WEAVE 100 - PAPER I

MD Psychiatry theory. DNB revision fits too.

Brain, Mind and Method

One hundred ten-mark questions, solved as answer maps

Eight examining boards. Seventy-eight sittings. A count of where the marks have actually gone in the paper that holds the brain a psychiatrist is examined on, the psychology the clinic runs on, and the arithmetic that decides whether a finding is real: neuroanatomy and imaging, neurochemistry, genetics, learning and development, personality theory, culture, psychometry, research method and statistics, and the descriptive psychopathology underneath every diagnosis. Twelve areas, weighted by what was asked, then one hundred questions drawn in that proportion and solved as one-page answer maps. Every question in this book was set by a board. None was invented.

Dr. Wilfred Dsouza · Dr. Niharika Reddy

WEAVE POWERED BY TRIJOG

A NOTE ON WHO MADE THIS

Weave, powered by Trijog

Weave is a psychiatry practice and a publishing house for the things clinicians and families actually need to read. Trijog is the mental-health organisation Weave partners with for therapy delivery at scale.

WHAT WEAVE DOES	Integrative psychiatry, online and in Mumbai, and a free library for residents, clinicians and families.
WHAT TRIJOG ADDS	A therapy network with the reach to see the person you refer, quickly, and to keep seeing them.
WHAT THAT MEANS HERE	This book is free because the practice pays for it. There is nothing to buy on any page of it.

WEAVE 100 - PAPER I

Brain, Mind and Method

One hundred ten-mark questions, solved as answer maps

THE WEAVE 100 SERIES

TITLE

Weave 100, Paper I: Brain, Mind and Method

Book one of Weave 100, one hundred solved ten-mark questions for each paper of the MD psychiatry written examination.

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EDITION

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SOURCES

Question records were read from the published papers and archives of the boards named on the back cover. Board names appear throughout because the count is only meaningful with them. No board has endorsed this book and none was asked to.

Contents

HOW TO USE THIS BOOK

One question, one page

Every page after the weightage chapter is one question and its answer map. Nothing continues onto a second page, so a map can be read in the time you have rather than the time it deserves. Five blocks, always in the same order.

1	**Question band**	The slot number, the question as a board set it, and three chips: the mark split, the area, and where the question has been seen.
2	**Skeleton**	Three to five rows. The mark column sums to ten, so you can see the allocation before you read a word of content.
3	**Body**	Two to four components, chosen for the question: a comparison table, a flowchart, a plate, typed lines, at most one pitfall box.
4	**What earns the marks**	Three or four things an examiner ticks. Each one names something that is on the page above it.
5	**Links**	The related slots in this book, and the Weave Sprint day that teaches the underlying topic in full.

The colour code

Structure	The shape of the answer: headers, rules, table heads, the question band. When a line is indigo it is telling you how the answer is organised.
Key number	A figure to carry exactly: a mark split, a threshold, a count, a cut-off. Amber means memorise the number, not just the shape of it.
Trap	The mistake the examiner is waiting for. When a line is red it is warning you off an error that looks right until it costs you the mark.

Two ways to work. Read an area straight through, banner first, to see how the boards have carved it up. Or use the ten sets at the back: each is ten questions and one hundred marks, drawn one from each rank decile, so a set is a paper-shaped sitting rather than ten easy questions or ten hard ones.

The chips are traceable. A recurrence chip names the boards and gives a count against a stated denominator. If it says three of seventy-eight sittings, that is three sittings out of the seventy-eight this book counted, and the boards are listed by name so you can check.

THE WEIGHTAGE CHAPTER, PAGE ONE OF THREE

Where the marks have gone

Nothing in this book is a prediction. It is a count of 78 sittings from 8 examining boards, and a statement of where those boards have put their marks in Paper I.

Twelve areas, weighted. The university boards are pooled by how much each is trusted, DNB is held out and given a fixed thirty percent, and the resulting weight becomes a share of one hundred and then a whole number of slots. The ledger of which boards were counted, how their papers reached us, and how far each one is trusted, is on the page opposite.

AREA, IN WEIGHT ORDER	UNIV POOL	DNB	WEIGHT	SHARE	SLOTS
01 Neurochemistry and neurophysiology	0.455	0.532	0.479	14.52%	13
02 Functional neuroanatomy and neuroimaging	0.456	0.422	0.446	13.53%	12
03 Personality theory and psychodynamics	0.321	0.407	0.347	10.52%	10
04 Learning, memory and general psychology	0.335	0.304	0.326	9.89%	9
05 Psychometry and assessment	0.318	0.254	0.299	9.07%	9
06 Genetics in psychiatry	0.252	0.396	0.295	8.96%	9
07 Social, cross-cultural and evolutionary psychology	0.326	0.098	0.257	7.81%	8
08 Biostatistics and epidemiology	0.232	0.295	0.251	7.61%	8
09 Research methodology	0.115	0.311	0.174	5.27%	6
10 Developmental psychology and attachment	0.192	0.110	0.168	5.09%	6
11 Nosology, classification and descriptive psychopathology	0.051	0.422	0.162	4.92%	6
12 Neuroendocrinology and basic psychopharmacology	0.077	0.127	0.092	2.79%	4

Univ pool is the trust-weighted verdict of the seven university boards; DNB is the national paper, held out. Weight = 0.70 times the pool plus 0.30 times DNB. Share is that weight as a fraction of Paper I. Slots are the share turned into whole questions by largest remainder, with a floor of two and a cap of twenty; Paper I reached neither.

THE WEIGHTAGE CHAPTER, PAGE TWO OF THREE

How the hundred were picked

BOARD	SITTINGS COUNTED	HOW THE PAPERS REACHED US	TRUST
RGUHS	12	Hand transcription and public mirrors of the printed papers	0.806
KUHS	9	Official university portal PDFs	0.600
TN-MGRMU	35	Public archive of the printed papers, exact sitting dates recovered	0.480
KNRUHS	3	Official university portal PDFs	0.300
MUHS	2	Two watermark-confirmed papers, read by OCR. A third-party recurrence compilation was tested against them, matched none, and was removed from the sitting count	0.267
NTRUHS	2	Mirror text of the printed papers	0.160
WBUHS	2	Public archive of the printed papers	0.180
DNB	10	Official NBE material; 32 of 40 files verified by checksum against the board's own published path	fixed 0.30

Trust is how far a board sets the university pool: its standing, its number of sittings, and the quality of its evidence. DNB sits outside the pool. A third-party compilation claiming thirteen further MUHS sittings was tested against the only confirmed MUHS papers inside its own window, matched none of them, and was removed from this ledger; its topic distribution still contributes to area mass at reduced quality.

1 Count the sittings

78 sittings from 8 boards, each weighted so that it halves roughly every six years and is reduced again when the paper reached us as a mirror, an archive or a transcription rather than an official file.

2 Weight the areas

Each board reads each area twice: breadth, how often it examines there, and mark share, how much of its marks land there. The two are averaged, the boards are pooled by trust, and DNB is held out at a fixed thirty percent.

3 Turn weight into slots

The twelve weights become shares of one hundred, then whole slots by largest remainder, with a floor of two and a cap of twenty so no area disappears and none swallows the book.

4 Score every eligible question

Four components: weighted recurrence (45 per cent), recency (30), how many boards have asked it (15), and how well it is shaped as a ten-mark question (10). Normalised across all four papers, so one score means one thing everywhere.

5 Fill the slots

The highest scorers in each area, up to that area's slot count, near-duplicates merged first. All 100 were filled from questions actually set: 11 anchor, 18 high, 3 recurrent, 68 single-appearance.

THE WEIGHTAGE CHAPTER, PAGE THREE OF THREE

What this book cannot promise

A frequency count is a strong prior, not a prophecy. Here is exactly what that sentence costs, stated before you rely on it.

It does not predict a paper. Nothing here says what your board will set. It says where the marks have gone across 78 sittings, which is a different claim and a smaller one. A board that changes its examiner, its syllabus or its habits can leave every number in this book behind in one sitting.

Most questions were seen once. Of the 403 selectable Paper I questions in the record, 342 appeared in a single sitting. That is not a defect in the counting, it is what happens when eight boards set different papers: the same TOPIC returns constantly while the same QUESTION rarely does. So the selection is driven by AREA weight, where the signal is dense, and each map prints a band rather than a frequency claim about its own question.

The band on a map is a description, not a forecast. Anchor means this question has come back across boards and years. Single means it was set once in the record we hold. A single-appearance question is here because its AREA is heavy, and it is a fair worked example of that area, which is the only thing it is offered as.

Nothing here is invented. All 100 slots in this book are questions a board actually set. Where a paper in this series cannot fill its slots from observed questions, the shortfall is authored to syllabus and labelled as such on the map. Paper I has no such slots.

The record is uneven, and openly so. Two of the eight boards set papers in a legacy structure that does not map onto the modern four, so their sittings are counted against all of their papers rather than one. Two more contribute only two sittings each. The trust column on the first page of this chapter is where that unevenness is priced in, and it is the number to argue with if you want to argue with the weighting.

No board has endorsed this. Board names appear on every page because a count that hides its sources is not a count. None of them was asked, and none of them is responsible for anything in this book.

AREA 01 OF 12 · P1-A02

Neurochemistry and neurophysiology

13	14.5%	I-01	I-13
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 2 · HIGH 2 · SINGLE 9

REVISION ORDER

The memory block first and together: four slots share one answer and two of them carry the anchor weight in this area. Sleep next, as a set of three, and the classification slot last of the three because it is the only one that turns clinical. The dopamine pathways after that, short and set often. The three transmitter slots, the electrophysiology slot and the markers slot are independent and each stands alone.

WHAT IS IN THIS AREA

Memory in four slots (the neurobiology with its clinical relevance, the cortical contribution with memory impairment across illnesses, the types of memory with rehabilitation in chronic illness, and synaptic plasticity). Sleep in three (the neurobiology of sleep and wakefulness, polysomnography with the stages and their EEG findings, and the classification with restless legs and a benzodiazepine taper). Single transmitters in three: nitric oxide, glutamate and the endocannabinoids. The dopamine pathways, the electrophysiological abnormalities of four disorders, and the biological-markers slot.

I - 01

Describe the Neuro-biology of memory and its relevance in clinical practice. [10]

10 NEUROCHEMISTRY AND NEUROPHYSIOLOGY

KUHS, MUHS, RGUHS, TN-MGRMU - 6 APPEARANCES, 6 OF 78 SESSIONS

SKELETON

2	The four stages	Encoding, consolidation, storage, retrieval, each a separate failure
3	Systems and their anatomy	One structure per system, not one memory centre
3	Cellular basis	Long term potentiation, and what makes a trace permanent
2	Clinical relevance	The bedside uses that follow from the anatomy

THE LINE WORTH WRITING FIRST

Memory is not one faculty in one place. It is several parallel systems, each with its own anatomy, its own timescale and its own way of failing. An answer that names the systems separately has already earned more than one that describes memory in general.

SYSTEM	ANATOMY	WHAT IT HOLDS	WHERE IT FAILS
Working	Dorsolateral prefrontal cortex	Seconds of actively held material	Schizophrenia, ADHD, delirium
Episodic	Hippocampus, medial temporal lobe	Events with a time and a place	Alzheimer disease, Korsakoff, ECT
Semantic	Anterolateral temporal cortex	Facts stripped of their context	Semantic dementia
Procedural	Striatum and cerebellum	Skills and habits	Parkinson disease; spared in amnesia

The last row is the one that separates the bands: procedural learning survives dense amnesia, which is what proves the systems are separate.

- **Cellular basis.** Long term potentiation at glutamatergic synapses. Strong activity relieves the magnesium block on the NMDA receptor, calcium enters, kinases move more AMPA receptors into the membrane, and the synapse is stronger.
- **Making it last.** Early potentiation needs no new protein. Late potentiation does: calcium signalling reaches CREB, new proteins are made, spines change shape. That gene step is why consolidation takes hours and why it can be blocked.
- **Clinical relevance.** Sparing of procedural learning guides rehabilitation; ECT produces retrograde and anterograde deficits that mostly recover; benzodiazepines and alcohol block encoding rather than erase storage, which is what blackout means.

WHAT EARNS THE MARKS

- **Four stages named before any anatomy.** It lets you say which stage a drug or a lesion damages.
- **Each system with its own structure,** and procedural memory identified as the spared one.
- **Relevance made concrete.** Blackout, ECT and rehabilitation are the three the examiner expects.

SEE ALSO Slot I - 10 types of memory and rehabilitation Slot I - 13 synaptic plasticity SPRINT Day 1, Functional Neuroanatomy and Neurophysiology

I - 02

Describe the role of cortex in memory formation. Discuss about memory impairment in various psychiatric illnesses. [5+5]

5 + 5 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB, TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 **Which cortex, for what**	Prefrontal for control, sensory cortex for the trace
3 **Systems consolidation**	How a trace moves from hippocampus to neocortex
3 **Impairment by illness**	Which stage fails, and whether it persists
2 **What it changes**	Assessment and rehabilitation that follow

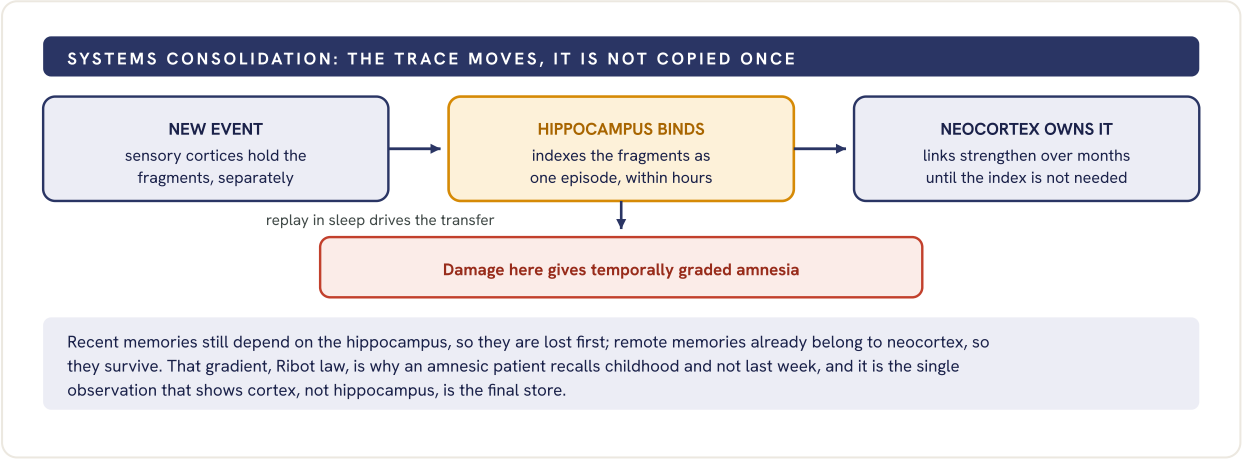

Fig 002.1 Consolidation drawn as a handover: cortex holds the fragments throughout, the hippocampus indexes them, and the index is gradually made unnecessary.

- **Which cortex does what.** Dorsolateral prefrontal cortex runs encoding strategy and retrieval search; ventrolateral prefrontal cortex selects among competitors; the sensory cortices are where the content itself is finally stored.
- **Impairment by illness.** Schizophrenia: verbal declarative and working memory deficits present from onset and largely independent of symptoms. Depression: effortful encoding fails with attention and motivation, plus overgeneral autobiographical recall. Bipolar disorder: deficits persist into euthymia.
- **The reversible causes, kept separate.** Delirium, benzodiazepines and alcohol, ECT, thiamine deficiency, hypothyroidism and dissociative amnesia. Naming which of these are treatable before describing the untreatable is the marked move.

WHAT EARNS THE MARKS

- **Cortex named as the final store,** with the hippocampus as a temporary index.
- **The temporal gradient, and what it proves.** It is the evidence, not just a clinical sign.
- **Deficits described by stage,** encoding against retrieval, rather than as poor memory.
- **Reversible causes listed separately** from the disorders themselves.

SEE ALSO Slot I - 01 neurobiology of memory Slot I - 23 hippocampal formation **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

I - 03

Describe in brief neurobiology of sleep and wakefulness. [10]

10 NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB, KUHS, RGUHS, WBUHS - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

3	**Ascending arousal system**	Two branches out of the brainstem, and their transmitters
2	**Sleep promoting system**	The preoptic switch, GABA and galanin
2	**Flip-flop switch**	Mutual inhibition, and what orexin does to it
2	**REM sleep**	The cholinergic and aminergic reciprocal model
1	**Drug correlates**	Each sedating drug named to the system it acts on

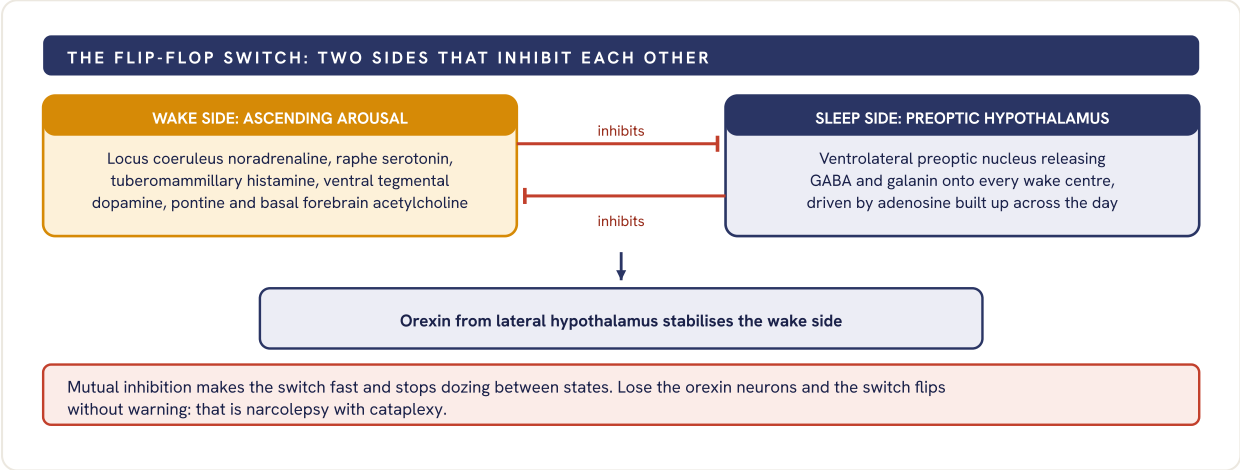

Fig 003.1 Wake and sleep drawn as two mutually inhibiting populations with orexin as the stabiliser, which is why the state changes abruptly rather than by degrees.

- **REM sleep.** The reciprocal interaction model: cholinergic pontine neurons fire to generate REM while noradrenergic and serotonergic neurons fall silent. Loss of that aminergic tone with active cholinergic drive gives the vivid dreams and the muscle atonia.
- **Two clocks, not one.** A homeostatic drive from accumulating adenosine, and a circadian drive from the suprachiasmatic nucleus signalling through melatonin. Sleep happens where the two align.

DRUG	WHAT IT ACTS ON	THE EFFECT THIS PREDICTS
Benzodiazepines, Z drugs	GABA-A receptor, the sleep side	Faster sleep onset, less slow wave sleep
Sedating antihistamines	Histamine, a wake transmitter	Sedation, and daytime hangover
Caffeine	Adenosine receptor blockade	Removes the homeostatic sleep signal
Melatonin agonists	MT1 and MT2, the circadian arm	Shifts timing rather than sedating

WHAT EARNS THE MARKS

- **Both sides of the switch, with transmitters attached.** A list of nuclei without transmitters is half the answer.
- **Mutual inhibition explained as a mechanism,** and orexin given its stabilising role.
- **Drugs mapped back onto the diagram,** which is what makes this a pharmacology answer too.

SEE ALSO Slot I - 09 polysomnography and sleep stages Slot I - 11 classification of sleep disorders **SPRINT** Day 1, Functional Neuroanatomy and Neurophysiology

I - 04

Describe the synthesis, mechanism of action and neuropathological role of nitric oxide. [3+3+4]

3 + 3 + 4 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**Synthesis**	One substrate, three enzymes, and what triggers them
3	**Mechanism of action**	Why it breaks every rule of a classical transmitter
4	**Neuropathological role**	The same molecule as signal and as poison

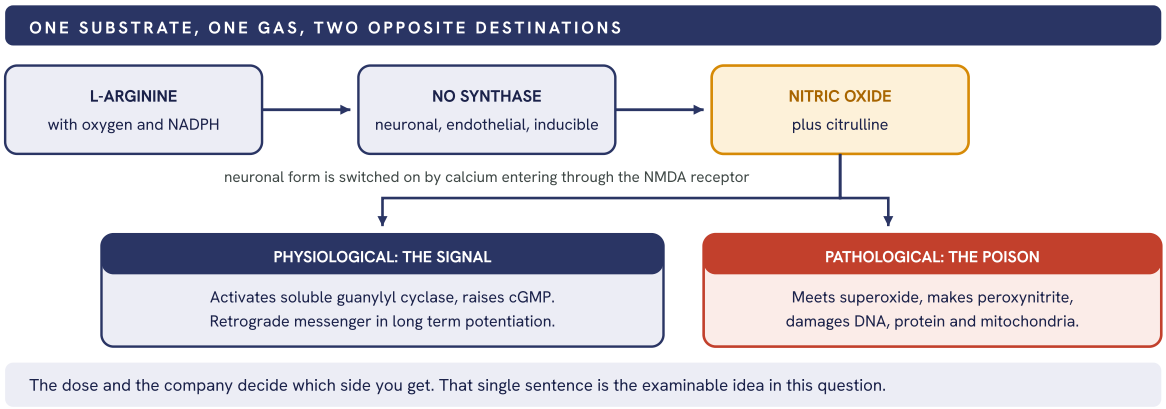

Fig 004.1 Nitric oxide drawn once and used twice: the same synthetic step feeds a signalling arm and an excitotoxic arm, and only the local conditions choose between them.

- **Why it is unusual.** A gas: not stored, not exocytosed, no membrane receptor. Made on demand, it crosses membranes freely and acts within microns for seconds, signalling backwards from the postsynaptic cell to the terminal that fed it.
- **Where it turns pathological.** Excess glutamate opens NMDA receptors, calcium floods in, neuronal NO synthase runs hard, and peroxynitrite forms. This is the final common path of excitotoxicity in stroke, neurodegeneration and, as an inducible form in microglia, in neuroinflammation.

THE PSYCHIATRIC HOOK

Nitric oxide signalling is downstream of the NMDA receptor, which is the same receptor implicated in the glutamate model of schizophrenia and targeted by ketamine. It is also the mediator of penile erection, which is why phosphodiesterase inhibitors treat one of the commonest antidepressant side effects.

WHAT EARNS THE MARKS

- **The synthetic step written out,** arginine with oxygen and NADPH to nitric oxide and citrulline, and all three synthase isoforms named.
- **The four ways it breaks transmitter rules.** Not stored, not exocytosed, no receptor, membrane permeable.
- **Signal and poison as one mechanism,** separated by amount and by the presence of superoxide.

SEE ALSO Slot I - 06 glutamate receptors and pathways Slot I - 13 synaptic plasticity

I - 05

Describe the electrophysiological abnormalities in the following conditions. a) Schizophrenia. b) Catatonia. c) Obsessive compulsive disorder. d) Delirium. [3+2+2+3]

3 + 2 + 2 + 3 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	Schizophrenia	Evoked potentials and gating, not the resting record
2	Catatonia	Mostly to exclude something, and what that something is
2	Obsessive compulsive disorder	The error signal, and frontal rhythm
3	Delirium	The one diagnosis where EEG genuinely decides

CONDITION	RESTING EEG	EVOKED AND EVENT RELATED	WHAT IT IS USED FOR
Schizophrenia	Non-specific; no diagnostic pattern	Reduced P300 amplitude with delayed latency, P50 gating failure, reduced mismatch negativity, weak gamma response	Endophenotypes for research, not diagnosis
Catatonia	Normal, or diffuse non-specific slowing	Not diagnostically useful	To exclude non-convulsive status epilepticus
Obsessive compulsive disorder	Increased frontal beta in some series	Enhanced error related negativity, present before onset and in unaffected relatives	A candidate trait marker
Delirium	Diffuse slowing, loss of alpha, more theta and delta; triphasic waves in hepatic causes	Slowing tracks severity and settles as the patient clears	Separates delirium from a functional psychosis

Amber cells are the discriminators. Only one row on this page describes a test that changes what you do next, and saying which one is itself a mark.

- **What each measure means.** P50 gating asks whether the brain suppresses a repeated click; mismatch negativity, whether it notices an odd tone unattended; P300, whether it registered a rare target. All three are weak in schizophrenia and attention independent, which is why they survive as endophenotypes.
- **Delirium, in practice.** Slowing appears early and is graded, so a normal record in a confused patient argues against delirium and toward a functional cause.
- **The catatonia trap.** Catatonia and non-convulsive status epilepticus look alike at the bedside, and a benzodiazepine improves both. The EEG is what separates them, so it is ordered for exclusion rather than for confirmation.

WHAT EARNS THE MARKS

- **Resting EEG kept apart from evoked potentials.** Merging them is the commonest error on this question.
- **Each finding named with its purpose,** research marker or clinical decision.
- **Non-convulsive status epilepticus named under catatonia,** which is the safety point the question is testing.
- **Delirium identified as the one diagnostic use,** with the direction of the change stated.

SEE ALSO Slot I - 08 biological markers Slot I - 09 sleep stages and EEG **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

I - 06

Describe the synthesis, receptors and major pathways of glutamate in brain. [3+3+4]

3 + 3 + 4 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 **Synthesis and disposal**	The glutamine cycle, and why the astrocyte is essential
3 **Receptors**	Three ionotropic, three metabotropic groups, with their jobs
4 **Pathways and relevance**	Four named tracts, then the psychiatric use

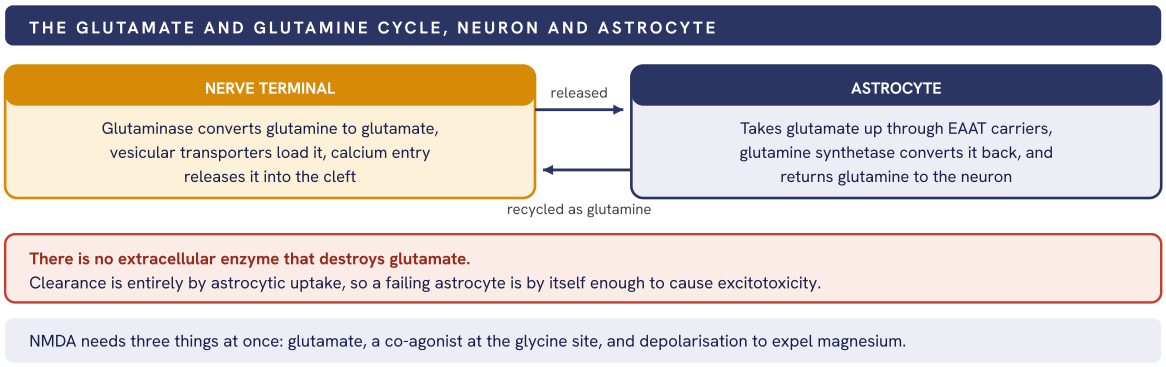

Fig 006.1 Synthesis and clearance as one loop between neuron and astrocyte, with the two facts that follow: no enzymatic breakdown, and a receptor that fires only on coincidence.

RECEPTOR	TYPE	WHAT IT DOES	DRUG THAT ACTS HERE
AMPA	Ionotropic, sodium	Fast excitation, moment to moment	Perampanel in epilepsy
NMDA	Ionotropic, calcium	Coincidence detection, plasticity	**Ketamine, memantine, alcohol**
Kainate	Ionotropic	Modulates release, some fast excitation	Topiramate in part
mGluR groups I to III	Metabotropic, G protein	Tunes gain rather than carrying the signal	Investigational only

- **Major pathways.** Corticocortical association fibres, the corticostriatal projection, reciprocal corticothalamic and thalamocortical fibres, and inside the temporal lobe the perforant path and Schaffer collaterals.
- **Why psychiatry cares.** Ketamine and phencyclidine block the NMDA receptor and reproduce positive, negative and cognitive symptoms in healthy volunteers, the strongest argument for the glutamate model of schizophrenia. The same receptor carries ketamine's rapid antidepressant effect.

WHAT EARNS THE MARKS

- **The astrocyte named as the only clearance route.** It is the fact the whole excitotoxicity story rests on.
- **Ionotropic and metabotropic separated,** with all three ionotropic receptors named.
- **The three NMDA requirements listed together.** Glutamate, co-agonist, depolarisation.
- **Named pathways, not just named receptors,** since the question asks for both.

SEE ALSO Slot I - 04 nitric oxide Slot I - 13 synaptic plasticity

I - 07

Describe the biosynthesis, transmission and implications in psychiatric diagnosis of endocannabinoids. [2+3+5]

2 + 3 + 5 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Biosynthesis	Two ligands, made on demand from membrane lipid
3 Transmission	Backwards across the synapse, then broken down locally
5 Psychiatric implications	Cannabis and psychosis first, then the honest limits

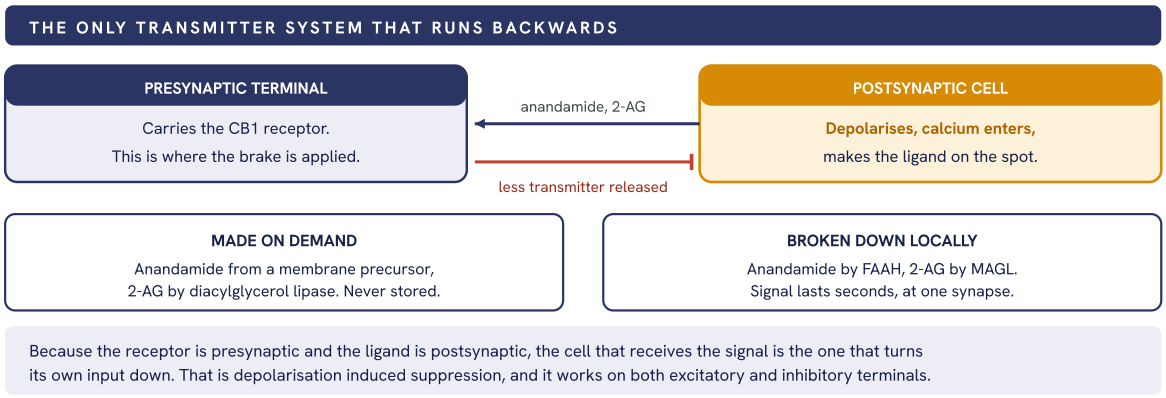

Fig 007.1 The endocannabinoid synapse: ligand made postsynaptically on demand, receptor sitting presynaptically, and a brake applied by the receiving cell onto its own input.

- **The two ligands and the two receptors.** Anandamide and 2-arachidonoylglycerol are the ligands, both derived from arachidonic acid in membrane phospholipid. CB1 is the brain receptor, dense in hippocampus, basal ganglia, cerebellum and cortex and sparse in the brainstem, which is why cannabis is not respiratory depressant. CB2 sits mainly on immune cells and microglia.
- **Cannabis and psychosis.** Tetrahydrocannabinol is a partial CB1 agonist and reproduces positive and cognitive symptoms in volunteers. Prospective cohorts show a dose dependent rise in later psychosis, larger for early and for high potency use. Cannabidiol is not psychotomimetic and has been studied as an antipsychotic.
- **Where the evidence stops.** No endocannabinoid measure is a diagnostic test. Raised anandamide in cerebrospinal fluid and altered CB1 binding are reported in schizophrenia but are not specific. Causation in the cannabis association is supported by dose response and by genetic studies, but reverse causation and confounding are not fully excluded.

WHAT EARNS THE MARKS

- **Retrograde signalling stated as the defining property,** with the receptor placed presynaptically.
- **Made on demand, not stored,** and each ligand paired with the enzyme that ends it.
- **The sparse brainstem CB1 density explained,** since it is why overdose does not kill.
- **Diagnosis answered honestly.** No test exists, and saying so with the reason earns the top of this band.

SEE ALSO Slot I - 06 glutamate receptors Slot I - 08 biological markers

I - 08

Discuss the biological markers of any two major psychiatric illnesses. [5+5]

5 + 5 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Define and classify	State against trait, and the four properties a marker needs
3	Schizophrenia	Electrophysiological, imaging, ocular, biochemical
3	Depression	Endocrine, sleep, inflammatory
2	The verdict	Why none is yet a diagnostic test

DEFINE IT BEFORE YOU LIST ANYTHING

A biological marker is an objectively measured biological characteristic that indicates a normal process, a disease process, or a response to treatment. A state marker changes with the episode; a trait marker persists in remission and in unaffected relatives, which is what makes it an endophenotype.

DOMAIN	SCHIZOPHRENIA	DEPRESSION	STATE OR TRAIT
Electrophysiology	P50 gating failure, reduced P300 and mismatch negativity	Shortened REM latency, increased REM density	**Mostly trait**
Endocrine	Blunted growth hormone response to dopamine agonists	Dexamethasone non-suppression, raised cortisol	State
Imaging	Enlarged ventricles, reduced temporal grey matter	Reduced hippocampal volume with recurrence	**Trait for the first, cumulative for the second**
Ocular	Smooth pursuit eye movement abnormality	Not established	**Trait, present in relatives**
Inflammatory	Raised inflammatory markers in a subgroup	Raised interleukin 6 and C reactive protein	State
Neurotrophic	Reduced BDNF reported inconsistently	Low serum BDNF that rises with treatment	State

Amber cells are the trait markers. They are the ones that survive recovery and appear in well relatives, which is what makes them useful for genetics rather than for diagnosis.

- **The four properties a marker must have.** Sensitivity, specificity, reliability across labs, and independence from treatment. Almost every marker above fails at least one, most often specificity.
- **The verdict.** No biological marker is currently diagnostic in psychiatry. They separate groups, not individuals, and the distributions of ill and well people overlap. The working uses today are research stratification, endophenotypes for genetics, and monitoring rather than diagnosis.

WHAT EARNS THE MARKS

- **Two illnesses chosen and named at the top,** since the question allows a choice and expects you to make it.
- **State separated from trait,** applied to each marker rather than stated once.
- **Markers grouped by domain,** which keeps ten items readable as two answers.
- **The honest closing verdict.** Claiming a diagnostic test loses the last marks.

SEE ALSO Slot I - 05 electrophysiological abnormalities Slot I - 99 personalised medicine

I - 09

What is polysomnography? Briefly describe various stages of sleep along with their EEG findings. Discuss the neurobiology of sleep and wakefulness. [2+3+5]

2 + 3 + 5 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Polysomnography	The three channels that stage sleep, then the rest
3 The stages	Each with its signature waveform and its share of the night
5 Neurobiology	The switch, the two drives, and the REM mechanism

STAGE	EEG SIGNATURE	EYES AND MUSCLE TONE	SHARE OF THE NIGHT
N1	Posterior alpha of wake drops out, low voltage mixed frequency, vertex sharp waves	Slow rolling eye movements	Roughly a twentieth
N2	**Sleep spindles and K complexes**	Eyes still, tone reduced	**About half**
N3	**Delta, under 4 hertz, high amplitude**	Eyes still, tone low	About a fifth, front loaded
REM	Low voltage mixed frequency, saw-tooth waves	**Rapid eye movements with atonia**	About a fifth, back loaded

- **What polysomnography is.** A multi-channel overnight recording. Three channels stage it: EEG, electro-oculogram, chin electromyogram; the rest measure breathing, oxygen, heart, legs and position.
- **Neurobiology.** Wake is held by the ascending arousal system: noradrenaline, serotonin, histamine, dopamine, acetylcholine. Sleep is imposed by the ventrolateral preoptic nucleus using GABA and galanin. The two inhibit each other; orexin stabilises wake, and adenosine with the suprachiasmatic clock sets timing.
- **REM, and what shortens its latency.** Pontine cholinergic neurons generate REM while aminergic firing falls silent, giving dreams with atonia. Short REM latency, increased REM density and reduced slow wave sleep is the classic depression profile. Sleep onset REM points to narcolepsy.

Fig 009.1 The architecture of one night, with the parasomnias placed in the half of the night whose sleep stage produces them.

WHAT EARNS THE MARKS

- **Three staging channels named,** EEG, eye movement and chin tone, before the rest of the montage.
- **Spindles and K complexes tied to N2,** delta to N3, sawtooth to REM.
- **Both drives named,** homeostatic adenosine and the circadian clock, not sleep pressure alone.

SEE ALSO Slot I - 03 neurobiology of sleep and wakefulness Slot I - 11 classification of sleep disorders **SPRINT** Day 1, Functional Neuroanatomy and Neurophysiology

I - 10

What are the various type of the memory? Describe neurobiology of the memory. Describe rehabilitation of memory problems in chronic mental illness. [2+3+5]

2 + 3 + 5 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The classification	Name the axis, then divide: awareness first, duration second
3 Neurobiology	Anatomy per branch, then the shared cellular mechanism
5 Rehabilitation	Restore, compensate, adapt, and what the evidence supports

BRANCH	SUBTYPE	WHAT IT HOLDS	ANATOMY
Declarative	Episodic	Events, with their time and place	**Hippocampus, medial temporal**
Declarative	Semantic	Facts, stripped of context	Anterolateral temporal cortex
Non-declarative	Procedural	Skills and habits	**Striatum, cerebellum**
Non-declarative	Priming	Easier processing after exposure	Neocortex
Non-declarative	Conditioning	Learned associations, including fear	Amygdala, cerebellum
By duration	Sensory, short term, working, long term	The second axis, cutting across the first	Prefrontal for working memory

The axis is awareness. Duration is a second, independent axis, so working memory is not a stage on the way to long term memory.

- **The shared mechanism.** Whatever the branch, the cellular event is the same: long term potentiation, made permanent by new protein synthesis and structural change at the spine.
- **Rehabilitation, three strategies.** Restorative training of the impaired function; compensatory training that routes around it; and environmental adaptation that removes the demand. In chronic mental illness the compensatory and adaptive routes carry most of the benefit.

WHAT THE EVIDENCE SUPPORTS IN CHRONIC ILLNESS

Errorless learning and spaced retrieval, leaning on intact procedural memory. External aids: alarms, pill boxes, written routines. Cognitive remediation improves cognition in schizophrenia. Treat the reversible contributors first: sedative load, anticholinergic burden, depression, poor sleep, thyroid and vitamin B12.

WHAT EARNS THE MARKS

- **The axis of division stated before the tree.** A list with no axis reads as recall.
- **Duration named as a second, independent axis,** not a deeper level of the first.
- **Errorless learning justified by the anatomy:** intact procedural carrying damaged declarative.
- **Reversible contributors named first.** Anticholinergic burden is most often missed.

SEE ALSO Slot I - 01 neurobiology of memory Slot I - 02 memory impairment in psychiatric illness **SPRINT** Day 1, Functional Neuroanatomy and Neurophysiology

I - 11

Classify sleep disorders. How would you manage a case of restless leg syndrome? How do you manage benzodiazepine dependence in a chronically mentally ill person? [2+4+4]

2 + 4 + 4 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Classification**	Seven categories, named, with one example each
4	**Restless legs**	Confirm, correct the cause, then treat in order
4	**Benzodiazepine taper**	Assess, switch, taper slowly, support throughout

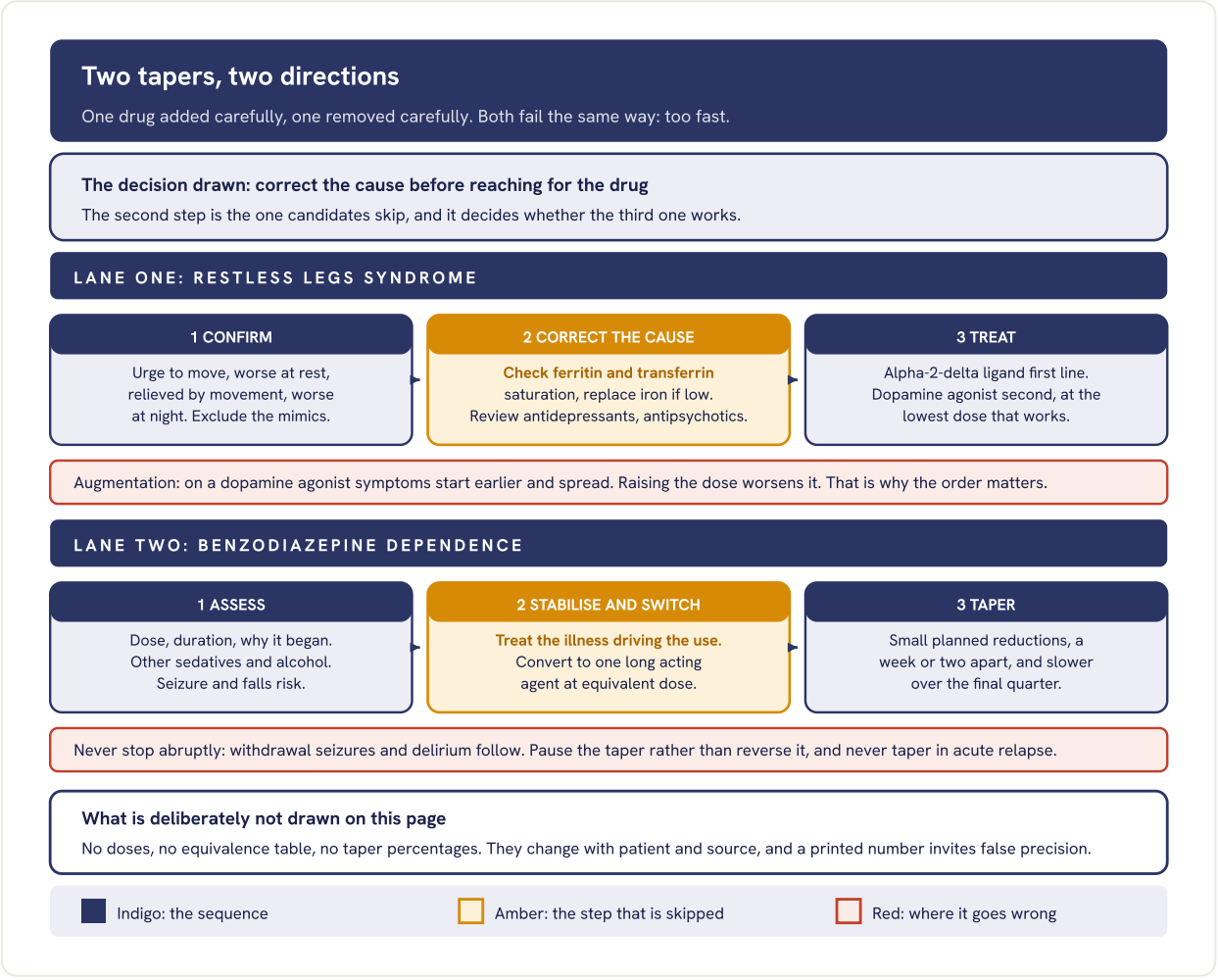

Fig 011.1 Both managements as three steps of the same shape, each with its failure mode below.

CATEGORY	ONE EXAMPLE EACH
Insomnia disorders	Chronic insomnia disorder
Sleep related breathing disorders	Obstructive sleep apnoea
Central disorders of hypersomnolence	Narcolepsy
Circadian rhythm sleep-wake disorders	Delayed sleep-wake phase disorder
Parasomnias	Sleepwalking, REM sleep behaviour disorder
Sleep related movement disorders	**Restless legs syndrome**

Six of the seven categories; the seventh is a residual other. Filing restless legs under movement disorders, not insomnia, is the classification mark.

- **The one that catches people out.** Antipsychotics and antidepressants can cause or worsen restless legs, and akathisia is the differential. Akathisia is generalised inner restlessness at any hour; restless legs is in the legs, at rest, in the evening, and eased by walking.

WHAT EARNS THE MARKS

- **Named categories,** with restless legs filed under movement disorders.
- **Iron studies before any drug,** and augmentation as the reason for the order.
- **The illness treated and a long acting agent substituted before tapering.**
- **Akathisia offered as the differential.** It is this question's discriminator.

SEE ALSO Slot I - 03 neurobiology of sleep Slot I - 09 polysomnography **SPRINT** Day 17, Eating and sleep-wake disorders

I - 12

Name various dopamine pathways of brain. Describe their significance in Psychiatry in detail. [3+7]

3 + 7 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY

DNB, NTRUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

3	The four pathways	Origin and destination for each, named precisely
3	The blockade problem	One drug, four pathways, four different consequences
2	Receptors and synthesis	D1 and D2 families, and where the transmitter comes from
2	Beyond blockade	Salience, addiction, and partial agonism

PATHWAY	FROM, TO	WHAT IT NORMALLY DOES	WHAT BLOCKADE PRODUCES
Mesolimbic	Ventral tegmental area to nucleus accumbens	Assigns salience and reward value	**Positive symptoms settle; this is the therapeutic pathway**
Mesocortical	Ventral tegmental area to prefrontal cortex	Working memory, motivation, drive	**Negative and cognitive symptoms worsen**
Nigrostriatal	Substantia nigra pars compacta to dorsal striatum	Selects and releases movement	**Parkinsonism, dystonia, akathisia, tardive dyskinesia**
Tuberoinfundibular	Arcuate nucleus to anterior pituitary	Tonically inhibits prolactin release	**Hyperprolactinaemia, galactorrhoea, amenorrhoea, sexual dysfunction**

Every amber cell is produced by the same molecule at the same receptor. An antipsychotic cannot choose its pathway, which is why the side effect profile is predictable from this table alone.

- **Synthesis and receptors.** Tyrosine to DOPA by tyrosine hydroxylase, the rate limiting step, then to dopamine by DOPA decarboxylase. Receptors fall into a D1 family that stimulates adenylyl cyclase and a D2 family that inhibits it. Antipsychotic potency tracks D2 affinity.
- **Salience, not pleasure.** Mesolimbic dopamine marks what matters rather than what feels good. Aberrant salience gives ordinary events undue significance, which is a mechanism for how delusions form, and explains why blockade dampens conviction before it removes the belief.
- **Where the simple model fails.** Blockade is immediate but the antipsychotic response takes weeks, so receptor occupancy cannot be the whole story. Clozapine works best with relatively low D2 occupancy, and partial agonists such as aripiprazole stabilise rather than block, which is why they raise prolactin less.

WHAT EARNS THE MARKS

- **Four pathways with both ends named.** Mesolimbic and mesocortical share an origin, and saying so shows the pathways are understood.
- **One drug, four consequences,** laid out as a single table rather than as a side effect list.
- **Salience rather than pleasure** as the account of mesolimbic function.
- **The delay problem raised honestly.** It is the strongest evidence that blockade alone is insufficient.

SEE ALSO Slot I - 21 basal ganglia pathways Slot I - 97 pineal gland and prolactin rhythm

I - 13

Define synaptic plasticity. How is it important in psychiatry? [2+8]

2 + 8 | NEUROCHEMISTRY AND NEUROPHYSIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Definition and forms**	One sentence, then short term against long term, functional against structural
3 **The mechanism**	Induction, expression, and what makes it last
3 **Disorders**	Where plasticity is excessive, deficient, or misdirected
2 **Treatments**	Every effective treatment as a plasticity intervention

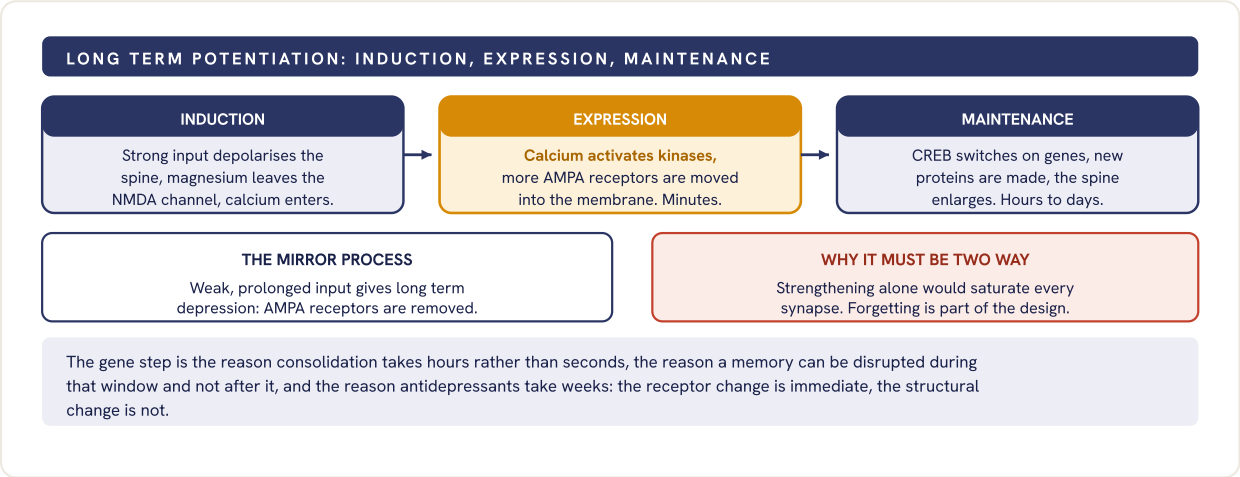

Fig 013.1 Potentiation in three stages with its mirror process alongside, and the gene step marked as the source of every clinically important delay.

- **The definition.** The capacity of a synapse to change the strength of its transmission in response to activity. It is functional when receptor number or release probability changes, and structural when spines and connections are added or lost.
- **Where it goes wrong.** Excessive synaptic pruning in adolescence, with complement C4 implicated, in schizophrenia. Stress and glucocorticoids retract dendrites in hippocampus and prefrontal cortex while the amygdala grows, in depression. Misdirected strengthening of reward circuits in addiction, and of fear circuits in post-traumatic stress.
- **Every treatment is a plasticity treatment.** Antidepressants raise BDNF signalling and restore spine density over weeks. Ketamine produces rapid synaptogenesis through mTOR. Electroconvulsive therapy and repetitive transcranial stimulation induce plasticity directly. Psychotherapy and exposure work by extinction learning, which is plasticity in prefrontal to amygdala pathways.

WHAT EARNS THE MARKS

- **A one sentence definition first,** since only two marks are on offer for it.
- **Potentiation and depression given as a pair.** Naming only strengthening misses why the system works.
- **The gene step used to explain a clinical delay.** That single link is the top band on this question.
- **Psychotherapy included among plasticity treatments,** not only the drugs.

SEE ALSO Slot I - 01 neurobiology of memory Slot I - 06 glutamate receptors

AREA 02 OF 12 · P1-A01

Functional neuroanatomy and neuroimaging

12	13.5%	I-14	I-25
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 5 · RECURRENT 1 · SINGLE 6

REVISION ORDER

The limbic group first and as one block: the Papez circuit, the limbic system, the amygdala and the two hippocampal slots overlap almost completely. The cortical group next, prefrontal, frontal, cerebral cortex and occipital, and the frontal slot is the long one. The caudate nucleus is asked twice and is one answer at two widths. Thalamus, corpus callosum and basal ganglia last, and all three are short.

WHAT IS IN THIS AREA

Twelve structures, close to one each: the corpus callosum, the Papez circuit with the caudate nucleus, the prefrontal cortex, the amygdala, the cerebral cortex with its cellular organisation and circuitry, the thalamus, the limbic system, the basal ganglia with the hippocampus, the frontal lobe with three injury syndromes and parietal atrophy, the hippocampal formation with its subfield volumes, the caudate nucleus on its own, and the occipital cortex. The registered name of this area also carries neuroimaging. Imaging is a limb of one slot here rather than the subject of any, and the area is described by what its slots actually ask.

I - 14

Discuss the role of Corpus callosum. [10]

10 FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 **What it is**	The largest commissure, and its five named segments
3 **Fibre topography**	Which cortex each segment joins, front to back
3 **What it does**	Transfer, lateralisation, bimanual and interhemispheric control
2 **When it fails**	Agenesis, callosotomy, and the disconnection signs

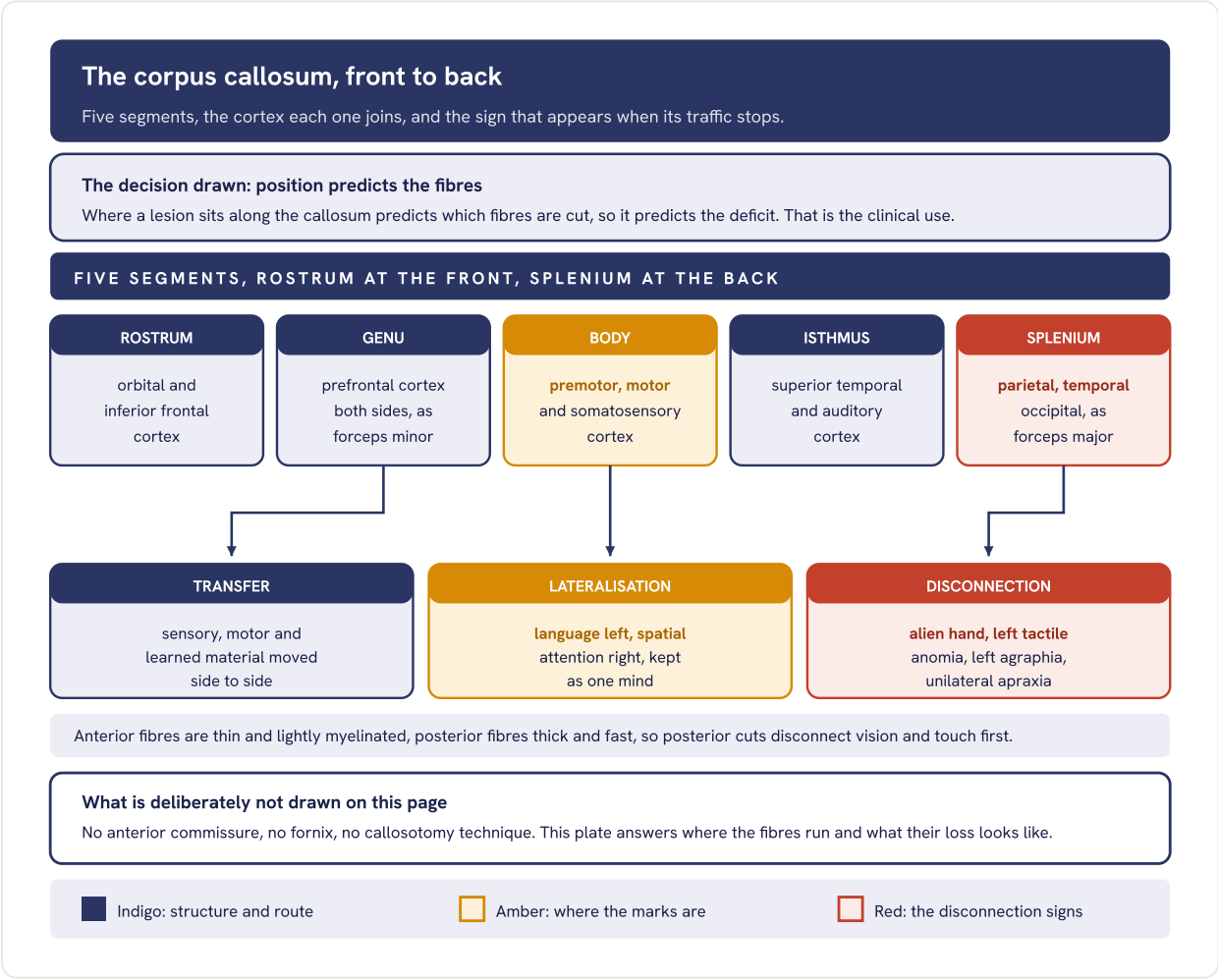

Fig 014.1 The callosum drawn as five segments with the cortex each joins, and the three consequences that follow from the traffic they carry.

THE DEFINITION

The largest white matter commissure of the brain, a sheet of myelinated axons joining homologous and heterologous cortical areas of the two hemispheres. It is topographically ordered: rostrum and genu in front carry frontal fibres, the splenium behind carries parieto-occipital fibres.

- **Transfer.** Sensory, motor and learned information crosses here, which is why a split-brain patient cannot name an object felt only by the left hand.

- **Integration with lateralisation.** Language sits left and spatial attention right; the callosum lets specialised hemispheres act as one, and coordinates bimanual movement.
- **Callosal syndromes.** Agenesis (often with other midline defects), surgical callosotomy for intractable epilepsy, and Marchiafava-Bignami disease from chronic alcohol use.

WHAT EARNS THE MARKS

- **The five segments named in order.** Rostrum, genu, body, isthmus, splenium, each with the cortex it joins.
- **Topography stated as a rule** before any sign is listed: front carries frontal, back carries posterior sensory.
- **Disconnection signs tied to a segment.** Left tactile anomia and left agraphia belong to anterior fibres, hemialexia to the splenium.
- **The three failure states,** agenesis, callosotomy and Marchiafava-Bignami, named as such.

SEE ALSO Slot I - 16 prefrontal cortex Slot I - 22 frontal and parietal lobe syndromes **SPRINT** Day 1, Functional Neuroanatomy and Neurophysiology

I - 15

Neuroanatomy of Papez circuit and a brief mention about the caudate nucleus. [10]

10 FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

KNRUHS, TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	The proposal	Papez 1937, and what the loop was meant to explain
4	The loop in order	Six stations, hippocampus back to hippocampus
2	What it explains	Diencephalic amnesia sits on this ring
2	Caudate, briefly	Position, loop membership, two clinical uses

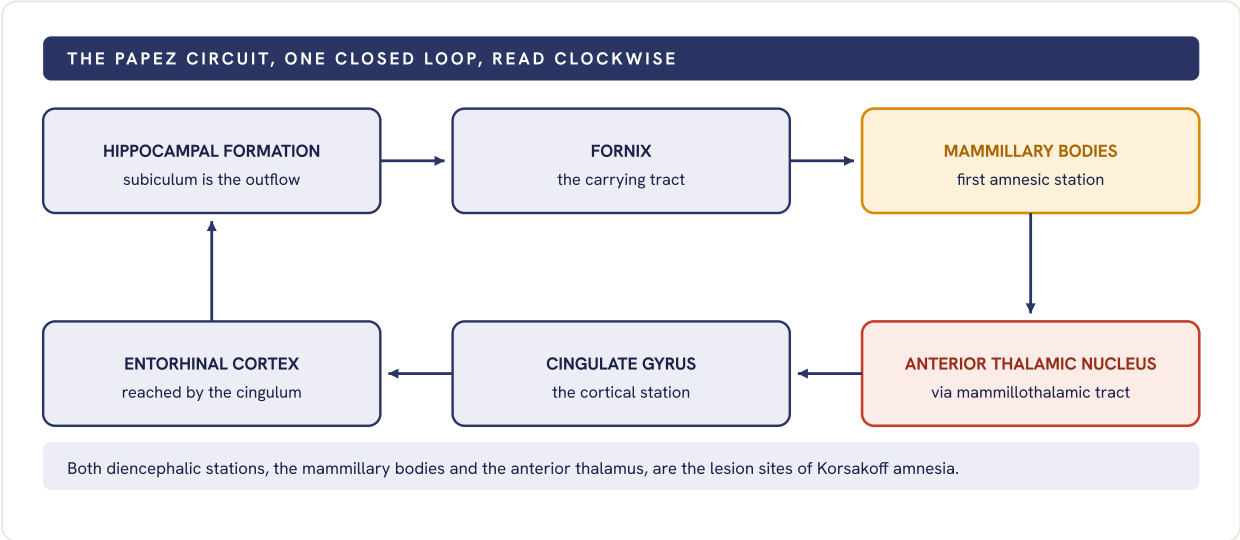

Fig 015.1 The circuit as a closed ring, with the two diencephalic stations that carry the amnesia marked out from the four that do not.

- **What Papez proposed.** In 1937 a cortical to hypothalamic loop offered as the substrate of emotional experience. MacLean later widened it into the limbic system.
- **Why the anatomy outlived the claim.** It is now taught as an episodic memory circuit: thiamine deficient Korsakoff amnesia, fornix section and anterior thalamic infarcts all sit on this ring.
- **Caudate nucleus.** A C-shaped mass with head, body and tail curving in the lateral ventricle wall, continuous with the putamen and forming the associative striatum.
- **Caudate, clinically.** Head atrophy with boxcar ventricles in Huntington disease, hypermetabolism in obsessive compulsive disorder that settles as the illness responds.

WHAT EARNS THE MARKS

- **All six stations, in order, with the two tracts named.** Fornix and mammillothalamic tract are the links most answers skip.
- **The circuit tied to amnesia, not only to emotion.** Saying where Korsakoff sits is the line that separates the bands.
- **One honest limit.** Emotion is not made by one ring; the amygdala and prefrontal circuits lie outside Papez.

SEE ALSO Slot I - 20 limbic system Slot I - 24 caudate nucleus SPRINT

Day 1, Functional Neuroanatomy and Neurophysiology

I - 16

Prefrontal cortex. [10]

10 FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

KNRUHS, MUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	What defines it	Granular cortex in front of premotor, defined by its thalamic input
4	The three subdivisions	Each with its job and the syndrome its loss makes
2	Connections	Loops with striatum, thalamus, amygdala and hippocampus
2	Psychiatric relevance	Late maturation, hypofrontality, and what is testable

THE DEFINITION

The granular association cortex of the frontal lobe lying anterior to the premotor areas. It is defined anatomically by its reciprocal connection with the mediodorsal nucleus of the thalamus, and functionally as the seat of executive control: holding a goal, selecting an action, and suppressing the rest.

SUBDIVISION	WHERE IT SITS	WHAT IT DOES	WHAT ITS LOSS LOOKS LIKE
Dorsolateral	Middle frontal gyrus, lateral surface	Working memory, planning, set shifting, abstraction	Dysexecutive: perseveration, concrete thinking, poor planning
Orbitofrontal	Above the orbit, with gyrus rectus	Reward valuation, impulse control, social judgement	Disinhibited: tactless, impulsive, socially coarse
Ventromedial and anterior cingulate	Medial wall	Motivation, drive, error monitoring, autonomic tone	Apathetic: abulia, and akinetic mutism when severe

Three syndromes, not one frontal syndrome. Naming which subdivision produced the picture is what the examiner is checking.

- **Connections.** Five parallel cortico-striato-thalamo-cortical loops run through the caudate and thalamus, with heavy dopaminergic input from the ventral tegmental area and direct links to amygdala and hippocampus.
- **Development.** It myelinates last, into the third decade, which is the anatomical reason adolescent judgement and impulse control mature after appetite and emotion do.
- **Psychiatric relevance.** Reduced dorsolateral activation on working memory tasks in schizophrenia, subgenual cingulate overactivity in depression, and orbitofrontal loop overactivity in obsessive compulsive disorder.

WHAT EARNS THE MARKS

- **The mediodorsal thalamic connection as the definition.** It is what makes prefrontal cortex a region and not a direction.
- **Three syndromes, each tied to its subdivision.** A single undifferentiated frontal syndrome is a mid-band answer.
- **One bedside test per subdivision.** Verbal fluency and Wisconsin sorting for dorsolateral, go and no-go for orbitofrontal.

SEE ALSO Slot I - 22 frontal lobe syndromes Slot I - 19 thalamic nuclei **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

I - 17

Write briefly about the functional neuroanatomy of amygdala. Discuss the psychiatric implications related with amygdala. [5+5]

5 + 5 | FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

DNB, MUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	Position and nuclei	Three nuclear groups, and which one is the gate
3	Input and output	Two roads in, four autonomic routes out
3	Disorders	Anxiety, trauma, mood, psychosis, and the direction of change
2	What treatment does	Prefrontal control as the mechanism of extinction

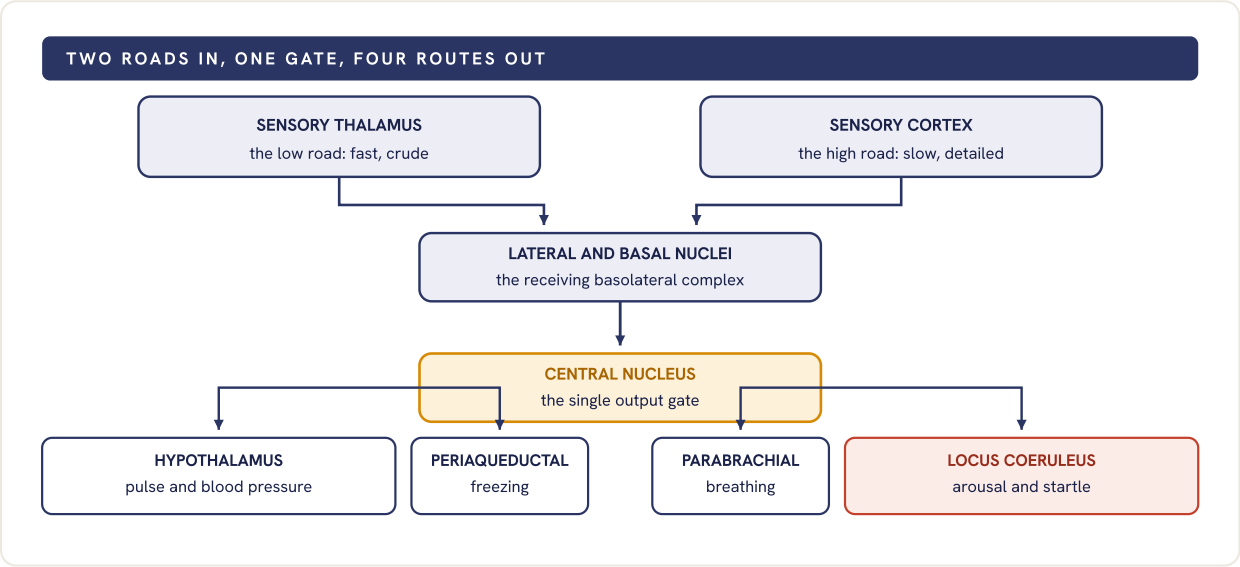

Fig 017.1 The fear pathway drawn as two afferent roads converging on the basolateral complex, one central output gate, and the four brainstem and hypothalamic routes that produce the bodily signs.

- **Position and nuclei.** An almond of grey matter in the medial temporal lobe, in front of the hippocampal head. Three groups: basolateral (input), centromedial (output), and cortical with the olfactory link.
- **What it computes.** Assigns emotional value to a stimulus, then drives the body to match. Fear conditioning is learned here, and the prefrontal cortex learns extinction by inhibiting it.
- **Across disorders.** Overactive to threat and to fearful faces in anxiety and post-traumatic stress with weak prefrontal restraint. Overactive to negative faces in depression. Reduced volume and abnormal face processing in schizophrenia and in autism.
- **The lesion picture.** Bilateral loss gives Kluver-Bucy syndrome, with placidity, hyperorality and loss of fear, and abolishes the startle potentiation that fear normally produces.

WHAT EARNS THE MARKS

- **The three nuclear groups, with their traffic direction.**
- **Two roads in and named routes out.** Each output tied to the sign it produces earns more than a list of nuclei.
- **The direction of change per disorder,** up or down, not simply involvement.

SEE ALSO Slot I - 20 limbic system in psychiatric disorders SPRINT

Day 1, Functional Neuroanatomy and Neurophysiology

I - 18

Describe the neuroanatomy and cellular organization of cerebral cortex. Describe its functional circuitry with a suitable diagram. [3+3+4]

3 + 3 + 4 | FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Gross organisation	Lobes, allocortex against neocortex, Brodmann mapping
3 Cellular organisation	Six layers, two cell classes, the column as the unit
4 Functional circuitry	The diagram: what enters at four, what leaves at five and six

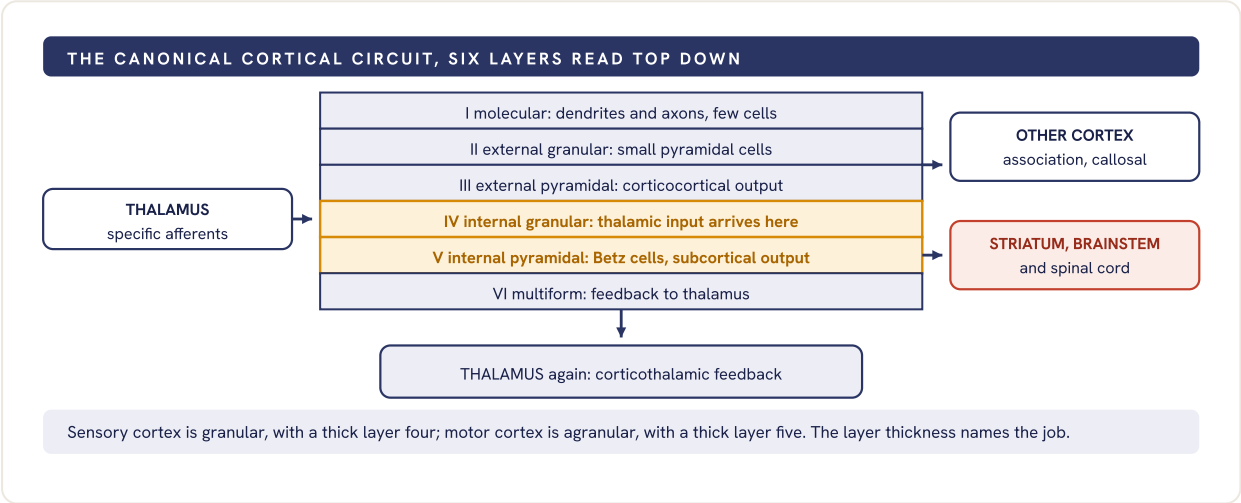

Fig 018.1 The six laminae with the traffic each carries: input at layer four, corticocortical output from layers two and three, subcortical output from layer five, thalamic feedback from layer six.

- **Gross organisation.** Neocortex is six layered and covers most of the surface; allocortex, meaning the hippocampal and olfactory cortex, has three layers. Brodmann divided the surface by cytoarchitecture, and those numbers are still the working map.
- **Two cell classes.** Pyramidal cells are excitatory, glutamatergic and projecting; interneurons are inhibitory, GABAergic and local. Parvalbumin interneurons pace gamma rhythm, and their loss is a leading account of cognitive symptoms in schizophrenia.

UNIT	WHAT IT IS	WHY IT MATTERS
Minicolumn	A vertical chain of about a hundred cells sharing one receptive field	The smallest repeating processing unit
Cortical column	Minicolumns grouped across all six layers	Same circuit everywhere, tuned by its input; a Brodmann number names a set of them

WHAT EARNS THE MARKS

- **Six layers named in order,** with the traffic of each, not simply the list.
- **The diagram, since the question asks for one.** Input at four, output at five, feedback at six is the whole circuit.
- **Granular against agranular cortex** explained by layer thickness rather than asserted.

SEE ALSO Slot I - 19 thalamic nuclei Slot I - 16 prefrontal cortex **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

I - 19

Describe functions of Thalamus. [10]

10 FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

KNRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 **What it is**	The gateway to cortex, and the one sense that bypasses it
4 **Nuclei and their traffic**	Grouped by the lamina, each with its cortical target
2 **Beyond relay**	Gating, arousal, and the generation of sleep rhythms
2 **Clinical correlates**	Amnesia, thalamic pain, and the schizophrenia findings

NUCLEUS	MAIN INPUT	PROJECTS TO	WHAT ITS LOSS SHOWS
Anterior	Mammillary bodies, by the mammillothalamic tract	Cingulate gyrus	Amnesia, on the Papez ring
Mediodorsal	Amygdala, olfactory and limbic cortex	Prefrontal cortex	Amnesia, executive loss
Ventral posterior	Medial lemniscus, spinothalamic, trigeminal	Postcentral gyrus	Hemisensory loss, thalamic pain
Geniculate bodies	Optic tract, inferior colliculus	Visual and auditory cortex	Field defect, deafness
Reticular and intralaminar	Cortex and brainstem collaterals	Thalamus itself and diffuse cortex	Impaired arousal and attention

The reticular nucleus is the exception in the table: it is GABAergic, it projects to the thalamus rather than to cortex, and it is the gate the others pass through.

- **More than a relay.** Every sense but olfaction is routed here, and the relay is not passive: the reticular nucleus gates what reaches cortex, which is the anatomy behind selective attention.
- **Rhythm generation.** Thalamocortical loops with the reticular nucleus produce sleep spindles and the slow oscillations of deep sleep, so thalamic damage disturbs sleep architecture as well as sensation.
- **Psychiatric correlates.** Reduced mediodorsal volume and disrupted thalamocortical connectivity are among the more replicated structural findings in schizophrenia, and the pulvinar is implicated in the attention deficits.

THE LINE WORTH WRITING FIRST

The thalamus is the obligatory gateway to the cerebral cortex for all sensory modalities except olfaction, and every cortical area sends a return projection to the thalamic nucleus that supplies it. Relay is reciprocal, never one way.

WHAT EARNS THE MARKS

- **The olfaction exception, stated as an exception.** It is the cheapest mark on the page.
- **Nuclei grouped, each with its cortical target.** A bare list of names without destinations is recall.
- **Gating and rhythm named alongside relay,** which is what turns a list into a function answer.
- **Two clinical anchors,** diencephalic amnesia and thalamic pain, each tied to its nucleus.

SEE ALSO Slot I - 15 Papez circuit Slot I - 18 cortical circuitry **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

I - 20

a) Write about functional neuroanatomy of limbic system. b) Discuss role of limbic system in psychiatric disorders. [5+5]

5 + 5 | FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

DNB - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	What the term covers	Broca to MacLean, and why the boundary is disputed
3	The components	Three tiers, cortical, subcortical, diencephalic
3	Disorder by disorder	Which node, which direction of change
2	What follows for treatment	Targets that are actually used in practice

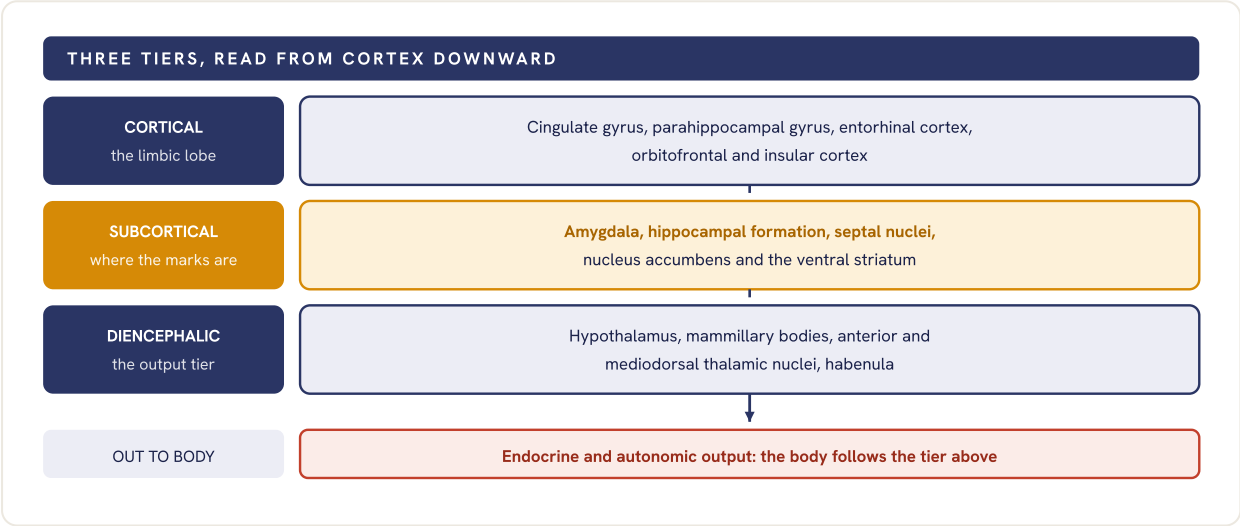

Fig 020.1 The limbic system laid out as three tiers rather than a ring, so that the cortical, subcortical and diencephalic members can be named separately and the output route stays visible.

- **What the term covers.** Broca named the limbic lobe as the ring of cortex around the corpus callosum; Papez gave it a circuit and MacLean gave it a name. The boundary has never been settled, so name your list before you use it.
- **Across disorders, and where they are targeted.** Amygdala overactivity with weak prefrontal restraint in anxiety and post-traumatic stress; smaller hippocampal volume in recurrent depression; subgenual cingulate overactivity in depression, where deep brain stimulation remains investigational; ventral striatal underactivity to reward in anhedonia and negative symptoms. Cingulotomy and capsulotomy are last resort options in severe refractory obsessive compulsive disorder.
- **Do not stop at the emotional brain.** It also carries episodic memory, olfaction, reward and endocrine control, and the triune brain framing MacLean built around it is no longer supported.

WHAT EARNS THE MARKS

- **A defined membership list before any function.** Saying the boundary is disputed reads as command, not hedging.
- **Three tiers rather than one heap,** with the output route to hypothalamus named.
- **Direction of change per disorder.** Amygdala up, hippocampus down, ventral striatum down to reward.

SEE ALSO Slot I - 15 Papez circuit Slot I - 17 amygdala **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

I - 21

Describe the functional neuroanatomy of basal ganglia. Describe different parts of hippocampus. Discuss the psychiatric implications of basal ganglia and hippocampus. [3+2+5]

3 + 2 + 5 | FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Basal ganglia	The nuclei, then the direct and indirect pathways
2 Hippocampal parts	Dentate, the four CA fields, subiculum
5 Implications	Structure by structure, with the direction of the finding

THE ORGANISING IDEA

Both structures work as loops rather than as centres. Cortex projects to the striatum, the striatum acts on the pallidum and thalamus, and the thalamus returns to the same cortical area. The hippocampus runs a comparable loop through entorhinal cortex. Naming the loop is worth more than naming the parts.

- **Basal ganglia, the parts.** Caudate and putamen make the striatum, globus pallidus has an external and an internal segment, with subthalamic nucleus and substantia nigra completing the set.
- **The two pathways.** The direct pathway carries D1 receptors and releases movement; the indirect pathway carries D2 receptors and suppresses it. Antipsychotic blockade of striatal D2 receptors tips the balance toward suppression, which is parkinsonism.
- **Hippocampal parts.** Dentate gyrus, the cornu ammonis fields CA1 to CA4, and the subiculum, together with entorhinal cortex forming the hippocampal formation. CA1 is the field most vulnerable to hypoxia.

STRUCTURE	THE FINDING	WHERE IT IS SEEN	WHAT IT MEANS CLINICALLY
Caudate	Overactivity in the orbitofrontal loop	Obsessive compulsive disorder	Settles as the illness responds
Caudate	Head atrophy, boxcar ventricles	Huntington disease	Chorea with psychiatric onset
Striatum	D2 blockade above the therapeutic range	Antipsychotic treatment	Parkinsonism, akathisia, dystonia
Hippocampus	Reduced volume, most in CA1 and CA3	Recurrent depression, post-traumatic stress	Partly reversible with recovery

WHAT EARNS THE MARKS

- **Direct against indirect pathway, with D1 and D2 attached.** It is the line that explains extrapyramidal side effects.
- **All hippocampal parts named,** dentate, four CA fields and subiculum, with CA1 flagged as the vulnerable one.
- **Findings given a direction,** up or down, and a disorder. Involvement alone earns little.

SEE ALSO Slot I - 23 hippocampal subfields Slot I - 24 caudate nucleus **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

I - 22

Discuss the functions of frontal lobe with a suitable diagram. Write 3 syndromes associated with injury to frontal lobe. Describe signs and symptoms of parietal lobe atrophy. [3+4+3]

3 + 4 + 3 | FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Frontal functions	The diagram: motor at the back, executive in front
4 Three syndromes	Each named, sited, and given its picture
3 Parietal atrophy	Dominant against non-dominant, then the bilateral case

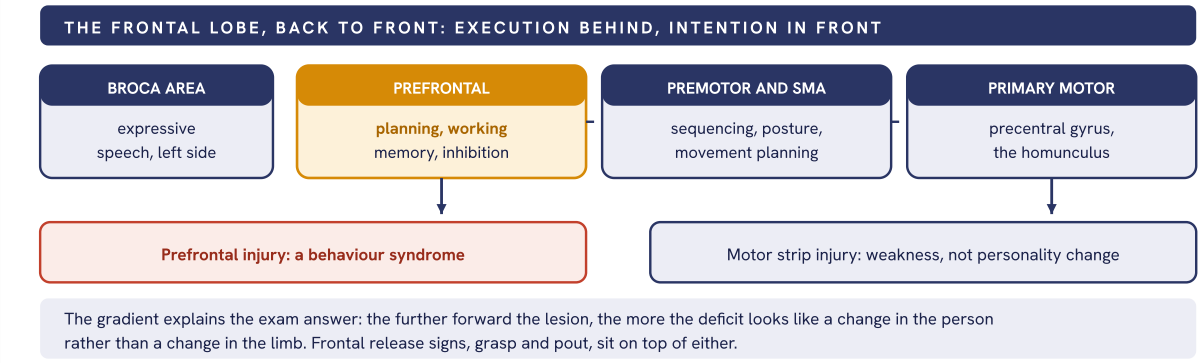

Fig 022.1 The frontal lobe as a back to front gradient, execution at the central sulcus and intention at the pole, with the two kinds of injury picture that follow from it.

SYNDROME	WHERE THE LESION SITS	THE PICTURE
Dysexecutive	Dorsolateral prefrontal	Poor planning and sequencing, perseveration, concrete thinking, reduced verbal fluency
Disinhibited	Orbitofrontal	Tactless, impulsive, jocular, socially coarse, utilisation behaviour, preserved formal testing
Apathetic	Medial frontal and anterior cingulate	Abulia, loss of initiative, sparse speech, akinetic mutism when the lesion is large and bilateral

- **Parietal atrophy, dominant side.** Gerstmann syndrome, meaning agraphia, acalculia, finger agnosia and right to left disorientation, with ideomotor apraxia and receptive language difficulty.
- **Parietal atrophy, non-dominant side.** Contralateral neglect and anosognosia, dressing and constructional apraxia, and loss of topographical sense. Bilateral posterior atrophy gives Balint syndrome: optic ataxia, oculomotor apraxia and simultanagnosia.

WHAT EARNS THE MARKS

- **The diagram, since the question asks for one,** drawn as a back to front gradient rather than a list of areas.
- **Exactly three syndromes, each with its site.** The question counts them, so the answer should too.
- **Parietal signs split by side** before the bilateral case. An unsided list loses the localising mark.

SEE ALSO Slot I - 16 prefrontal cortex Slot I - 25 occipital cortex **SPRINT**

Day 34, Stroke, TBI, delirium and focal brain syndromes

I - 23

What is hippocampal formation? Discuss its role in learning. Describe hippocampal subfield volumes and its use in psychiatry. [2+3+5]

2 + 3 + 5 | FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	Dentate, CA1 to CA4, subiculum, entorhinal: a formation, not a nucleus
3 Role in learning	The trisynaptic circuit, long term potentiation, consolidation
5 Subfield volumetry	How it is measured, what it shows, and what it cannot do

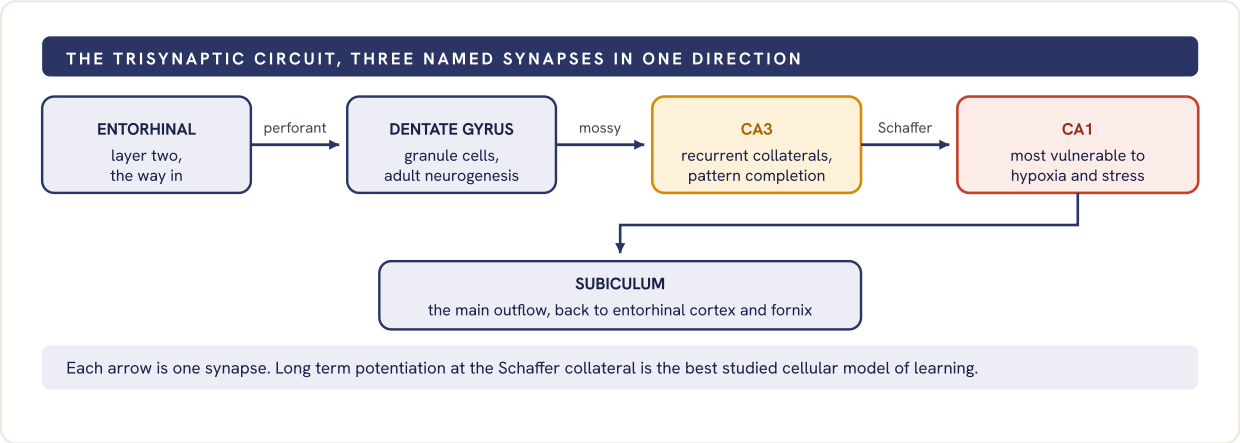

Fig 023.1 The trisynaptic circuit as a one way chain, with the field that completes patterns in amber and the field that dies first in red.

- **Role in learning.** It binds a new episode into a single trace, then hands it to neocortex over months, which is systems consolidation. Damage gives dense anterograde amnesia with graded retrograde loss and intact procedural learning, the pattern first described in the patient H M.

SUBFIELD VOLUMETRY: WHAT IT SHOWS, AND WHAT IT CANNOT

High resolution imaging with automated segmentation gives per field volumes rather than one hippocampal number. CA1 and subiculum loss tracks early Alzheimer disease; CA3 and dentate loss tracks chronic stress and recurrent depression. But values overlap heavily between the ill and the well, and depend on the software and field strength. It does not diagnose an individual, and saying so is a mark.

WHAT EARNS THE MARKS

- **The formation listed as a set,** with entorhinal cortex included.
- **Three synapses named,** perforant path, mossy fibres, Schaffer collaterals, with long term potentiation attached.
- **Subfields tied to specific conditions,** with the limits of the measure stated. An answer that claims diagnostic use has overreached.

SEE ALSO Slot I - 21 basal ganglia and hippocampus Slot I - 13 synaptic plasticity **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

I - 24

Describe Caudate nucleus and its functions. [10]

10 FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Anatomy	Shape, parts, relations, and what it is made of
2 Connections	Where its input comes from and where its output goes
4 Functions by loop	Motor, executive, limbic and oculomotor, one line each
2 Clinical correlates	The four conditions that turn on this nucleus

LOOP	CORTEX IT SERVES	WHAT THE LOOP DOES	WHEN IT FAILS
Motor	Motor and premotor cortex	Selects and releases a wanted movement	Chorea, bradykinesia
Executive	Dorsolateral prefrontal cortex	Set shifting, habit learning, sequencing	Perseveration, poor planning
Limbic	Orbitofrontal and anterior cingulate	Weighs reward, stops an unwanted act	Obsessions, compulsions, apathy
Oculomotor	Frontal eye fields	Directs voluntary saccades	Impaired saccade control

One nucleus, four parallel loops. Naming the loop that failed is what turns a movement sign or a psychiatric sign into the same explanation.

- **Anatomy.** A C-shaped grey mass with head, body and tail, curving along the lateral ventricle. The head bulges into the frontal horn; the tail ends near the amygdala. It joins the putamen across the internal capsule through grey bridges, and together they are the striatum.
- **Cells and transmitters.** Mostly GABAergic medium spiny neurons, receiving glutamate from cortex and dopamine from the substantia nigra pars compacta, with local cholinergic interneurons. D1 cells begin the direct pathway, D2 cells the indirect.
- **The four clinical anchors.** Head atrophy with boxcar ventricles in Huntington disease, caudate and orbitofrontal overactivity in obsessive compulsive disorder, caudate involvement in Sydenham chorea, and reduced volume in Tourette syndrome.

PITFALL

Do not treat the caudate as a purely motor structure. Its largest loops serve prefrontal cortex, which is why a caudate answer in a psychiatry paper must reach obsessive compulsive disorder, apathy and habit learning, and not stop at chorea.

WHAT EARNS THE MARKS

- **The C shape with head, body and tail,** and its relation to the lateral ventricle.
- **Medium spiny neurons with their three inputs.** Cortical glutamate, nigral dopamine, local acetylcholine.
- **Four loops, each with its cortex,** which is the structure of the four-mark half.
- **Both a movement disorder and a psychiatric disorder** among the clinical correlates.

SEE ALSO Slot I - 21 basal ganglia Slot I - 16 prefrontal cortex SPRINT

Day 1, Functional Neuroanatomy and Neurophysiology

I - 25

Functions of the occipital cortex. [10]

10 FUNCTIONAL NEUROANATOMY AND NEUROIMAGING

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Anatomy**	Striate and extrastriate cortex, and the retinotopic map
3 **The two streams**	Dorsal where, ventral what, and what each computes
3 **Lesion syndromes**	Field defects, then the agnosias, sited
2 **Psychiatric relevance**	Visual hallucination, and what its shape tells you

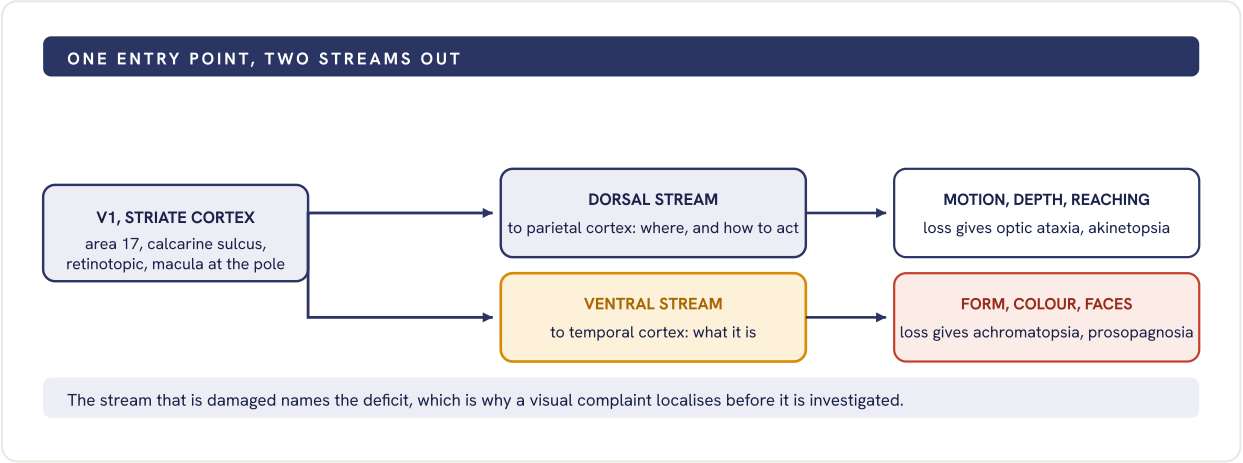

Fig 025.1 Visual cortex as one retinotopic entry point feeding two streams, each with the recognisable deficit that follows when it is cut.

- **Anatomy.** Striate cortex, area 17, lines the calcarine sulcus and is named for the stria of Gennari, an unusually thick layer four. Extrastriate areas 18 and 19 surround it and carry the higher processing.
- **Psychiatric relevance.** Occipital and temporal lesions give formed visual hallucinations with insight preserved. Charles Bonnet syndrome, formed hallucinations in an eye-diseased older person with intact cognition, is the presentation not to mistake for psychosis.

LESION	THE SIGN	WHAT IT TELLS YOU
Unilateral occipital	Homonymous hemianopia with macular sparing	Posterior cerebral territory, dual blood supply at the pole
Bilateral occipital	Cortical blindness, sometimes denied	Anton syndrome, blindness with anosognosia
Left occipital with splenium	Alexia without agraphia	A disconnection, not a language cortex lesion
Ventral occipitotemporal	Prosopagnosia, achromatopsia	The what stream, not the eye

WHAT EARNS THE MARKS

- **Retinotopy stated, with macular sparing explained.** It is the classic clinical consequence of the anatomy.
- **Two streams named and separated,** each with the deficit its loss makes.
- **Charles Bonnet syndrome named,** since this is a psychiatry paper and the differential is the point.

SEE ALSO Slot I - 14 corpus callosum Slot I - 22 parietal lobe signs **SPRINT**

Day 1, Functional Neuroanatomy and Neurophysiology

AREA 03 OF 12 · P1-A07

Personality theory and psychodynamics

10	10.5%	I-26	I-35
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 HIGH 1 SINGLE 8

REVISION ORDER

The two defence-mechanism slots together, they are one answer at two widths, and the Anna Freud slot sits directly on top of them. The three neo-Freudian slots next, where Jung is most of all three. The topographical model and the concept of self after that. Transactional analysis is short and independent. The family-theories slot is the one here that needs reading of its own: it is a critical evaluation, not a description.

WHAT IS IN THIS AREA

Defence mechanisms in two slots, one asking for them and one asking for their classification and their classical use in treatment. The neo-Freudians in three, twice with Carl Jung named inside the question and once with Jung alone. Anna Freud, the topographical model with primary and secondary process thinking, the concept of self, transactional analysis, and the slot that asks whether the family theories of schizophrenia are still relevant.

I - 26

Ego defence mechanisms. [10]

10 PERSONALITY THEORY AND PSYCHODYNAMICS

KUHS, NTRUHS, RGUHS - 6 APPEARANCES, 6 OF 78 SESSIONS

SKELETON

2	**Definition**	What the ego defends against, and with whose authority
2	**The organising frame**	Vaillant's four levels, named as levels
4	**The mechanisms**	Each at its own level, one line, one example
2	**The confusable pairs**	The four separations that mark a good answer

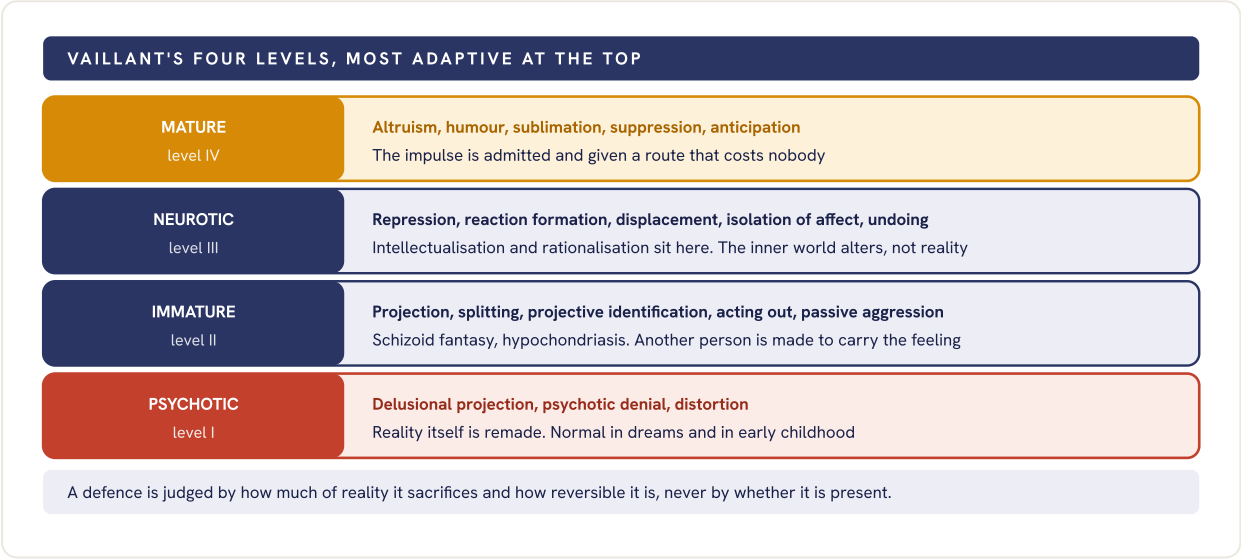

Fig 026.1 The four levels as rungs, the mature rung in amber because it is the one most often left out, the psychotic rung in red because only there does reality testing itself fail.

- **The definition, with its authority.** An unconscious operation of the ego that keeps an unacceptable impulse, affect or idea out of awareness and so holds anxiety down. Anna Freud gave the first systematic account in The Ego and the Mechanisms of Defence, 1936; her father had named repression in 1894 as the prototype from which the rest follow.
- **How they are recognised.** A defence is inferred from what is absent: the affect that fails to arrive with the content, or the theory offered in place of a feeling.
- **The four separations.** Projection puts one's own impulse into another; projective identification also makes the other feel and act it. Denial refuses a perception; repression removes an idea that was once conscious. Isolation of affect keeps the idea and strips the feeling; intellectualisation puts a theory where the feeling was. Suppression is conscious, repression is not.

WHAT EARNS THE MARKS

- **The definition attributed.** Anna Freud 1936, repression as Freud's prototype.
- **Four levels, named as levels,** with the mechanisms inside them rather than in one flat list. A list with no level is the standard mid-band answer.
- **The confusable pairs separated.** Projection against projective identification is the one examiners probe.

SEE ALSO Slot I - 29 classifying the defences and using them in treatment Slot I - 34 Anna Freud SPRINT Day 5, social, learning and personality psychology

I - 27

Relevance of family theories of Schizophrenia today. [10]

10 PERSONALITY THEORY AND PSYCHODYNAMICS

KNRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

1	The claim they made	The family as cause, in its own era
5	The theories	Each with author, year and verdict
2	What survived	Expressed emotion, and why it lived
2	Relevance today	From blame to burden

THEORY	WHAT IT CLAIMED	WHOSE, AND WHEN	WHERE IT STANDS TODAY
Schizophrenogenic mother	A cold, rejecting mother produces the illness	Fromm-Reichmann, 1948	Abandoned; it harmed a generation of families
Double bind	Contradictory injunctions, no escape, comment forbidden	Bateson, Jackson, Haley, Weakland, 1956	Not causal; survives as a description of confusing talk
Marital schism and skew	Open chronic conflict, or one partner's dominance accepted	Lidz, 1957	Not causal; discord is a stressor like any other
Pseudomutuality and rubber fence	A surface of harmony that forbids real difference	Wynne, Ryckoff, Day and Hirsch, 1958	Not causal; the boundary idea survives clinically
Communication deviance	Fragmented, unclear attention in family speech	Singer and Wynne, from 1963	Predicts thought disorder in high-risk samples
Expressed emotion	Criticism, hostility and over-involvement predict relapse	Brown, Birley and Wing, 1972; Vaughn and Leff, 1976	**Stands; replicated, and the basis of family intervention**

The fourth column answers the word today. Five rows are history; the amber row is the one still in the guidelines.

- **Why expressed emotion survived when the rest did not.** It was measured rather than asserted, on the Camberwell Family Interview, and it made a prospective claim about relapse rather than a retrospective claim about cause. It predicts relapse; it does not predict onset. Vaughn and Leff set high contact at more than thirty-five hours a week face to face.
- **The error the stem is testing.** Treating expressed emotion as a modern version of the schizophrenogenic mother. One is a blaming aetiological claim with no evidence; the other is a measured predictor of course that says nothing about who caused the illness.
- **The Indian evidence, and its limit.** Expressed emotion runs lower in Indian families than in the British samples it was derived from, and the World Health Organization comparative studies reported a better course in centres in developing countries. Both are contested on method, and neither licenses the claim that the joint family is protective in itself.

WHAT EARNS THE MARKS

- **Each theory with its author and decade.** The stem is historical, so unattributed theories earn little.
- **A verdict against every row,** not a description of every row.
- **Expressed emotion separated from blame.** Predicting relapse is not claiming cause, and the shift from blame to burden is what the answer ends on.

SEE ALSO Slot I - 30 the Neo-Freudians and their use in psychotherapy SPRINT

Day 5, social, learning and personality psychology

I - 28

What is the concept of self? Discuss its perspective in psychiatric disorders. [4+6]

4 + 6 | PERSONALITY THEORY AND PSYCHODYNAMICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What the self is	The knower and the known
2	Formal features	Jaspers' four, named as four
4	Failure by disorder	Each against the condition that breaks it
2	The psychodynamic self	What Kohut and Winnicott add

THE DEFINITION

The self is the person as known to himself: at once the subject that experiences and the object experienced. William James, 1890, separated the I, the knower, from the Me, the known, and divided the Me into material, social and spiritual selves.

ASPECT OF THE SELF	HOW IT FAILS	WHERE THE FAILURE IS SEEN	WHOSE ACCOUNT
Awareness of activity	Thought and act are experienced as authored from outside	**Thought insertion and passivity in schizophrenia**	Jaspers
Awareness of unity	More than one self is experienced at one time	Dissociative identity presentations	Jaspers
Awareness of continuity	The thread back to the earlier self is cut	Dissociative amnesia and fugue	Jaspers
Awareness of boundaries	Self and world stop being demarcated	Ego boundary loss in schizophrenia; drug and ecstatic states	Jaspers
The bodily and narrative self	The self feels unreal, or will not hold one shape across roles	Depersonalisation; borderline identity diffusion	Schilder, Winnicott

- **The four accounts worth naming.** James, 1890, the I and the Me. Cooley, 1902, the looking-glass self, assembled from what we believe others see. Mead, 1934, the self arising in social interaction through the generalised other. Jaspers, 1913, four formal characteristics of self-awareness.
- **The psychodynamic self.** Kohut made the self, not the drive, the unit: built by selfobject responses of mirroring, idealisation and twinship, and fragmenting when they fail. Winnicott separated a true self, rooted in the body, from a false self built to comply.
- **Three words that are not synonyms.** Self-esteem is the value placed on the self, self-concept is its content, self-awareness is the formal fact of having one. Only the third breaks in schizophrenia.

WHAT EARNS THE MARKS

- **The I and the Me, attributed to James.** Two of the four concept marks live in that one sentence.
- **Jaspers' four characteristics named as four,** because the disorder half is hung on them.
- **A named disorder against each characteristic,** not a general claim that the self is disturbed in mental illness.

SEE ALSO Slot I - 31 Carl Jung and the Self as archetype Slot I - 33 the topographical theory of mind **SPRINT** Day 5, social, learning and personality psychology

I - 29

How do you classify the defense mechanisms? Describe their classical use in treatment of psychiatric disorders. [3+7]

3 + 7 | PERSONALITY THEORY AND PSYCHODYNAMICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	The classifications	Three schemes, and what each one is for
4	The treatment decision	Support, interpret or convert, decided by level
2	The sequence	Resistance before content, defence before impulse
1	The limit	Where interpreting a defence does harm

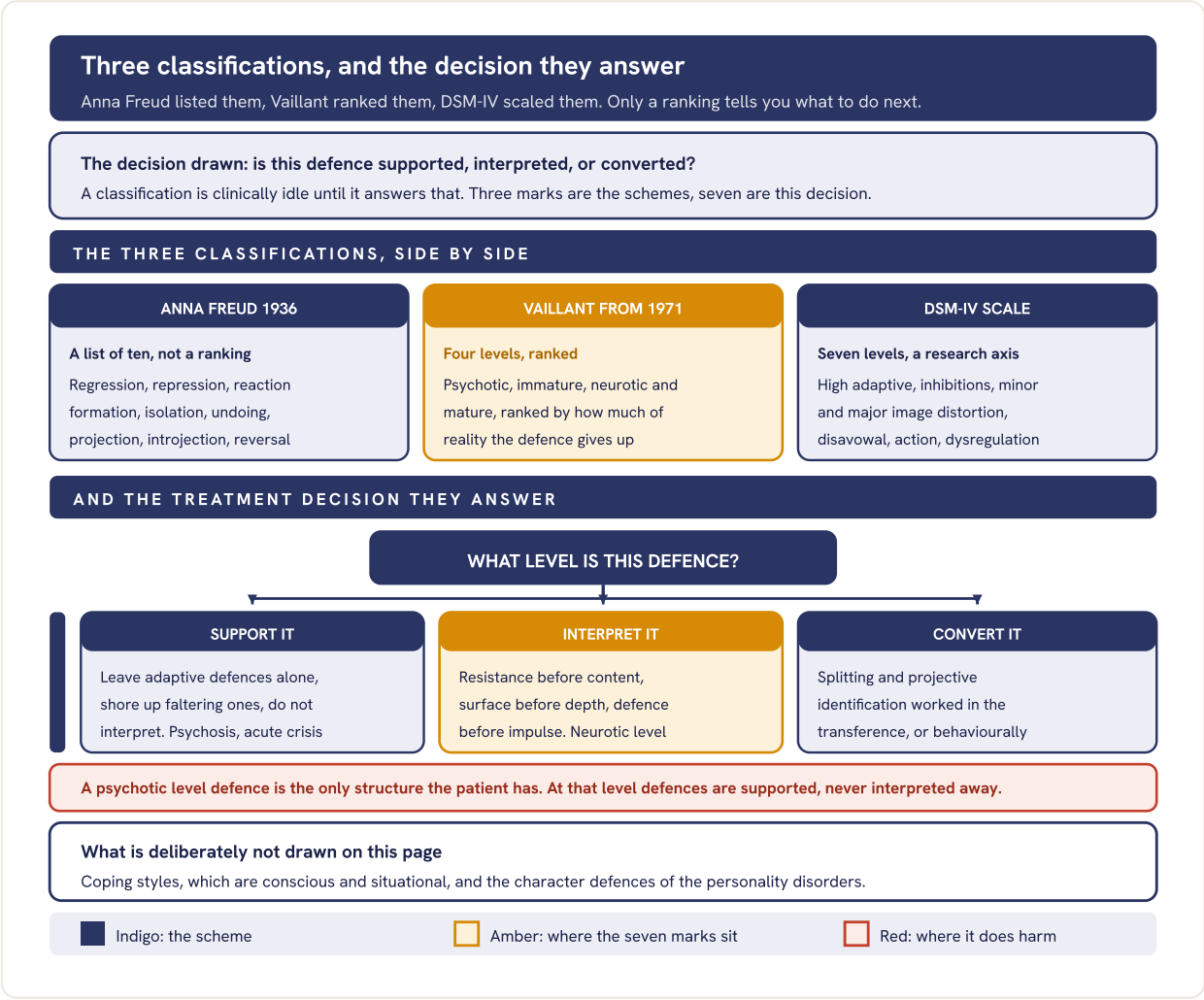

Fig 029.1 The three schemes across the top, the level question as the single node, and the three treatment lanes below it, with the interpretive lane in amber.

- **The sequence, which is the classical use.** Interpret resistance before content, surface before depth, and the defence before the impulse it hides. The aim is never to remove a defence but to replace it with a more adaptive one doing the same job.
- **What each scheme is for.** Anna Freud's list is what a clinical description draws on. Vaillant's levels are what a formulation and a prognosis draw on, because only a ranking supports a decision.

WHAT EARNS THE MARKS

- **Three schemes with their authors,** not one undifferentiated list of mechanisms.
- **The level used to make a decision.** The stem says classical use, so stopping at the classification throws away seven marks.
- **The interpretive sequence, in order.** Resistance, then content; defence, then impulse.
- **The red strip.** Supporting rather than interpreting a psychotic level defence is what shows clinical use.

SEE ALSO Slot I - 26 the ego defence mechanisms described Slot I - 34 Anna Freud **SPRINT**

Day 5, social, learning and personality psychology

I - 30

Describe various theories of Neo-Freudians. Discuss their use in psychotherapy. [5+5]

5 + 5 | PERSONALITY THEORY AND PSYCHODYNAMICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

1	The axis	What makes a theorist Neo-Freudian rather than Freudian
4	The theories	Each with its break from Freud and its signature concept
4	Use in psychotherapy	The mechanism each one gave the consulting room
1	The honest limit	Where the label itself is loose

THEORIST	THE BREAK WITH FREUD	SIGNATURE CONCEPTS	WHAT IT GAVE PSYCHOTHERAPY
Alfred Adler	Social striving replaces the sexual drive	Inferiority and compensation, style of life, social interest	**Encouragement and goal-focused brief work; an ancestor of cognitive therapy**
Carl Jung	Libido is general psychic energy	Collective unconscious, archetypes, individuation, introversion and extraversion	Dream and symbol work; the tasks of the second half of life
Karen Horney	Basic anxiety, not libido, is the root of neurosis	Moving toward, against and away from people; the tyranny of the should	Work on the idealised self; the founding critique of Freud on women
Harry Stack Sullivan	Personality exists only between people	Self-system; good me, bad me, not me; parataxic distortion	**The therapist as participant observer; the parent of interpersonal psychotherapy**
Erich Fromm	Society and the burden of freedom form character	Escape from freedom, productive orientation	Existential and humanistic therapies
Erik Erikson	Development is psychosocial and runs the whole life span	Eight stages, epigenetic principle, identity, moratorium	Identity work in adolescence; life review in later life

The fourth column is the second half of the stem. Amber marks the two whose method survives under its own name in current practice.

- **The axis, stated before the list.** Every one of them kept the unconscious and the developmental frame and gave up the primacy of the sexual drive, putting culture, relationship or social striving in its place. Otto Rank and Wilhelm Reich are sometimes counted with them.
- **The five psychotherapy marks, which most answers lose.** Name a mechanism per theorist rather than a school: encouragement and goal setting from Adler, symbol work from Jung, the idealised self from Horney, the participant observer from Sullivan, identity and moratorium from Erikson.

WHAT EARNS THE MARKS

- **The axis in one line before any name.** A list of theorists with no stated criterion reads as recall.
- **Each theorist tied to what he or she abandoned,** not merely to a concept.
- **A therapeutic mechanism per row.** The stem splits five and five, so the last column carries half the paper.

SEE ALSO Slot I - 32 Neo-Freudians against Post-Freudians Slot I - 31 Carl Jung in full **SPRINT**

Day 5, social, learning and personality psychology

I - 31

Carl Jung. [10]

10 PERSONALITY THEORY AND PSYCHODYNAMICS

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The man and the break	Dates, and the one revision that made him Jungian
4	Structure of the psyche	Collective unconscious, archetypes, complexes
2	Typology and method	Attitudes, functions, word association, amplification
2	Individuation	The goal, and why it belongs to the second half of life

THE MAN, AND THE ONE REVISION

Carl Gustav Jung, 1875 to 1961, Swiss psychiatrist, Freud's designated successor until the break of 1913. He founded analytical psychology on a single revision: libido is general psychic energy rather than sexual energy, and the unconscious is collective as well as personal.

DOMAIN	THE CONCEPT	WHAT JUNG MEANT BY IT	WHERE IT SHOWS UP
Structure	Collective unconscious	An inherited layer common to all people, beneath the personal unconscious	The recurrence of the same myths and symbols
Structure	Archetypes	Inherited forms that organise experience: persona, shadow, anima and animus, the Self	**Persona and shadow are the two most often asked for**
Structure	Complexes	Feeling-toned clusters of ideas in the personal unconscious	Shown on his own Word Association Test
Typology	Attitudes and functions	Introversion and extraversion, crossed with thinking, feeling, sensation, intuition	The ancestor of later personality typologies
Development	Individuation	Lifelong integration of the unconscious into a whole centred on the Self	The task of the second half of life
Method	Amplification	A symbol elaborated through myth rather than reduced to biography	Dream work, and active imagination

- **The two confusions that cost marks.** The Self is not the ego: the ego is the centre of consciousness, the Self is the centre and the totality of the whole psyche and is the goal of individuation. The shadow is not the evil in a person but everything unlived and unacknowledged, undeveloped good included.
- **Where he still touches practice.** Symbol and dream work in analytical psychotherapy, midlife presentations read as a developmental task rather than a decline, and the word association method, which was an early objective demonstration that unconscious material can be measured.

WHAT EARNS THE MARKS

- **The revision named, not just the split.** Libido as general psychic energy is what makes him Jungian.
- **Four archetypes named,** and the Self distinguished from the ego.
- **Typology given as two attitudes crossed with four functions,** which is the form Jung used.
- **Individuation defined as a goal,** not as a synonym for growing up.

SEE ALSO Slot I - 32 Neo-Freudians against Post-Freudians Slot I - 28 the concept of self SPRINT

Day 5, social, learning and personality psychology

I - 32

Who are Neo-Freudians? How they are different from Post-Freudians? Write about contributions of Carl Jung. [2+3+5]

2 + 3 + 5 | PERSONALITY THEORY AND PSYCHODYNAMICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Who they are**	The names, and the criterion that groups them
3 **The difference**	Broke away against built upon, domain by domain
4 **Jung's contributions**	Five, each named in his own terms
1 **The caveat**	Where the two labels overlap

DOMAIN	NEO-FREUDIANS	POST-FREUDIANS	WHAT SETTLES IT
Who	Adler, Jung, Horney, Sullivan, Fromm	Anna Freud, Hartmann, Klein, Winnicott, Fairbairn, Bion, Kohut	**Whether drive theory was abandoned or extended**
Relation to Freud	Broke away, usually in person as well as in theory	Stayed inside psychoanalysis and built on it	**Broke away against built upon**
What drives behaviour	Social striving, culture, inter-personal security	The drive kept, with the ego or the internal object added	The place still given to libido
Central unit	The person in a social field	The ego and its functions, or the internal object	Field against internal structure
Period	Roughly 1910 to 1940	Roughly 1930 onward	Suggestive, not decisive
Technique	Shorter, more active, more directive	The analytic frame kept, inter-pretation deepened	How far the method moved

The amber cells are the discriminators. The rest of the grid supports them, and saying which is which is itself a mark.

- **Jung's contributions, which carry five of the ten marks.** The collective unconscious and its archetypes, persona, shadow, anima and animus, and the Self. Complexes, demonstrated on the Word Association Test. A typology of two attitudes crossed with four functions. Individuation as the task of the second half of life. And amplification: the symbol elaborated rather than reduced.
- **The caveat that earns the last mark.** The label is loose. Jung and Adler are counted as Neo-Freudian because they broke away; Erikson is often counted with them although he never left. State the criterion you are sorting on before you sort the names.

WHAT EARNS THE MARKS

- **Both lists of names given,** because the stem asks who before it asks how.
- **The discriminator identified as the discriminator.** Abandoned against extended, not six differences of equal weight.
- **Five Jung contributions, separately named.** The last third of the stem is half the paper.

SEE ALSO Slot I - 31 Carl Jung in full Slot I - 30 the Neo-Freudian theories in psychotherapy SPRINT

Day 5, social, learning and personality psychology

I - 33

What is primary and secondary process of thinking as proposed by Freud? Write a brief note on topographical theory of mind. [4+6]

4 + 6 | PERSONALITY THEORY AND PSYCHODYNAMICS

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Primary process**	The mode of the unconscious
2	**Secondary process**	The mode of the ego
4	**The topography**	Three systems, and what bars each door
2	**Its successor**	What the structural model added in 1923

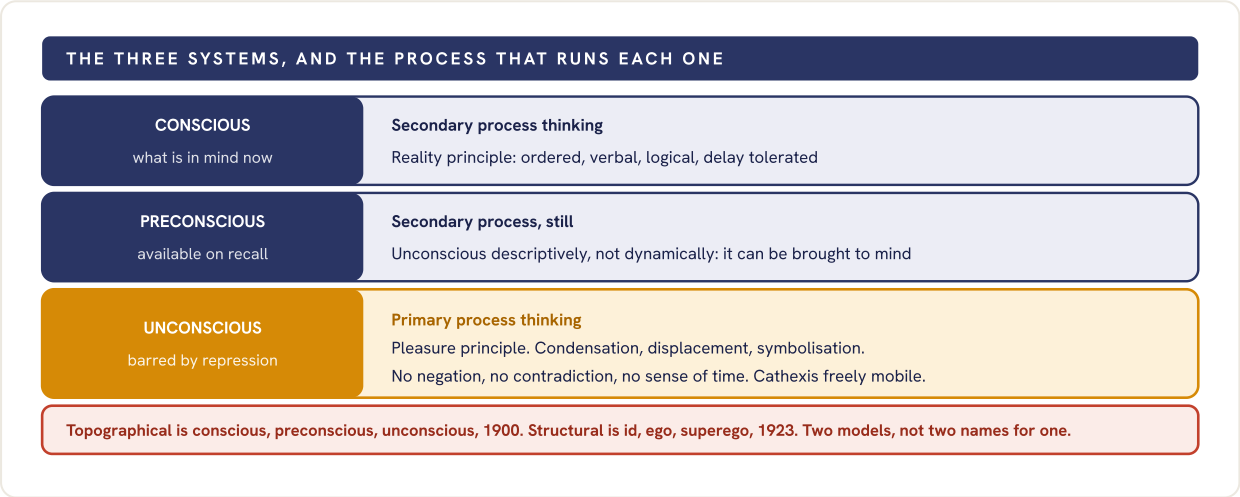

Fig 033.1 The three systems as strata with the correct mode of thinking hung on each. Amber marks the unconscious stratum, where the primary process marks are won; red marks the model most often confused with this one.

- **The two processes, as Freud defined them.** Primary process is the mode of the unconscious and of the infant: immediate discharge under the pleasure principle, cathexis freely mobile, working by condensation, displacement and symbolisation. Secondary process is the mode of the ego: cathexis bound, the reality principle, logic, language, and delay tolerated.
- **Where each is seen.** Primary process shows in dreams, in free association, in slips and jokes, and in the formal thought disorder of psychosis. Secondary process is ordinary waking thought, and its loss is what makes psychotic speech read as dreamlike.
- **Why it needed a successor.** The topographical model was supplemented in 1923, not abandoned. Freud found that much of the ego and all of the superego are themselves unconscious, which a model built on the single axis of awareness cannot hold.

WHAT EARNS THE MARKS

- **Both processes defined by their principle,** pleasure against reality, not by their qualities alone.
- **Condensation, displacement and symbolisation named.** They are what make primary process specific.
- **Preconscious separated from unconscious properly.** Descriptive against dynamic is the distinction examiners check.

SEE ALSO Slot I - 29 classifying the defences and using them in treatment Slot I - 28 the concept of self **SPRINT** Day 5, social, learning and personality psychology

I - 34

Anna Freud. [10]

10 PERSONALITY THEORY AND PSYCHODYNAMICS

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The person	Dates, position, and the book
4	The contributions	Each stated in its own terms
2	The dispute with Klein	What each held, and what it split
2	What survives	Defence analysis, developmental profile

THE PERSON, AND THE BOOK

Anna Freud, 1895 to 1982, youngest child of Sigmund Freud, a lay analyst who founded child psychoanalysis in the British tradition and, with Hartmann, ego psychology. The book every answer must name is The Ego and the Mechanisms of Defence, 1936.

CONTRIBUTION	WHAT SHE ACTUALLY DID	THE CONCEPT TO NAME	WHERE IT STILL SHOWS
Defence, made systematic	Gathered scattered ideas into one account of how the ego wards off anxiety	**Ten mechanisms, and defence analysis as a technique**	Every later classification descends from it
Ego psychology	Moved analytic attention from the id to the ego and its adaptation	The ego as an agent with its own development	Formulation in terms of ego strength
Child analysis	Analysed children through play, education and a real working alliance	The child's ego needs support before it can bear interpretation	Child psychotherapy technique
Developmental lines	Traced development as continuous strands rather than stages alone	From dependency to emotional self-reliance, and others	Developmental profile in child assessment
Direct observation	Ran wartime residential nurseries for separated children in London	Observation admitted as psychoanalytic evidence	The empirical strand Bowlby then took up

- **Her definition of defence, which is the quotable line.** An unconscious operation by which the ego wards off an unacceptable instinctual demand, affect or idea, holding anxiety within bounds. She listed ten: regression, repression, reaction formation, isolation, undoing, projection, introjection, turning against the self, reversal and sublimation.
- **Anna Freud against Melanie Klein, one line each.** Anna Freud held the child's ego too immature for classical interpretation, so the work opens with an educational and supportive alliance. Klein held play to be the child's free association, and interpreted deeply and early. The disagreement split British psychoanalysis into three groups.

WHAT EARNS THE MARKS

- **The 1936 book named with its year.** On a named-person stem the title is a mark on its own.
- **Contributions kept separate,** not merged into one paragraph about child analysis, and ego psychology credited to her and Hartmann together.
- **The Klein dispute given as two positions,** with what it split, rather than as a personal quarrel.

SEE ALSO Slot I - 26 the ego defence mechanisms described

Slot I - 29 classifying the defences and using them in treatment **SPRINT** Day 5, social, learning and personality psychology

I - 35

Transactional analysis. [10]

10 PERSONALITY THEORY AND PSYCHODYNAMICS

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The frame	Berne, and the four levels of analysis
3	Ego states	Three, with divisions and voice
3	Transactions	Complementary, crossed, ulterior
2	Positions and games	Life positions, strokes, the script

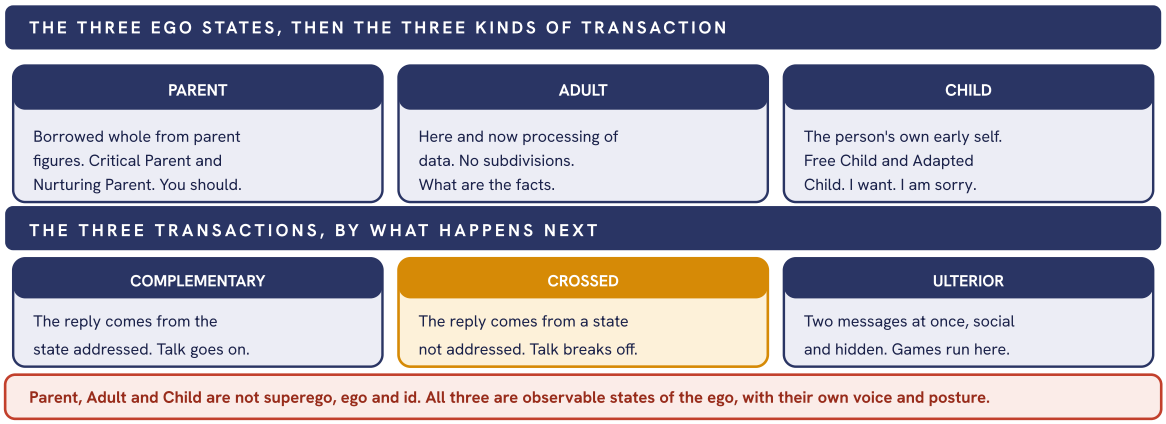

Fig 035.1 Ego states above, transactions below, sorted by what happens to the conversation. Amber marks the crossed transaction, the one this question asks to be illustrated.

LIFE POSITION	STANCE	WHERE IT IS SEEN
I am OK, you are OK	Get on with	Healthy and mutual; the therapy goal
I am OK, you are not OK	Get rid of	Blaming; paranoid and antisocial
I am not OK, you are OK	Get away from	Commonest; depressive, dependent
I am not OK, you are not OK	Get nowhere	Futility; chronic hopelessness

- **The frame, and its four levels.** Eric Berne, from 1958, built a contractual therapy in plain language. Structural analysis names the ego states, transactional analysis reads the exchanges, game analysis names repeated ulterior sequences with a predictable payoff, set out in Games People Play, 1964, and script analysis reads the life plan laid down in childhood.
- **Strokes and time.** A stroke is a unit of recognition, and people take negative strokes over none, which is what keeps a game running. Berne ranked six ways of structuring time: withdrawal, ritual, pastime, activity, game, intimacy.

WHAT EARNS THE MARKS

- **The four levels of analysis named as four.** Structural, transactional, game, script.
- **The three transactions with an example each,** crossed illustrated rather than merely defined.
- **Ego states kept apart from the structural model.** It is the discrimination this question is built on.

SEE ALSO Slot I - 30 the Neo-Freudian theories in psychotherapy Slot I - 33 the topographical theory of mind SPRINT Day 5, social, learning and personality psychology

AREA 04 OF 12 · P1-A05

Learning, memory and general psychology

9	9.9%	I-36	I-44
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 4 · HIGH 2 · RECURRENT 1 · SINGLE 2

REVISION ORDER

The learning block first: four slots and largely one answer, with the two conditioning slots almost interchangeable. The two emotion slots next, likewise, and the James-Lange slot carries the evaluation the other does not ask for. Intelligence after that, theories first and then the Indian test. Emotional intelligence and social learning theory are short and stand on their own.

WHAT IS IN THIS AREA

Learning in four slots: the definition with the models and their clinical importance, classical against operant conditioning with an example of each, Pavlovian conditioning inside behaviour therapy, and social learning theory. Emotion in two, the basic emotions with the theories, and the theories again with James-Lange critically evaluated. Intelligence in three, the theories with their application, emotional intelligence against IQ, and the Indian standardised test. This is the anchor-heaviest area in the book: four of its nine slots have come back across boards and years.

I - 36

Theories of intelligence and their application in Psychiatry. [10]

10 LEARNING, MEMORY AND GENERAL PSYCHOLOGY

KNRUHS, RGUHS, TN-MGRMU - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	Definition	What the construct is, and the general factor debate
4	The theories	Each theorist, his claim, the structure proposed
2	How it is measured	Ratio and deviation quotient, and what a test yields
2	Use in psychiatry	Where the figure changes a clinical decision

THE DEFINITION

Wechsler defined intelligence as the global capacity to act purposefully, to think rationally and to deal effectively with the environment. Binet called it judgement applied to circumstances. Both are functional definitions: intelligence is inferred from performance, never observed directly.

THEORIST	THE CLAIM	STRUCTURE PROPOSED	WHAT IT IS USED FOR
Spearman, 1904	One general ability runs through every task	Two factors, general and specific	The rationale for a single quotient
Thurstone, 1938	There is no single ability, only several	Seven primary mental abilities	Profile testing, subtest scatter
Cattell and Horn	Ability splits by how it ages	Fluid and crystallised intelligence	Separating decline from a low baseline
Guilford, 1967	Ability is a three way classification	Operations by contents by products	Divergent thinking and creativity
Gardner, 1983	Intelligences are plural and independent	Seven, later eight, intelligences	Strengths based educational planning
Sternberg, 1985	Intelligence is what succeeds in context	Analytic, creative and practical	Vocational and rehabilitation planning

- **Measurement.** Stern's ratio quotient, mental age over chronological age times a hundred, fails in adults because mental age plateaus. Wechsler replaced it with the deviation quotient, mean a hundred, standard deviation fifteen. Vernon's hierarchical model is the shape modern batteries take.
- **Application, and its limit.** Intellectual disability needs a low quotient **and** impaired adaptive functioning; the older bands read mild fifty to sixty nine, moderate thirty five to forty nine, severe twenty to thirty four, profound below twenty. Fluid ability falls in dementia while crystallised ability holds, which is what makes that pair useful.

WHAT EARNS THE MARKS

- **A definition with a name attached,** written out before the theories start.
- **Every row of the grid carrying a theorist and a date.** Theories listed without authors read as recall.
- **The amber row, fluid against crystallised,** because it is the one that does clinical work at the bedside.
- **Adaptive functioning named alongside the quotient.** A number alone does not make the diagnosis.

SEE ALSO Slot I - 43 the Indian standardised intelligence test Slot I - 38 emotional intelligence and its relation to quotient

SPRINT Day 4, general and developmental psychology

I - 37

Social learning theory. [10]

10 LEARNING, MEMORY AND GENERAL PSYCHOLOGY

KUHS, RGUHS - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2 The claim	Learning by watching, without doing and without reward
3 The four processes	Drawn in order, with the point where learning is complete
3 The constructs	Reciprocal determinism, self efficacy, vicarious reinforcement
2 Clinical use	The treatments it produced, and the harms it explains

THE CLAIM

Behaviour is acquired by observing a model, held in symbolic form, and performed later when the incentive is right. Direct reinforcement of the learner is not required, and that is what separates this from operant theory.

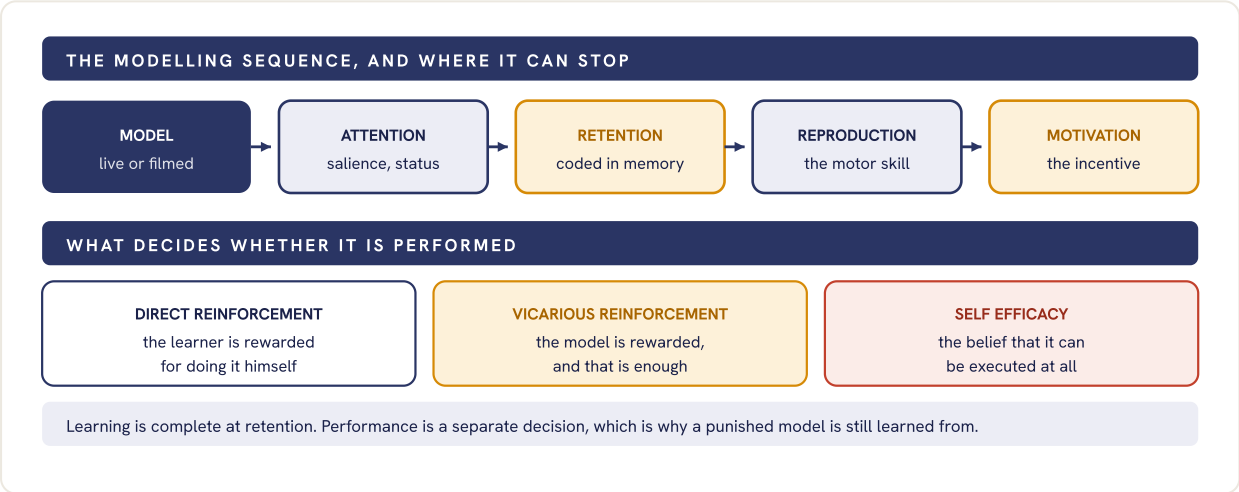

Fig 037.1 The four mediational processes in order, with the two amber stages that carry the theory's whole argument, and the three influences that decide whether what was learned is ever shown.

- **The evidence and the constructs.** Children who watched an adult attack an inflatable doll reproduced the aggression; those who saw the model punished performed less, yet showed on incentive that they had learned as much. Reciprocal determinism holds that person, behaviour and environment each shape the other two.
- **Clinical use, both directions.** Participant modelling in phobia work, behavioural rehearsal in social skills training, video modelling with children. The same mechanism explains learned illness behaviour in a family and imitative self harm after media coverage.

WHAT EARNS THE MARKS

- **Learning separated from performance,** which is the sentence the whole theory turns on.
- **All four mediational processes named in order,** exactly as the figure draws them.
- **Self efficacy named and defined,** built from mastery, vicarious experience, persuasion and bodily state.

SEE ALSO Slot I - 40 the models of learning classified Slot I - 41 classical and operant conditioning SPRINT Day 5, social, learning and personality psychology

I - 38

What is emotional intelligence? Describe components of emotional intelligence. Mention the relationship between emotional intelligence and I.Q. [2+4+4]

2 + 4 + 4 | LEARNING, MEMORY AND GENERAL PSYCHOLOGY

DNB - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 Definition	What it is, and who named it in which year
4 The components	Three rival models, each with its own component list
3 Against the quotient	What each predicts, and what the overlap really is
1 Clinical bearing	The disorders that sit at the low end

THE DEFINITION

Salovey and Mayer, who named the construct in 1990, defined it as the ability to monitor one's own and other people's feelings, to discriminate among them, and to use that information to guide thinking and action. Goleman's 1995 book widened it well beyond that.

MODEL	COMPONENTS	HOW IT IS MEASURED	STANDING
Ability, Salovey and Mayer	Perceiving emotion, using emotion to aid thought, understanding emotion, managing emotion	Performance test with scored right answers	**The only model that behaves like an intelligence**
Mixed, Goleman	Self awareness, self regulation, motivation, empathy, social skill	Self report and rating by others	Popular, evidence weaker than the claims
Social and emotional, Bar-On	Intrapersonal, interpersonal, adaptability, stress management, general mood	Self report inventory	Broad, close to wellbeing
Trait, Petrides	Emotional self perceptions sited within personality	Self report questionnaire	Overlaps heavily with personality

- **The relationship with the quotient.** Positively but only modestly related, and not substitutes. The quotient remains much the stronger predictor of academic attainment and of cognitively demanding work. Ability emotional intelligence adds a small increment for interpersonal outcomes once quotient and personality are accounted for; trait and mixed measures add least, being largely personality under another name.
- **Clinical bearing, and the trap.** The low end is recognisable in clinic: alexithymia, the empathy profile of autism, the intact cognitive with impaired affective empathy of antisocial personality, the emotion dysregulation of borderline personality. The trap is not naming which model you mean, because a performance test and a self report questionnaire do not measure the same thing.

WHAT EARNS THE MARKS

- **The 1990 definition with both authors,** kept separate from Goleman's later version.
- **Components given model by model,** because the models disagree about what they are.
- **The amber row.** Only the ability model is measured the way an intelligence is measured.

SEE ALSO Slot I - 36 theories of intelligence Slot I - 39 basic emotions and theories of emotion SPRINT

Day 4, general and developmental psychology

I - 39

What are the basic emotions? Discuss briefly the theories of emotion. [2+8]

2 + 8 | LEARNING, MEMORY AND GENERAL PSYCHOLOGY

DNB, TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	Basic emotions	The criteria first, then the named sets
5	The theories	Each one as a sequence, with its support and its flaw
2	The appraisal turn	What cognition added, and what it displaced
1	Why it matters	One line linking the theory to a treatment

THEORY	THE SEQUENCE PROPOSED	ITS STRONGEST SUPPORT	WHERE IT FAILS
James and Lange, 1884	Bodily change first, emotion is its reading	Facial feedback; blunting after cord injury	Visceral change too slow, too alike
Cannon and Bard, 1927	Thalamus fires to cortex and body at once	Emotion survives loss of feedback	The thalamus is too simple a relay
Schachter and Singer, 1962	Arousal, then a label taken from context	Adrenaline felt as anger or elation by context	Replication has been uneven
Lazarus, 1966	Appraisal first, and it can be automatic	One event, different appraisals, different emotions	Appraisal is hard to measure apart
Papez and MacLean	A closed circuit carries the experience	The connections named are real	Superseded as an account of emotion

- **What makes an emotion basic.** Universal recognition across cultures, a distinct facial expression, quick onset and brief course, a distinct physiological signature, automatic appraisal, a counterpart in other primates.
- **The three sets worth naming.** Ekman gives six, from universally recognised facial expressions: happiness, sadness, fear, anger, disgust, surprise. Plutchik gives eight as four opposed pairs on a wheel, joy against sadness, trust against disgust, fear against anger, surprise against anticipation. Panksepp names seven systems from the circuits themselves: seeking, rage, fear, lust, care, panic and grief, play.
- **The clinical pay-off.** Schachter and Singer is the panic model in embryo. Benign arousal receives a catastrophic label, and it is the label, not the arousal, that interoceptive exposure changes.

PITFALL

These theories are not a league table with a winner. Each was framed against the one before it, and the position now held is that bodily change, appraisal and context all contribute. Writing one as simply correct throws away the discussion marks.

WHAT EARNS THE MARKS

- **The criteria for basic, before any list.** A list with no criteria is recall.
- **Each theory written as a sequence,** exactly as the second column sets it out.
- **Named sets with their authors,** six, eight and seven, not a single unattributed list.

SEE ALSO Slot I - 42 the theories of emotion evaluated at depth Slot I - 38 emotional intelligence SPRINT Day 4, general and developmental psychology

I - 40

Define learning. Write in brief about the basic tenets of different models of learning and their clinical importance in psychiatry. [2+8]

2 + 8 | LEARNING, MEMORY AND GENERAL PSYCHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Definition**	What counts as learning, and what is excluded
3	**The models drawn**	One axis, three branches, the theorists attached
3	**The basic tenets**	The mechanism of each model, one line each
2	**Clinical importance**	A named therapy standing under every branch

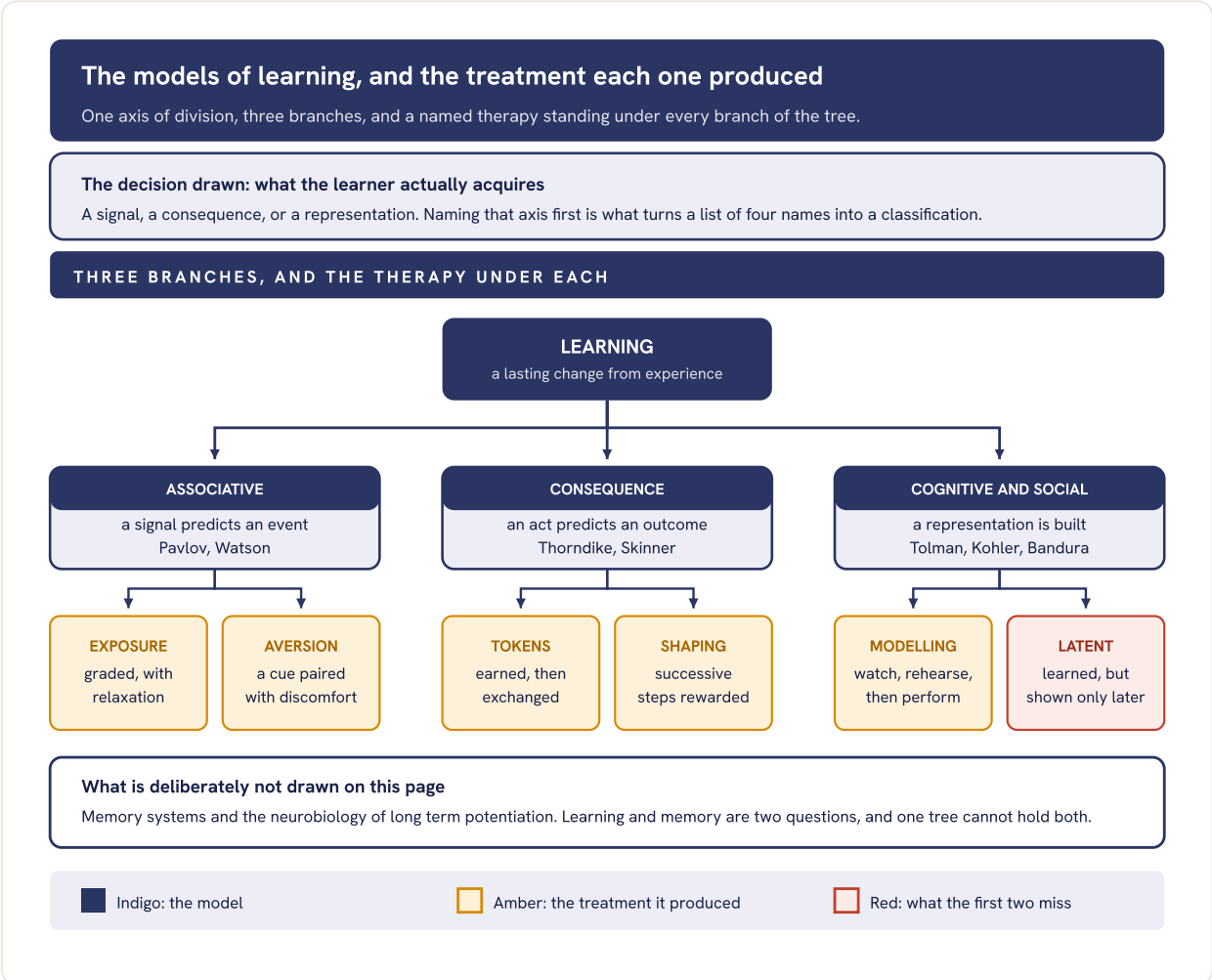

Fig 040.1 The models of learning divided on what the learner acquires, with the therapy each branch produced set in amber beneath it, and latent learning marked in red because it is the finding the associative and consequence models cannot account for.

- **The tenets, one line each.** Classical conditioning pairs a neutral stimulus with one that already elicits a reflex until it elicits that reflex alone. Operant conditioning makes a behaviour more or less likely by what follows it. Observational learning is acquired by watching a model and stored before it is performed. Cognitive learning builds an internal representation, shown by latent learning and by insight.

- **The definition, and what it excludes.** Learning is a relatively permanent change in behaviour, or in the capacity for it, following experience. Change from fatigue, maturation, injury or a drug is not learning.

PITFALL

Naming the models without naming the treatment each produced answers half the question. Clinical importance carries the second eight marks, and every branch has a named therapy under it.

WHAT EARNS THE MARKS

- **The definition with its exclusions,** before the classification begins.
- **The axis stated in one line.** A tree that does not name its axis is a list.
- **Every branch with its theorists,** Pavlov, Skinner, Tolman, Bandura.
- **A named therapy under each branch.** The amber leaves are the clinical marks.

SEE ALSO Slot I - 41 classical and operant conditioning compared Slot I - 44 Pavlovian conditioning in behaviour therapy

SPRINT Day 5, social, learning and personality psychology

I - 41

Classical conditioning and Operant conditioning. Discuss their application in clinical setting with one example for each. [10]

10 | ASKED AT 15 MARKS | LEARNING, MEMORY AND GENERAL PSYCHOLOGY

KUHS, TN-MGRMU - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2 Classical	The paradigm, and the four terms in their order
2 Operant	The four consequences, named without error
4 Applications	A named treatment and one example for each
2 Shared phenomena	Extinction, generalisation, discrimination, schedules

FEATURE	CLASSICAL	OPERANT	WHAT SETTLES IT
What is learned	A signal predicts an event	An act produces an outcome	**Is the behaviour elicited or emitted**
The response	Reflexive, autonomic, involuntary	Skeletal, voluntary, acting on the world	Could the person withhold it
Where the event sits	The signal comes before the response	The consequence comes after it	**Which side of the behaviour the event falls**
The law invoked	Contiguity of two stimuli, Pavlov and Watson	Effect of the consequence, Thorndike and Skinner	Pairing against payoff
Clinical example	Graded desensitisation for a phobia of heights	A token economy on a rehabilitation ward	Is the cue changed, or the consequence

- **The operant terms, used correctly.** Reinforcement raises the rate of a behaviour and punishment lowers it; positive means a stimulus is added and negative means one is removed. That gives four cells, not two. Negative reinforcement is therefore not a punishment, and it is precisely what maintains avoidance in every anxiety disorder.
- **What both paradigms share.** Acquisition, extinction, spontaneous recovery, stimulus generalisation and discrimination all appear on either side. Among the operant schedules a variable ratio produces the behaviour hardest to extinguish, which is why the machine rather than the salary is the model for gambling.

PITFALL

The commonest error is calling negative reinforcement a punishment. The second is offering exposure as the operant example: graded exposure works by extinguishing a conditioned response and belongs on the classical side of this table. Aversion therapy sits there too.

WHAT EARNS THE MARKS

- **Both paradigms defined before the comparison begins,** each with its theorists named.
- **The two amber cells,** elicited against emitted, and which side of the behaviour the event falls.
- **One named treatment and one concrete example for each,** which is what the stem asks for twice.

SEE ALSO Slot I - 40 the models of learning classified Slot I - 44 Pavlovian conditioning in behaviour therapy **SPRINT** Day 5, social, learning and personality psychology

I - 42

Discuss various theories of emotions and critically evaluate James-Lange theory and its implications. [10]

10 LEARNING, MEMORY AND GENERAL PSYCHOLOGY

KUHS, RGUHS, TN-MGRMU - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	The field in brief	Four positions, one line each, in date order
2	The claim itself	James and Lange stated so it can be tested
4	The evaluation	Test by test, with a verdict on each
2	Implications	What survives, and what it is used for now

THE CLAIM, STATED SO IT CAN BE TESTED

James held that bodily change is not the expression of emotion but its cause: we are sad because we weep. Lange said the same of vasomotor change. Stated strongly, peripheral feedback is necessary and sufficient for feeling; stated weakly, it only modulates intensity.

THE TEST APPLIED	WHAT WAS FOUND	HOW THE THEORY STANDS	VERDICT
Cutting visceral feedback	Emotional behaviour persists in animals	Feedback is not necessary for it	**Strong form refuted**
Comparing autonomic patterns	Fear and anger look largely alike	Some patterning since demonstrated	Partly answered
Timing the visceral response	Viscera are slow, sparsely innervated	Facial and muscular feedback is faster	Partly answered
Injecting adrenaline	Arousal without any felt emotion	A cognitive label must be added	**Reframed by Schachter**
Studying spinal cord injury	Higher lesions, less intense feeling	Feedback modulates intensity	Weak form supported

- **The field, in one line each.** Cannon and Bard put the thalamus at the centre, sending to cortex and body at once. Schachter and Singer made emotion arousal plus a label. Lazarus put appraisal first. Papez and MacLean supplied the circuit, not the psychology. LeDoux separated a fast subcortical route from a slower cortical one.
- **Implications, the second half of the stem.** Facial feedback and its therapeutic offshoots; relaxation working on the body to change the feeling; beta blockade for performance anxiety, where removing the peripheral signal lowers the fear; interoceptive exposure in panic; and Damasio's somatic marker account of how bodily states bias decision.

WHAT EARNS THE MARKS

- **The strong and weak forms separated in the definition box.** Critical evaluation is impossible until the claim is stated precisely.
- **A verdict in every row,** not a list of criticisms with no conclusion attached.
- **The implications answered separately.** They carry their own marks and most answers omit them entirely.

SEE ALSO Slot I - 39 basic emotions and the theories in brief Slot I - 38 emotional intelligence SPRINT

Day 4, general and developmental psychology

I - 43

What do you understand by the term intelligence? Describe the one test commonly used for measuring intelligence which has been standardized for use in India. [10]

10 | ASKED AT 15 MARKS | LEARNING, MEMORY AND GENERAL PSYCHOLOGY

KUHS, TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

3	What intelligence is	A definition, and the quotient it is expressed as
2	The Indian tests	Named, with the one you will describe marked
3	The chosen test	Structure, administration, scoring
2	Interpretation	What the figure settles, and what it cannot

TEST	AUTHOR AND YEAR	AGE RANGE	WHERE IT IS USED
Malin's Intelligence Scale for Indian Children	Malin, 1969	Six to fifteen years	**The standard child scale, described below**
Binet-Kamat Test of Intelligence	Kamat, 1934	Childhood into early adulthood	Indian adaptation of the Binet tradition
Bhatia Battery of Performance Tests	Bhatia, 1955	Eleven years and above	Wholly performance based, so language and literacy do not limit it
Vineland Social Maturity Scale, Indian adaptation	Malin	Infancy to adulthood	Social maturity, not intelligence; used alongside, never instead

- **Structure of the chosen test.** An Indian adaptation of the Wechsler children's scale, with Indian norms and content. Eleven subtests in two halves. Six verbal: information, general comprehension, arithmetic, analogies and similarities, vocabulary, digit span. Five performance: picture completion, block design, object assembly, coding, mazes. It yields verbal, performance and full scale quotients.
- **Administration and scoring.** Individually administered by a trained examiner in about an hour, alternating the two halves to hold attention. Raw scores convert to scaled scores, and those to a deviation quotient of mean a hundred, standard deviation fifteen. Subtest scatter is read alongside the total.

INTERPRETATION, AND THE HONEST LIMITS

Used for intellectual disability certification, for specific learning disorder where a verbal and performance discrepancy matters, and for documenting decline against a baseline. The limits belong in the answer: norms drift, schooling inflates the verbal half, sensory and motor impairment depress the performance half, and a quotient never diagnoses disability alone.

WHAT EARNS THE MARKS

- **A definition before the test.** The first part of the stem carries its own marks.
- **One test chosen and named,** with author and year, rather than a survey of five.
- **The subtests in their two halves,** which is the body of the description.

SEE ALSO Slot I - 36 theories of intelligence Slot I - 49 rating scales and their psychometric properties SPRINT Day 4, general and developmental psychology

I - 44

Discuss Pavlovian conditioning. Discuss its role in behaviour therapy. [5+5]

5 + 5 | LEARNING, MEMORY AND GENERAL PSYCHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The paradigm	Four terms, in the order in which they occur
3	The phenomena	Extinction onward, each with its named effect
3	In behaviour therapy	Each treatment traced back to a phenomenon
2	Why relapse happens	Extinction is inhibition, never erasure

THE PARADIGM

An unconditioned stimulus already produces an unconditioned response. A neutral stimulus presented repeatedly just before it comes to produce a similar response alone; it is then a conditioned stimulus, its effect a conditioned response. Rescorla's correction is worth a line: what is learned is prediction, not mere pairing.

- **The phenomena to name.** Acquisition, extinction when the signal comes without the event, spontaneous recovery after a rest. Generalisation and discrimination. Higher order conditioning. Experimental neurosis, from demanding a discrimination that cannot be made. Blocking, where a predictive cue prevents a new one being learned. Preparedness, which is why snakes condition readily and sockets do not. Taste aversion, learned in one trial across hours.
- **The therapy lineage.** Watson and Rayner conditioned fear in an eleven month old infant; Jones then removed a child's fear by pairing the animal with food and with unafraid children, the first behaviour therapy on record. Wolpe formalised it as systematic desensitisation: a graded hierarchy, a response incompatible with anxiety, exposure to each step until it settles. Response prevention in obsessive compulsive disorder, flooding, aversion therapy, cue exposure for craving, the bell and pad for enuresis and interoceptive exposure in panic all descend from it.
- **Conditioning as pathogenesis, not only treatment.** Conditioned craving and conditioned withdrawal explain relapse on returning to the place of use. Anticipatory nausea before chemotherapy is a conditioned response to the clinic. Agoraphobic avoidance begins as generalisation from a single panic.

PITFALL

Extinction does not erase the original learning. It builds a new inhibitory association tied to its context, which is why fear returns with time, in a new setting, or after one unexpected exposure.

WHAT EARNS THE MARKS

- **The four terms in the right order,** with prediction rather than pairing named as what is learned.
- **Named phenomena, not a paraphrase.** Blocking, preparedness and taste aversion are the ones most answers miss.
- **The relapse paragraph.** Context dependent extinction is the discriminating point on this stem.

SEE ALSO Slot I - 41 classical and operant conditioning compared Slot I - 40 the models of learning classified SPRINT Day 5, social, learning and personality psychology

AREA 05 OF 12 · P1-A09

Psychometry and assessment

9	9.1%	I-45	I-53
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 4 SINGLE 5

REVISION ORDER

The three projective slots together: they are one body of material asked at three widths, and the catalogue slot half writes the other two. The two rating-scale slots next, they share their psychometrics. The mini mental state examination and the structured assessment slot after that, both short. The personality-tools slot and the socioeconomic slot are independent of everything else in this area.

WHAT IS IN THIS AREA

Projective testing in three slots: the Thematic Apperception Test in full, the classification with any one instrument described in detail, and the catalogue. The mini mental state examination. The classification of rating scales with the psychometric properties and the standardisation of a newly built intelligence test. The scales of depression, with one described as used in practice. Structured clinical assessment, and a slot that wants five theories of personality evaluated with four assessment tools. One slot here asks for the determination of socioeconomic strata in India. That is a social classification question the boards set inside an assessment area, and it is answered on its page as it was set.

I - 45

What is apperception? Describe administration and interpretation of Thematic Apperception Test. [2+4+4]

2 + 4 + 4 | PSYCHOMETRY AND ASSESSMENT

DNB, KNRUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 Apperception	Perception plus the meaning the perceiver adds
4 Administration	Cards, instruction, sessions, the inquiry
3 Interpretation	Hero, need, press, thema, across stories
1 Standing	A battery supplement, never a diagnosis

THE DEFINITION

Apperception is perception interpreted: a new percept absorbed into, and given meaning by, the perceiver's past experience, needs and expectations. Make the stimulus ambiguous enough and the meaning can come only from the perceiver, which is the idea Murray built the test on.

STAGE	WHAT THE TESTER DOES	WHAT IS RECORDED
Selection	Picks a subset from the card series using the age and sex codes each card carries, one card being blank	Which cards, and in what order
Instruction	Asks for a story with what led up to the scene, what is happening, what the figures think and feel, and how it ends	**The story, verbatim, in the subject's own words**
Sessions	Around twenty cards over two sittings classically; about ten in ordinary clinical use	Latency to first word, time per card
Inquiry	Returns afterwards to ask about vague endings, omitted figures and refusals	Blocks, evasions and corrections

The cards are copyrighted. Describe the procedure and the codes, never the pictures and never a story.

- **Murray's frame.** Identify the hero, the hero's needs, the press acting on the hero from outside, and the outcome. Needs and press together give the thema.
- **Read across, not down.** A single story means little. Recurrent theme, repeated conflict, constant emotional tone and stereotyped resolution across the set are the data.
- **The limit.** Formal systems such as Bellak's exist, but routine use stays qualitative, so interpreter dependence is the stated weakness. It yields no diagnosis and supplements a battery when self-report is guarded.

WHAT EARNS THE MARKS

- **Apperception defined as meaning added to a percept,** not as a synonym for perception.
- **The instruction written out in its four parts.** Past, present, thoughts and feelings, ending.
- **Hero, need, press and thema named as the interpretive frame,** with Murray attached.
- **The honest limit.** Supplement, not a diagnostic test, and interpreter dependent.

SEE ALSO Slot I - 48 projective tests classified Slot I - 53 projective tests, short note **SPRINT**

Day 6, psychometry, assessment and descriptive psychopathology

I - 46

Describe the methods of determination of socioeconomic strata in India. [10]

10 PSYCHOMETRY AND ASSESSMENT KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Why it is measured	A covariate in every Indian clinical study
3	The household-head route	Kuppuswamy: education, occupation, income
3	The per capita route	Prasad, and why it needs re-indexing
2	Rural and asset routes	Pareek, standard of living, wealth index

SCALE	UNIT IT SCORES	DOMAINS SCORED	WHERE IT FITS
Modified Kuppuswamy	Head of the family	Education, occupation, monthly family income	**Urban and semi-urban, the default in Indian psychiatry papers**
B G Prasad	Per capita monthly income	Income alone, in five graded classes	**Rural and urban, single-variable, quick**
Udai Pareek	The rural household	Caste, occupation, education, land, housing, possessions, social participation	Village and agrarian samples
Standard of living index	Household assets	Housing, sanitation, fuel, durables, land	Large surveys where income recall fails
Wealth index	Household assets	Same asset set, weighted by principal component analysis	National survey work and quintile comparison

Amber marks the two an examiner expects you to be able to apply, not merely name.

- **Kuppuswamy, the structure.** Three components scored separately: education one to seven, occupation one to ten, family income one to twelve. The total runs from three to twenty-nine and falls into five classes, upper, upper middle, lower middle, upper lower and lower.
- **The number you must not memorise.** The income slabs of both Kuppuswamy and Prasad are re-indexed every year to the consumer price index for industrial workers. Quote the year of the revision you used, never a remembered rupee figure.
- **Choosing one.** Head of family for urban clinic samples, per capita for community samples, asset based where income is under-reported or seasonal.
- **Where they fail.** Joint families dilute per capita income, agricultural income is seasonal and under-declared, and none of these scales measures caste disadvantage or debt.

WHAT EARNS THE MARKS

- **Named scales, not the phrase socioeconomic status.** Four names on the page is four ticks.
- **Kuppuswamy's three components with their score ranges,** and the five classes they produce.
- **The re-indexing rule stated explicitly.** It is the single point that separates a current answer from a copied one.
- **One line on choosing between them** for a stated study setting.

SEE ALSO Slot I - 50 structured clinical assessment Slot I - 83 the National Mental Health Survey

I - 47

Mini mental state examination. [10]

10 PSYCHOMETRY AND ASSESSMENT KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 What it is	Author, year, and the one job it does
4 Structure	Seven domains, thirty points, the allocation
2 Interpretation	The traditional cut, and what shifts it
2 Limits in India	Literacy load, and the adaptation to use instead

THE DEFINITION

The Mini Mental State Examination (Folstein, Folstein and McHugh, 1975) is a brief clinician-administered screen of global cognitive function, scored out of thirty and completed in about five to ten minutes. It screens. It does not diagnose, it does not localise, and it is not a neuropsychological battery.

DOMAIN	POINTS	WHAT IT SAMPLES
Orientation to time	5	Temporal orientation, the earliest to go
Orientation to place	5	Spatial orientation across nested levels
Registration	3	Immediate encoding of new material
Attention and calculation	5	Sustained attention and working memory
Recall	3	Short-term retention after interference
Language	8	Naming, repetition, comprehension, reading, writing
Construction	1	Visuoconstructional copying

Thirty points across seven domains. The amber rows are where delirium and early dementia declare themselves first.

- **The cut.** Below twenty-four traditionally suggests cognitive impairment, with twenty-four to thirty read as unimpaired, eighteen to twenty-three as mild and seventeen or below as severe. Age and education move the threshold, so the cut is a prompt to test further, never a diagnosis.
- **What it misses.** It is insensitive to mild impairment and largely blind to executive and visuospatial failure: a patient can score full marks and still be grossly dysexecutive. The Montreal Cognitive Assessment, also out of thirty, is preferred when early change is the question.
- **The Indian caveat.** It is language and literacy loaded, so Western cut-offs misclassify non-literate patients. Use a validated vernacular adaptation such as the Hindi Mental State Examination, and screen in a language the patient reads.

WHAT EARNS THE MARKS

- **The point allocation reproduced,** domain by domain, summing to thirty.
- **The cut given with its qualifier.** A bare "less than twenty-four" without age and education loses the second mark.
- **Screen, not diagnosis,** written as a sentence rather than assumed.
- **The vernacular adaptation named.** It is the Indian mark on this question.

SEE ALSO Slot I - 50 structured clinical assessment Slot I - 95 disorders of consciousness SPRINT

Day 6, psychometry, assessment and descriptive psychopathology

I - 48

What do you understand by projective tests? Name different projective tests and describe any one in detail. [2+2+6]

2 + 2 + 6 | PSYCHOMETRY AND ASSESSMENT

DNB, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 **The projective hypothesis**	Ambiguous stimulus, hidden key, broad band
2 **The classification**	Five response modes, named tests under each
4 **One in detail**	Rorschach: material, phases, scoring codes
2 **Standing**	What it is good for, and the honest weakness

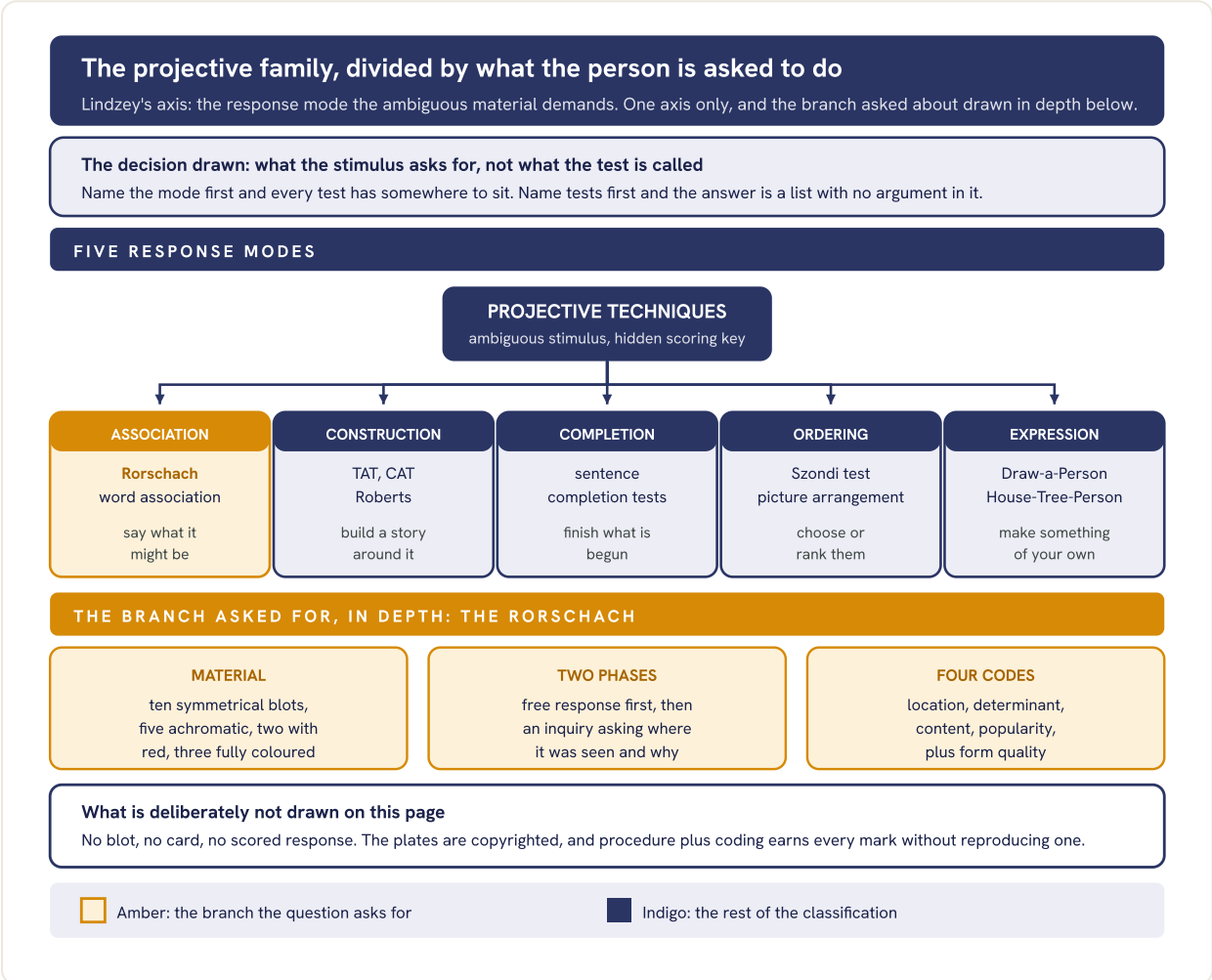

Fig 048.1 The projective techniques divided on one axis, the response mode, with the association branch opened out into the material, the phases and the coding of the Rorschach.

- **The projective hypothesis.** When the stimulus is ambiguous and the scoring key hidden, the structure the person imposes must come from within, so needs, conflicts and defences are read off it. Sampling is broad band and it is harder to fake than self-report.
- **Rorschach in one line.** Hermann Rorschach, Psychodiagnostik, 1921; scored today by the Exner Comprehensive System or its successor, the Rorschach Performance Assessment System.

- **The honest weakness.** Inter-rater and criterion validity are contested, norms are culture bound, administration is long. It supplements a battery when thought process is the referral question, never standing alone.

WHAT EARNS THE MARKS

- **The axis named before the list.** Response mode, then the five branches, then the tests.
- **Every branch of the plate reproduced.** Five modes with a named test under each is the two-mark listing part complete.
- **Depth only in the branch chosen,** material, phases and coding, and nowhere else.
- **The contested psychometrics named,** not defended and not hidden.

SEE ALSO Slot I - 45 the Thematic Apperception Test Slot I - 53 projective tests, short note **SPRINT**

Day 6, psychometry, assessment and descriptive psychopathology

I - 49

Classify psychiatry rating scales. What are the psychometric properties of a psychiatric scale? Describe different procedures of standardization of a newly developed intelligence test. [2+2+6]

2 + 2 + 6 | PSYCHOMETRY AND ASSESSMENT | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Classify the scales	By rater, by purpose, by breadth, by scaling
2	The properties	Reliability, validity, and sensitivity to change
5	Standardisation	Items, pilot, fixed procedure, norms, IQ scale
1	Release	What has to be true before it is used

STEP	WHAT IS DONE	EVIDENCE IT YIELDS	PROPERTY SECURED
Blueprint and items	Define the construct, write a surplus of items, pilot them	Difficulty and discrimination	Content validity
Fix the procedure	Freeze instructions, order, timing, stop rules, scoring key	One administration everywhere	Standardisation
Norming	A large sample stratified by age, sex, education, region	Age-graded norm tables	Comparability
Transformation	Raw score to a deviation quotient, fixed mean and spread	The deviation IQ	Interpretability
Reliability, validity	Re-test, split-half, alternate forms, inter-rater; then correlate against an established test	A coefficient per method	Both, in that order

STATUS: WHAT HAS TO BE TRUE BEFORE IT IS USED

A test becomes usable when the procedure is frozen, the norms come from the population it will be used on, and a manual exists that a second examiner can follow to the same score. An imported test used without local norms is unstandardised, whatever its reputation abroad.

- **Classifying the scales.** By rater, self, clinician or informant. By purpose, screening, diagnostic, severity or outcome. By breadth, global against symptom specific. By scaling, categorical, ordinal, Likert or visual analogue.
- **The properties.** Reliability is consistency: re-test, internal consistency, alternate forms, inter-rater agreement. Validity is accuracy: face, content, criterion and construct evidence. A severity scale needs one more, sensitivity to change.
- **The order that matters.** Reliability is necessary and not sufficient, since a test can be consistently wrong. Validity evidence comes after reliability, never instead of it.

WHAT EARNS THE MARKS

- **A classification with a stated axis,** not an unsorted list of scale names.
- **Reliability and validity each broken into their named subtypes.**
- **Norming written as a step separate from procedure fixing.** They are the two halves of standardisation and most answers merge them.

SEE ALSO Slot I - 52 scales in research and depression instruments Slot I - 80 reliability and validity in study design

SPRINT Day 6, psychometry, assessment and descriptive psychopathology

I - 50

What is structured clinical assessment? Discuss its role and limitations in psychiatry. [2+5+3]

2 + 5 + 3 | PSYCHOMETRY AND ASSESSMENT | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	Fixed questions, fixed probes, operational scoring
3 What it fixes	Information variance and criterion variance
2 Where it is used	The named instruments, by setting
3 Limitations	Time, rigidity, culture, and what it cannot do

THE DEFINITION

Structured clinical assessment replaces the free interview with fixed questions in a fixed order, with defined probes and operational rules for scoring each response against diagnostic criteria. A fully structured instrument fixes the wording and can be given by a trained lay interviewer; a semi-structured one fixes coverage and criteria but leaves judgement in the rating, so it needs a clinician.

- **The two variances it removes.** Information variance, two clinicians eliciting different facts because they asked different questions. Criterion variance, two clinicians applying different thresholds to the same facts. Structuring the interview removes the first; operational criteria remove the second. Together they are the main source of unreliable diagnosis.
- **The instruments, by setting.** Semi-structured and clinician-rated for research diagnosis and for schedules built on a glossary. Fully structured and lay-administrable for large epidemiological surveys, which is how national and cross-national prevalence figures are generated at all. Brief structured instruments for screening in busy clinical services, and child versions for developmental work.
- **What else it buys.** A reproducible case definition for a trial, a teachable framework for a trainee, and a defensible record where the diagnosis may be examined later.
- **The limits.** Long, needing training and re-calibration. It rates presence rather than severity or function, is rigid with comorbid and subthreshold presentations, and inherits every error in the classification it encodes. Translated instruments need re-validation, not only translation.

PITFALL

Treating the schedule as the assessment. It produces a diagnosis, not a formulation. Nothing in it asks why this person became ill now, and the rapport lost to a questionnaire is the same rapport the treatment will need.

WHAT EARNS THE MARKS

- **Structured and semi-structured told apart,** by who may administer and what is fixed.
- **Information variance and criterion variance named as terms.** They are the reason the whole method exists.
- **Instruments placed by setting,** research, survey, clinic, rather than listed loose.
- **Diagnosis separated from formulation** in the limitations, which is where the last marks sit.

SEE ALSO Slot I - 47 the mini mental state examination Slot I - 49 rating scales and their properties SPRINT

Day 6, psychometry, assessment and descriptive psychopathology

I - 51

a) What are the 5 major theories of personality? b) Critically evaluate those major theories. c) Briefly write about any four such personality assessment tools related to those theories. [2+4+4]

2 + 4 + 4 | PSYCHOMETRY AND ASSESSMENT | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Name the five	One line each, with the theorist attached
4 Critical evaluation	One strength, one failure, per theory
3 Four tools	Each tied to the theory that generated it
1 The verdict	Which framework the evidence now favours

THEORY	CORE CLAIM	THE CRITIQUE THAT STICKS	TOOL IT GENERATED
Psychoanalytic	Personality is structure and unconscious conflict, laid down early	Retrospective, hard to falsify, weak predictive evidence	**Rorschach, Thematic Apperception Test**
Trait and dispositional	Personality is a small set of stable, measurable dimensions	Describes without explaining; underrates the situation	**16PF, NEO-PI-R, Eysenck questionnaire**
Behavioural and social learning	Personality is learned behaviour, cued and reinforced by situations	Under-explains cross-situational consistency and inner life	**Behavioural analysis, locus of control scale**
Humanistic	Personality is the self, organised around growth and self-actualisation	Optimistic, poorly operationalised, thin on psychopathology	Q-sort, self-concept measures
Cognitive and constructivist	Personality is the system of personal constructs used to anticipate events	Narrow range, limited clinical purchase	**Repertory grid**

Amber marks the four tools that answer part c. Naming a tool without the theory behind it answers half the question.

- **The evaluation, in one argument.** The five differ on where personality is located: inside, in traits, in situations, in the self, or in constructs. The trait framework has won on measurement because it alone produced replicable dimensions with normative data; the psychoanalytic framework has kept its clinical usefulness in formulation while losing the measurement argument.
- **The trap in part c.** A projective and an inventory are not interchangeable tools. Projective tools follow from the psychoanalytic claim that the important material is not available to self-report; inventories follow from the trait claim that it is.
- **Indian use.** Trait inventories need local norms before a raw profile means anything, and translated inventories need re-validation rather than translation alone.

WHAT EARNS THE MARKS

- **Five named theories with a theorist each,** before any evaluation begins.
- **One critique per theory,** written as a limitation rather than as dislike.
- **Four tools, each bolted to its parent theory.** That link is what part c is testing.
- **A stated verdict** on which framework the measurement evidence supports.

SEE ALSO Slot I - 48 projective tests classified Slot I - 53 projective tests, short note SPRINT
Day 5, social, learning and personality psychology

I - 52

a) What are the types of scales used in psychiatry from research point of view? b) Name the rating scales and questionnaires for depression and describe one rating scale in detail that you are using in your clinical practice. [4+6]

4 + 6 | PSYCHOMETRY AND ASSESSMENT | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4 Types of scales	Measurement level first, then rater, then purpose
2 Name them	Clinician-rated and self-rated depression instruments
3 One in detail	Structure, range, bands, response and remission
1 Its bias	Where the chosen scale reads high, and why

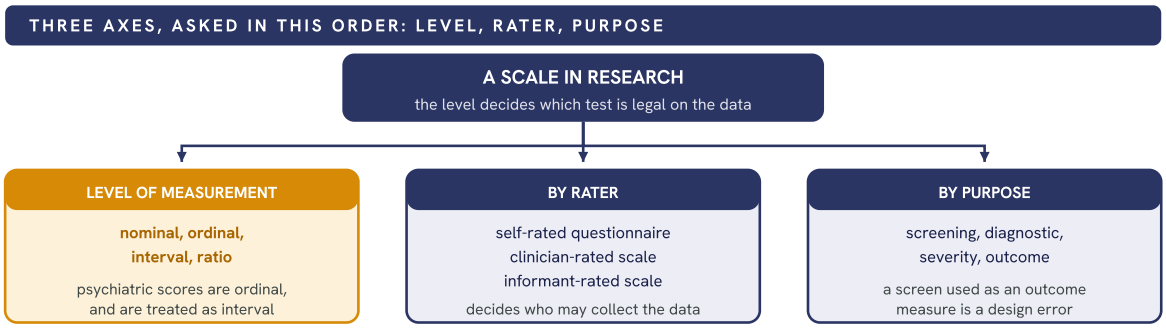

Fig 052.1 The three axes a research scale is classified on, the measurement level in amber because it decides which statistical test the data will allow.

ONE SCALE IN DETAIL: THE HAMILTON DEPRESSION RATING SCALE

Hamilton, 1960. Clinician-rated at interview over the past week, usually in its seventeen-item form: nine items score zero to four and eight zero to two, so the total runs zero to fifty-two. Bands: zero to seven none, eight to sixteen mild, seventeen to twenty-three moderate, twenty-four and above severe. In trials, response is a fall of half and remission a total of seven or below.

- **Name them.** Clinician-rated: Hamilton, Montgomery Asberg. Self-rated: Beck Depression Inventory, the nine-item Patient Health Questionnaire, Zung. Population specific: Geriatric Depression Scale, Edinburgh postnatal scale.
- **Where it reads high.** Hamilton is weighted to sleep, appetite and somatic items, so it inflates in physical illness and in the elderly. Montgomery Asberg was built less somatic and is preferred there.
- **The one rule.** A rating scale measures severity, never diagnosis. A high score is a reason to interview.

WHAT EARNS THE MARKS

- **Measurement level answered first,** with the consequence for test choice stated.
- **Both rater classes named for depression,** with instruments under each.
- **Its known bias stated.** The somatic weighting reads as practice rather than recall.

SEE ALSO Slot I - 49 classifying rating scales and standardisation Slot I - 80 reliability and validity in study design

SPRINT Day 6, psychometry, assessment and descriptive psychopathology

I - 53

Projective tests. [10]

10 PSYCHOMETRY AND ASSESSMENT RGUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	The hypothesis	Ambiguity forces the answer to come from within
5	The catalogue	Test, material, task, what is read from it
2	Clinical use	The referral questions they actually answer
1	Standing	One honest sentence on the evidence

TEST	MATERIAL	THE TASK	WHAT IS READ FROM IT
Rorschach	Ten symmetrical inkblots	Say what it might be, then show where	Perceptual organisation, thought process, reality testing
Thematic Apperception Test	Ambiguous interpersonal scenes	Tell a story with a past and an ending	Needs, external press, object relations
Children's Apperception Test	Animal or human scenes, ages three to ten	Tell a story	Childhood conflict around feeding, rivalry, control
Sentence completion	Incomplete written stems	Finish each stem with the first response	Attitudes to defined areas of life
Draw-a-Person	Blank paper and a pencil	Draw a person, then one of the other sex	Body image and self concept

Amber marks the two an examiner will follow up on. The materials are copyrighted and are described in words only.

- **The projective hypothesis.** The stimulus is unstructured and the scoring key is hidden, so the person has nothing external to lean on and the structure they impose is taken to reveal needs, conflicts and defences. Sampling is broad band, the whole personality rather than one trait.
- **What they are actually used for.** Where self-report is guarded or defended, where object-relational and motivational material is the referral question, and as one element of a battery in personality assessment. Never as a stand-alone diagnostic instrument.
- **Graded transparency.** Sentence completion is the most structured and most transparent, so the easiest to fake; the inkblot is the least. Word association, the oldest of the group, reads complexes from blocking and long latency.

PITFALL

Calling a projective test objective because it has a scoring system. The two families measure personality by opposite logic: ambiguous stimulus with inferential scoring against fixed-choice items with an empirical key. The reliability weakness follows from that logic and better scoring has not removed it.

WHAT EARNS THE MARKS

- **The hypothesis stated before any test is named.** A bare list is a mid-band answer.
- **Material and task given separately for each test.** That pairing is what the grid is for.
- **The referral question each one answers,** rather than a claim that they diagnose.
- **One honest line on the contested psychometrics.**

SEE ALSO Slot I - 48 projective tests classified and the Rorschach in detail Slot I - 45 the Thematic Apperception Test

SPRINT Day 6, psychometry, assessment and descriptive psychopathology

AREA 06 OF 12 · P1-A04

Genetics in psychiatry

9	9.0%	I-54	I-62
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 HIGH 1 SINGLE 7

REVISION ORDER

The two mapping slots together, they are the same answer asked twice, and the association-study slot sits directly on top of them. Twin and adoption designs next, as a pair. The schizophrenia slot after that: it is the anchor here and it reuses the family-risk ladder. Epigenetics, the gene-environment slot and the genetic-correlation slot are independent and each is short.

WHAT IS IN THIS AREA

The methods in five slots: twin studies with the bipolar findings, adoption designs with schizophrenia, the gene-mapping strategies with their strengths and limits, gene mapping set against linkage and association, and the genome-wide association studies. Then the genetics of schizophrenia, epigenetics, gene and environment in neuropsychiatric disease, and the slot that runs genetic correlation and pleiotropy across substance use and psychiatric disorders.

I - 54

Genetics of Schizophrenia. [10]

10 GENETICS IN PSYCHIATRY KNRUHS, RGUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2 The architecture	Highly heritable and highly polygenic, said in one line
3 Family, twin, adoption	The risk gradient, and what each design controls for
3 Molecular findings	Common variants, then the rare ones with big effects
2 The models	Two-hit and neurodevelopmental, and where they meet

THE LINE WORTH WRITING FIRST

Schizophrenia is among the most heritable of psychiatric disorders and among the most polygenic. Twin studies put heritability at about eighty per cent. Thousands of common variants of very small effect carry most of that, and a small number of rare copy number variants carry large effects in a few families.

RELATIONSHIP TO AN AFFECTED PERSON	GENES SHARED	APPROXIMATE LIFETIME RISK
No relation, general population	Baseline	About 1 in 100
Sibling	One half	About 9 in 100
Child of one affected parent	One half	About 13 in 100
Dizygotic co-twin	One half	About 17 in 100
Monozygotic co-twin	Essentially all	**About 48 in 100**

The pooled family study figures. The last two rows are the argument: both twin types share a womb and a household, only the genes differ, and the risk trebles. Note also that a monozygotic co-twin is far from certain to be affected, which is where environment enters.

- **What molecular work added.** The 2014 consortium study named 108 genome-wide significant loci, and later analyses several hundred. The strongest signal sits in the major histocompatibility complex on chromosome 6 and is driven by complement C4, which tags synaptic pruning. Rare copy number variants, the 22q11.2 deletion above all, carry far larger individual risk.
- **The two models to close with.** Two-hit: genetic vulnerability is the first hit, an environmental insult the second. Neurodevelopmental: early insults stay latent until adolescent synaptic pruning unmasks them. The C4 finding is where the two models meet.

WHAT EARNS THE MARKS

- **The monozygotic against dizygotic comparison stated as an argument,** not offered as two loose numbers.
- **Polygenic architecture named before any single gene.** A candidate-gene answer reads as dated.
- **Complement C4 tied to pruning.** It is the one molecular finding with a mechanism attached.

SEE ALSO Slot I - 58 twin studies in bipolar affective disorder Slot I - 60 adoption study designs SPRINT Day 3, Psychiatric Genetics

I - 55

Explain the genetic and environmental factors in neuropsychiatric diseases. [5+5]

5 + 5 | GENETICS IN PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**The genetic side**	Complex and polygenic is the rule; name the exceptions
2	**Heritability, read correctly**	What the number is and is not about
3	**The environmental side**	Ordered by timing, prenatal to adult
2	**How the two meet**	Interaction, correlation, epigenetic mediation

CONDITION	GENETIC SIDE	ENVIRONMENTAL SIDE	WHAT THE PAIR SHOWS
Huntington disease	One dominant gene, a CAG repeat	Almost none	**Genes can be sufficient**
Schizophrenia	High heritability, polygenic	Obstetric events, cannabis, urban birth, migration	Neither side alone is sufficient
Depression	Moderate heritability	Childhood adversity, loss events, chronic illness	Environment carries more weight
Alcohol use disorder	Moderate heritability	Availability, price, peer group	**Policy can move the phenotype**

The amber cells are the two ends of the spectrum. Everything examined in psychiatry sits between a gene that is sufficient and an exposure that can be legislated.

- **Environment, ordered by timing.** Prenatal: maternal infection, famine, hypoxia, valproate. Perinatal: obstetric complications. Childhood: abuse, neglect, separation, urban upbringing, migration. Later: cannabis, stimulants, alcohol, head injury, social defeat.
- **Three ways genes and environment meet.** Interaction, where an exposure harms only the vulnerable. Correlation, where genes shape which exposures a person meets, passively, evocatively or by active choice. Mediation, where the exposure leaves an epigenetic mark on expression.

PITFALL

High heritability is not fixity. Heritability describes how much of the variance in one population at one time is genetic; it says nothing about how modifiable an individual is. Phenylketonuria is fully genetic and fully preventable by diet, and that one example settles the point.

WHAT EARNS THE MARKS

- **Complex inheritance named as the rule,** with Mendelian conditions given as the exception rather than the model.
- **Exposures ordered by timing,** which shows a developmental frame rather than a memorised list.
- **All three modes of interplay,** not interaction alone.
- **The heritability caveat.** It is the sentence that separates a top answer here.

SEE ALSO Slot I - 54 genetics of schizophrenia Slot I - 59 epigenetics in psychiatry **SPRINT** Day 3, Psychiatric Genetics

I - 56

What are Genome Wide Association Studies? Describe their contribution to understanding etiology of psychiatric disorders. [10]

10 | GENETICS IN PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	Hypothesis-free, common variants, cases against controls
3 How it is done	The four steps, and the threshold that guards them
3 What it has contributed	Architecture, shared risk, and one drug-target lead
2 What it cannot do	The honest limits, stated as limits

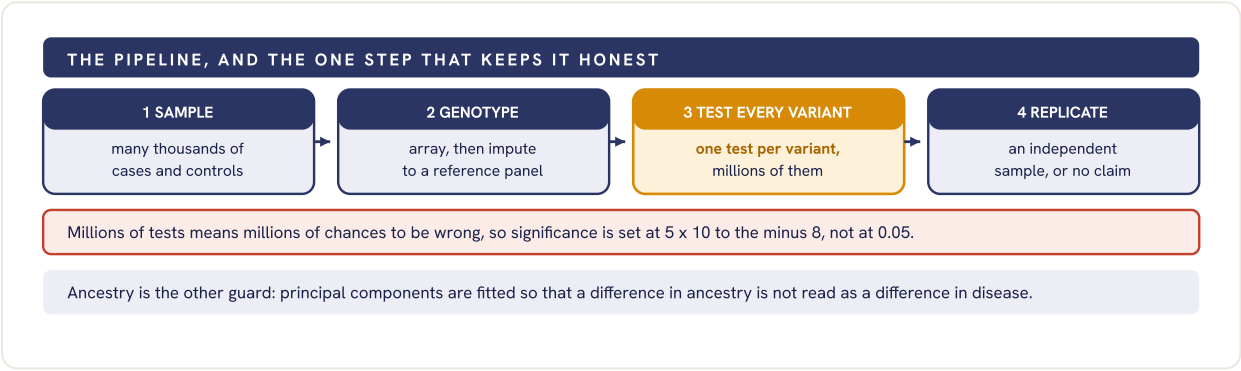

Fig 056.1 The four steps, with the two safeguards that make the output interpretable printed underneath rather than buried inside the method.

- **What it has contributed.** It settled the architecture: psychiatric disorders are highly polygenic, built from many common variants of tiny effect, not from a few broken genes. It showed substantial shared genetic risk across schizophrenia, bipolar disorder and depression. It produced polygenic risk scores, useful for research. In schizophrenia it delivered the complement C4 lead and pointed at calcium channel genes.
- **What it cannot do.** Each hit explains almost nothing on its own, and together they still leave heritability unaccounted for. The signal is a region, not a gene, because of linkage disequilibrium. Rare variants and copy number variants are invisible to it. Most samples are of European ancestry, so risk scores transfer poorly. Nothing here is a clinical test.

WHAT EARNS THE MARKS

- **Hypothesis-free stated explicitly,** and contrasted with the candidate-gene era it replaced.
- **The genome-wide threshold given with its reason.** Multiple testing, not convention.
- **Contribution framed as architecture, not as a gene list.** Polygenicity and cross-disorder overlap are the findings.
- **Limits named as limits,** including the ancestry bias, which is the one most candidates miss.

SEE ALSO Slot I - 61 gene mapping against linkage and association Slot I - 62 genetic correlation and pleiotropy

SPRINT Day 3, Psychiatric Genetics

I - 57

What are the different gene mapping strategies? Write down the basic idea, strength and limitations of the strategies. [3+2+2+3]

3 + 2 + 2 + 3 | GENETICS IN PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**The strategies**	Name the axis, then the four families of method
2	**Basic idea of each**	One sentence per strategy, no more
2	**Strengths**	What each one is uniquely good at finding
3	**Limitations**	The failure mode of each, and the one they share

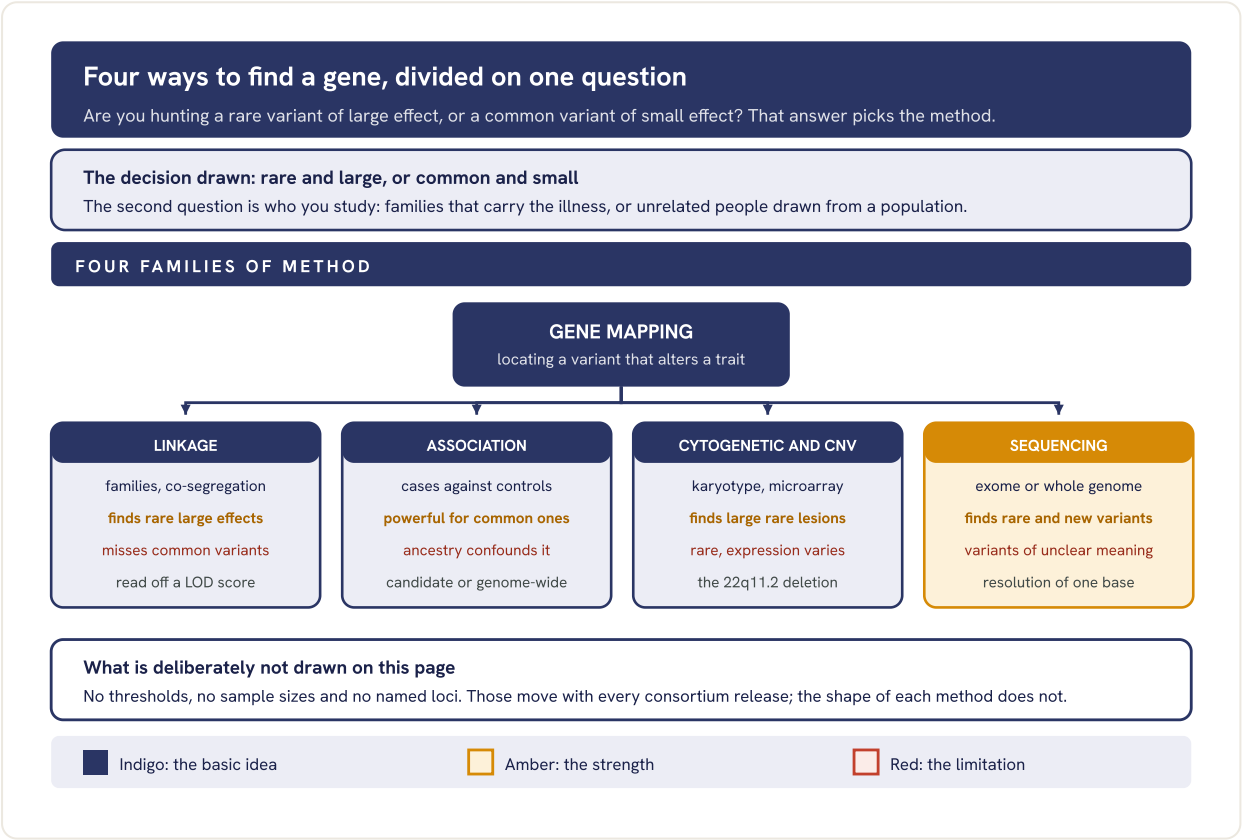

Fig 057.1 One axis, four branches, and inside each branch the same four lines in the same order, so the strategies can be read down the page as well as across it.

- **The basic ideas in one line each.** Linkage asks whether a marker travels with the illness through a family. Association asks whether an allele is commoner in cases than controls. Cytogenetics and microarray look for missing or duplicated stretches of chromosome. Sequencing reads the bases and compares them.
- **How they hand over to each other.** Linkage narrows a large region; association and sequencing resolve it to a variant. Practice today runs association genome-wide first, and keeps linkage for pedigrees with a striking inheritance pattern.

PITFALL

A genome-wide hit is not the gene. Linkage disequilibrium means the associated marker usually stands in for a nearby causal variant, and fine mapping and functional work are separate jobs that often fail. Saying so is the limitation mark that most answers leave on the table.

WHAT EARNS THE MARKS

- **The axis stated before the list.** Rare and large against common and small is what organises the four.
- **Idea, strength and limitation for every strategy,** in that order, because the stem asks for all three.
- **Linkage and association placed under gene mapping,** not offered as its alternatives.
- **The shared limitation.** A located region is not a mechanism.

SEE ALSO Slot I - 61 gene mapping against linkage and association Slot I - 56 genome wide association studies

SPRINT Day 3, Psychiatric Genetics

I - 58

Twin studies in Bipolar affective disorder. [10]

10 GENETICS IN PSYCHIATRY KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The twin method	Why two kinds of twin answer a genetic question
3	Design and assumptions	Zygosity, concordance, and the modelling that follows
3	What it shows in bipolar	The pattern, the heritability, and the spectrum finding
2	Limitations	The assumption that carries the whole method

THE METHOD IN TWO SENTENCES

Monozygotic twins share essentially all their segregating genes, dizygotic twins about half, and both kinds share a womb and a household. So any excess of monozygotic over dizygotic concordance is attributed to genes, provided the two kinds of twin really do share their environment equally.

ELEMENT	WHAT IT MEANS	WHAT IT DELIVERS	WHERE IT IS CHALLENGED
Zygosity	Established by genotyping, not by looks	The two comparison groups	Misclassification dilutes the estimate
Probandwise concordance	Counts each affected twin as a proband	The figure comparable with population risk	**Pairwise counting gives a lower number for the same data**
Equal environments	Both twin types share family environment equally	Makes the excess interpretable as genetic	**Identical twins are treated and treat each other more alike**
Variance modelling	Splits variance into genes, shared and unique environment	The heritability estimate	Assumes no gene-environment interaction

Single concordance percentages are not printed here on purpose: they swing with how wide the diagnostic boundary is drawn and with how the pairs were counted. Quote the pattern and the assumption, and you cannot be caught out on the number.

- **What twin studies show in bipolar disorder.** Monozygotic concordance is several times dizygotic concordance, and heritability estimates sit around eighty per cent, among the highest in psychiatry. Concordance is nowhere near complete, so environment and chance still matter.
- **The finding examiners want beyond the numbers.** Co-twins who are not bipolar are still at raised risk of unipolar depression and of other affective illness, which is evidence that what is inherited is a broad affective liability rather than a diagnosis.

WHAT EARNS THE MARKS

- **The logic of the method before any figure.** Two twin types, one difference, one inference.
- **The equal environments assumption named,** and named as the method's weak point.
- **Incomplete concordance used as an argument,** not treated as a failure of the genetic case.
- **The broadened phenotype in co-twins,** which is what makes twin work informative about nosology.

SEE ALSO Slot I - 54 genetics of schizophrenia Slot I - 60 adoption study designs SPRINT

Day 3, Psychiatric Genetics

I - 59

Define epigenetics. Discuss its contribution and implications in Psychiatry. [2+4+4]

2 + 4 + 4 | GENETICS IN PSYCHIATRY | DNB, MUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 Definition	Change in expression without change in sequence
4 Mechanisms	Three marks, each with its direction of effect
2 Contribution	What it explains that genetics alone could not
2 Implications and limits	Targets and biomarkers, and why to be careful

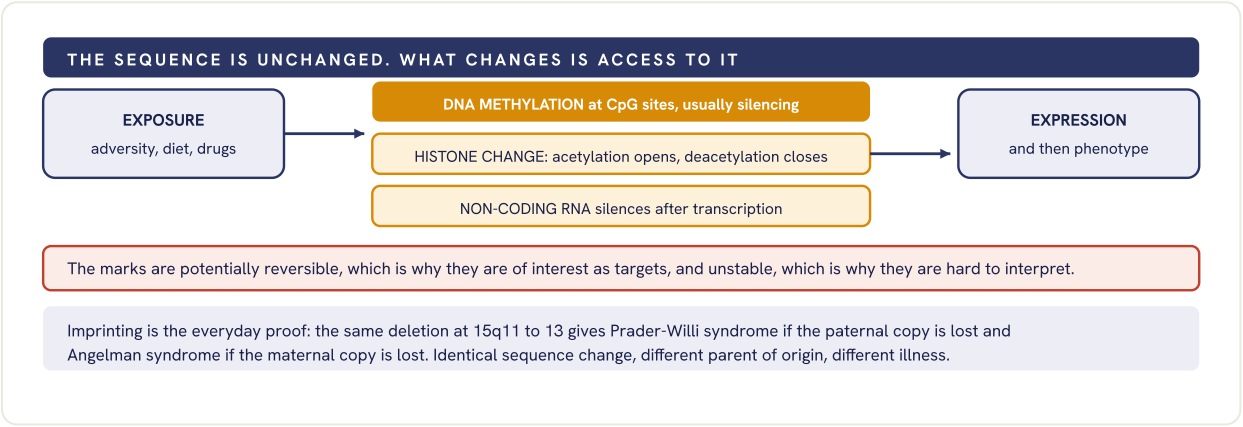

Fig 059.1 The three marks drawn as one layer between exposure and expression, with imprinting placed beneath as the demonstration that parental origin alone can change a phenotype.

- **Contribution to psychiatry.** It supplies a mechanism for gene-environment interplay, so early adversity can leave a lasting mark on stress-axis gene expression. It helps explain why monozygotic twins are discordant. Valproate is a histone deacetylase inhibitor, and folate and vitamin B12 feed the methyl supply, so the mechanism already touches prescribing.
- **The limits to state plainly.** Most human work measures blood, and blood is not brain. Direction of causation is rarely established: illness and its treatment change methylation too. Findings are strongly tissue and cell specific, and few replicate. No epigenetic test is in clinical use in psychiatry.

WHAT EARNS THE MARKS

- **The definition tied to sequence.** Expression changes, sequence does not, and heritable through cell division.
- **All three mechanisms, with a direction each.** Methylation alone is the commonest half answer.
- **One worked example.** Imprinting at 15q11 to 13 is the cleanest one available.
- **Reversibility named as the reason for interest,** and instability named as the reason for caution.

SEE ALSO Slot I - 55 genetic and environmental factors Slot I - 54 genetics of schizophrenia **SPRINT** Day 3, Psychiatric Genetics

I - 60

What do you understand by adoption studies? Describe different designs of adoption studies with special reference to Schizophrenia. [3+7]

3 + 7 | GENETICS IN PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 What they are	The one thing adoption separates that twins cannot
4 The four designs	Who is sampled first decides which design it is
3 Findings and limits	What schizophrenia work established, and what to concede

DESIGN	WHO IS SAMPLED FIRST	WHAT IT SEPARATES	WHAT SCHIZOPHRENIA WORK SHOWED
Adoptee study	Adopted-away children of affected biological parents	Genes from rearing, prospectively	**Raised rates despite rearing away from the affected parent**
Adoptee's family method	Adopted people who are already ill	Biological relatives from adoptive relatives	**Excess illness in biological relatives only**
Cross-fostering	Children of well parents reared by affected adoptive parents	The effect of rearing on its own	Being reared by an affected parent did not raise risk
Adoptive family study	High-risk and low-risk adoptees, with the adoptive family assessed	Interaction between the two	High genetic risk mattered mainly in disturbed adoptive families

The amber rows are the two designs that carry the genetic claim, from opposite ends. The fourth put interaction back into a literature that had been read as proof of genes alone.

- **What adoption adds that twins cannot.** A twin pair shares its rearing, so a twin study cannot fully separate genes from family environment. Adoption breaks the two apart: the child carries the genes of one family and grows up in another.
- **The spectrum finding.** Biological relatives of adopted people with schizophrenia showed an excess not only of schizophrenia but of schizotypal and paranoid presentations. That is the evidence base for the idea of a schizophrenia spectrum, and it came from adoption work.
- **Limitations to concede.** Placement is not random and adoptive homes are screened, which narrows the environmental range. Late placement leaves early rearing shared. The biological mother still supplies the prenatal environment. Numbers are small, and adoption itself selects an unusual population.

WHAT EARNS THE MARKS

- **The comparison with twin studies made explicit,** since it is the reason the design exists.
- **All four designs named and told apart by who is sampled first.**
- **The spectrum finding credited to adoption work,** which is the "special reference to schizophrenia" the stem is asking for.
- **The prenatal environment named among the limits.** It is the one candidates most often miss.

SEE ALSO Slot I - 54 genetics of schizophrenia Slot I - 58 twin studies in bipolar affective disorder **SPRINT** Day 3, Psychiatric Genetics

I - 61

What is gene mapping? How is it different from linkage analysis and association studies? [3+7]

3 + 7 | GENETICS IN PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Gene mapping defined	The umbrella, with the other two placed under it
4 The grid	Domain by domain, with the discriminators marked
3 Overlap and what follows	Where the two methods meet, and how they are sequenced

DOMAIN	LINKAGE ANALYSIS	ASSOCIATION STUDY	WHAT SETTLES IT
Who is studied	Families with several affected members	Unrelated cases and controls, or trios	**Related people or strangers**
What is tracked	Co-segregation of a marker with illness within a family	Allele frequency difference between groups	**Within a pedigree or across a population**
Distance it works over	Long, tens of millions of bases	Very short, the span of linkage disequilibrium	**Resolution: a region against a variant**
Statistic	The LOD score	A p value against a genome-wide threshold	Different traditions, same logic
Best suited to	Rare variants of large effect	Common variants of small effect	**Effect size and allele frequency**
Main failure mode	Underpowered for common small effects	Confounded by population stratification	Each fails where the other works

Amber cells are the discriminators. Each reduces to the same pair of questions: whose DNA is compared, and how big is the effect being hunted.

- **The overlap.** Both are gene mapping, and family-based association tests sit in between: the transmission disequilibrium test compares the alleles a parent passes on with the alleles the same parent does not, so it is an association test that cannot be confounded by ancestry.
- **What follows from the difference.** Linkage narrows the search to a region; association resolves the region to a variant; sequencing reads it. They are stages, not rivals, and saying so answers the stem's hidden question.

PITFALL

The stem invites you to treat all three as parallel alternatives. They are not. Gene mapping is the goal; linkage and association are two ways of reaching it. Opening with that sentence earns the first three marks before any comparison begins.

WHAT EARNS THE MARKS

- **Gene mapping defined as the umbrella,** in the first line, not implied by the layout.
- **A grid, not two paragraphs.** The comparison has to be readable across a row.
- **The discriminators identified as discriminators,** rather than a flat list of six differences.
- **The family-based association test offered as the overlap,** which is the depth mark on this question.

SEE ALSO Slot I - 57 gene mapping strategies Slot I - 56 genome wide association studies SPRINT Day 3, Psychiatric Genetics

I - 62

Genetic correlation, pleiotropy, and associations between substance use and psychiatric disorders. [10]

10 | GENETICS IN PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	The two terms	Defined apart, because they are not the same claim
2	How they are measured	Twin designs first, then summary-statistic methods
3	Substance use findings	What overlaps with what, and the measurement caveat
2	What it does not prove	Correlation, direction, and how direction is tested

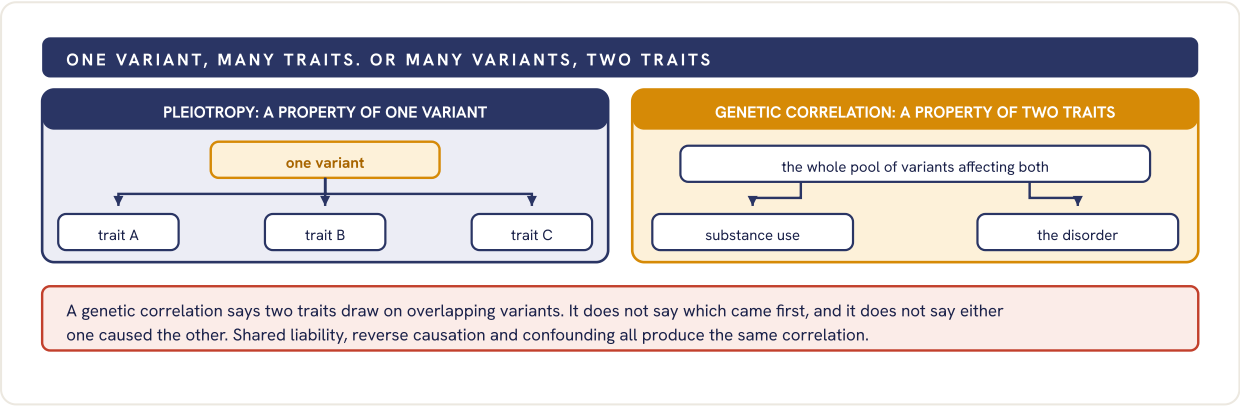

Fig 062.1 The two constructs drawn side by side because they are routinely merged: pleiotropy is a statement about one variant, genetic correlation a statement about two traits.

- **How they are measured, and what is found.** Twin designs give cross-twin cross-trait correlations; modern estimates come from genome-wide summary statistics by linkage disequilibrium score regression. Substantial genetic correlation runs across schizophrenia, bipolar disorder, depression and ADHD, and substance use traits correlate with all of them, most strongly with ADHD and with depression.
- **The measurement caveat, and the causal test.** How much a person drinks and whether the drinking has become a disorder are genetically different traits that correlate differently with psychiatric illness, so say which you mean. For direction the tool is Mendelian randomisation, run both ways between cannabis and schizophrenia.

PITFALL

Do not use the two terms as synonyms. Separate horizontal pleiotropy, one variant affecting two traits independently, from vertical, acting on the second only through the first. Mendelian randomisation is valid only under the horizontal assumption.

WHAT EARNS THE MARKS

- **Two separate definitions,** one about a variant and one about a pair of traits.
- **Horizontal separated from vertical pleiotropy,** which is the discriminator this question is built around.
- **Consumption distinguished from disorder** when reporting any substance use finding.
- **Causation addressed explicitly,** with Mendelian randomisation named as the method that tests it.

SEE ALSO Slot I - 56 genome wide association studies Slot I - 54 genetics of schizophrenia SPRINT Day 3, Psychiatric Genetics

AREA 07 OF 12 · P1-A08

Social, cross-cultural and evolutionary psychology

8	7.8%	I-63	I-70
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 7

REVISION ORDER

Build one culture answer properly and four of the eight slots open. Take the definitional slot first and the culture-bound syndromes last of the four, because the syndromes are a list and the other three are an argument. The social theories of suicide next, it is the one that needs its own reading. Religion, marriage and imprinting are independent and all three are short.

WHAT IS IN THIS AREA

Culture in four slots: its role in psychiatry, the definition with illness behaviour and symptomatology, the culture-bound syndromes with five Indian ones and the cultural theories of mental illness, and the slot that runs culture through expression, assessment and treatment and adds immigration. Then the social theories of suicide, religion and psychiatry, marriage as an institution, and imprinting, which is the one ethology slot in this book.

I - 63

What is culture bound syndrome? Describe any five Indian culture bound syndrome. Discuss different theories of mental illness from cultural perspective. [1+5+4]

1 + 5 + 4 | SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

1	Definition	The term, and what DSM-5 put in its place
5	Five Indian	Each with its region and its presentation
3	The theories	Etic against emic, and the category fallacy
1	The caution	Where the concept is criticised

THE DEFINITION

A recurrent, locality-specific pattern of aberrant behaviour and distressing experience that is recognised as an illness within its own culture and often has no direct counterpart in the international classifications.

SYNDROME	WHERE	THE PRESENTATION	BEST UNDERSTOOD AS
Dhat	North and west India, South Asia widely	Fatigue, weakness and anxiety attributed to semen passed in urine	**An idiom of distress; usually depression or anxiety**
Koro	Epidemics in Assam and West Bengal	Acute terror that the genitals are retracting, with fear of death	Acute anxiety, spread by contagion
Possession states	Across India, more often in women	Trance, altered identity, a spirit speaking through the person	Dissociative trance and possession disorder
Ascetic syndrome	Described in India by Neki	Adolescent, usually male: withdrawal, severe religiosity, self-imposed austerity	Often the prodrome of schizophrenia
Gilhari syndrome	Rajasthan	A swelling believed to move under the skin and to kill on reaching the neck	A culturally shaped conversion presentation

- **The theories, which are the last four marks.** The universalist or etic position holds disorders to be the same everywhere, with culture only colouring them. The relativist or emic position holds categories to be meaningful only inside the culture that made them. Kleinman named the category fallacy, the error of carrying a category built in one culture into another where it has no coherence, and set the patient's explanatory model against the professional one.
- **What DSM-5 did to the term.** It retired culture-bound syndrome and put three things in its place: cultural syndromes, cultural idioms of distress, and cultural explanations of distress. Saying so is worth a mark.

WHAT EARNS THE MARKS

- **Exactly five, each with a region and a presentation.** The stem says five, so a sixth earns nothing and a fourth loses a mark.
- **Etic and emic named as positions,** not described as attitudes.
- **The category fallacy, attributed.** It is the single highest-value term in the last section.

SEE ALSO Slot I - 70 culture, illness behaviour and symptomatology

Slot I - 67 the role of culture across the clinical pathway **SPRINT** Day 5, social, learning and personality psychology

I - 64

Concept of imprinting in ethology. [10]

10 SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **The definition**	Rapid, timed, unrewarded, lasting
3 **The classic work**	Lorenz, the geese, and the window
3 **The two kinds**	Filial and sexual, and their separate windows
2 **Use in psychiatry**	The line from ethology to attachment

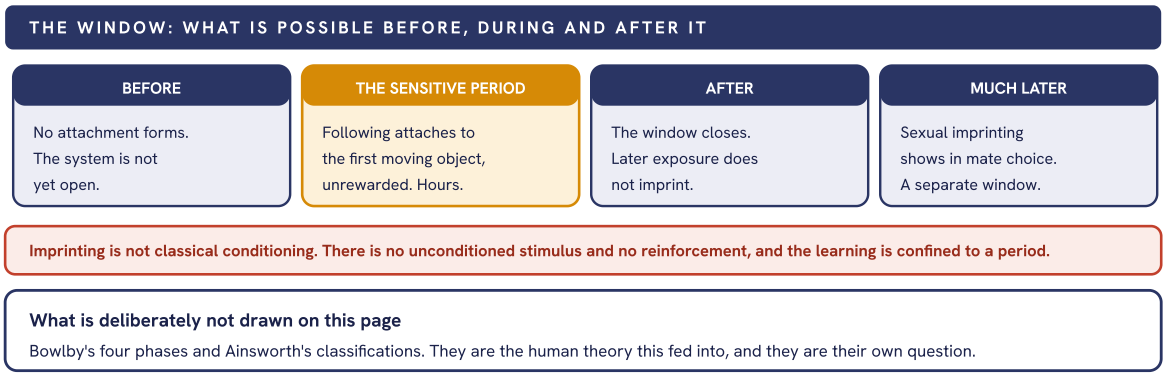

Fig 064.1 Imprinting drawn as a window rather than a process. Amber marks the sensitive period itself, which is the claim the concept stands or falls on; the fourth panel is the separate, much later window for sexual imprinting.

- **The definition, with its authority.** A rapid form of learning, confined to a sensitive period early in life, by which a young animal forms an attachment to a moving object, usually the parent, without reward and with lasting effect. Konrad Lorenz established it in greylag geese from 1935, the observation having been made by Spalding in the previous century. Lorenz shared the 1973 Nobel Prize with Tinbergen and von Frisch.
- **Why psychiatry keeps it.** It supplied the biological argument Bowlby used: attachment as a species-typical behavioural system with its own sensitive period, not a by-product of feeding. Harlow's monkeys made the same point from the other side, preferring the cloth mother to the one that fed them.

WHAT EARNS THE MARKS

- **All four defining features.** Rapid, period-bound, without reinforcement, and persistent.
- **Lorenz and the geese, with the year.** The classic experiment is the answer's evidence.
- **Filial and sexual imprinting separated,** with their windows named as separate.
- **The bridge to Bowlby.** On a psychiatry paper an ethology answer that never reaches attachment is incomplete.

SEE ALSO Slot I - 89 attachment theory SPRINT Day 5, social, learning and personality psychology

I - 65

Critically evaluate marriage as an institution. [10]

10 SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The institution	What marriage is for, stated as functions
4	The case both ways	Domain by domain, for and against
2	The evidence	What the mental health data support
2	The verdict	A position, defended, not a fence

DOMAIN	THE CASE FOR	THE CASE AGAINST	WHAT THE EVIDENCE SHOWS
Mental health	Less psychiatric morbidity and lower suicide rates among the married	A bad marriage is worse than none	**Protection is larger for men; selection and causation both operate**
Social function	Legitimises children, transmits property, organises care of the old and ill	Concentrates unpaid care on women	The function is real, its distribution is contested
Support in illness	A confiding relationship buffers against depression	The spouse can be the source of the stress	A confidant protects; marriage does not guarantee one
Indian context	Near universal; family support is built in	Dowry, early marriage and domestic violence carry direct harm	Both hold at once and belong in one answer
Stability	Divorce is uncommon	Low divorce is not high satisfaction	Stability and quality are separate measures

The fourth column is what makes this a critical evaluation rather than a list. Amber marks the finding the verdict rests on.

- **The verdict, which is what the verb asks for.** Marriage is associated with better mental health; the association is stronger for men than for women; it is partly selection rather than protection; and the quality of the marriage predicts more than the fact of it. Commit to that and defend it, rather than balancing two lists and stopping.
- **The psychiatric handles.** Marital status is a standard variable in suicide and depression epidemiology, marital discord is a recognised focus of clinical attention, and couple therapy is an evidence-based treatment for depression where discord maintains it.

WHAT EARNS THE MARKS

- **A stated verdict.** Critically evaluate is marked on whether a position was taken and defended.
- **Both sides of at least four domains,** which is what stops the answer becoming an opinion.
- **Selection distinguished from protection.** It is the methodological point that lifts the answer.
- **The Indian material named,** not implied by the setting.

SEE ALSO Slot I - 66 social theories of suicide SPRINT Day 5, social, learning and personality psychology

I - 66

Social theories of suicide. [10]

10 SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The claim	Suicide rates as social facts
4	Durkheim's four	Two axes, each failing in two directions
2	After Durkheim	Status integration, the critique, contagion
2	The limit	Rates against individuals

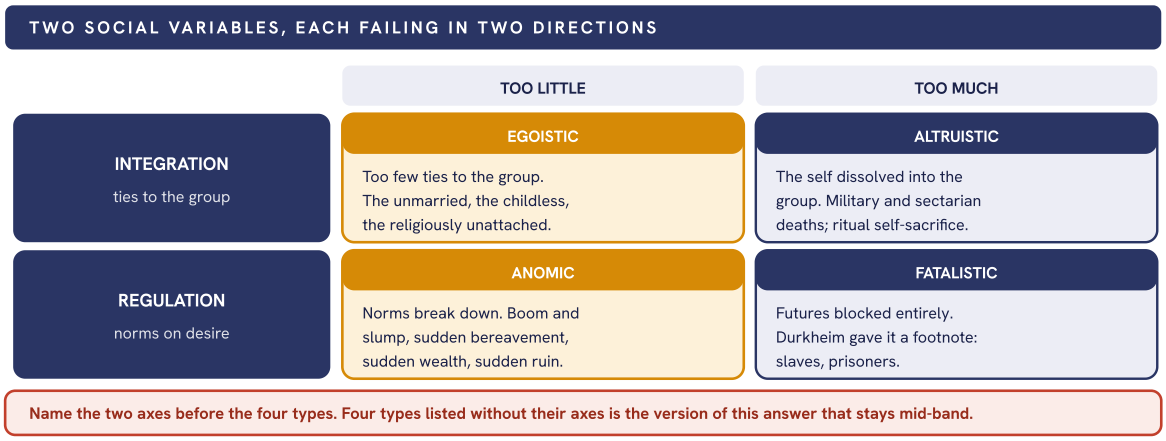

Fig 066.1 Durkheim's typology drawn as one grid: two social variables down the side, too little and too much across the top. Amber marks the two types that carry most of the modern evidence.

AFTER DURKHEIM	THE CLAIM	WHOSE
Status integration	Rates fall where a person's statuses are compatible and role conflict is low	Gibbs and Martin
The statistics critique	Official suicide rates are produced by coroners, so they are social constructions rather than raw facts	Douglas, 1967
Ecological studies	Suicide clusters in socially disorganised districts, whoever lives in them	Sainsbury, 1955
Media contagion	Reported suicides are followed by a rise, the Werther effect; responsible reporting reverses it, the Papageno effect	Phillips, 1974; Niederkrotenthaler, 2010

- **The limit, which is the last two marks.** Social theories explain rates, not individuals. They cannot say which patient will die, and an answer that offers Durkheim as a clinical risk assessment has mistaken the level of explanation.

WHAT EARNS THE MARKS

- **The two axes named before the four types,** and fatalistic included, since it is the type most often dropped.
- **At least two theories after 1897,** with their authors, since the stem says theories rather than Durkheim.
- **The level-of-explanation limit,** stated rather than implied.

SEE ALSO Slot I - 65 marriage as an institution SPRINT Day 5, social, learning and personality psychology

I - 67

Role of culture in Psychiatry: [10]

10 SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

KUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 The claim	Culture at every step, not only at diagnosis
4 The pathway	Each step, with its clinical consequence
2 The instrument	The cultural formulation, and what it asks
2 The two errors	Category fallacy and stereotyping

THE CLAIM TO OPEN WITH

Culture enters at every step of the clinical pathway, not only at the point of diagnosis. It shapes what counts as illness, how distress is spoken, who is consulted first, what treatment is accepted, and what outcome follows.

STEP	WHAT CULTURE DOES THERE	THE CLINICAL CONSEQUENCE
Aetiology	Alters exposure to risk and protection: family structure, migration, discrimination, drinking norms	Risk profiles differ between populations
Expression	Supplies the idioms of distress, and the content of delusion and hallucination	Somatic presentation is the rule in much of Indian practice
Help-seeking	Sets the pathway: faith healer, family elder, general practitioner, then psychiatrist	Long untreated intervals; the first contact is rarely psychiatric
Diagnosis	Decides whether a behaviour reads as normal, deviant or ill	**Risk of category fallacy; the Cultural Formulation Interview exists to check it**
Treatment	Governs adherence, and who makes the decision	Consent and disclosure are family matters more often than individual ones
Outcome	Shapes stigma, family tolerance, and re-entry to work and marriage	The WHO comparative studies reported a better course in developing-country centres

- **The instrument to name.** DSM-5 carries a Cultural Formulation Interview: the cultural definition of the problem, its perceived causes, supports and stressors, cultural factors in coping and past help-seeking, and factors in current help-seeking including the clinician relationship. Its predecessor is the DSM-IV Outline for Cultural Formulation.
- **Two errors, named.** The category fallacy is applying a category built in one culture where it has no coherence. Cultural stereotyping is reading a person as a specimen of a culture instead of asking what this person believes. The second is the commoner clinical error.

WHAT EARNS THE MARKS

- **A pathway, not a list of topics.** The structure is what turns six observations into an argument.
- **The formulation instrument named,** with at least three of the domains it asks about.
- **Both errors, kept apart.** One is a nosological error, the other is an interviewing error.

SEE ALSO Slot I - 69 cultural factors in expression, assessment and treatment Slot I - 63 culture-bound syndromes

SPRINT Day 5, social, learning and personality psychology

I - 68

Religion and Psychiatry. [10]

10 SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The two frames	Religion as a variable, and as a context
4	The evidence	Association, coping, and the boundary tests
2	The boundary	When belief becomes a clinical concern
2	The Indian setting	Faith healing, and how to work with it

DOMAIN	WHAT IS ESTABLISHED	WHAT TO DO WITH IT
Religiosity and outcome	Religious involvement is associated with less depression, less substance use and lower suicide rates; the effects are modest and confounded	Ask about it as a resource, not only as a symptom
Religious coping	Pargament separated positive coping, collaborative and benevolent, from negative coping, punishing God and spiritual struggle	**Negative coping predicts worse outcome; the kind matters, not the amount**
Belief against delusion	A belief is not delusional because it is religious; the test is whether the person's own community shares it and holds it in that manner	The community is the reference, not the clinician
Obsession against devotion	Religious obsessions are intrusive, resisted and unwanted; devout practice is none of these	Ask about resistance and distress, not content
Possession and trance	Classified as dissociative states when involuntary, distressing and outside accepted ritual	Involuntary and unwanted is the threshold

- **The two frames to name at the start.** Religion as a variable, studied for its association with outcome, and religion as a context, which sets what a community counts as normal. An answer with only the first is epidemiology; an answer with only the second is anthropology.
- **The error runs in both directions.** Reading every religious experience as pathology, and refusing to read any of it as pathology. Both substitute a prior position for an assessment.

THE INDIAN SETTING

Temple and dargah healing is a common first contact and often runs in parallel with treatment. Collaboration is more useful than confrontation: attacking the belief system costs the alliance and the adherence, and the family usually holds both frames at once without difficulty.

WHAT EARNS THE MARKS

- **The two frames separated in the opening lines.** It is what organises everything after.
- **The community test for delusion,** stated as a test rather than as a caution.
- **Positive and negative religious coping distinguished.** It is the finding examiners look for.

SEE ALSO Slot I - 67 the role of culture across the clinical pathway SPRINT

Day 5, social, learning and personality psychology

I - 69

Discuss in detail cultural factors involved in expression, assessment and treatment of mental illness in an individual. Add a note on immigration and mental health. [10]

10 | ASKED AT 15 MARKS | SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Expression	Idioms of distress, and what culture supplies
3	Assessment	Whose normality, and the formulation instrument
3	Treatment	Explanatory models, family, and adherence
2	Immigration	The excess, and the mechanism proposed

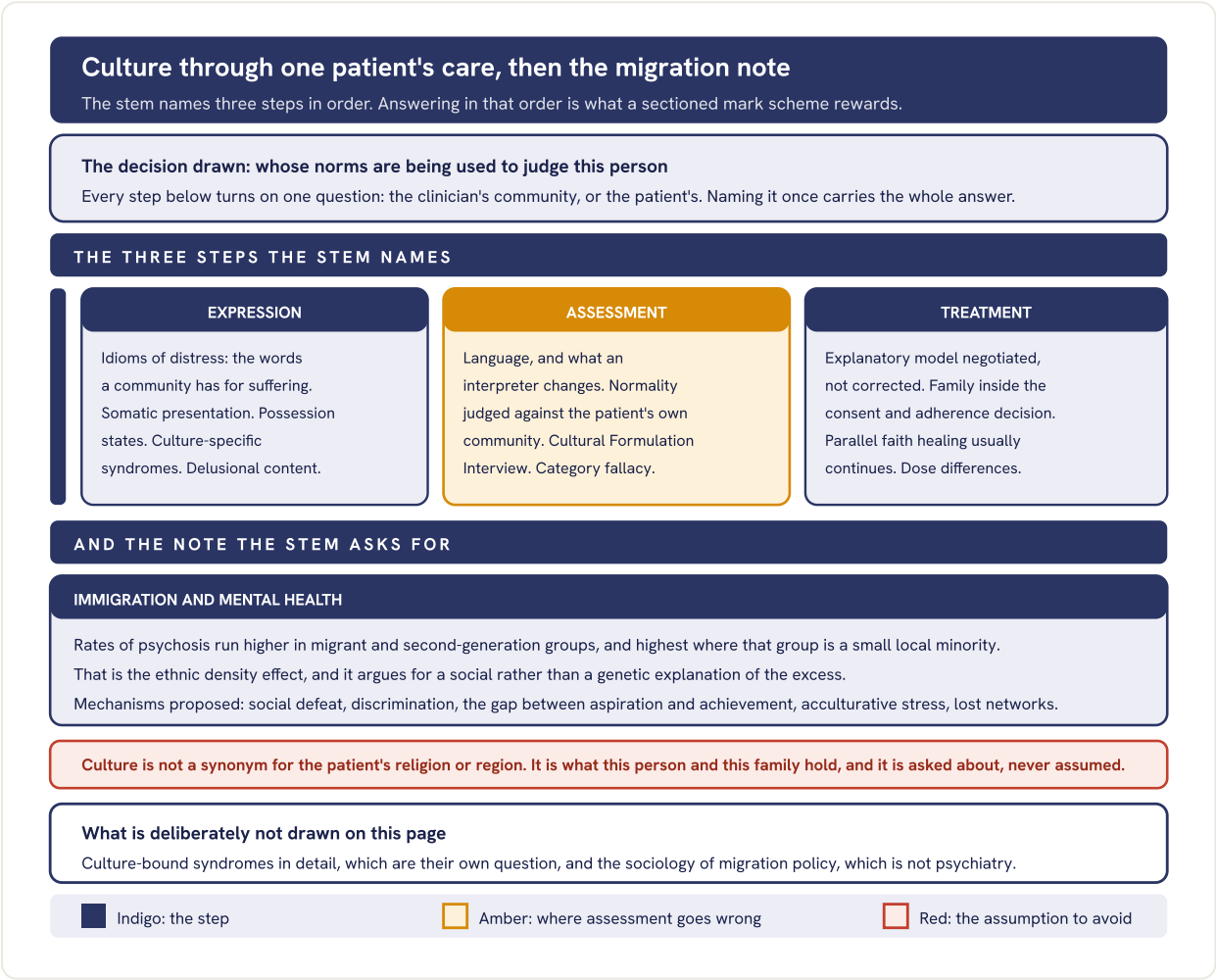

Fig 069.1 The three steps in the stem's own order along one spine, with the migration note as its own band below. Amber marks assessment, where the category fallacy is made.

- **Answer in the stem's order.** Expression, assessment, treatment, then the note. A fifteen-mark version of this question is marked by section, so a well-informed answer in the wrong order still loses marks it has earned.
- **The ethnic density effect, stated carefully.** Rates rise as the proportion of a migrant's own group in the local population falls. It is among the more robust findings in social psychiatry, and it is the sentence that turns a list of migration stressors into an argument.

WHAT EARNS THE MARKS

- **Three sections in the stem's order,** each headed, because the mark scheme is sectioned.
- **The formulation instrument named at the assessment step,** which is where its marks sit.
- **Migration answered as a mechanism,** not as a list of hardships.

SEE ALSO Slot I - 67 the role of culture across the clinical pathway

Slot I - 70 culture, illness behaviour and symptomatology **SPRINT** Day 5, social, learning and personality psychology

I - 70

Define culture. Describe the role of culture in shaping illness behaviour and symptomatology in mental disorders. [2+8]

2 + 8 | SOCIAL, CROSS-CULTURAL AND EVOLUTIONARY PSYCHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Define culture**	Learned, shared, transmitted, changing
4	**How culture acts**	The six effects, named as mechanisms
2	**Illness behaviour**	Mechanic and Parsons, and what each adds
2	**Symptomatology**	What is shaped, and what is not

THE DEFINITION

Culture is the shared, learned and transmitted system of meanings, values and practices by which a group interprets experience. It changes, and it is not inherited.

EFFECT	WHAT CULTURE DOES TO THE DISORDER	EXAMPLE
Pathogenic	Culture causes the disorder by supplying the belief	**Koro and dhat: the belief is the illness**
Pathoselective	Culture selects which reaction is chosen under stress	Amok; family suicide in some societies
Pathoplastic	Culture shapes the content of a universal disorder	Delusional content: technology or spirits
Pathoelaborating	Culture intensifies a universal behaviour into an extreme form	Trance elaborated through ritual
Pathofacilitative	Culture raises the frequency without changing the form	Alcohol use disorders where drinking is normative
Pathoreactive	Culture governs the reaction to the illness and what it is called	Stigma; a psychosis named as possession

The six are Tseng's. Amber marks the only row where culture is a cause, not a shaper.

- **Illness behaviour, defined and attributed.** Mechanic's term for the way symptoms are perceived, evaluated and acted upon, which varies between cultures independently of the disease. Parsons' sick role sets what a society exempts the sick person from, and what it asks in return: to seek help, and to want to get well.
- **The eight marks are the mechanisms, not the examples.** Name the effect, then attach the example. An answer built from colourful syndromes with no typology behind them stays mid-band however many it lists.

WHAT EARNS THE MARKS

- **A definition with all four properties.** Learned, shared, transmitted, changing. Two marks in one sentence.
- **The effects named as effects,** in the technical vocabulary, with one example each.
- **Illness behaviour attributed to Mechanic,** separated from the sick role, and one line on what culture does not change.

SEE ALSO Slot I - 63 culture-bound syndromes Slot I - 69 cultural factors in expression, assessment and treatment

SPRINT Day 5, social, learning and personality psychology

AREA 08 OF 12 · P1-A11

Biostatistics and epidemiology

8	7.6%	I-71	I-78
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 · RECURRENT 1 · SINGLE 6

REVISION ORDER

The two selector slots first and together: they open the other three, and the paired and unpaired t-tests belong inside them. Chi square next, it is the anchor here and it is short. Analysis of variance after that. The two odds-ratio slots are one pair and should be prepared as one. Regression stands alone and is the longest here.

WHAT IS IN THIS AREA

The tests of significance in five slots: the selector question twice, once opened out on the paired test and once on the unpaired, then chi square, the t-test and analysis of variance each alone. The odds ratio in two, once against relative risk and once with confounding, effect modification and mediation. Regression analysis takes the eighth.

I - 71

Chi Square test. [10]

10 BIOSTATISTICS AND EPIDEMIOLOGY

KNRUHS, KUHS, NTRUHS, RGUHS - 5 APPEARANCES, 5 OF 78 SESSIONS

SKELETON

2 What it is	Observed against expected, for categorical data
4 The working	Expected counts, the statistic, the degrees of freedom
2 Assumptions	And the exact test that replaces it when they fail
2 Its three uses	Association, goodness of fit, homogeneity

THE DEFINITION AND THE FORMULA

A non-parametric test of whether two categorical variables are associated, built entirely on counts. The statistic is the sum over all cells of observed minus expected, squared, divided by expected. Each expected count is its row total times its column total, divided by the grand total, and the degrees of freedom are rows minus one times columns minus one.

CELL, ILLUSTRATIVE COUNTS	OBSERVED	EXPECTED	CONTRIBUTION
Adherent, relapsed	20	30	3.33
Adherent, no relapse	80	70	1.43
Not adherent, relapsed	40	30	3.33
Not adherent, no relapse	60	70	1.43
Total	200	200	Chi square 9.52, one degree of freedom

Made-up counts, arithmetic real. Row totals 100 and 100, column totals 60 and 140, so every expected count is 30 or 70. At one degree of freedom the statistic clears both the 3.84 cut at five per cent and the 6.63 cut at one per cent.

- **Assumptions.** Independent observations, raw counts and never percentages, and adequate expected counts: in a two by two no expected cell below five. When that fails, use Fisher's exact test. Paired categorical data need McNemar's test, not this one.
- **Its three uses.** Test of association between two categorical variables; goodness of fit against an expected distribution; and test of homogeneity of a proportion across several groups.
- **What it does not tell you.** A significant result says the variables are associated, not how strongly and not in which direction. Report an effect measure, an odds ratio or a relative risk, alongside it.

WHAT EARNS THE MARKS

- **The expected-count formula written out,** not just the chi square formula.
- **Degrees of freedom derived from the table shape,** with the number stated.
- **The expected-count-below-five rule with Fisher's exact test as the remedy.**
- **The three uses separated,** and McNemar named for paired data.

SEE ALSO Slot I - 74 tests of significance Slot I - 78 odds ratio and relative risk SPRINT

Day 7, research methods, biostatistics and epidemiology

I - 72

Enumerate various statistical tests used to measure the differences. Describe the paired t-test in detail with an example. Describe any two non-parametric tests used for group difference. [4+2+4]

4 + 2 + 4 | BIOSTATISTICS AND EPIDEMIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4	The enumeration	Drawn as a selector, by data type and pairing
3	Paired t-test	What is tested, the statistic, an example
2	Two non-parametric	Mann-Whitney and Wilcoxon, and what each replaces
1	The choosing rule	Pairing first, then distribution

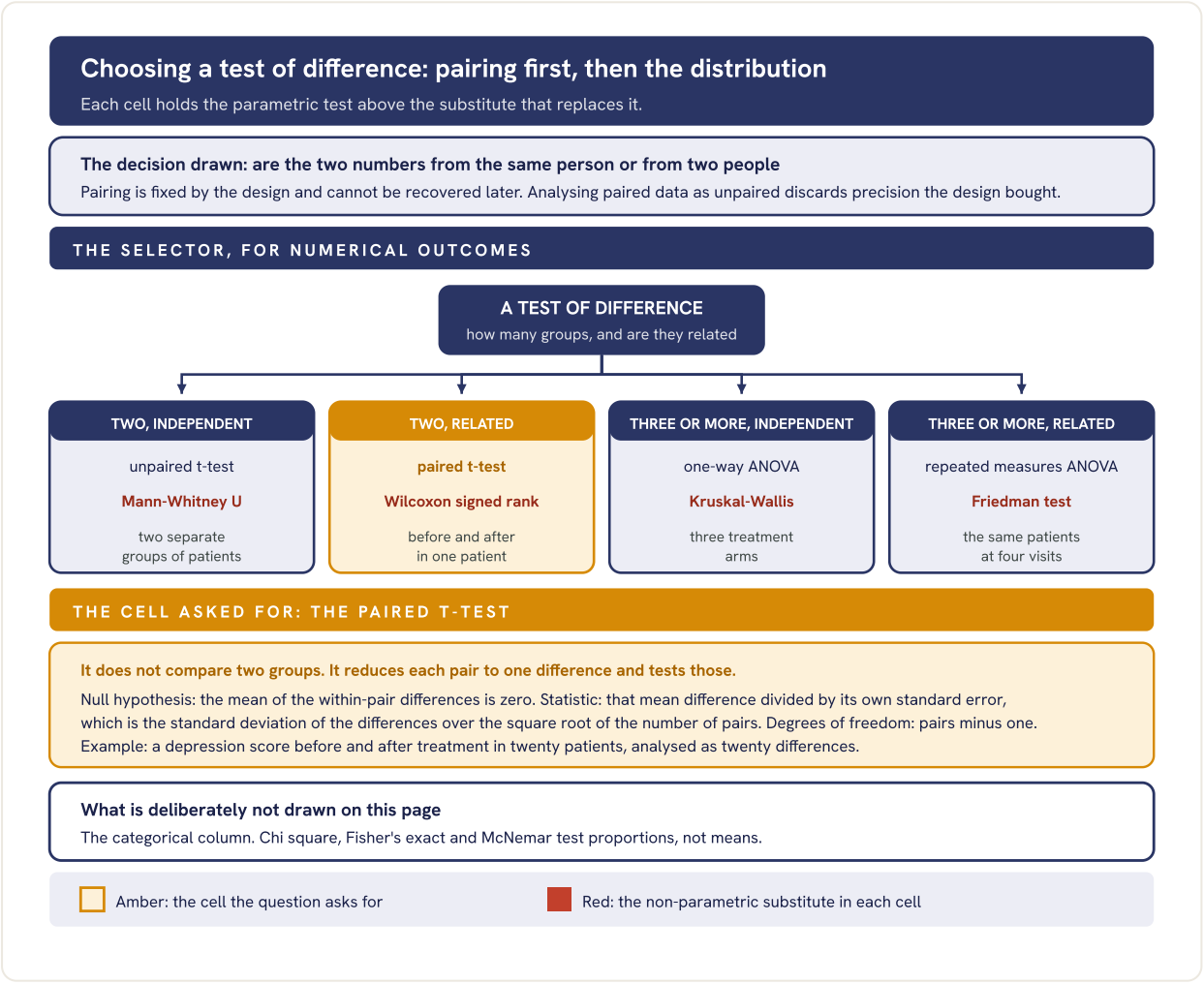

Fig 072.1 The difference tests as a two-way selector, groups against pairing, with the paired cell opened out below.

- **The two non-parametric tests.** Mann-Whitney U pools the observations, ranks them, and asks whether high ranks cluster in one group; it replaces the unpaired t-test. Wilcoxon signed rank ranks the absolute within-pair differences and compares positive against negative rank sums; it replaces the paired t-test.
- **The choosing rule.** Settle pairing from the design first, then distribution. Non-parametric tests need no normality, cost a little power when it does hold, and answer a question about ranks rather than means.

WHAT EARNS THE MARKS

- **The enumeration drawn as a selector,** so each test arrives with the situation it belongs to.
- **The paired test described as a one-sample test on differences,** which is what it actually is.
- **Degrees of freedom given as pairs minus one,** not as total observations minus two.
- **Each non-parametric test matched to the parametric one it replaces.**

SEE ALSO Slot I - 76 the unpaired t-test worked through Slot I - 77 analysis of variance **SPRINT** Day 7, research methods, biostatistics and epidemiology

I - 73

What is odd's ratio? Write briefly about confounding, effect modification and mediation. [4+2+2+2]

4 + 2 + 2 + 2 | BIOSTATISTICS AND EPIDEMIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4	Odds ratio	Definition, formula, design, interpretation
2	Confounding	A distortion, and how it is removed
2	Effect modification	A real finding, reported not removed
2	Mediation	On the causal path, so never adjusted away

THE DEFINITION

The odds ratio is the odds of the outcome among the exposed divided by the odds among the unexposed, computed from a two by two table as a times d over b times c. One means no association, above one a positive association, below one a protective one. It is the measure a case-control study can produce, because such a study fixes the number of cases and so cannot yield a risk. Read it with its confidence interval: an interval containing one is not a significant result.

QUESTION	CONFOUNDING	EFFECT MODIFICATION	MEDIATION
Where it sits	Beside the exposure, linked to both exposure and outcome	Beside the exposure, changing how strongly it acts	**On the causal path, between exposure and outcome**
What it does	Distorts the estimate, up or down	Splits one true estimate into two real ones	Carries part of the true effect
How you find it	The estimate moves when you adjust for it	**The stratum estimates differ from each other**	The effect shrinks when you adjust, and that is expected
What you do	Remove it: restrict, match, randomise, or adjust	Report it: give the estimate stratum by stratum	**Do not adjust it away if the total effect is the question**

Amber marks the discriminators. One question separates all three: is the third variable on the causal path, beside it, or changing its strength.

- **The one-line test.** Adjusting for a confounder corrects the answer. Adjusting for a mediator destroys it. Adjusting for an effect modifier hides the finding, because the finding is that the effect differs.
- **Where it goes wrong.** Randomisation controls confounding by variables you never measured, which is its whole advantage; adjustment controls only what you thought to record. Effect modification is often reported as a subgroup result without a formal interaction test, which is how spurious subgroup claims enter the literature.

WHAT EARNS THE MARKS

- **The formula written as a times d over b times c,** with the design it comes from named.
- **The confidence interval mentioned** in the same breath as the point estimate.
- **All three third variables told apart by position,** on the path or beside it.
- **The remove, report, do-not-adjust triad,** which is the whole second half of the question.

SEE ALSO Slot I - 78 odds ratio against relative risk Slot I - 80 internal and external validity SPRINT Day 7, research methods, biostatistics and epidemiology

I - 74

Discuss different types of test of significance in research. [10]

10 | BIOSTATISTICS AND EPIDEMIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The axis	Data type, number of groups, pairing, distribution
5	The selector	Parametric and non-parametric, side by side
2	Assumptions	What a parametric test demands before it is legal
1	Reading the result	What a p value is, and what it is not

WHAT IS COMPARED	PARAMETRIC TEST	NON-PARAMETRIC TEST	DATA NEEDED
Two independent groups	Unpaired t-test	Mann-Whitney U	Numerical
Two related measures	Paired t-test	Wilcoxon signed rank	Numerical
Three or more independent	One-way analysis of variance	Kruskal-Wallis	Numerical
Three or more related	Repeated measures analysis of variance	Friedman	Numerical
Two proportions, independent	Z test for proportions	Chi square, or Fisher's exact when cells are sparse	Categorical
Two proportions, paired	Not applicable	McNemar	Categorical
Association of two numerical variables	Pearson correlation	Spearman rank correlation	Numerical

Amber marks the categorical rows, because they are where a candidate who has memorised the t-test family runs out.

- **What a parametric test assumes.** Numerical data on an interval scale, approximately normally distributed, with comparable variances across groups, and independent observations. Fail any of these and the non-parametric column is not a compromise, it is the correct test.
- **Reading the result.** A p value is the probability of a result at least this extreme if the null hypothesis were true. It is not the probability that the null is true, and it says nothing about the size of the effect. Report the effect with its confidence interval alongside it.
- **The two errors.** A type one error rejects a true null, its rate set by the significance level. A type two error misses a real effect, its rate falling as power rises. Multiple testing inflates the first, which is why a correction or a pre-specified primary outcome is required.

WHAT EARNS THE MARKS

- **A selector, not a list.** Every test arrives attached to the comparison it serves.
- **Parametric and non-parametric paired row by row,** rather than in two separate lists.
- **The four parametric assumptions named.**
- **The p value defined correctly,** which is the single most commonly lost mark on this question.

SEE ALSO Slot I - 72 the selector drawn, and the paired test Slot I - 71 the chi square test SPRINT Day 7, research methods, biostatistics and epidemiology

I - 75

What is regression analysis? In which situations it is indicated? Discuss giving examples, how multiple regression analysis is conducted. [2+2+6]

2 + 2 + 6 | BIOSTATISTICS AND EPIDEMIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	Modelling one outcome from one or more predictors
2 When it is indicated	Predict, adjust, quantify a dose-response
5 How it is conducted	Specify, check, fit, report, in that order
1 The caution	Association modelled is not causation shown

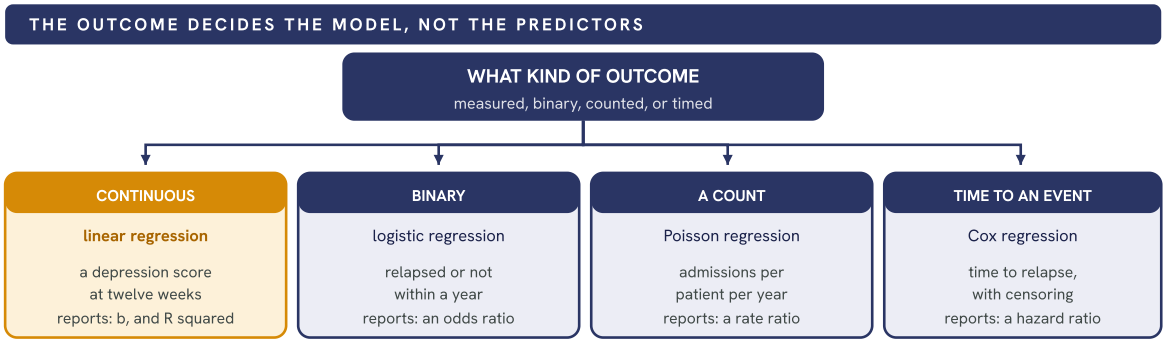

Fig 075.1 The regression family selected on the type of the outcome variable, each branch naming the effect measure it reports, with the linear case in amber as the one the question opens with.

HOW A MULTIPLE REGRESSION IS ACTUALLY CONDUCTED

Specify the outcome and choose predictors from clinical reasoning, never from a screening of p-values. Check the assumptions: a linear relation, independent residuals with constant spread and roughly normal distribution, and no severe collinearity between predictors. Keep enough data for the model, about ten observations for every predictor entered. Fit the model, entering variables together or in pre-planned blocks. Report each adjusted coefficient with its confidence interval, and the variance explained.

- **When it is indicated.** To predict an outcome from several variables, to adjust an exposure effect for measured confounders, and to quantify a dose-response relation across a range of exposure.
- **Two cautions.** Correlation quantifies the strength of a relation and makes no distinction between the two variables; regression names one as outcome and predicts it. And a stepwise search over many candidate predictors overfits, producing a model that does not replicate.

WHAT EARNS THE MARKS

- **The outcome type named as the thing that selects the model.**
- **Each model tied to the effect measure it reports,** coefficient, odds ratio, rate ratio, hazard ratio.
- **Assumptions checked before fitting,** written as a step of the method.

SEE ALSO Slot I - 73 confounding, effect modification and mediation Slot I - 83 Firth's penalized logistic regression

SPRINT Day 7, research methods, biostatistics and epidemiology

I - 76

Enumerate various statistical tests used to measure the differences. Describe the t-test in detail with an example. [4+6]

4 + 6 | BIOSTATISTICS AND EPIDEMIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 **Enumerate**	By pairing and by number of groups, in one line
2 **What the t-test is**	A signal to noise ratio, with its three forms
4 **The worked example**	Pooled spread, standard error, t, degrees of freedom
1 **Assumptions**	And the substitute when they fail

THE ENUMERATION, AND THE THREE FORMS OF THE T-TEST

Two independent groups take the unpaired t-test or Mann-Whitney U; two related measures take the paired t-test or Wilcoxon signed rank; three or more groups take analysis of variance or Kruskal-Wallis; proportions take chi square, Fisher's exact or McNemar. The t-test itself has three forms: one sample against a known value, two independent samples, and paired. Each is a difference divided by the standard error of that difference.

STEP	WORKING, ON ILLUSTRATIVE DATA	VALUE
The data	Ten patients per arm; means 18.0 and 22.0, standard deviations 4.0 and 5.0	n equals 10 and 10
Difference in means	22.0 minus 18.0	4.00
Pooled variance	Nine times 16, plus nine times 25, all over 18	20.50
Pooled standard deviation	The square root of 20.50	4.53
Standard error of the difference	4.53 times the square root of one fifth	2.02
The statistic and the verdict	4.00 divided by 2.02, on 18 degrees of freedom, against a two-sided cut of 2.10	**t equals 1.98, not significant**

Made-up data, arithmetic real. A four-point difference that looks clinically worth having fails to reach significance at this spread and this sample size, which is the lesson the example is here to teach.

- **Read the failure correctly.** Not significant does not mean no difference. It means this study could not distinguish the observed difference from chance, which is a statement about the study's power as much as about the treatment.
- **Assumptions.** Numerical outcome, roughly normal within each group, comparable variances, independent observations. Unequal variances take the Welch correction; non-normality with small samples takes Mann-Whitney U instead.

WHAT EARNS THE MARKS

- **The enumeration organised by pairing and group number,** not as an unsorted list.
- **The three forms of the t-test named,** which most answers reduce to two.
- **Arithmetic shown line by line** to the degrees of freedom and the critical value.
- **A verdict written in words,** including what a non-significant result does not prove.

SEE ALSO Slot I - 72 the selector drawn, and the paired t-test Slot I - 77 analysis of variance **SPRINT** Day 7, research methods, biostatistics and epidemiology

I - 77

Analysis of Variance (ANOVA). Its role in research in behavioral Medicine. [10]

10 | ASKED AT 7 MARKS | BIOSTATISTICS AND EPIDEMIOLOGY

TN-MGRMU - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 What it is	Variance partitioned, then compared as a ratio
3 The working	The F ratio and its two degrees of freedom
2 Its forms	One-way, two-way, repeated measures, covariance
3 Role in the field	Where a behavioural trial needs it, and post hoc

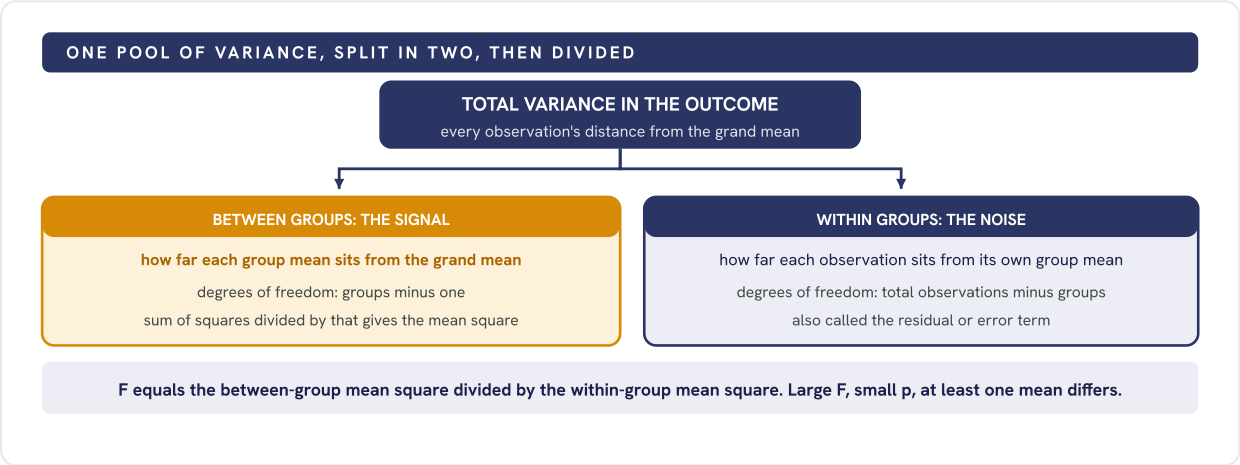

Fig 077.1 Analysis of variance drawn as what its name says: the total variation split into a between-group part and a within-group part, the ratio of the two being the test statistic.

- **Why not repeated t-tests.** Three comparisons at five per cent each carry about a fourteen per cent chance of a false positive. One F test holds it at five.
- **What a significant F does not say.** Not all means are equal, never which pair differs. Only then a post hoc test: Tukey, Bonferroni or Scheffe.
- **Its forms and its assumptions.** One-way for one factor; two-way for two, which makes an interaction testable; repeated measures over time; covariance to adjust for baseline. It assumes independence, normal residuals, comparable variances.

ITS ROLE IN BEHAVIOURAL RESEARCH

Behavioural work rarely compares two things once. It compares three or more arms, or the same participants at baseline, end of treatment and follow-up. Two-way and repeated measures forms are what let a trial test an interaction.

WHAT EARNS THE MARKS

- **The partition named,** between-group against within-group, before the F ratio.
- **Both degrees of freedom given,** groups minus one and observations minus groups.
- **The inflated error rate quantified,** not just asserted.
- **Interaction named as what two-way analysis buys.**

SEE ALSO Slot I - 74 tests of significance Slot I - 76 the t-test worked through SPRINT
Day 7, research methods, biostatistics and epidemiology

I - 78

What is odds ratio? How does it differ from relative risk? Describe the differences giving examples. [2+8]

2 + 8 | BIOSTATISTICS AND EPIDEMIOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Both, defined**	Odds against risk, and the table both come from
4	**The grid**	Design, denominator, meaning, behaviour
3	**Worked both ways**	A common outcome, then a rare one
1	**The rule**	When the two may be spoken of as one

DOMAIN	RELATIVE RISK	ODDS RATIO	WHAT SETTLES IT
What it divides	Risk in the exposed by risk in the unexposed	Odds in the exposed by odds in the unexposed	**Risk uses everyone as denominator, odds uses the non-affected**
Design it comes from	Cohort study or randomised trial	Case-control, and also cohort or trial	**A case-control fixes the cases, so it can yield no risk**
How it is read aloud	Directly: twice as likely	As odds, which is not the same as likely	Only relative risk may be spoken as times the chance
Common outcome	Stays interpretable	Sits further from one than the risk ratio does	**Above about ten per cent, the two separate**
Rare outcome	Close to the odds ratio	Approximates the relative risk	The rare disease assumption

WORKED TWICE, ON ILLUSTRATIVE COUNTS

Common outcome. Of 100 exposed, 30 develop the outcome; of 100 unexposed, 15 do. Risks are 0.30 and 0.15, so the relative risk is 2.00. Odds are 30 over 70 and 15 over 85, so the odds ratio is 2.43. The odds ratio has overstated the risk ratio. Rare outcome. Of 1000 exposed, 3 develop it; of 1000 unexposed, 1 does. The relative risk is 3.00 and the odds ratio is 3.01. Now they agree.

- **The rule the two examples prove.** The odds ratio approximates the relative risk only when the outcome is uncommon. When the outcome is common, the odds ratio exaggerates the effect in whichever direction it points, and reporting it as though it were a risk ratio overstates the finding.
- **Where this bites in practice.** Logistic regression returns odds ratios whatever the frequency of the outcome, so an adjusted odds ratio from a study of a common outcome is routinely read as a risk ratio and routinely overstated. Say odds, and give the confidence interval.

WHAT EARNS THE MARKS

- **Odds distinguished from risk in the first two lines,** by what sits in the denominator.
- **The design constraint stated,** why a case-control study cannot report a risk.
- **Numbers on the page.** The stem asks for examples, so the two worked sets are the examples.
- **The rare disease assumption named as an assumption,** with the condition under which it fails.

SEE ALSO Slot I - 73 the odds ratio and confounding Slot I - 75 regression and the measures it reports SPRINT Day 7, research methods, biostatistics and epidemiology

AREA 09 OF 12 · P1-A10

Research methodology

6	5.3%	I-79	I-84
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 5

REVISION ORDER

Reliability and validity first, three of the other five lean on it. Sampling and sample size next. The qualitative slot and the research-question slot after that, both of them arguments rather than lists. The survey slot and the registry slot are independent, and each carries a second half that has to be prepared separately from the first.

WHAT IS IN THIS AREA

Qualitative research set against quantitative. Reliability with its types, and internal against external validity. Sampling with the principles of sample-size calculation for two designs. The research question with the types of hypothesis and a critique of meta-analysis and network analysis. The National Mental Health Survey with Firth's penalised logistic regression. The clinical trials registry with the steps to be completed before a trial opens.

I - 79

What do you understand about qualitative research? How does it differ from quantitative research in terms of essential features, strength and limitations? [2+3+3+2]

2 + 3 + 3 + 2 | RESEARCH METHODOLOGY | DNB - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	**Both, defined**	Meaning and process against magnitude and frequency
4	**The grid**	Question, sampling, data, analysis, rigour
2	**Strengths and limits**	What each buys and what each cannot say
2	**Choosing**	The question decides, and mixed methods

DOMAIN	QUALITATIVE	QUANTITATIVE	WHAT SETTLES IT
The question	What is it like, how does it work, why does it happen	How many, how much, how strongly associated	**Does the question ask for meaning or for a number**
Sampling	Purposive, small, continued until saturation	Probability based, size fixed by a power calculation	**Saturation against a power calculation**
Data	Interviews, focus groups, observation, documents	Measurements, scores, counts	Words against numbers
Analysis	Coding into themes, framework analysis, grounded theory	Statistical tests and effect estimates	Interpretive against inferential
Rigour	Credibility, transferability, dependability, confirmability	Internal validity, external validity, reliability, precision	**Trustworthiness against validity**
Output	Theory and thick description, transferable not generalisable	A population estimate with a confidence interval	Whether a number can be quoted from it

Amber cells are the discriminators an examiner is checking for. The rest of the column supports them.

- **Strengths of the qualitative arm.** It reaches what a questionnaire cannot: illness explanation, treatment refusal, stigma, family decision-making. It generates the hypothesis that a later trial will test, and it explains why a trial that worked elsewhere failed here.
- **Its limits, stated honestly.** No prevalence, no effect size, no generalisation to a population. It is labour intensive, and the analyst is part of the instrument, which is why reflexivity, triangulation, member checking and an audit trail are demanded in its place.
- **Mixed methods.** Sequential where qualitative work builds the instrument a survey then uses, or explains a trial result; concurrent where both run inside one study and are integrated at interpretation.

WHAT EARNS THE MARKS

- **A grid, not two paragraphs.** The comparison has to be readable across a row.
- **Saturation named against power.** It is the sampling discriminator and most answers miss it.
- **The four trustworthiness criteria named,** rather than a claim that qualitative work has no rigour standard.
- **Mixed methods offered as the resolution,** not as a hedge.

SEE ALSO Slot I - 80 reliability and validity Slot I - 81 sampling and sample size SPRINT

Day 7, research methods, biostatistics and epidemiology

I - 80

What is reliability? Discuss the different types of reliability with examples. What is internal validity and external validity of a study? [2+4+4]

2 + 4 + 4 | RESEARCH METHODOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Reliability defined	Consistency, and how it differs from accuracy
4	The four types	Each with its statistic and one example
2	Internal validity	Selection, information, confounding
2	External validity	Generalisability, and the trade-off between the two

THE DEFINITION

Reliability is the consistency of a measurement: the same instrument on the same unchanged subject gives the same answer again. Validity is accuracy, whether the instrument measures what it claims. A scale that always reads five kilograms heavy is perfectly reliable and wholly invalid, which is why reliability is necessary and never sufficient.

TYPE	WHAT IT ASKS	STATISTIC	EXAMPLE
Test-retest	Is the score stable over time in a stable subject	Correlation coefficient	The same scale re-given after two weeks
Inter-rater	Is the score independent of who rates it	Cohen's kappa for categories	Two clinicians rating one interview
Internal consistency	Do all items measure one construct	Cronbach's alpha, split-half	Odd items correlated with even items
Parallel forms	Are two versions interchangeable	Correlation between forms	Two equivalent versions given in turn

Split-half correlations are corrected upward by the Spearman-Brown formula; alpha is the average of all possible split halves.

- **Internal validity.** Whether the association observed inside this study is real for the people studied. It is threatened by selection bias, information and measurement bias, confounding, and chance. Randomisation, blinding, standardised measurement and adjustment are the defences.
- **External validity.** Whether the finding transfers to other patients, settings and times. It is judged from the sampling frame, the inclusion and exclusion criteria, the setting and the comparator used.
- **The trade-off.** They pull against each other. A tightly criteria-bound trial buys internal validity by excluding the comorbid, elderly and substance-using patients who fill an actual clinic, and pays for it in external validity. Internal validity is judged first: a biased study does not generalise, it only travels.

WHAT EARNS THE MARKS

- **Reliability separated from validity in the first two lines,** with the consistently-wrong example.
- **Four types, each with a statistic and an example.** The statistic is the mark most answers drop.
- **Internal validity given as its three named threats,** not as a definition alone.
- **The trade-off stated,** with internal validity judged first.

SEE ALSO Slot I - 49 psychometric properties of a scale Slot I - 79 qualitative and quantitative research SPRINT Day 7, research methods, biostatistics and epidemiology

I - 81

Describe different type of sampling in psychiatric research. What is the sample size? Describe principle and essential components of sample size calculation for any two types of research designs? [2+2+6]

2 + 2 + 6 | RESEARCH METHODOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 The sampling tree	Probability against non-probability, named methods
2 Sample size	The smallest n that can answer the question
4 Two designs	A prevalence survey and a two-arm trial
1 Inflation	Attrition, and the cluster design effect

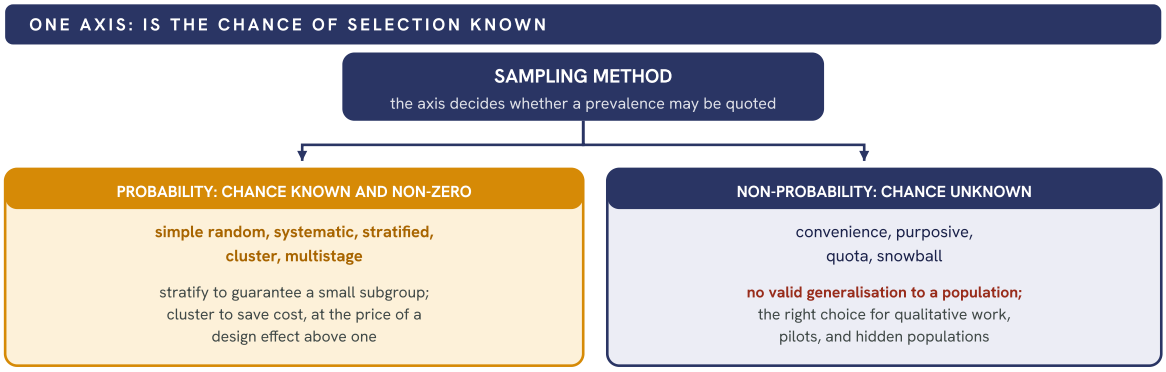

Fig 081.1 Sampling divided on the one axis that matters, whether the probability of selection is known, with the branch that permits a population estimate in amber.

TWO DESIGNS, THE COMPONENTS SPELLED OUT

A prevalence survey needs the expected proportion, the absolute precision wanted either side of it, and the confidence level, giving the familiar form n equals Z squared times p times one minus p, divided by d squared. A two-arm trial comparing means needs the smallest difference worth detecting, the standard deviation of the outcome, the significance level, usually five per cent two-sided, and the power, usually eighty or ninety per cent.

- **The principle.** Sample size is the smallest number that gives an acceptable chance of detecting an effect that is real and worth detecting. It is fixed before recruitment, from the primary outcome only, and never revised because the result was disappointing.
- **Then inflate it.** Add for expected attrition, and multiply by the design effect in any cluster or multistage design. An unadjusted cluster sample is over-precise and its confidence intervals are too narrow.

WHAT EARNS THE MARKS

- **The axis stated before the list,** known against unknown probability of selection.
- **Power and significance level both named,** with their conventional values.
- **The design effect,** which separates a competent answer from a memorised formula.

SEE ALSO Slot I - 79 qualitative and quantitative research Slot I - 82 research question and hypothesis SPRINT Day 7, research methods, biostatistics and epidemiology

I - 82

How to generate research question? Describe different types of hypothesis in research. Discuss advantages and disadvantages of meta analysis and network analysis. [2+2+6]

2 + 2 + 6 | RESEARCH METHODOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Generating the question	Where it comes from, then the two frameworks
2	Types of hypothesis	Null and alternative, directional, simple, complex
4	Meta-analysis	What pooling buys, and what it cannot repair
2	Network meta-analysis	Indirect comparison, and the assumption it rests on

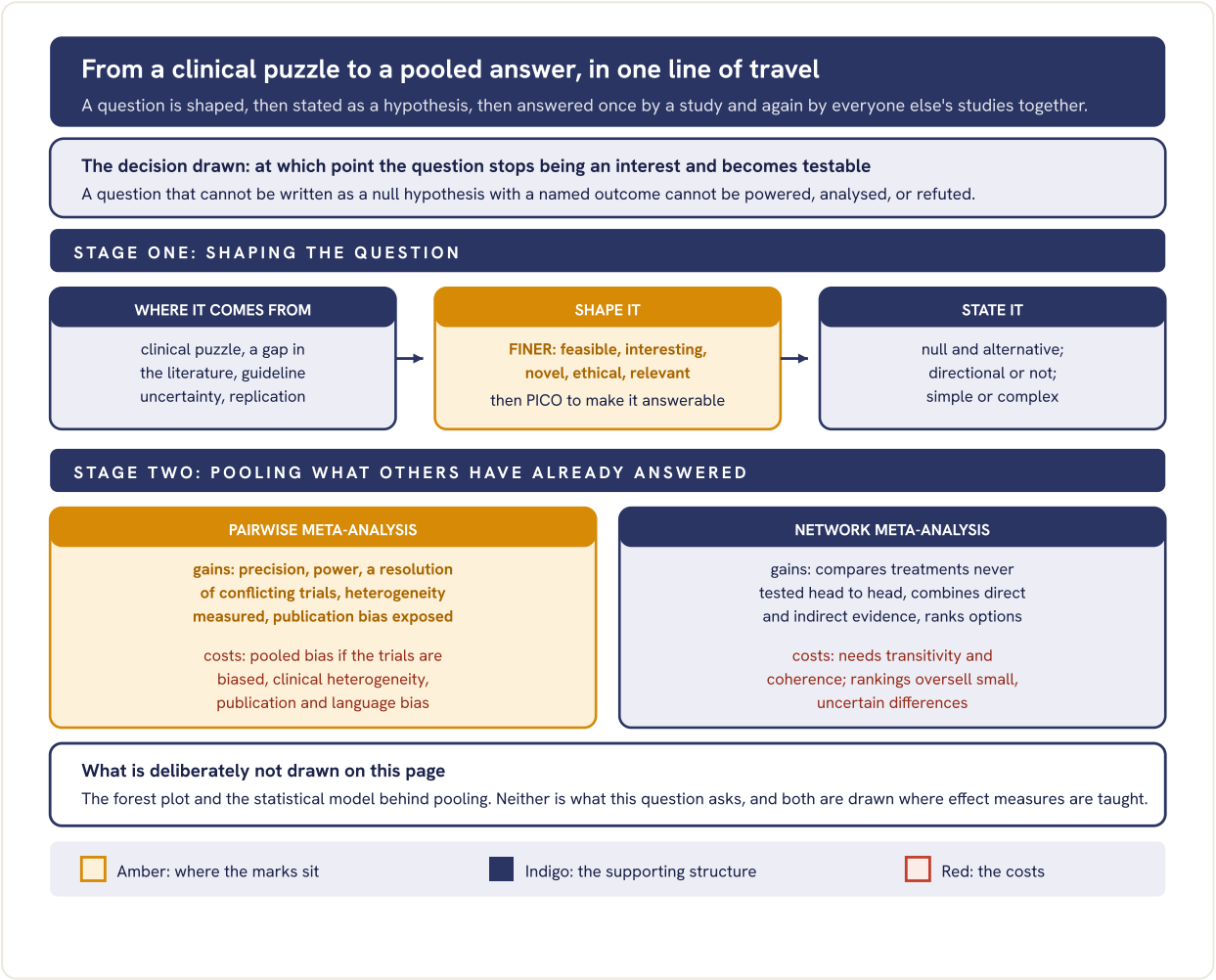

Fig 082.1 The question shaped and stated in the upper band, then answered a second time by synthesis in the lower band, with gains and costs kept apart inside each method.

- **The hypothesis types.** Null against alternative; directional, tested one-sided, against non-directional, tested two-sided; simple, one predictor and one outcome, against complex, several of either; and the research hypothesis, written in words, against the statistical hypothesis, written about parameters.
- **The other reading.** If the examiner means symptom network analysis rather than network meta-analysis, say so and answer briefly: symptoms modelled as nodes with partial correlations as edges, with central symptoms proposed as treatment targets, and stability of the estimated network the standing criticism.

WHAT EARNS THE MARKS

- **A named framework for shaping the question,** not a paragraph on curiosity.
- **Null and alternative written out,** with one-sided and two-sided attached to directionality.
- **Gains and costs kept in separate lists for each method,** as the plate keeps them.
- **Transitivity named** as the assumption a network analysis stands or falls on.

SEE ALSO Slot I - 81 sampling and sample size Slot I - 84 the clinical trials registry and pre-trial steps **SPRINT** Day 7, research methods, biostatistics and epidemiology

I - 83

Discuss various findings of National Mental Health Survey of India 2016. What is Firth's penalized logistic regression used in this survey? [6+4]

6 + 4 | RESEARCH METHODOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What the survey was	Design and scope, before any number
4	The findings	Burden, the treatment gap, what it cannot say
3	Firth's method	The problem it solves in a rare-outcome model
1	Why it matters here	Rare disorders in small subgroups

WHAT IS ASKED	WHAT THE SURVEY REPORTED	WHY IT MATTERS
Design and scope	A cross-sectional multistage stratified random household survey across twelve states, run in 2015 to 2016	**Known selection probabilities, so a national estimate is legitimate**
Current burden	Weighted current prevalence of any mental disorder about 10.6 per cent	Roughly one adult in ten is ill now
Lifetime burden	Lifetime prevalence near 13.7 per cent	The denominator for any service projection
Treatment gap	**Above 80 per cent overall, and widest for substance use**	The policy headline, and the reason the survey is quoted at all
What it cannot say	These are prevalence figures, a stock of existing cases	Prevalence is roughly incidence times duration, so it cannot show whether new cases are rising

FIRTH'S PENALIZED LOGISTIC REGRESSION

Ordinary logistic regression fits by maximum likelihood, which is biased in small samples and breaks outright under separation, where a predictor perfectly or almost perfectly divides the outcome and the estimated odds ratio runs to infinity with an unusable standard error. Firth's method adds a penalty term to the likelihood, an invariant prior on the parameters, which removes most of the small-sample bias and always returns a finite estimate with a usable interval.

- **Why a prevalence survey reaches for it.** Individual disorders are uncommon and subgroup cells are small, so a model of a rare disorder against a rare exposure can easily contain a zero cell. The stem states the survey used it; what it buys, in general, is a finite and interpretable odds ratio where the ordinary fit would give none.
- **Read the survey as a design, not a table.** The examinable point is that this design, cross-sectional, multistage and probability sampled, is what makes those percentages quotable at all. A convenience sample of clinic attenders could not have produced a national figure whatever its size.

WHAT EARNS THE MARKS

- **The design named before the numbers,** cross-sectional and multistage.
- **Current and lifetime prevalence given as two separate figures,** with the treatment gap third.
- **Separation named** as the specific failure Firth's penalty repairs.
- **The prevalence-is-not-incidence line,** which is the honest limit of the whole dataset.

SEE ALSO Slot I - 81 sampling and sample size Slot I - 75 regression analysis SPRINT

Day 7, research methods, biostatistics and epidemiology

I - 84

What is clinical trial registry of India? Describe its role in research. What are the steps to be followed before starting a clinical trial in your hospital? [2+3+5]

2 + 3 + 5 | RESEARCH METHODOLOGY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What it is	Who runs it, since when, and its standing
3	Its role	Transparency, selective reporting, publication
4	The steps	Protocol, ethics, regulator, registration, consent
1	The line	Nothing is enrolled before all of it is done

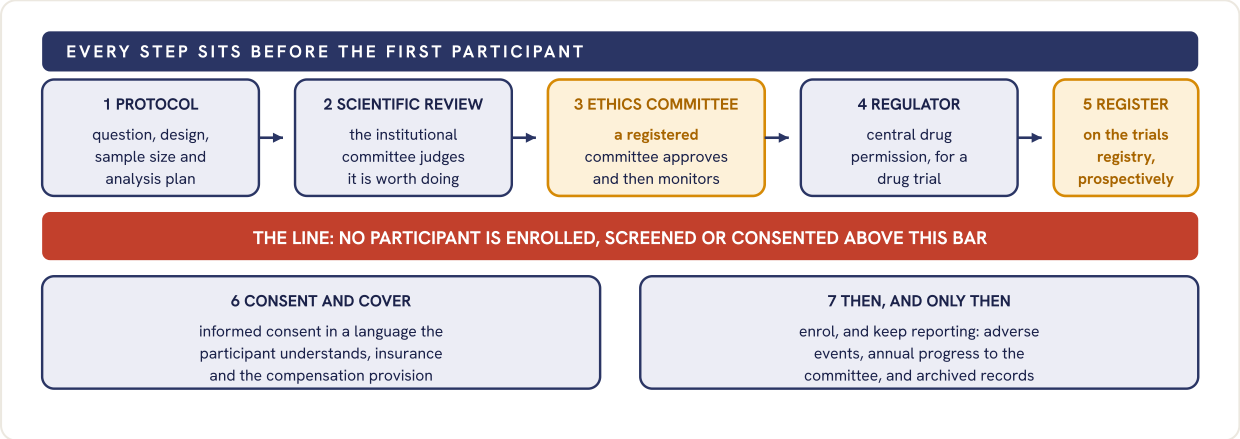

Fig 084.1 The pre-trial sequence with the enrolment bar in red, because the marks in the second half of this question are for putting each step on the correct side of it.

- **What the registry is.** The Clinical Trials Registry of India, launched in 2007 and run by the national medical statistics institute under the Indian Council of Medical Research. It is a primary registry of the World Health Organization platform, and prospective registration has been mandatory since June 2009.
- **Its role.** A public record of what was planned before any result existed, which makes selective outcome reporting and quiet abandonment visible, and which journals now require.
- **The Indian frame.** Human research is governed by the national ethical guidelines of 2017, a registered ethics committee being mandatory; a drug trial adds the new drugs and clinical trials rules of 2019.

PITFALL

Registering after recruitment has begun. Retrospective registration defeats the purpose of a registry, is refused by most journals, and cannot be repaired later.

WHAT EARNS THE MARKS

- **The registry placed institutionally,** who runs it and under whose platform.
- **Prospective registration named as the rule,** with the reason it exists.
- **Steps in order with the enrolment line drawn,** and both the 2017 guidelines and the 2019 rules cited for a drug trial.

SEE ALSO Slot I - 82 research question and hypothesis Slot I - 81 sampling and sample size SPRINT
Day 7, research methods, biostatistics and epidemiology

AREA 10 OF 12 · P1-A06

Developmental psychology and attachment

6	5.1%	I-85	I-90
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 2 SINGLE 4

REVISION ORDER

The three Erikson slots together and in that order: the full life cycle first, then the childhood stages, then the biographical slot, which the first two have already written most of. Piaget next, and it is the highest-scoring question in this book. Attachment theory and theory of mind last, and both are short.

WHAT IS IN THIS AREA

Erikson in three slots: the eight stages of the life cycle, the childhood half of them, and the man with his contribution. Piaget's stages of cognitive development. Attachment theory. Theory of mind, with its place in normal development and in disorder.

I - 85

Describe the Piaget's theory and the various stages of cognitive development. [2+8]

2 + 8 | DEVELOPMENTAL PSYCHOLOGY AND ATTACHMENT

DNB, KUHS, RGUHS, TN-MGRMU - 6 APPEARANCES, 6 OF 78 SESSIONS

SKELETON

2	The theory	Adaptation, and the four invariants that drive it
5	The four stages	Age, defining operation, and the error still made
2	Clinical use	Explanation, consent, delay, a child's account
1	Criticism	One honest limit, named rather than hinted at

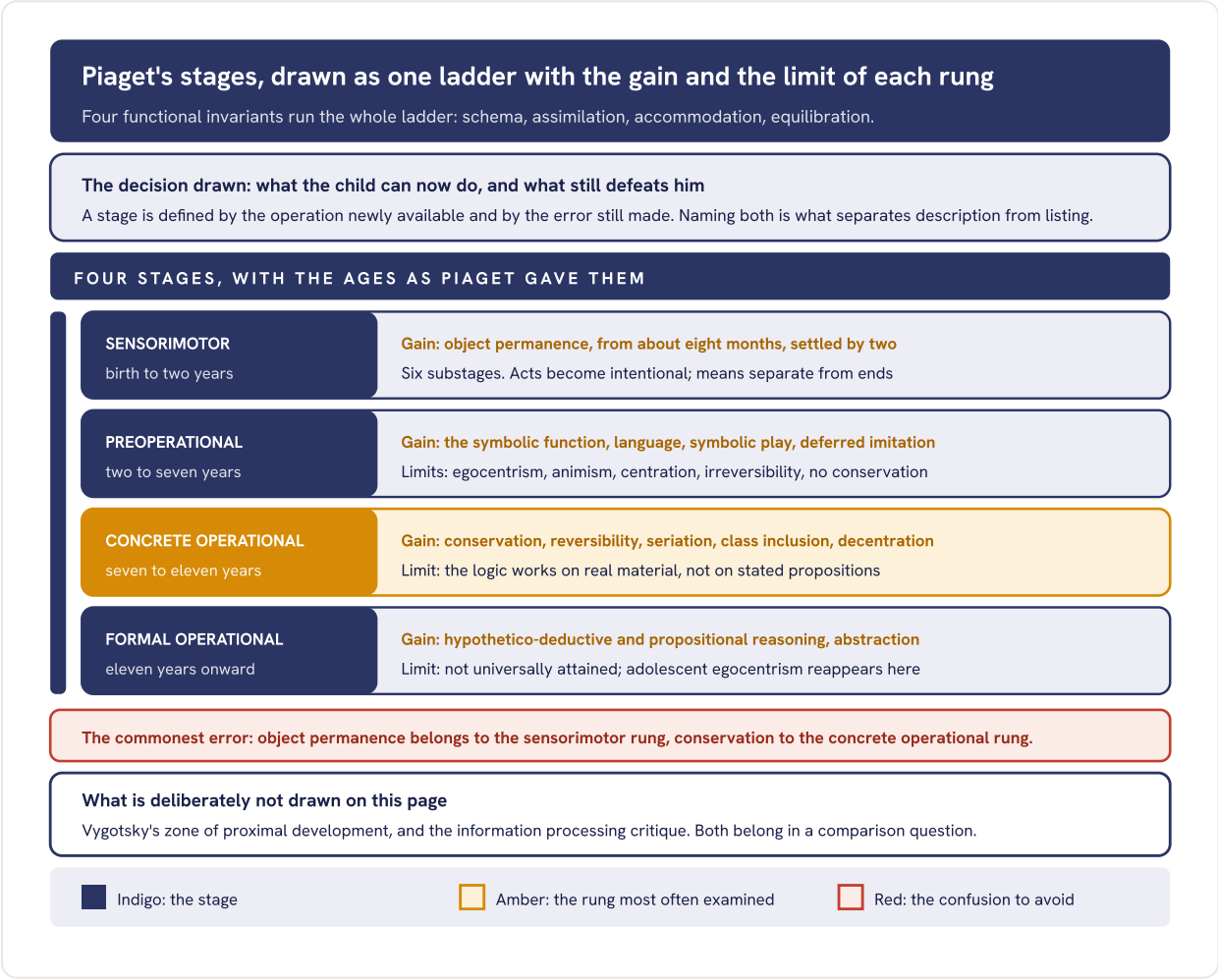

Fig 085.1 The four stages as rungs on a single spine, each carrying its age range, the operation it makes available in amber, and the error that still defeats the child at that rung.

- **The theory, not only the stages.** Intelligence is adaptation. The schema is the unit; assimilation fits experience into an existing schema, accommodation alters the schema to fit experience, equilibration drives the alternation. The sequence is invariant, the rate is not, and each stage is a different logic, not more of the last.
- **Clinical use, and the honest criticism.** The stages set how illness is explained to a child, the threshold for assent, and the weight given to a child's account. Piaget underestimated infants because his tasks demanded motor and verbal responses, underplayed teaching and culture, and formal operations are not universally reached.

WHAT EARNS THE MARKS

- **The four invariants named before the ladder.** Schema, assimilation, accommodation, equilibration.
- **Every rung with its age and its defining operation.** Ages without the operation is where the eight marks go.
- **The limit of each stage, not only its gain.**
- **The red strip.** Object permanence and conservation belong to different rungs.

SEE ALSO Slot I - 87 theory of mind and its developmental timetable Slot I - 88 Erikson's theory of child development

SPRINT Day 4, general and developmental psychology

I - 86

Erik H.Erikson - Stages of Life Cycle. [10]

10 ASKED AT 15 MARKS DEVELOPMENTAL PSYCHOLOGY AND ATTACHMENT

TN-MGRMU, WBUHS - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2 The frame	Epigenetic principle, and the crisis as a ratio
6 The eight stages	Age, crisis, virtue, and the pathology if unresolved
2 Clinical use	Where the life cycle enters a formulation

STAGE AND AGE	CRISIS	VIRTUE	IF UNRESOLVED
Infancy, to one year	Trust against mistrust	Hope	Withdrawal, suspicion
Early childhood, one to three	Autonomy against shame and doubt	Will	Compulsion, self doubt
Play age, three to six	Initiative against guilt	Purpose	Inhibition, guilt
School age, six to eleven	Industry against inferiority	Competence	Inertia, inferiority
Adolescence, twelve to eighteen	Identity against role confusion	Fidelity	Identity diffusion, repudiation
Young adulthood	Intimacy against isolation	Love	Exclusivity, isolation
Middle adulthood	Generativity against stagnation	Care	Rejectivity, self absorption
Late adulthood	Ego integrity against despair	Wisdom	Disdain, despair

- **The epigenetic principle.** Erik Homburger Erikson held that development unfolds on a ground plan, each stage arising at its proper time and the whole personality being the sum of all eight. A crisis is resolved as a ratio, never as a victory: enough trust to function alongside enough mistrust to stay safe.
- **Where it is used.** Identity work in adolescent presentations, generativity in midlife depression, life review and integrity in late life. Erikson extended Freud's five psychosexual stages across the whole life span and made the conflict social rather than instinctual, which is the sentence that names his contribution.

WHAT EARNS THE MARKS

- **All eight stages, with the crisis written as a pair of opposites,** which is the form Erikson used.
- **The virtue attached to each resolved crisis.** Most answers give the crisis and stop there.
- **The epigenetic principle named.** It is what makes this a theory rather than a table.

SEE ALSO Slot I - 88 Erikson's theory of child development Slot I - 90 Erikson, the man and the contribution SPRINT Day 4, general and developmental psychology

I - 87

Define "Theory of Mind" (TOM). What is the significance of TOM in normal development of children? Describe its significance in various psychiatric disorders. [1+3+6]

1 + 3 + 6 | DEVELOPMENTAL PSYCHOLOGY AND ATTACHMENT

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

1 Definition	The capacity, and who named it in which year
3 Normal development	The timetable, drawn, with the task at each point
6 In disorder	Four conditions, each with the specific failure named

THE DEFINITION

The capacity to attribute mental states, beliefs, desires, intentions and knowledge, to oneself and to others, and to grasp that another person's may differ from one's own. Premack and Woodruff named it in 1978.

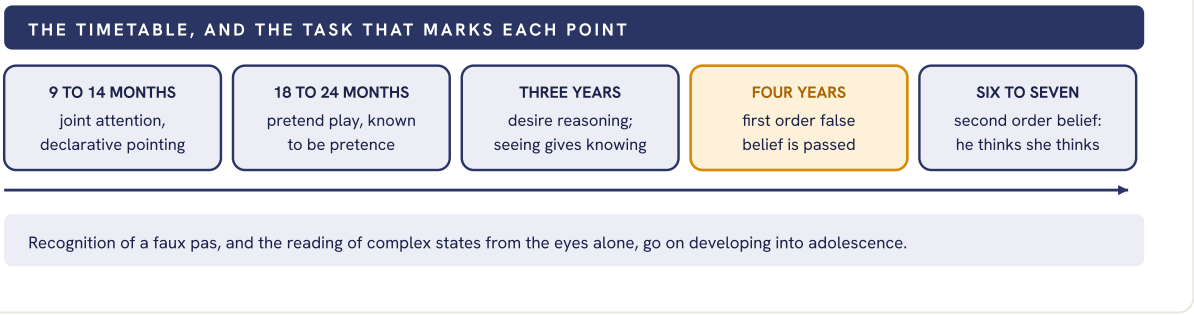

Fig 087.1 The developmental timetable, with the first order false belief task at four years marked in amber because it is the milestone every version of this question turns on.

- **Significance in normal development.** It is what makes deception, persuasion, teaching, keeping a secret, empathy and negotiated friendship possible. The false belief task of Wimmer and Perner is its standard marker, in the Sally-Anne form used by Baron-Cohen, Leslie and Frith.
- **Significance in disorder, named condition by condition.** Autism, where the false belief failure was first shown and framed as mindblindness. Schizophrenia, where mentalising failure tracks persecutory delusions and passivity phenomena. Antisocial personality, where cognitive theory of mind is intact while the affective side is not. It is reported too in attention deficit hyperactivity disorder, in bipolar disorder, in anorexia nervosa and in frontotemporal dementia.

WHAT EARNS THE MARKS

- **The definition in one sentence with its 1978 attribution.** One mark, thirty seconds.
- **The timetable with ages,** and the four year first order task named as the amber milestone.
- **A specific failure per disorder,** not a list of diagnoses said to have a deficit.

SEE ALSO Slot I - 85 Piaget and the stages of cognitive development Slot I - 89 attachment theory **SPRINT** Day 4, general and developmental psychology

I - 88

Erikson's theory of child development. [10]

10 DEVELOPMENTAL PSYCHOLOGY AND ATTACHMENT

RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The departure	Psychosocial against psychosexual, stated first
6 The childhood crises	Five, each with its age and its Freudian parallel
2 What the child needs	What each stage asks of the people around it

CRISIS	AGE	FREUD'S PARALLEL	SEEN IN CLINIC AS
Trust against mistrust	To one year	Oral	**Difficulty trusting any caregiver, later any clinician**
Autonomy against shame and doubt	One to three	Anal	Control battles; shame carried into adult self doubt
Initiative against guilt	Three to six	Phallic	Inhibited play and questioning; guilt at wanting
Industry against inferiority	Six to eleven	Latency	School refusal, inferiority, giving up on effort
Identity against role confusion	Twelve to eighteen	Genital	**Identity diffusion, and the borderline presentations near it**

- **How Erikson departed from Freud.** Freud's stages end at puberty and turn on the site of instinctual gratification. Erikson's run the whole life span and turn on a social task set by the widening circle of people around the child. The ego is autonomous and adaptive, not merely the servant of drive and reality.
- **What each stage asks of the environment.** Consistent responsive care in the first year; graded permission to act and to fail in the next two; tolerance of questions and noisy play in the preschool years; real tasks with real feedback through school; and in adolescence a moratorium in which roles can be tried without permanent cost.

PITFALL

The stem asks for child development, so the three adult stages are context and not content. Listing all eight and describing none of them is the commonest way this answer goes wrong.

WHAT EARNS THE MARKS

- **Psychosocial set against psychosexual in the opening lines.** It frames everything after it.
- **Five childhood crises with ages,** each written as a pair of opposites.
- **What the environment must supply at each stage,** which is what turns the table into developmental psychology.

SEE ALSO Slot I - 86 the eight stages of the life cycle Slot I - 85 Piaget and the stages of cognitive development

SPRINT Day 4, general and developmental psychology

I - 89

Attachment theory. [10]

10 DEVELOPMENTAL PSYCHOLOGY AND ATTACHMENT

RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The claim	A behavioural system, not a by-product of feeding
3 Development	Four phases, and the separation sequence
3 The patterns	Four classifications, and how each is elicited
2 Clinical use	The disorders, and what a pattern predicts

THE CLAIM

Bowlby held attachment to be a biologically based behavioural system, selected because proximity to a caregiver kept the infant safe. Its set goal is felt security. It is activated by fear, illness and separation, and terminated by contact.

PATTERN	IN THE STRANGE SITUATION	CAREGIVER PATTERN	ADULT INTERVIEW
Secure	Protests separation, is comforted, returns to play	Consistently available and responsive	Autonomous
Insecure avoidant	Little protest, ignores the return, play looks unaffected	Rejecting of distress, pushes independence	Dismissing
Insecure resistant	Intense protest, then angry and clingy, hard to settle	Inconsistent and unpredictable	Preoccupied
Disorganised	Contradictory approach, freezing, apprehension at the caregiver	Frightened or frightening; loss and trauma	Unresolved

- **The developmental frame.** Four phases: preattachment in the first weeks, attachment in the making to about six months, clear-cut attachment with separation protest and stranger wariness from then to two years, and goal corrected partnership after it. Separation runs protest, then despair, then detachment. What is laid down is an internal working model of self and other.
- **Evidence and clinical use.** Harlow's infant monkeys clung to the cloth mother rather than the one that fed them, which separated attachment from feeding. Ainsworth's Strange Situation, eight brief episodes with two separations, is the classification instrument; disorganised was added later by Main and Solomon; the Adult Attachment Interview is the adult counterpart. Clinically: reactive attachment disorder, disinhibited social engagement disorder, separation anxiety, and disorganised attachment as an antecedent of borderline presentations.

WHAT EARNS THE MARKS

- **Attachment named as a system with a set goal,** not as a synonym for a close bond.
- **All four patterns in the grid,** disorganised included, with what elicits each.
- **The instrument named,** Strange Situation for infants, Adult Attachment Interview for adults.
- **The internal working model,** which is what carries the childhood pattern into adult relationships.

SEE ALSO Slot I - 87 theory of mind and its developmental timetable

Slot I - 64 imprinting and the ethological background SPRINT Day 4, general and developmental psychology

I - 90

Eric Erikson. [10]

10 DEVELOPMENTAL PSYCHOLOGY AND ATTACHMENT

KUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 The man	Dates, training, and the phrase he gave the language
5 The contribution	Four ideas, each stated in its own terms
2 Standing today	Where it is used, and the criticism it carries

THE MAN

Erik Homburger Erikson, 1902 to 1994. He trained in Vienna in the analysis of children, with Anna Freud as his analyst, and held neither a medical nor a doctoral degree. He gave psychoanalysis the whole life span, gave the ego its own developmental line, and gave the language the phrase identity crisis.

- **The four contributions, named separately.** The epigenetic principle, that development unfolds on a ground plan with each part rising at its proper time. The eight psychosocial stages, which carried Freud's five past puberty to the end of life. Ego psychology, in which the ego has its own energies and adaptive tasks rather than serving drive and reality alone. And the ego virtues, one strength issuing from each crisis resolved.
- **Identity, which is his central idea.** Identity is a sense of sameness and continuity within oneself, matched by the recognition of that sameness by others. Its failure in adolescence is role confusion. He described the psychosocial moratorium, a sanctioned delay in which roles are tried without permanent commitment, and identity diffusion as the outcome when it does not close.
- **Psychohistory, and the criticism.** He applied the stage frame to whole lives, in studies of Luther and of Gandhi, arguing that a personal identity crisis and a historical moment can resolve each other. This is the part of the work most criticised, since a single life cannot be a controlled observation, and the stages themselves are hard to falsify.

WHERE ERIKSON IS STILL USED

Adolescent presentations read as identity work rather than as settled trait pathology. Midlife depression framed as stagnation against generativity. Life review in late life and in palliative care, where integrity against despair is the live question rather than a theoretical one.

WHAT EARNS THE MARKS

- **The epigenetic principle and the eight stages named as two contributions,** because they are two.
- **Identity defined rather than invoked.** Sameness within, recognised from without.
- **One honest criticism.** On a named-person stem it is what separates command from recitation.

SEE ALSO Slot I - 86 the eight stages of the life cycle Slot I - 34 Anna Freud SPRINT
Day 4, general and developmental psychology

AREA 11 OF 12 · P1-A12

Nosology, classification and descriptive psychopathology

6	4.9%	I-91	I-96
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

SINGLE 6

REVISION ORDER

The three phenomenology slots together: they share one definitional grammar and the marks are in the criteria, not in the length. The two classification slots next, the neo-Kraepelinians first because the revision slot argues from them. The spectrum slot last, and give it time: it is three questions in one.

WHAT IS IN THIS AREA

Three phenomenology slots: delusion with the primary forms and the dimensions, consciousness with its dimensions and quantitative changes, and hallucination with the true against pseudo distinction. Three nosology slots: the neo-Kraepelinians and their influence on the current systems, the purpose of revising DSM and ICD and how far that purpose has been met, and the spectrum concept with the mixed episode and the epilepsy spectrum. Every slot in this area was seen once in the record, which is the whole of what its chips claim.

I - 91

Who are the neo-Kraeplineans? How they influence the current classificatory system? [3+7]

3 + 7 | NOSOLOGY, CLASSIFICATION AND DESCRIPTIVE PSYCHOPATHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**Who they were**	The St Louis school, and why Kraepelin is the ancestor
2	**What they demanded**	Illness, boundaries, criteria, validation
4	**The influence**	Feighner, the RDC, DSM-III, and what ICD took
1	**The honest limit**	Reliability bought, validity still owed

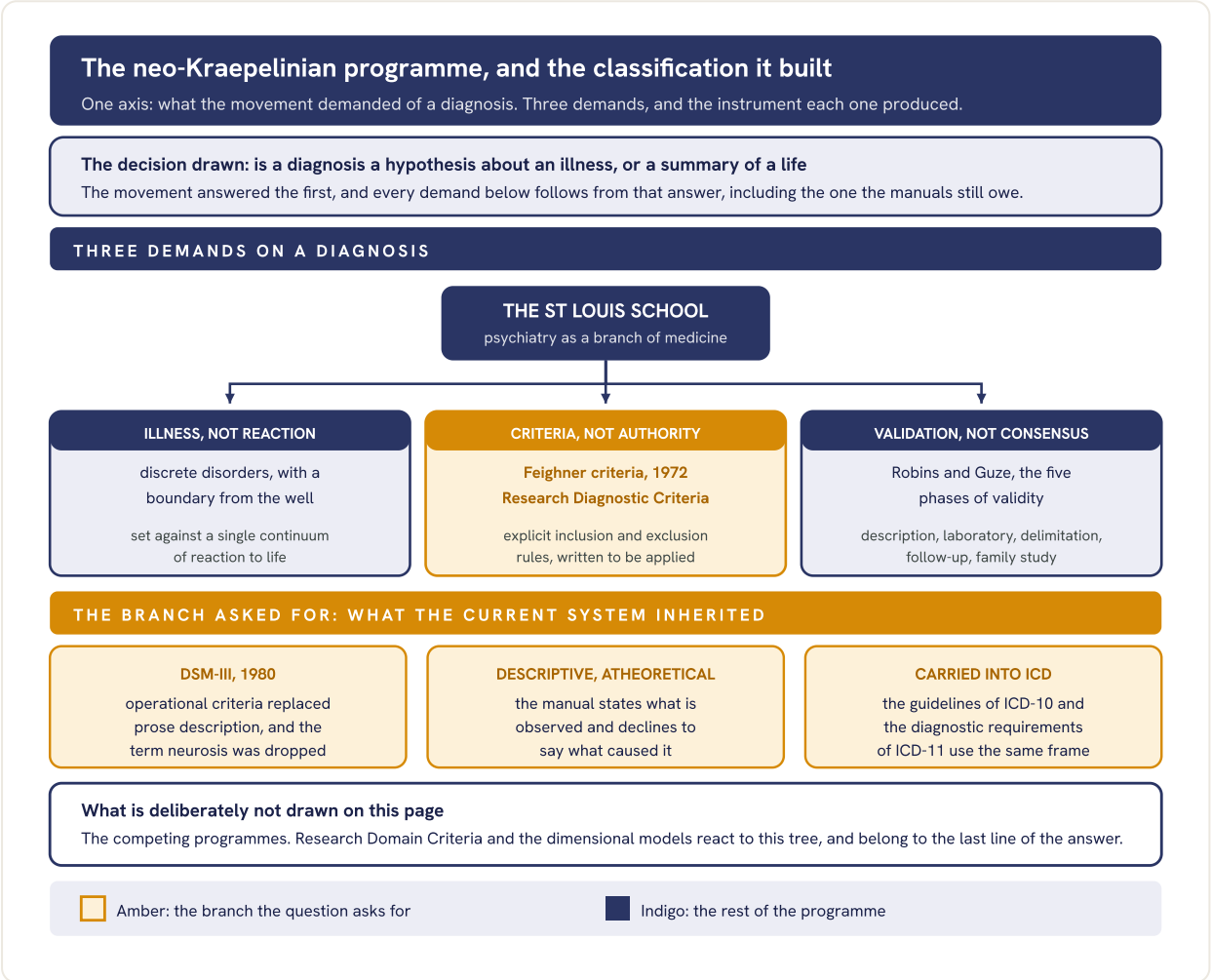

Fig 091.1 The neo-Kraepelinian programme drawn as three demands made on a diagnosis, with the criteria branch opened out into the architecture DSM-III built and both manuals still use.

- **Who they were.** A group working in St Louis from the late 1960s, Eli Robins, Samuel Guze and George Winokur, named neo-Kraepelinian by Gerald Klerman, who set out the movement's credo. Kraepelin is the ancestor because he classified by course and outcome rather than by presumed cause.
- **What it has not delivered.** Reliability improved; validity did not follow. No laboratory finding delimits these categories, comorbidity is the rule rather than the exception, and both manuals have answered by adding dimensions to a frame that stays categorical.

WHAT EARNS THE MARKS

- **The names attached to the movement.** Robins, Guze and Winokur, and Klerman who named it.
- **Operational criteria named as the inheritance,** rather than DSM-III named as an event.
- **The Robins and Guze validators listed.** They are the standard the system is still judged against.
- **The unfinished part stated.** Reliability without validity is the honest close.

SEE ALSO Slot I - 93 the purpose of revising a classification Slot I - 92 spectrum concepts in psychiatry **SPRINT** Day 6, psychometry, assessment and descriptive psychopathology

I - 92

Discuss the concept of spectrum disorder(s) in psychiatry with suitable diagrams. How has current understanding of mixed episode developed? What is epilepsy spectrum disorder? [3+5+2]

3 + 5 + 2 | NOSOLOGY, CLASSIFICATION AND DESCRIPTIVE PSYCHOPATHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	What a spectrum is	Two senses, drawn: one graded condition, one family
2	Why it is claimed	Family, genetic and dimensional evidence
3	Mixed episode	Kraepelin, DSM-IV, the DSM-5 specifier, ICD-11
2	Epilepsy spectrum	What the term covers, and where it stands

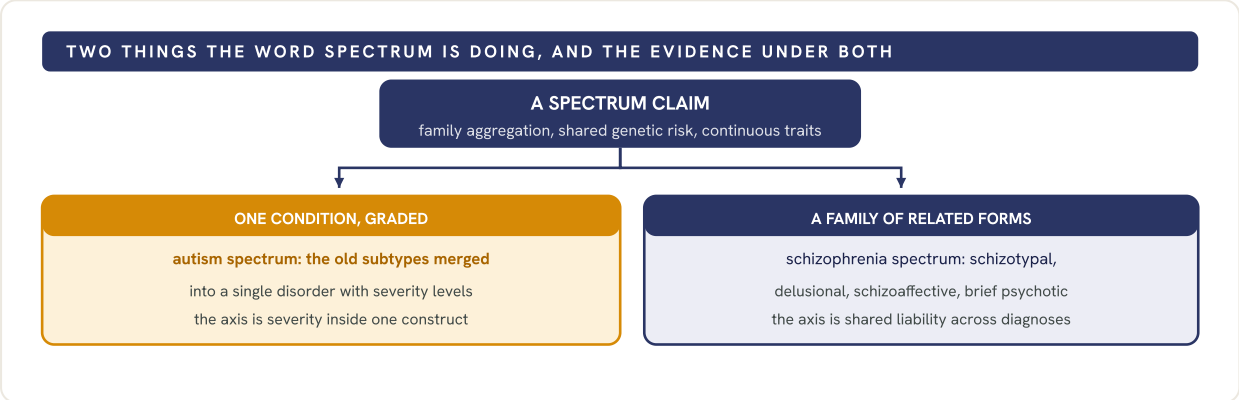

Fig 092.1 The two senses the word spectrum carries in psychiatry, severity graded within one construct on the left and shared liability across neighbouring diagnoses on the right, with the evidence both rest on named at the root.

- **How the mixed episode changed.** Kraepelin drew mixed states from the independent variation of mood, thought and volition. DSM-IV required full criteria for mania and for depression at once, a threshold so rarely met that most mixity went unrecorded.
- **Where the systems now part.** DSM-5-TR carries mixity as a with-mixed-features specifier, three non-overlapping symptoms of the opposite pole. ICD-11 keeps a mixed episode as an episode type. Mixed features predict poorer outcome and higher suicide risk.

WHAT THE THIRD PART IS ACTUALLY ASKING

Epilepsy spectrum disorder names no category in either manual. It is used for the temporolimbic psychiatric picture seen with epilepsy: interictal dysphoric disorder, postictal and interictal psychosis, and the interictal behaviour syndrome of hyperreligiosity, hypergraphia, viscosity and altered sexuality.

WHAT EARNS THE MARKS

- **Both senses of spectrum given, with an example under each.**
- **The mixed episode traced, not merely defined.** Kraepelin, DSM-IV, DSM-5, ICD-11.
- **The specifier stated exactly.** Three non-overlapping symptoms of the opposite pole.
- **The honest line on the third part.** The term names no category in either manual.

SEE ALSO Slot I - 91 the neo-Kraepelinians and the current system Slot I - 93 the purpose of revising a classification

I - 93

There have been many revisions of diagnostic systems (e.g. DSM, ICD) in psychiatry. In your view, what is the purpose of the exercise of revision and to what extent the purpose has been achieved by DSM-5 and ICD-11? [5+5]

5 + 5 | NOSOLOGY, CLASSIFICATION AND DESCRIPTIVE PSYCHOPATHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	Why a system is revised	Evidence, usability, harm, and the users served
3	What the two did	Regrouping, dimensions, and what was removed
2	Where it was met	Organisation and utility, field tested
2	Where it was not	No validators, comorbidity, threshold creep

PURPOSE OF REVISING	WHAT DSM-5 AND DSM-5-TR DID	WHAT ICD-11 DID	HOW FAR IT WAS MET
Follow the evidence	Chapters regrouped by shared mechanism	The same regrouping; subtypes dropped	**Architecture moved, validators did not**
Raise reliability	Field trials in routine clinics, reported openly	Field studies across many countries	**Measured honestly, and modest**
Serve the user	Kept criteria with counts and durations	Essential features written as prose	**ICD-11 chose the clinician**
Admit dimensions	Severity specifiers; trait model kept apart	Personality rebuilt as severity plus traits	Carried further by ICD-11
Reduce harm	Bereavement exclusion removed	Gender incongruence moved out of the chapter	Met in part, contested in both

- **What a revision is for.** To move the manual closer to nature where evidence allows, to keep it usable by the people who must apply it, and to stop it doing harm. The three pull against each other, so every revision is a compromise.
- **The charge that sticks.** Neither revision produced a diagnostic validator, and comorbidity is still the rule, which is what a valid categorical system should not produce.

PITFALL

Answering with a list of what changed. The stem asks for a purpose and then for a verdict, so every change named has to be tied to the purpose it served and judged against it. A changed manual is not by itself an achieved purpose.

WHAT EARNS THE MARKS

- **Purposes named before any change is described.** The stem asks for a view, and a view needs a criterion.
- **The structural contrast stated,** counted criteria against prose essential features.
- **A verdict per purpose,** rather than one verdict on the whole revision.
- **The missing validator named.** It is the honest answer to the second half.

SEE ALSO Slot I - 91 the neo-Kraepelinians and the current system Slot I - 92 spectrum concepts in psychiatry

SPRINT Day 6, psychometry, assessment and descriptive psychopathology

I - 94

Define delusion. What are primary delusions? Discuss dimensions of delusions. [2+3+5]

2 + 3 + 5 | NOSOLOGY, CLASSIFICATION AND DESCRIPTIVE PSYCHOPATHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The definition	Three criteria, stated as criteria
3 Primary delusions	Un-understandable, and the forms it takes
4 The dimensions	Each one varying free of the others
1 Why they matter	Insight, function, and where the risk sits

THE DEFINITION

A false belief held with extraordinary conviction and incomparable subjective certainty, not open to counter-argument or to the evidence of experience, and out of keeping with the person's social, cultural and educational background. Jaspers named three external criteria: certainty, incorrigibility, impossibility of content.

- **Delusional mood.** An oppressive sense that something momentous is under way, before any belief has formed. It settles when the delusion crystallises.
- **Delusional perception.** A normal percept given a private and unmediated significance: two members, the percept and the meaning. A first-rank symptom.
- **Sudden idea, and delusional memory.** A belief arriving fully formed with no trigger, one member only; and a real memory recalled as carrying a meaning it never had.
- **Primary against secondary.** A primary delusion is un-understandable: it cannot be derived from mood, personality or circumstance. A secondary one can, and is better called a delusion-like idea.

DIMENSION	WHAT IT RANGES OVER	WHY IT IS ASKED FOR
Conviction	Passing doubt through to unshakeable certainty	**Tracks insight, and predicts response to treatment**
Extension	One corner of life through to every part of it	Predicts functional impairment
Bizarreness	Culturally possible through to physically impossible	**Rated unreliably, so it no longer stands alone as a criterion**
Disorganisation	A single fragment through to a systematised structure	Systematised or fragmentary, and how far it is elaborated
Pressure	An occasional thought through to constant, driven preoccupation	**Later work added distress and action, the item that carries risk**

WHAT EARNS THE MARKS

- **The three criteria named as criteria.** Stopping at false belief is half a mark.
- **Un-understandability given as what makes a delusion primary.**
- **The five dimensions named as a set:** conviction, extension, bizarreness, disorganisation, pressure.
- **Independence stated.** Total conviction with no distress and no action is common.

SEE ALSO Slot I - 96 true and pseudo hallucinations Slot I - 95 consciousness and its disturbances

I - 95

Define consciousness. Write the dimensions and quantitative changes in consciousness. [2+4+4]

2 + 4 + 4 | NOSOLOGY, CLASSIFICATION AND DESCRIPTIVE PSYCHOPATHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The definition	Awareness of self and surroundings, on two axes
3 The dimensions	Level, content, field, and awareness of self
4 Quantitative changes	The ladder, each rung told from the one above
1 How it is scored	Three subscales, and the total they give

THE DEFINITION

Consciousness is awareness of the self and of the environment. Clinically it runs on two axes that vary independently: the level of arousal, a brainstem and thalamic function of the ascending reticular activating system, and the content of awareness, which is cortical.

- **The dimensions.** Level of arousal, the quantitative axis. Content of awareness, the qualitative axis. The field, which narrows in twilight and dissociative states. And awareness of self: Jaspers' activity, unity, identity over time, and self against not-self.
- **How it is scored.** The Glasgow Coma Scale rates eye opening one to four, verbal response one to five and motor response one to six, giving a total of three to fifteen.

LEVEL	AROUSAL AND RESPONSE	WHAT TELLS IT FROM THE RUNG ABOVE
Full alertness	Awake, attention sustained and directable	The baseline the rest are measured against
Clouding	Threshold raised, attention and comprehension fluctuate	**Fluctuation across hours: the core of delirium**
Drowsiness	Rousable by ordinary speech, then drifts back	Rouses without a physical stimulus
Stupor	Responds only to vigorous or painful stimulation	**Reflex and postural responses are kept**
Coma	Unrousable, no purposeful response to anything	**No sleep-wake cycle at all**

THE WORD STUPOR

Psychiatry and neurology use it differently: in psychiatry akinesis and mutism with consciousness preserved, as in catatonia; in neurology a depressed level of arousal. Rate arousal and awareness separately, as the locked-in syndrome shows.

WHAT EARNS THE MARKS

- **Arousal and awareness separated in the definition itself.**
- **Every rung named with what distinguishes it** from the rung above.
- **Jaspers' four aspects of self-awareness listed,** the part most answers omit.

SEE ALSO Slot I - 47 the mini mental state examination Slot I - 94 delusion, defined and dimensioned

I - 96

Define hallucinations. How do true and pseudo hallucinations differ? What is their clinical significance? [2+5+3]

2 + 5 + 3 | NOSOLOGY, CLASSIFICATION AND DESCRIPTIVE PSYCHOPATHOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Both, defined**	A percept without an object, and the two exclusions
4	**The grid**	Space, quality, insight, and where each is seen
2	**The disagreement**	Two traditions, and they do not overlap
2	**Clinical significance**	Modality points to cause; not all are illness

THE DEFINITION

A hallucination is a percept without an object: a sensory experience arising with no external stimulus, in external objective space, carrying the force and clarity of a true perception. It is not an illusion, which misperceives something real.

DOMAIN	TRUE HALLUCINATION	PSEUDOHALLUCINATION	WHAT SETTLES IT
Where it is located	External objective space	Inner subjective space	**The space it occupies, Jaspers' test**
Sensory quality	Concrete, as a real percept is	Figurative, without substance	**Concreteness, not vividness**
Insight	Usually absent, held as real	Usually present	**Separates them in the British usage only**
Where it is seen	Psychotic and organic states	Dissociative states, bereavement, trauma	Suggestive, never decisive

Amber marks the discriminators. Both are involuntary, so control settles nothing between them.

- **Two traditions, not one.** Jaspers separates the two by sensory quality and by the space they occupy, not by insight. The later British usage separates them by preserved insight. The two do not pick out the same experiences.
- **Modality points to the cause.** Auditory verbal in schizophrenia; visual with fluctuating consciousness in delirium; olfactory and gustatory in temporal lobe epilepsy; tactile in stimulant use and alcohol withdrawal; complex visual with insight kept in Charles Bonnet syndrome.
- **Not all are pathological.** Hypnagogic and hypnopompic hallucinations, those of bereavement and those of sensory deprivation occur in health.

WHAT EARNS THE MARKS

- **A grid, not two paragraphs.** The comparison has to be readable across a row.
- **The two traditions named and told apart.** One definition is not enough here.
- **Modality tied to aetiology,** which is most of the significance mark.
- **Command hallucinations flagged for risk,** and the healthy forms kept out.

SEE ALSO Slot I - 94 delusion, defined and dimensioned Slot I - 95 consciousness and its disturbances **SPRINT** Day 6, psychometry, assessment and descriptive psychopathology

AREA 12 OF 12 · P1-A03

Neuroendocrinology and basic psychopharmacology

4	2.8%	I-97	I-100
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 3

REVISION ORDER

The two stress slots together, they are one answer split at the immune limb. The pineal gland next, and it is short. The evidence-based medicine slot last: it is three terms in one question and it rewards precision rather than length.

WHAT IS IN THIS AREA

Stress in two slots, one asking for the neurobiology with a critical evaluation of coping, and one for the types with their effect on the immune system. The pineal gland. And the slot that runs evidence-based medicine into personalised medicine and pharmacogenetics. The registered name of this area carries basic psychopharmacology, and its one pharmacology slot is a pharmacogenetics question rather than a drug-class question. Four slots, and the area is described here by what they hold.

I - 97

Pineal gland. [10]

10 NEUROENDOCRINOLOGY AND BASIC PSYCHOPHARMACOLOGY

KNRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 What it is	Position, and the two things that make it unusual
3 Light to melatonin	The pathway, then the synthetic chain
3 Receptors and actions	Two receptors, and what each one does
2 Clinical relevance	The drugs, the disorders, the tumour

THE DEFINITION

A small unpaired midline endocrine gland of the epithalamus, sitting behind the third ventricle. Two features carry the answer: it has no direct nerve supply from the brain, and it lies outside the blood brain barrier. It converts a light signal into a hormonal one.

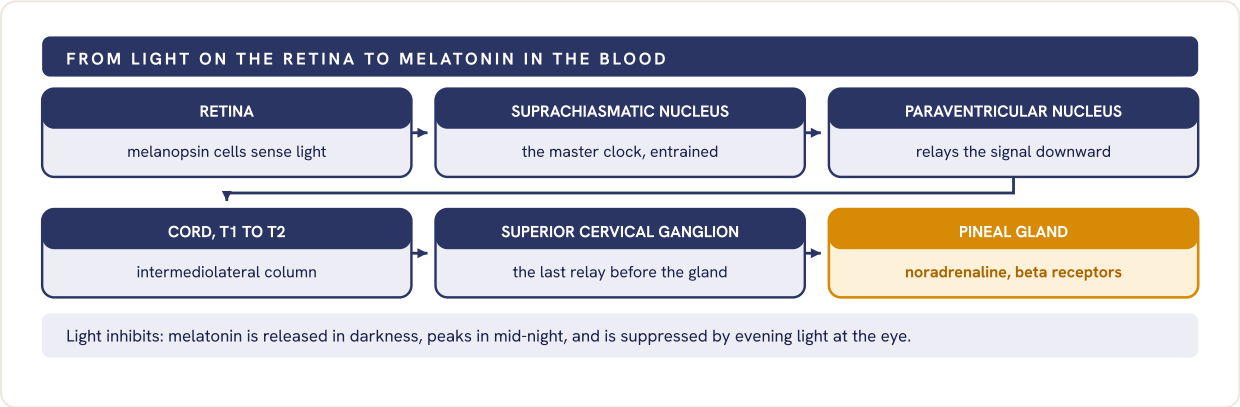

Fig 097.1 A sympathetic pathway with a hypothalamic beginning: the only route from the eye to the gland runs out of the brain, down the cord and back up the neck.

- **Synthesis.** Tryptophan to serotonin, then serotonin to N-acetylserotonin by arylalkylamine N-acetyltransferase, the rate limiting and dark-driven step, then to melatonin. Melatonin is not stored: it diffuses out as it is made.
- **Receptors and actions.** MT1 and MT2, both inhibitory and G protein coupled. MT1 damps the wake signal; MT2 shifts the phase. Melatonin re-times sleep more than it sedates.
- **Where it appears in exams.** Melatonin and ramelteon at MT1 and MT2; agomelatine, an agonist there and a 5-HT2C antagonist. Delayed sleep-wake phase disorder, free-running rhythm in blindness, jet lag. Calcification as a midline marker, and pineal tumours causing Parinaud syndrome.

WHAT EARNS THE MARKS

- **The two unusual features stated early.** No direct central innervation, and outside the blood brain barrier.
- **The whole pathway, eye to gland,** including the trip through the cord and the superior cervical ganglion.
- **The rate limiting enzyme named,** and the point that darkness drives it.
- **Phase shifting separated from sedation.** It is why melatonin is dosed by clock time, not by severity.

SEE ALSO Slot I - 03 neurobiology of sleep and wakefulness Slot I - 09 sleep stages and polysomnography **SPRINT** Day 2, Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology

I - 98

Define stress. Discuss neurobiology of stress. Critically evaluate the efficacy of different coping mechanisms in stress management. [2+3+5]

2 + 3 + 5 | NEUROENDOCRINOLOGY AND BASIC PSYCHOPHARMACOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Definition	One physiological, one transactional
3 Neurobiology	A fast axis, a slow axis, and the brake on both
5 Coping, evaluated	Match to controllability, then judge the evidence

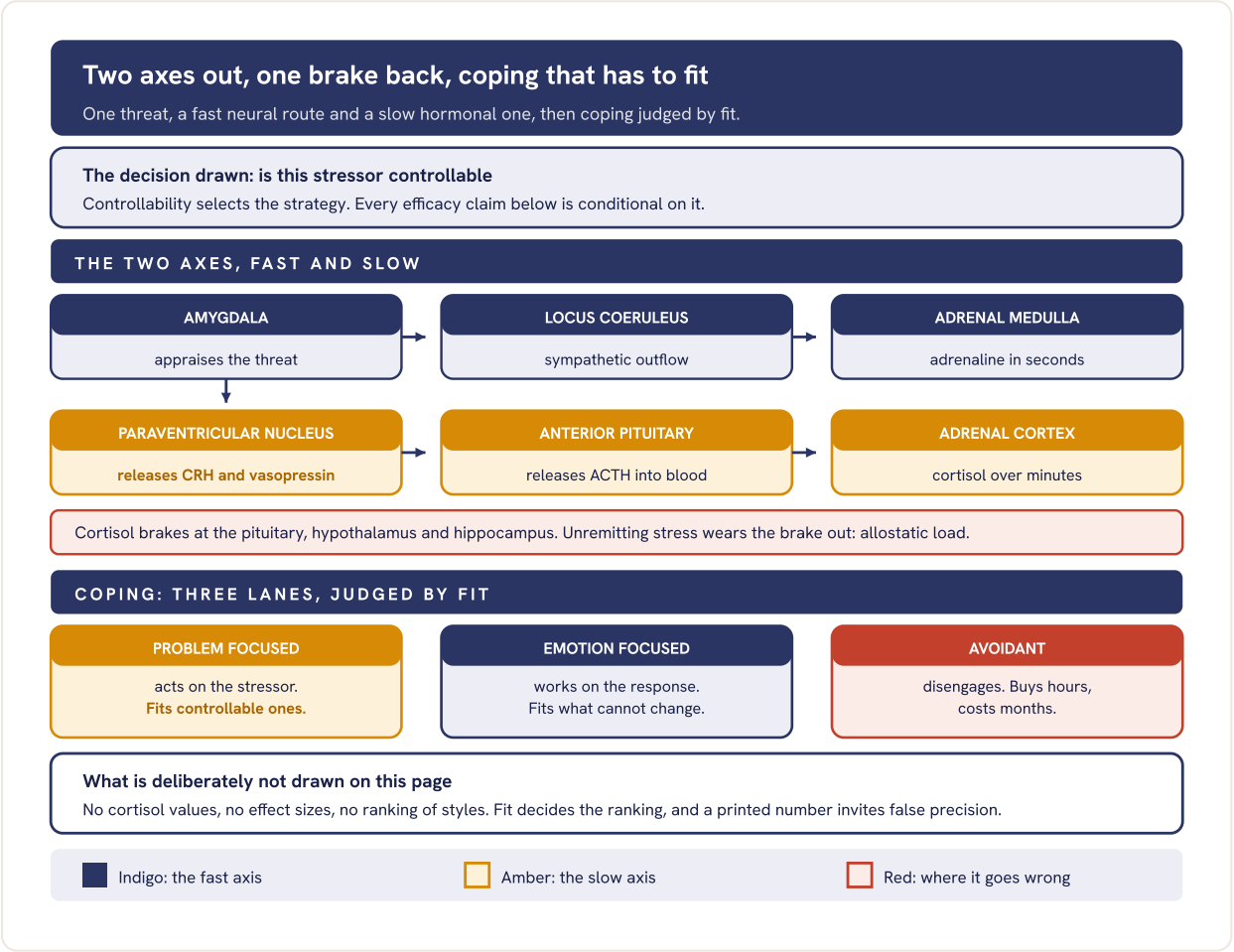

Fig 098.1 The two efferent axes drawn to one width so their timescales compare, with the brake and the coping lanes below.

COPING STYLE	WHAT THE PERSON DOES	WHERE IT WORKS	WHERE IT FAILS
Problem focused	Plans, seeks information, acts	**Controllable stressors: workload, illness self-management**	Effort on what cannot change raises distress
Emotion focused	Reappraises, accepts, relaxes, seeks support	**Uncontrollable ones: bereavement, terminal illness**	When it replaces action that was possible
Meaning focused	Reframes within values and goals	Chronic adversity, caregiving	When it minimises a real threat
Avoidant	Denies, distracts, uses substances	Briefly, in the first hours	**Over time: the most consistent predictor of poor outcome**

The amber cells are the goodness of fit rule. Avoidance reverses: what helps at hour one harms by month three.

- **The two definitions to give.** Physiological: the non-specific response of the body to any demand, through alarm, resistance and exhaustion. Transactional: a relationship appraised as taxing or exceeding the person's resources.
- **What critically evaluating means here.** Most coping research is cross-sectional and self-reported, the categories overlap, and stress management trials report modest, short-lived effects. Then say what stands: exercise, relaxation, mindfulness based stress reduction, stress inoculation.

WHAT EARNS THE MARKS

- **Both definitions.** The response without the appraisal is a half answer.
- **Both axes with their timescales,** and the brake named at all three levels.
- **The goodness of fit rule,** not styles ranked best to worst.
- **One honest limit on the evidence.** The word critically is asking for it.

SEE ALSO Slot I - 100 stress, immunity and psychiatric disorder Slot I - 29 classification of defence mechanisms

SPRINT Day 2, Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology

I - 99

What is evidence based medicine? What is personalized medicine in psychiatry? Discuss pharmacogenetics. [2+3+5]

2 + 3 + 5 | NEUROENDOCRINOLOGY AND BASIC PSYCHOPHARMACOLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Evidence based medicine**	The definition, its three components, the five steps
3	**Personalised medicine**	What it means, and what it is in psychiatry today
3	**Pharmacogenetics that works**	Metabolism genes, and the one safety gene
2	**Where the evidence stops**	Response prediction, and why it is not in clinic

THE DEFINITION TO WRITE FIRST

Evidence based medicine is the conscientious, explicit and judicious use of current best evidence in making decisions about the care of individual patients. Three components, all required: research evidence, clinical expertise, and the patient's values.

GENE	WHAT IT CHANGES	WHAT FOLLOWS CLINICALLY	HOW STRONG THE EVIDENCE IS
CYP2D6	Clearance of risperidone, aripiprazole, venlafaxine, tricyclics	Poor metabolisers accumulate drug; ultrarapid metabolisers are underexposed	**Established, with published dosing guidance**
CYP2C19	Clearance of escitalopram, citalopram, sertraline, some tricyclics	Same direction, so dose is adjusted at the two extremes	**Established, with published dosing guidance**
HLA-B*15:02	Risk of carbamazepine Stevens-Johnson syndrome	Test before starting carbamazepine in people of South and East Asian ancestry	**Established, testing recommended**
SLC6A4, HTR2A	Proposed prediction of antidepressant response	Not usable to choose a drug	Inconsistent across studies

The amber column is the whole answer: pharmacokinetic genes and one safety gene have earned their place, response prediction has not.

- **Evidence based medicine in five steps.** Ask an answerable question, acquire the best evidence, appraise it for validity and effect size, apply it alongside expertise and patient values, and assess the outcome.
- **What personalised medicine is not.** It is not a genetic test that picks the drug. In psychiatry it is mostly clinical: staging, phenotype, previous response, family history, comorbidity and measurement based care, with pharmacogenetics contributing at the metabolic extremes and one pre-treatment safety test.

WHAT EARNS THE MARKS

- **All three components of the definition.** Evidence alone is the commonest half answer.
- **Pharmacokinetic genes separated from pharmacodynamic ones,** and response prediction called not yet usable rather than promising.
- **HLA-B*15:02 before carbamazepine,** named as the one test with a clear ancestry-based indication.

SEE ALSO Slot I - 56 genome wide association studies Slot I - 84 clinical trial registry of India **SPRINT** Day 3, Psychiatric Genetics

I - 100

Enumerate the types of stress and its effect on the immune system and psychiatric disorders. [10]

10 | ASKED AT 15 MARKS | NEUROENDOCRINOLOGY AND BASIC PSYCHOPHARMACOLOGY

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The types	Name the axis first: duration, and whether there is an endpoint
3	How stress reaches immunity	Three routes, hormonal, neural and behavioural
3	What changes	Direction depends on the type, and that is the mark
2	Psychiatric consequence	Association, then the one clean causal example

TYPE OF STRESS	WHAT IT LOOKS LIKE	IMMUNE DIRECTION	EXAMPLE
Acute time limited	Minutes, with a clear end	Innate immunity mobilised, natural killer cells rise	A viva, a public talk
Brief naturalistic	Days, a real short stressor	Shift away from cellular toward humoral immunity	Examination week
Event sequences	One event with a train of consequences	Mixed, and it changes over the course	Bereavement, job loss
Chronic	No endpoint in sight, identity altered	**Broad suppression: killer cell cytotoxicity and lymphocyte proliferation fall, wounds heal slowly**	Caregiving, poverty
Distant	Long past, effects persist	**Low grade inflammation with glucocorticoid resistance**	Childhood adversity

The axis is duration and whether the stressor has an endpoint. The amber rows are where the direction reverses, and stating that reversal is what separates this answer from a list. Never write that stress suppresses immunity: it depends on the row.

- **How stress reaches the immune system.** Three routes. Hormonal, through cortisol on lymphocytes. Neural, through sympathetic fibres that directly innervate the spleen, thymus and lymph nodes. Behavioural, because stressed people sleep less, drink more, eat worse and attend less.
- **The paradox worth stating.** Cortisol is anti-inflammatory, so chronic stress ought to damp inflammation. It does the opposite, because immune cells become glucocorticoid resistant and inflammatory signalling comes off the brake.
- **Psychiatric consequence.** Raised C reactive protein and interleukin 6 are associated with depression and with poorer antidepressant response. The cleanest causal evidence is different: interferon alfa given for hepatitis produces depressive illness in previously well people.

WHAT EARNS THE MARKS

- **The types enumerated with a stated axis,** duration and whether the stressor has an end.
- **Both directions given.** Up in acute, down in chronic, is the discriminating sentence.
- **The three routes named,** hormonal, neural and behavioural, not cortisol alone.
- **Association separated from causation,** with interferon alfa offered as the causal case.

SEE ALSO Slot I - 98 neurobiology of stress and coping Slot I - 08 biological markers in psychiatric illness **SPRINT** Day 2, Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology

APPENDIX, PAGE ONE OF TWO

Ten sets, ten sittings

The same hundred questions, dealt into ten papers of ten. Each cell gives the slot and the page it is answered on.

Read down a row to sit a paper. The deal is not random: the hundred are ranked by score and dealt one question from each rank decile into each set, so every set holds one of the ten strongest, one of the ten weakest, and one from each band between. That is why the sets are comparable to each other and why none of them is an easy paper.

SET	Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	Q10
I-S1	I-85 p 113	I-46 p 64	I-19 p 32	I-21 p 34	I-29 p 43	I-31 p 46	I-94 p 125	I-65 p 87	I-75 p 100	I-89 p 118
I-S2	I-71 p 95	I-15 p 28	I-08 p 18	I-09 p 19	I-10 p 20	I-42 p 59	I-95 p 126	I-24 p 37	I-61 p 82	I-88 p 117
I-S3	I-45 p 63	I-05 p 15	I-55 p 75	I-48 p 66	I-63 p 85	I-64 p 86	I-86 p 115	I-44 p 61	I-51 p 70	I-78 p 103
I-S4	I-36 p 52	I-04 p 14	I-20 p 33	I-39 p 55	I-11 p 21	I-13 p 24	I-43 p 60	I-74 p 99	I-52 p 71	I-70 p 93
I-S5	I-79 p 105	I-18 p 31	I-28 p 42	I-47 p 65	I-23 p 36	I-30 p 45	I-32 p 47	I-67 p 89	I-62 p 83	I-77 p 102
I-S6	I-01 p 11	I-17 p 30	I-98 p 130	I-38 p 54	I-49 p 68	I-12 p 23	I-50 p 69	I-34 p 49	I-53 p 72	I-76 p 101
I-S7	I-02 p 12	I-27 p 41	I-06 p 16	I-72 p 96	I-99 p 132	I-41 p 58	I-56 p 76	I-66 p 88	I-35 p 50	I-87 p 116
I-S8	I-03 p 13	I-16 p 29	I-07 p 17	I-92 p 123	I-81 p 107	I-84 p 111	I-57 p 77	I-59 p 80	I-68 p 90	I-69 p 91
I-S9	I-54 p 74	I-26 p 40	I-91 p 121	I-22 p 35	I-40 p 56	I-83 p 110	I-73 p 98	I-25 p 38	I-60 p 81	I-90 p 119
I-S10	I-14 p 26	I-97 p 129	I-37 p 53	I-80 p 106	I-93 p 124	I-82 p 108	I-33 p 48	I-58 p 79	I-96 p 127	I-100 p 133

No set repeats a question, a merged variant of a question, or a near-duplicate, and no set takes more than three questions from any one area.

APPENDIX, PAGE TWO OF TWO

How to sit one of these

SET	MARKS	MEAN SCORE	AREAS	ANCHOR OR HIGH	HEAVIEST AREAS IN THE SET
I-S1	100	0.416	7	2	Developmental psychology 2; Functional neuroanatomy 2; Personality theory 2
I-S2	100	0.410	7	3	Neurochemistry 3; Functional neuroanatomy 2; Biostatistics 1
I-S3	100	0.402	7	3	Psychometry 3; Social 2; Neurochemistry 1
I-S4	100	0.401	6	3	Learning 3; Neurochemistry 3; Functional neuroanatomy 1
I-S5	100	0.402	7	3	Personality theory 3; Functional neuroanatomy 2; Research methodology 1
I-S6	100	0.402	7	4	Psychometry 3; Neurochemistry 2; Functional neuroanatomy 1
I-S7	100	0.402	8	3	Neurochemistry 2; Personality theory 2; Biostatistics 1
I-S8	100	0.402	6	3	Neurochemistry 2; Research methodology 2; Genetics in psychiatry 2
I-S9	100	0.392	8	2	Genetics in psychiatry 2; Functional neuroanatomy 2; Personality theory 1
I-S10	100	0.392	7	3	Neuroendocrinology 2; Research methodology 2; Nosology 2

Mean score is the selection score of the ten questions in that set, on the same scale used across all four books. The spread between the strongest and the weakest set is small by construction.

Three hours, ten questions, one hundred marks. That is the shape of the real paper, so it is the shape of these. Eighteen minutes a question leaves you twenty minutes at the end, which is roughly what you will have and roughly what you will need.

Write the skeleton first, for all ten. Spend the first twenty minutes writing nothing but mark splits and section headings across all ten answers. The map on each page shows what that skeleton should look like, so you can mark your own against it before you have written a line of content. Most marks lost in this examination are lost to allocation, not to ignorance.

Then mark yourself against the anchors. Every map ends with three or four things an examiner ticks. Those are the marking scheme, as close as this book gets to one. Count how many you hit before you read the page, because reading it first will convince you that you knew it.

Sit them in order, or sit them backwards. Set one holds the strongest question in the book and set ten the weakest, but the decile deal keeps the mean scores close, so any set is a fair paper. In order simply means the first one you sit is the one most like the paper you will get.

What a set is not. It is not a mock paper from any board, it is not a leak, and it is not a prediction. It is ten questions a board set, arranged so that ten of them look like a paper. Sit five and you will have written a hundred answers in the shape the examination wants, which is the only preparation that has ever reliably worked.

Index

Every entry points at the map that answers it. A subject asked in several places carries up to three pages, and the one in bold is the map whose question is about it.

E

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WEAVE SPRINT

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4

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TAP OR SCAN TO OPEN

STUDY TOGETHER

A hundred questions is a shortlist, not a syllabus.

This counting started as a room full of residents comparing papers and arguing about what actually comes. That room is still open. Bring a question we missed, a paper we have not read, and the corrections. Every one of them makes the next count better.

Join the study group

https://chat.whatsapp.com/H7TsLGK2wFl4bSe2r0Zfzn?mode=gi_t

CREDITS

Who made this, and from what

Dr. Wilfred Dsouza	Counting, area weighting, selection, and the answer maps. Lead author of the Weave 100 series.
Dr. Niharika Reddy	Clinical review of the personality, developmental and cross-cultural areas, and of the psychometry maps. Co-author.

The record. Every question in this book was read off a paper set by DNB, KNRUHS, KUHS, MUHS, NTRUHS, RGUHS, TN Dr. MGR Medical University or WBUHS. Where a paper reached us as a mirror, an archive or a transcription rather than an official file, that session carries a lower weight in the count, and the weightage chapter says by how much.

The residents. Papers, corrections and photographs of question papers came from residents across several states who had no reason to send them except that somebody else would need them. This book is mostly their work.

The tools. The counting, weighting, selection and typesetting all run as code in this repository, so any number in this book can be traced back to the row it came from.

PRINTING AND DISTRIBUTION

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WEAVE 100 - PAPER I

Eight boards were counted: DNB, KNRUHS, KUHS, MUHS, NTRUHS, RGUHS, TN Dr. MGR Medical University and WBUHS. Seventy-eight sittings of theory papers, weighted by how recent they are and how well the paper is evidenced.

From that count, twelve areas were weighted and one hundred questions were drawn in proportion. Each is solved as a one-page answer map: mark split, skeleton, the content that earns the marks, and what the examiner ticks. Ten ready-made sets at the back turn the hundred into ten sittings.

A frequency count is a strong prior, not a prophecy.

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