisal task**: given a study or a forest plot, name the design, its measure of association, its main bias, and whether the conclusion is justified.

Q As an **applied number**: read a 2x2 for sensitivity and predictive value, interpret a p-value and a confidence interval, or compute a relative risk or an NNT.

Q As an **Indian-data prompt**: quote the National Mental Health Survey prevalence and the treatment gap to frame a public-health answer.

Q As **research ethics**: the four principles, informed consent and the ethics committee within a scenario, most often in the forensic and community papers.

Prepare this material to be used, not just remembered. The two selector tables (choosing a design, choosing a test) and the four worked appraisal cases in this note are deliberately built to that examinable skill. The disorder-specific epidemiology that recurs in clinical essays is developed in the clinical chapters of Paper II; the professional and forensic ethics that the ethics questions usually target are developed in the mental health legislation and forensic psychiatry notes; this note owns the general research toolkit, the statistics, and the national survey numbers.

Quick review

The whole subject in one table	
EVIDENCE-BASED MEDICINE	The five steps (Ask, Acquire, Appraise, Apply, Assess); the focused question via PICO; the null and alternative hypotheses. The evidence pyramid ranks designs by control of bias: case report, cross-sectional, case-control, cohort, randomised controlled trial, then systematic review and meta-analysis at the apex.
OBSERVATIONAL DESIGNS	Cross-sectional is a snapshot giving prevalence. Case-control traces outcome back to exposure, gives the odds ratio, and is efficient for rare outcomes. Cohort follows exposure forward to outcome, gives incidence and the relative risk, and suits rare exposures. Ecological studies compare populations (mind the ecological fallacy).
THE RANDOMISED CONTROLLED TRIAL	Randomisation balances known and unknown confounders; allocation concealment hides the next assignment; blinding hides the treatment; intention-to-treat analyses everyone as randomised. Parallel, crossover, factorial and cluster designs. Drug-trial phases I (safety) to IV (post-marketing).
VALIDITY, BIAS, CONFOUNDING	Internal validity (true here) versus external validity (generalisable). Bias is systematic error: selection and information (including recall) bias. Confounding is a third variable on neither causal path, controlled at design (randomisation, restriction, matching) or analysis (stratification, regression). Distinct from psychometric reliability and validity.
SAMPLING	Probability sampling (simple random, systematic, stratified, cluster, multistage) permits generalisation; non-probability sampling (convenience, purposive, snowball) does not. Stratify to guarantee a small subgroup; clustering adds a design effect.
QUALITATIVE METHODS	Grounded theory, thematic analysis, phenomenology, ethnography; the focus group and the in-depth interview. Rigour through saturation, triangulation, reflexivity and an audit trail. For meaning and hypothesis generation, not statistical generalisation.
SYSTEMATIC REVIEW AND META-ANALYSIS	The reproducible protocol-driven review versus the statistical pooling that may sit inside it. Read the forest plot (point estimates, confidence intervals, the null line, the pooled diamond). Heterogeneity via I-squared; publication bias via the funnel plot; certainty via GRADE.
REPORTING GUIDELINES AND ETHICS	CONSORT for trials, PRISMA for reviews, STROBE for observational studies, GRADE for certainty. The four principles (autonomy, beneficence, non-maleficence, justice), Helsinki and Nuremberg, informed consent, the ethics committee, and the ICMR 2017 guidelines in India.
DATA TYPES AND SCALES	Nominal, ordinal, interval and ratio; categorical versus continuous (discrete or continuous). The measurement scale decides the legal summary

The whole subject in one table	
	statistic and the legal test, so it is the first thing to settle.
DESCRIBING DATA AND THE NORMAL CURVE	Central tendency (mean, median, mode) and dispersion (range, interquartile range, variance, standard deviation). The normal distribution: mean equals median equals mode, and the empirical rule that 68, 95 and 99.7 percent lie within one, two and three SDs. Skew and kurtosis; the z-score.
TESTS OF SIGNIFICANCE	Parametric tests for normal interval data: the t-test (one-sample, independent, paired), ANOVA with post-hoc for three or more groups. Non-parametric twins for ordinal or non-normal data: Mann-Whitney, Wilcoxon, Kruskal-Wallis; chi-square (Fisher exact for small cells) for categorical data.
CORRELATION, REGRESSION, FACTOR ANALYSIS	Pearson for linear association of continuous normal variables, Spearman for ranks. Linear regression predicts a continuous outcome (r-squared as variance explained); logistic regression predicts a binary one (adjusted odds ratios). Factor analysis reduces items to latent factors (exploratory versus confirmatory, eigenvalues, loadings, PCA).
DIAGNOSTIC TEST STATISTICS	From the 2x2: sensitivity and specificity are intrinsic (SnNOUT, SpPIN); the positive and negative predictive values are what the clinician wants but move with prevalence; the likelihood ratio shifts pre-test to post-test odds. At low prevalence a positive screening test is often a false alarm.
STATISTICAL INFERENCE	The p-value is the probability of the data (or more extreme) under the null; alpha is set at 0.05. The confidence interval gives the plausible range (crossing 0 for a difference, or 1 for a ratio, means non-significant). Type I error is a false positive (alpha), type II a false negative (beta); power is 1 minus beta, aim 80 percent. Sample size and effect size drive it.
SURVIVAL, NNT AND ROC	Kaplan-Meier plots event-free probability over time with censoring; the log-rank test compares curves and the hazard ratio quantifies the difference. NNT is one over the absolute risk reduction. The ROC curve plots sensitivity against one minus specificity, and the AUC grades discrimination (0.5 chance, 1.0 perfect).
DISEASE FREQUENCY AND ASSOCIATION	Incidence (new cases) versus prevalence (existing cases, point or period; roughly incidence times duration). Relative risk from a cohort, the odds ratio from a case-control (it approximates the RR when the outcome is rare), and the attributable risk as the excess in the exposed.
THE INDIAN PICTURE (NMHS)	The National Mental Health Survey 2015-16 gives India's current and lifetime prevalence and, its policy headline, the large treatment gap that varies by disorder. Read it beside the world mental health frame; it is an applied cross-sectional multistage survey. Every quoted number is verified against the source.

The whole subject in one table	
SCREENING	The Wilson and Jungner criteria (the condition, the test, the treatment, the programme). Lead-time bias (earlier diagnosis, not longer life), length bias (indolent disease over-sampled) and overdiagnosis. Screen with a sensitive test and confirm with a specific one; low prevalence lowers the predictive value.
DISCRIMINATE	Two selectors: the study-design chooser takes a question to a design (prevalence to cross-sectional, rare outcome to case-control, rare exposure or incidence to cohort, efficacy to RCT, totality to systematic review). The statistical-test chooser takes the data type, the number of groups and pairing to the correct parametric or non-parametric test.
APPLY AND CONSOLIDATE	Four worked appraisals: read a forest plot, choose the test for a dataset, compute sensitivity and PPV from a 2x2, and interpret a p-value with its confidence interval. Then the master mnemonic (design to data to disease frequency), answer-free retrieval prompts, and the three-branch synthesis map to redraw from memory.

References

The material is grounded in the standard texts (the source hierarchy below); statistical formulae and definitions are drawn from the medical-statistics references, and each landmark, contested or numeric claim carries a PubMed-verified journal citation. Every PMID here was checked against PubMed this build, and any citation whose identifier did not resolve to the cited paper was corrected or dropped. The National Mental Health Survey figures are taken from the primary results paper, not from a textbook summary.

SOURCE HIERARCHY (TEXTBOOKS AND STATISTICS REFERENCES)

1. Altman DG. Practical Statistics for Medical Research. London: Chapman & Hall/CRC; 1991. [the medical-statistics depth bar: distributions, tests, sample size, diagnostic statistics]
2. Bland M. An Introduction to Medical Statistics. 4th ed. Oxford: Oxford University Press; 2015. [describing data, significance testing, correlation and regression]
3. Johnstone EC, Owens DGC, Lawrie SM, McIntosh AM, Sharpe M, eds. Companion to Psychiatric Studies. 8th ed. Edinburgh: Churchill Livingstone/Elsevier; 2010. [the psychiatric research-methods and biostatistics chapter, the primary parsed source]
4. Geddes JR, Andreasen NC, Goodwin GM, eds. New Oxford Textbook of Psychiatry. 3rd ed. Oxford: Oxford University Press; 2020. [evidence-based medicine and clinical epidemiology]
5. Boland R, Verduin ML, eds. Kaplan & Sadock's Comprehensive Textbook of Psychiatry. 11th ed. Philadelphia: Wolters Kluwer; 2024. [the depth bar; psychiatric epidemiology and statistics]
6. Tasman A, Kay J, Lieberman JA, First MB, Riba MB, eds. Tasman's Psychiatry. Chichester: Wiley-Blackwell. [psychiatric epidemiology; the World Mental Health Survey Initiative]
7. Clinical Methods in Psychiatry. 1st ed. New Delhi: AIIMS Publication; 2024. [the mental-health survey figures and the Indian context]
8. Greenhalgh T. How to Read a Paper: The Basics of Evidence-Based Medicine and Healthcare. 6th ed. Oxford: Wiley-Blackwell; 2019. [critical appraisal, reading the literature]

FOUNDATIONAL SOURCES

1. Wilson JMG, Jungner G. Principles and Practice of Screening for Disease. WHO Public Health Papers No. 34. Geneva: World Health Organization; 1968. [the ten screening criteria]
2. Cochrane AL. Effectiveness and Efficiency: Random Reflections on Health Services. London: Nuffield Provincial Hospitals Trust; 1972. [the case for the randomised controlled trial and evidence-based care]
3. Higgins JPT, Thomas J, Chandler J, et al., eds. Cochrane Handbook for Systematic Reviews of Interventions. Version 6. London: Cochrane; 2023. [systematic-review method, heterogeneity and the I-squared interpretation bands]
4. World Medical Association. Declaration of Helsinki: Ethical Principles for Medical Research Involving Human Subjects. Adopted 1964, current revision 2013. [the research-ethics reference standard]
5. Indian Council of Medical Research. National Ethical Guidelines for Biomedical and Health Research Involving Human Participants. New Delhi: ICMR; 2017. [the Indian research-ethics framework]

JOURNAL REFERENCES (PUBMED-VERIFIED, 20)

1. Sackett DL, Rosenberg WM, Gray JA, Haynes RB, Richardson WS. Evidence based medicine: what it is and what it isn't. BMJ. 1996;312(7023):71-2. PMID: 8555924.
2. Evidence-Based Medicine Working Group. Evidence-based medicine. A new approach to teaching the practice of medicine. JAMA. 1992;268(17):2420-5. PMID: 1404801.
3. Chalmers TC, Celano P, Sacks HS, Smith H Jr. Bias in treatment assignment in controlled clinical trials. N Engl J Med. 1983;309(22):1358-61. PMID: 6633598.
4. Schulz KF, Chalmers I, Hayes RJ, Altman DG. Empirical evidence of bias. Dimensions of methodological quality associated with estimates of treatment effects in controlled trials. JAMA. 1995;273(5):408-12. PMID: 7823387.
5. Higgins JPT, Thompson SG, Deeks JJ, Altman DG. Measuring inconsistency in meta-analyses. BMJ. 2003;327(7414):557-60. PMID: 12958120.
6. Egger M, Davey Smith G, Schneider M, Minder C. Bias in meta-analysis detected by a simple, graphical test. BMJ. 1997;315(7109):629-34. PMID: 9310563.
7. Whittington CJ, Kendall T, Fonagy P, Cottrell D, Cotgrove A, Boddington E. Selective serotonin reuptake inhibitors in childhood depression: systematic review of published versus unpublished data. Lancet. 2004;363(9418):1341-5. PMID: 15110490.
8. Schulz KF, Altman DG, Moher D; CONSORT Group. CONSORT 2010 statement: updated guidelines for reporting parallel group randomised trials. BMJ. 2010;340:c332. PMID: 20332509.
9. Moher D, Liberati A, Tetzlaff J, Altman DG; PRISMA Group. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. BMJ. 2009;339:b2535. PMID: 19622551.
10. von Elm E, Altman DG, Egger M, Pocock SJ, Gotzsche PC, Vandenbroucke JP; STROBE Initiative. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement. Lancet. 2007;370(9596):1453-7. PMID: 18064739.
11. Guyatt GH, Oxman AD, Vist GE, et al. GRADE: an emerging consensus on rating quality of evidence and strength of recommendations. BMJ. 2008;336(7650):924-6. PMID: 18436948.
12. Stroup DF, Berlin JA, Morton SC, et al. Meta-analysis of observational studies in epidemiology: a proposal for reporting (MOOSE). JAMA. 2000;283(15):2008-12. PMID: 10789670.
13. Emanuel EJ, Wendler D, Grady C. What makes clinical research ethical? JAMA. 2000;283(20):2701-11. PMID: 10819955.
14. Appelbaum PS. Assessment of patients' competence to consent to treatment. N Engl J Med. 2007;357(18):1834-40. PMID: 17978292.
15. Stevens SS. On the theory of scales of measurement. Science. 1946;103(2684):677-80. PMID: 17750512.
16. Perneger TV. What's wrong with Bonferroni adjustments. BMJ. 1998;316(7139):1236-8. PMID: 9553006.
17. Sterne JAC, Davey Smith G. Sifting the evidence, what's wrong with significance tests? BMJ. 2001;322(7280):226-31. PMID: 11159626.
18. Fagan TJ. Letter: Nomogram for Bayes's theorem. N Engl J Med. 1975;293(5):257. PMID: 1143310.
19. Paykel ES, Scott J, Teasdale JD, et al. Prevention of relapse in residual depression by cognitive therapy: a controlled trial. Arch Gen Psychiatry. 1999;56(9):829-35. PMID: 12884889.
20. Gautham MS, Gururaj G, Varghese M, et al. The National Mental Health Survey of India (2016): Prevalence, socio-demographic correlates and treatment gap of mental morbidity. Int J Soc Psychiatry. 2020;66(4):361-72. PMID: 32126902.

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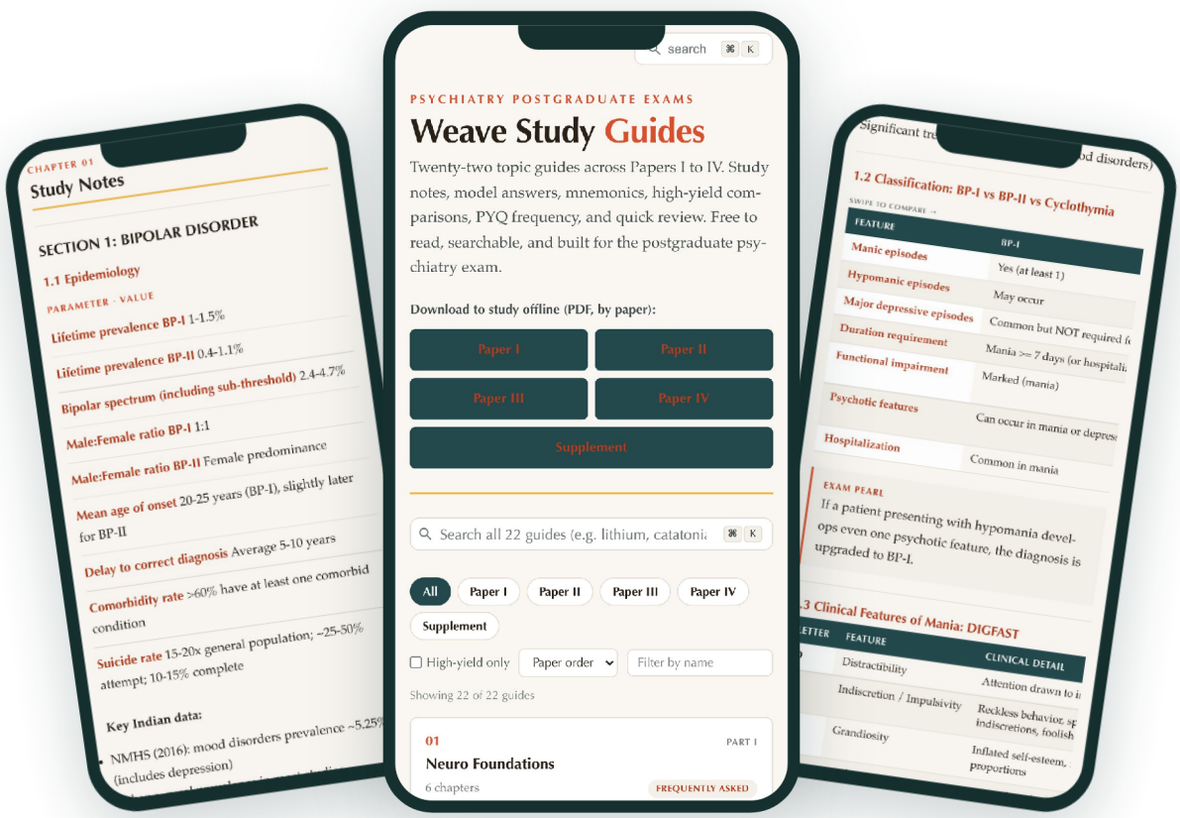
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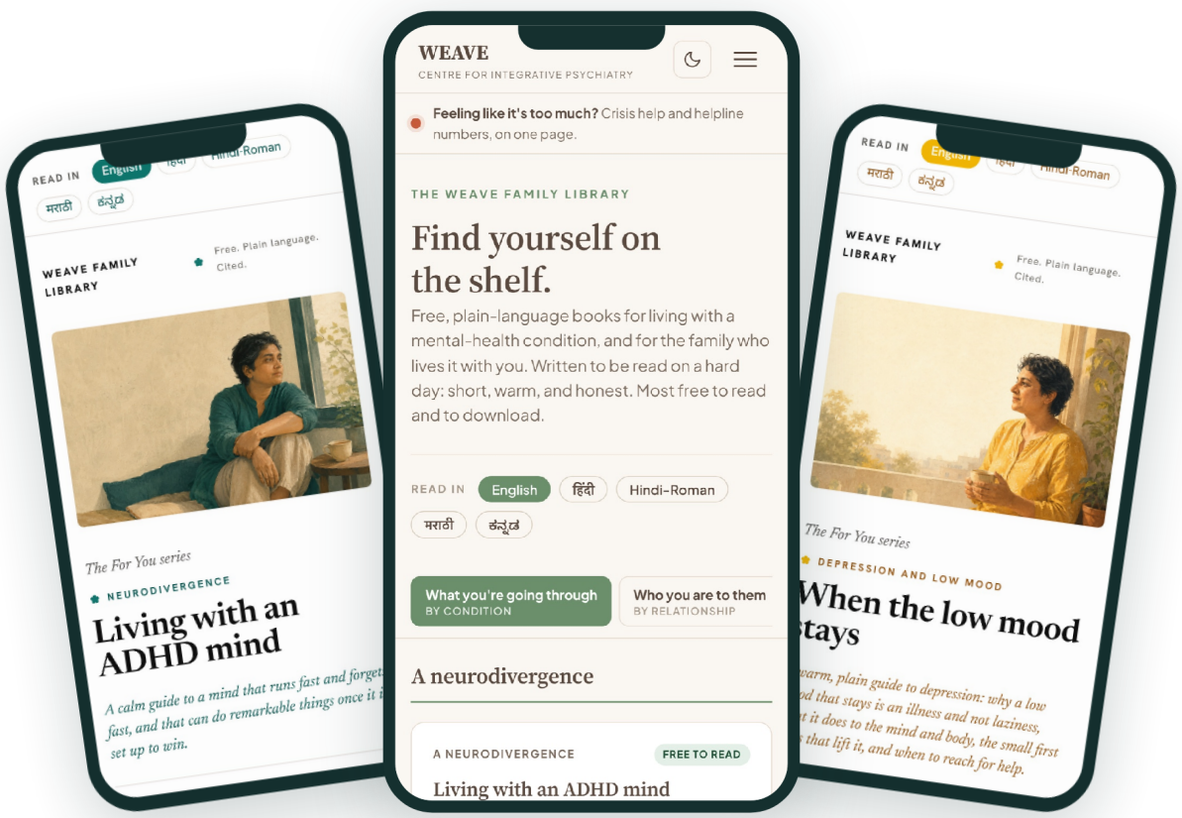
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