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Centre for Integrative Psychiatry

WEAVE 100 / PAPER IV

Brain, Drugs and the Community

One hundred ten-mark questions, solved as answer maps

MD Psychiatry theory. DNB revision fits too.



ILLUSTRATED REVIEW EDITION

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Dr. Wilfred Dsouza · Dr. Niharika Reddy

WEAVE

A NOTE ON WHO MADE THIS

Weave, Centre for Integrative Psychiatry

Weave is a psychiatry practice and a publishing house for the things clinicians and families actually need to read. Two consultant psychiatrists run it, online across India and in person in Mumbai.

WHAT WEAVE DOES	Integrative psychiatry, online and in Mumbai, and a free library for residents, clinicians and families.
WHO RUNS IT	Dr. Niharika Reddy and Dr. Wilfred D'souza, Consultant Psychiatrists.
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 IV

Brain, Drugs and the Community

One hundred ten-mark questions, solved as answer maps

THE WEAVE 100 SERIES

TITLE

Weave 100, Paper IV: Brain, Drugs and the Community

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

WRITTEN BY

Dr. Wilfred Dsouza and Dr. Niharika Reddy

PUBLISHED BY

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EDITION

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

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Illustrated edition, September 2026. Original answer maps with 139 selected plates. Page references updated for this edition.

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.

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

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THE HUNDRED, IN WEIGHT ORDER

01 **Community and public mental health** IV-01 to IV-11 · 11 slots · 13.3% 10

02 **Dementias: types and differential** IV-12 to IV-21 · 10 slots · 11.4% 40

03 **Consultation-liaison and systemic medicine psychiatry** IV-22 to IV-30 · 9 slots · 10.1% 68

04 **Clinical neurology, headache and sleep neurology** IV-31 to IV-38 · 8 slots · 8.0% 104

05 **Stroke, TBI and focal brain syndromes** IV-39 to IV-46 · 8 slots · 7.9% 123

06 **Movement and degenerative disorders in psychiatry** IV-47 to IV-53 · 7 slots · 6.7% 145

07 **Endocrine and metabolic psychiatry** IV-54 to IV-59 · 6 slots · 6.2% 164

08 **Recent advances and landmark studies** IV-60 to IV-65 · 6 slots · 5.9% 183

09 **Drug emergencies, interactions, TRD and neurostimulation** IV-66 to IV-71 · 6 slots · 5.6% 197

10 **Psychopharmacology: principles and drug classes** IV-72 to IV-76 · 5 slots · 5.0% 205

11 **Epilepsy and psychiatry** IV-77 to IV-81 · 5 slots · 4.9% 213

12 **Nutritional, toxic and metabolic encephalopathies** IV-82 to IV-86 · 5 slots · 4.2% 224

13 **Neuro-infective and autoimmune neuropsychiatry** IV-87 to IV-91 · 5 slots · 4.0% 242

14 **Delirium and amnestic syndromes** IV-92 to IV-96 · 5 slots · 3.9% 261

15 **Cognitive assessment and dementia management** IV-97 to IV-100 · 4 slots · 2.8% 271

BACK MATTER

Ten sets, ten sittings 279

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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 IV.

Fifteen 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 Community and public mental health	0.363	0.562	0.423	13.34%	11
02 Dementias: types and differential	0.388	0.304	0.362	11.44%	10
03 Consultation-liaison and systemic medicine psychiatry	0.364	0.214	0.319	10.07%	9
04 Clinical neurology, headache and sleep neurology	0.240	0.287	0.254	8.01%	8
05 Stroke, TBI and focal brain syndromes	0.267	0.215	0.251	7.93%	8
06 Movement and degenerative disorders in psychiatry	0.241	0.140	0.211	6.66%	7
07 Endocrine and metabolic psychiatry	0.262	0.048	0.198	6.25%	6
08 Recent advances and landmark studies	0.091	0.414	0.188	5.92%	6
09 Drug emergencies, interactions, TRD and neurostimulation	0.167	0.200	0.177	5.59%	6
10 Psychopharmacology: principles and drug classes	0.118	0.252	0.158	5.00%	5
11 Epilepsy and psychiatry	0.197	0.055	0.154	4.87%	5
12 Nutritional, toxic and metabolic encephalopathies	0.160	0.067	0.133	4.19%	5
13 Neuro-infective and autoimmune neuropsychiatry	0.181	0.000	0.127	4.00%	5
14 Delirium and amnestic syndromes	0.156	0.048	0.123	3.89%	5
15 Cognitive assessment and dementia management	0.128	0.000	0.090	2.84%	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 IV. Slots are the share turned into whole questions by largest remainder, with a floor of two and a cap of twenty; Paper IV 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	1	Watermark-confirmed printed papers, read by OCR; one of the two carries a Paper IV sitting. 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 fifteen 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: 18 anchor, 15 high, 2 recurrent, 65 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 394 selectable Paper IV questions in the record, 334 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 IV 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 15 · P4-A14

Community and public mental health

11	13.3%	IV-01	IV-11
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 3 SINGLE 8

REVISION ORDER

The two Act slots together, they are one answer split: the critique first, then the modes of admission. The three programme slots next as a block, District, one lakh and school, because they share a planning spine. Rehabilitation and the mental health promotion slot after that. The media, pandemic, adolescent suicide and LGBTQIA+ slots are independent of all of it and each stands alone.

WHAT IS IN THIS AREA

The District Mental Health Programme, a mental health programme planned for a population of one lakh, and the school mental health programmes with two Indian ones named. The Mental Healthcare Act in two slots, one a critique against the 1987 Act and one on the modes of admission. Then the principles and practice of rehabilitation, mental health defined with the strategies for promoting it, electronic media and its status in India, adolescent parasuicide and suicide with the legal and social issues, substance use and behavioural addictions during the pandemic with the theories of gambling, and the myths about LGBTQIA+ identities with the tenets of affirmative counselling.

IV - 01

Write a note on the District Mental Health Programme. [10]

10 COMMUNITY AND PUBLIC MENTAL HEALTH

DNB, KUHS, NTRUHS, RGUHS, TN-MGRMU - 7 APPEARANCES, 7 OF 78 SESSIONS

SKELETON

2	**Where it came from**	The national programme, the district pilot, the years
2	**What it is meant to do**	Objectives, stated as objectives
4	**The components**	The original core, then what was added
2	**The honest critique**	Manpower, funds, and where the service actually sits

THE LINEAGE, IN ONE BLOCK

The National Mental Health Programme was launched in 1982. Its district arm, the District Mental Health Programme, began in 1996 and was modelled on the district pilot run at Bellary in Karnataka from 1985. The idea both rest on is the same: mental health care delivered through the general health system, by people who are already there, rather than through a separate specialist service.

- **Objectives.** Minimum mental health care available and accessible to all, especially the underserved; mental health knowledge applied within general health care and social development; and community participation in the service rather than passive receipt of it.
- **The original components.** Early detection and treatment through the general health system; training of medical officers and health workers; public awareness through information, education and communication; monitoring and evaluation; and community participation.
- **What was added later.** A district mental health team with a psychiatrist, clinical psychologist, psychiatric social worker and psychiatric nurse; a small inpatient unit at the district hospital; life skills education and counselling in schools; college counselling; workplace stress programmes; and suicide prevention services.
- **The critique.** The manpower the design assumes does not exist in most districts, so the team is rarely complete. Allocated funds are frequently unspent. The service concentrates at the district hospital rather than at the primary health centre, which is the level the programme was written for. And the programme runs vertically, so it competes with the general health system it was meant to enter.

WHAT EARNS THE MARKS

- **Both years, and the district pilot named,** which almost no answer supplies.
- **Objectives kept separate from components.** Merging them is the commonest structural error here.
- **The later additions listed as additions,** which shows the programme is known as it stands rather than as first written.
- **A critique naming manpower and unspent funds,** not a closing line about the need for more awareness.

SEE ALSO Slot IV - 07 planning a programme for a population Slot IV - 08 promotion of mental health SPRINT Day 39, Community psychiatry and public health

IV - 02

Write about the principles and the practice of rehabilitation of psychiatric patients. [5+5]

5 + 5 | COMMUNITY AND PUBLIC MENTAL HEALTH

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

SKELETON

2	Definition and target	Impairment, disability, handicap, and which is treated
3	The principles	Early, functional, negotiated, in the real setting
3	The practice	Assessment, skills, family, work, and where it happens
2	The Indian frame	Family as the service, and the disability certificate

DEFINITION, AND WHAT IT IS AIMED AT

Rehabilitation helps a person with a psychiatric disability reach and hold the best level of independent functioning their illness allows. Impairment is the symptom, disability is the lost ability, handicap is the social disadvantage that follows. Treatment aims at the first; rehabilitation aims at the second and third. Saying so is the definition mark.

- **The principles.** Begin alongside treatment, not after it. Assess residual ability rather than diagnosis. Set the goal with the person, not for them. Train in the setting where the skill will be used. Work in the least restrictive place that will hold the person. Measure function, not symptoms. Expect years.
- **The practice.** Functional assessment and disability rating; skills training for daily living and for social situations; cognitive remediation where thinking is the barrier; family psychoeducation, which is the highest yield intervention available in most Indian settings; and vocational work running from sheltered activity to a real job with support.
- **Where it happens.** Day care centres, halfway homes, long stay homes for the small number who need them, self-help and peer groups, and, for almost everyone, the family home.
- **The Indian frame.** The family is the rehabilitation service, because institutional capacity is small, and the plan must be written for a family that has its own work to do. The Rights of Persons with Disabilities Act 2016 recognises mental illness as a disability, so certification, benefits and reservation are part of the rehabilitation plan and not an afterthought.

WHAT EARNS THE MARKS

- **The three level distinction stated before anything else,** because it defines what rehabilitation targets.
- **Principles written as principles,** not as a second list of activities.
- **The family named as the service,** which is what makes the answer Indian rather than translated.
- **Disability certification included in the plan,** which most answers omit entirely.

SEE ALSO Slot IV - 60 recent advances in psychological rehabilitation Slot IV - 01 the District Mental Health Programme

SPRINT Day 39, Community psychiatry and public health

IV - 03

Critically evaluate the Mental Health Care Act 2017. What are the major changes from the Mental Health Act 1987? [10]

10 | ASKED AT 15 MARKS | COMMUNITY AND PUBLIC MENTAL HEALTH

KUHS, TN-MGRMU - 5 APPEARANCES, 5 OF 78 SESSIONS

SKELETON

2	**The change of principle**	From licensing and custody to capacity and rights
4	**The journey, step by step**	The table, with the 1987 position beside it
2	**The critique**	Machinery, capacity assessment, and money
2	**What it does not cover**	The exclusions, stated plainly

STEP	UNDER THE 2017 ACT	UNDER THE 1987 ACT	WHO ACTS
1	Capacity to decide is presumed, and assessed when doubted	No capacity concept at all	The treating team
2	An advance directive and a nominated representative may be registered	Neither existed	The person
3	Independent admission where capacity is present	Voluntary admission, or a reception order	The person
4	Supported admission where capacity is absent, time limited	Reception order by a magistrate, open ended	Two independent examiners
5	The Mental Health Review Board reviews and hears appeals	The judicial magistrate	The Board

- **The other major changes.** Section 18 opens with a right to access mental healthcare rather than with the licensing of asylums. Section 21(4) requires insurers to cover mental illness on the same basis as physical illness. Section 95 bars electroconvulsive therapy without anaesthesia and muscle relaxants, and in minors without Board permission. Section 115 presumes severe stress in a person who attempts suicide, so that person is not tried or punished.
- **The critique.** The machinery is heavy: in several States the Boards and Authorities were slow to constitute and remain under-resourced. Advance directives are rarely made, and the route to registering and honouring one is thin. Capacity assessment asks for time and training a lone district psychiatrist does not have. A right of access created without matching funds shifts the failure onto the clinician who cannot deliver it.

WHAT THE ACT DOES NOT COVER

It does not fund the services it creates a right to. It says almost nothing about care outside registered establishments, which is where most patients are, and nothing about the family carer, who does most of the work. Disability benefits and certification sit under the Rights of Persons with Disabilities Act 2016, not here. Criminal responsibility remains a separate question entirely.

WHAT EARNS THE MARKS

- **Capacity named as the organising principle,** which is what actually changed.
- **Sections cited for the four headline provisions,** not paraphrased.
- **A critique with named weaknesses,** since critically evaluate is not satisfied by praise.
- **The exclusions strip,** which separates a top answer from a competent summary.

SEE ALSO Slot IV - 04 modes of admission under the Act **SPRINT** Day 39, Community psychiatry and public health

IV - 04

Discuss the modes of admission under the Mental Health Care Act. [10]

10 | COMMUNITY AND PUBLIC MENTAL HEALTH | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The organising principle	Capacity decides the route, not the diagnosis
4	The modes	Section, actor and limit, in order
2	Safeguards	Notice, review, appeal, and the right to be told
2	Outside the scheme	What no admission mode reaches

STEP	MODE	WHO ACTS	LIMIT OR CONDITION
1	Independent admission, Section 86	The person, who has capacity and asks	Discharge on request
2	Admission of a minor, Section 87	The nominated representative, after two assessments	Separate ward, an attendant stays, Board review
3	Supported admission, Section 89	Two independent examiners, one a psychiatrist	Up to thirty days, the Board notified
4	Supported admission beyond thirty days, Section 90	The Board, on continued need	Up to ninety days, then further reviewed periods
5	Emergency treatment, Section 94	Any registered medical practitioner	Up to seventy-two hours, to prevent harm

Steps 1 and 2 turn on who asks. Steps 3 and 4 turn on capacity being absent. Step 5 turns on time, and ends whether or not anything has been resolved.

- **The principle, and the safeguards.** The route follows capacity, never the diagnosis: a person with schizophrenia who retains capacity is admitted independently. Every supported admission is notified to the Mental Health Review Board, is time limited, and can be appealed by the person, the representative or any registered organisation. The person must be told the reason for admission and the right to appeal, in a language they understand.

WHAT THE ADMISSION SCHEME DOES NOT COVER

There is no route to compel treatment in an adult who retains capacity and refuses, however unwise the refusal looks. There is no community treatment order in Indian law. And these provisions govern admission to a registered mental health establishment, not admission to a general ward for a physical illness.

WHAT EARNS THE MARKS

- **Capacity named as the sorting rule** before the modes are listed.
- **Section numbers with the actor and the interval,** which is exactly what this archetype is checked on.
- **The seventy-two hour emergency provision,** which answers most often forget is an admission route at all.
- **The exclusions strip,** and particularly the capacitous refusal.

SEE ALSO Slot IV - 03 the Act critically evaluated SPRINT Day 39, Community psychiatry and public health

IV - 05

Discuss the role of electronic media in the management of psychiatric illness. Describe its status in India. [7+3]

7 + 3 | COMMUNITY AND PUBLIC MENTAL HEALTH | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What the term covers	Five distinct things, not one
3	What the evidence supports	Guided against unguided, and video consultation
3	Status in India	The 2020 guidelines and the national helpline
2	The limits	Prescribing, connectivity, privacy, examination

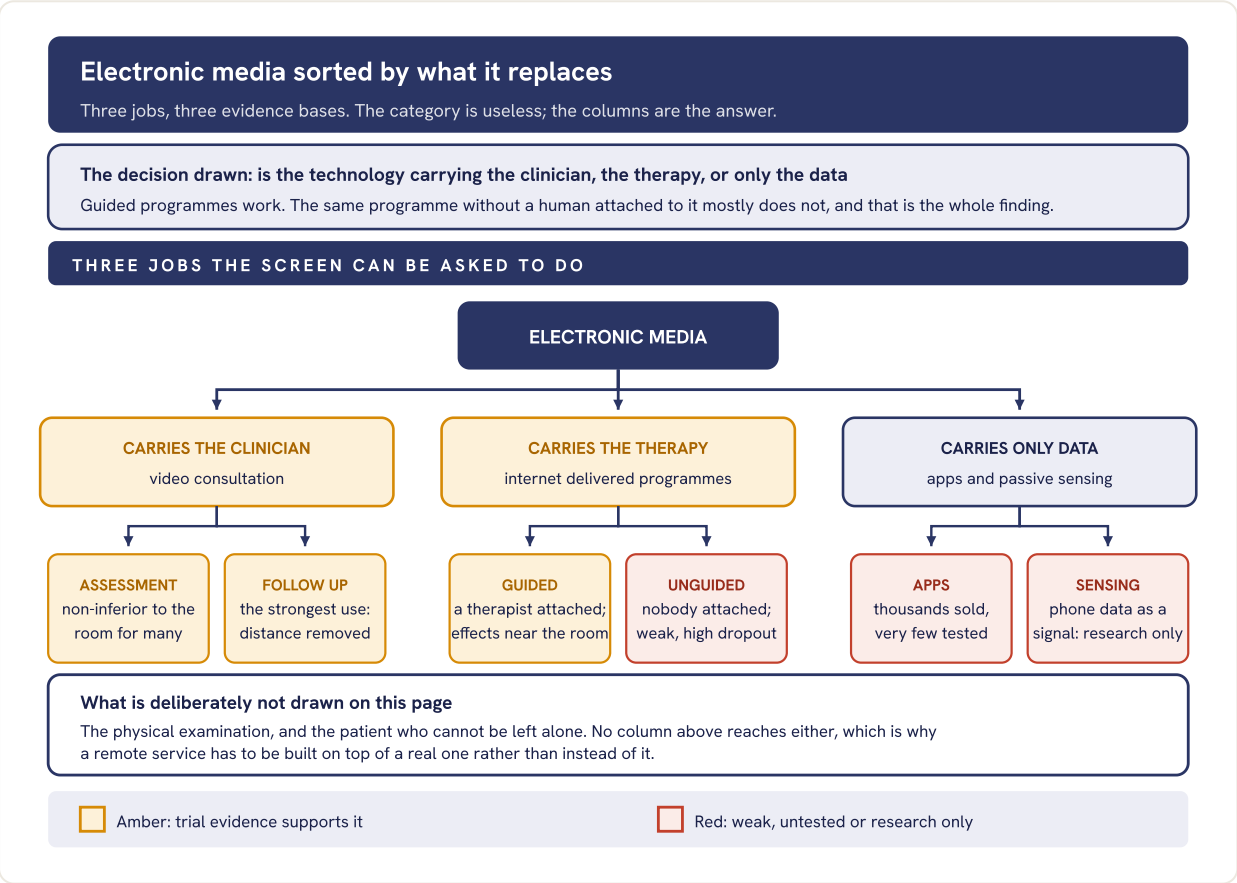


Fig 005.1 Guided and unguided sit in adjacent boxes and in different colours, because that single distinction carries more of the evidence than any other line on the page.

- **Status in India.** The Telemedicine Practice Guidelines of 2020 made remote consultation formally permissible and set out how a registered practitioner may consult and prescribe; telepsychiatry operational guidelines were issued the same year. Drugs on the prohibited list, which includes Schedule X, cannot be prescribed by teleconsultation at all. Tele MANAS, launched in 2022, runs a round the clock national tele mental health helpline with State cells.
- **The limits.** Connectivity and shared handsets, so the poorest patients gain least. No privacy in a one room house. Consent and record keeping requirements that a busy clinic will not meet. And no examination, which matters most in the patients who need it most.

WHAT EARNS THE MARKS

- **The category broken into three jobs** in the first sentence.
- **Guided separated from unguided,** which is the single most examinable finding here.
- **The 2020 guidelines and the prescribing restriction named,** which is the Indian status limb.
- **The limit that the poorest gain least,** rather than a closing line about the digital future.

SEE ALSO Slot IV - 07 planning a programme for a population Slot IV - 01 the District Mental Health Programme

SPRINT Day 39, Community psychiatry and public health

IV - 06

Discuss adolescent parasuicide and suicide. Discuss the legal and social issues related to it. [5+2+3]

5 + 2 + 3 | COMMUNITY AND PUBLIC MENTAL HEALTH

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 The clinical picture	Terms, who is at risk, and the means used
2 Assessment and management	Seen alone, means restricted, treatment named
3 The legal steps	Presumption, reporting duty, protection
2 The social issues	Stigma, school, family, media contagion

- **The clinical picture.** Self-harm without clear suicidal intent is far commoner than suicide and is the strongest single predictor of it. Attempts are commoner in girls, deaths in boys, and the difference is mostly method. Risk gathers around previous self-harm, depression, substance use, impulsivity, bullying including online, abuse, academic pressure, and easy access to means.

STEP	WHAT HAPPENS	WHO ACTS	CONDITION OR LIMIT
1	Medical stabilisation, and no discharge before a psychosocial assessment	Emergency team	Before any psychiatric decision
2	Psychosocial assessment, the adolescent seen alone and then with family	Psychiatrist or trained clinician	Every episode, not only severe ones
3	Section 115 applies: severe stress is presumed	The law, not the clinician	Not tried, not punished, care to be provided
4	Disclosed sexual abuse triggers mandatory reporting	The clinician, under POCSO 2012	Overrides the usual confidentiality
5	Safety plan, means restriction, treatment, follow up	Family, with the team	Follow up fixed before discharge

- **Treatment and the social side.** Treat the disorder, restrict means with the family in the room, and offer a therapy with adolescent trial evidence rather than general counselling. Socially: shame that stops the family returning, a school with no plan for the return, and reporting that names the method and seeds contagion.

WHAT THE LAW DOES NOT COVER

Section 115 removes punishment; it does not fund a pathway to care. No statute governs how a school responds or how a pupil returns after an attempt. Confidentiality for a mature minor is unsettled in Indian law. Media guidelines are advisory and unenforceable.

WHAT EARNS THE MARKS

- **The adolescent seen alone,** written as a step rather than assumed.
- **Section 115 stated correctly:** a presumption of severe stress, not blanket decriminalisation.
- **The reporting duty named,** because it is the conflict this question is really about.
- **Means restriction with the family present,** the one intervention with the best evidence per minute spent.

SEE ALSO Slot IV - 03 the Mental Healthcare Act critically evaluated Slot IV - 10 school mental health programmes

SPRINT Day 39, Community psychiatry and public health

IV - 07

How would you plan a mental health programme for a population of one lakh? [10]

10 | COMMUNITY AND PUBLIC MENTAL HEALTH | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Know the denominator	Who the lakh are, and what already serves them
2 Estimate the need	Survey data, not a figure from memory
4 Build into what exists	Train, supply, refer, reach out
2 Measure and sustain	Indicators, registers, and the money

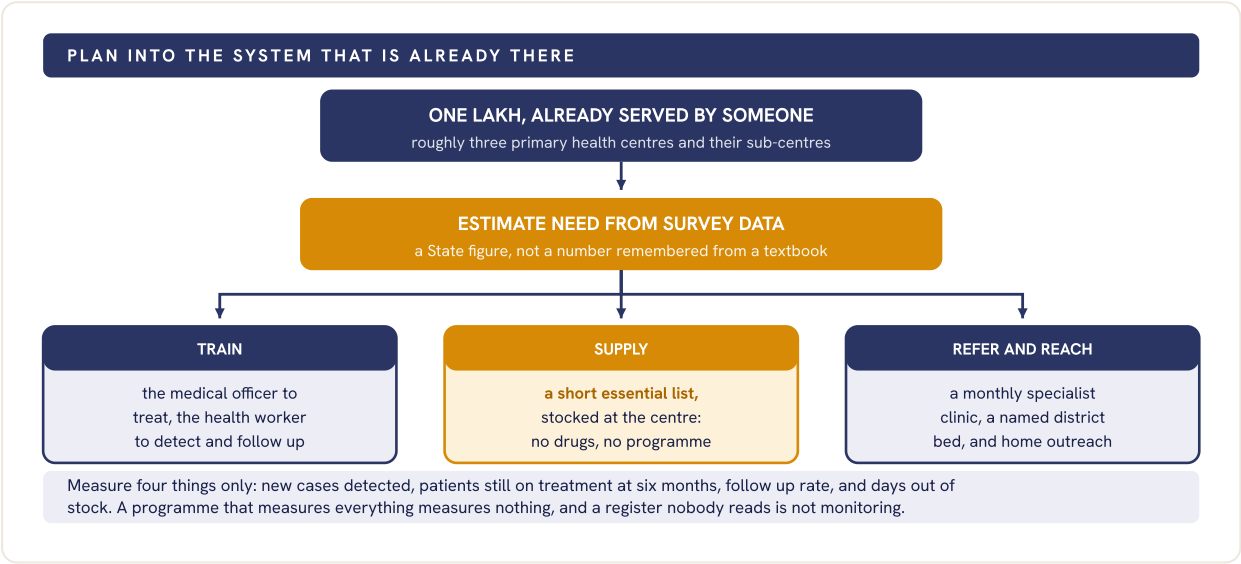


Fig 007.1 Supply sits in amber because a trained officer with an empty shelf produces referrals, not treatment, and that is the commonest way a district plan fails.

- **Know the denominator, then the need.** A lakh in the plains is about three primary health centres, twenty sub-centres, and a community health centre above them. Estimate need from the national survey rather than from memory: it found current mental morbidity in roughly one adult in ten, and a treatment gap above seventy per cent for most disorders. Those two numbers set the target and the reason for it.
- **Build in, not alongside.** Train the existing staff, stock a short essential list of an antipsychotic, an antidepressant, a mood stabiliser, an anticonvulsant, a benzodiazepine and an anticholinergic, run one specialist clinic a month, name the referral bed, and use the health worker for detection and follow up. Add school and workplace components once the clinical spine works, never before.

WHAT EARNS THE MARKS

- **The existing infrastructure counted first.** A plan that invents a new service has answered a different question.
- **Need estimated from a named source,** not asserted.
- **A drug list, by class,** which is the concrete detail examiners look for and answers rarely give.
- **Four indicators, named.** Monitoring described in general terms earns nothing.

SEE ALSO Slot IV - 01 the District Mental Health Programme Slot IV - 05 electronic media in management **SPRINT** Day 39, Community psychiatry and public health

IV - 08

Define mental health. Describe the strategies for promotion of mental health. What role can mental health professionals have in it? [2+5+3]

2 + 5 + 3 | COMMUNITY AND PUBLIC MENTAL HEALTH

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The definition	The wording, then what it is not
2	Promotion against prevention	Different target, different population
3	The strategies	By life stage, by setting, by determinant
3	The professional's role	Advise, train, deliver, evaluate, advocate

THE DEFINITION

Mental health is a state of well-being in which the individual realises his or her own abilities, can cope with the normal stresses of life, can work productively, and is able to contribute to the community. It is not the absence of illness: the two run on separate axes.

- **Promotion is not prevention.** Promotion raises positive mental health and acts on its determinants across a whole population, well or unwell. Prevention targets the occurrence of disorder, and is universal, selective or indicated by whom it reaches. The older primary, secondary and tertiary frame still earns its mark beside the newer one.
- **By life stage and setting.** Perinatal care and parenting programmes, early childhood nutrition and stimulation, life skills teaching in schools, workplace stress programmes, and social contact for older adults. The settings that carry them are the school, the workplace, primary care and the community, because that is where the population already is.
- **By determinant and instrument.** Poverty, education, housing, violence and harmful alcohol use. Then the law: the Mental Healthcare Act 2017 makes promotion and prevention a duty of government at Section 29, and awareness and stigma reduction a duty at Section 30. Anti-stigma work built on contact with people who have recovered outperforms leaflets.
- **The role, in five verbs.** Advise on policy and programme design. Train medical officers, health workers and teachers, so the work is shared rather than held. Deliver the clinical service that promotion cannot replace. Evaluate, so a programme is measured rather than asserted. Advocate with schools, employers, the media and the district administration.

WHAT EARNS THE MARKS

- **The definition written out,** with the line that mental health is not merely the absence of illness.
- **Promotion separated from prevention,** which is the axis this question turns on.
- **Strategies grouped by where they act,** not a loose list of good intentions.
- **An honest line where a figure would go,** since the treatment gap is large and unevenly measured.

SEE ALSO Slot IV - 01 the District Mental Health Programme Slot IV - 10 school mental health programmes SPRINT Day 39, Community psychiatry and public health

IV - 09

Discuss substance use disorder and behavioural addictions during the COVID-19 pandemic and the related movement restriction. What are the theories of gambling behaviour? [7+3]

7 + 3 | COMMUNITY AND PUBLIC MENTAL HEALTH | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **What restriction changed**	Supply, structure and treatment, all at once
3 **Substances**	Alcohol, opioids, tobacco, and the services
2 **Behavioural addictions**	Gaming, screen use, online gambling
3 **Theories of gambling**	The table, three accounts and one that joins them

THE ORGANISING SENTENCE

The pandemic did not create a new disorder. Movement restriction removed three things at once: supply, daily structure, and access to treatment. Which of the three moved first decides what was seen, and that sentence belongs before any list.

- **Alcohol.** Where sale stopped abruptly, restriction acted as an unplanned withdrawal experiment: withdrawal, seizures and delirium in the first days, with recourse to surrogate and illicit alcohol. Where sale continued or resumed, home drinking rose instead. Which pattern applied followed local sale policy, not the virus.
- **Opioids, tobacco, services.** Supply interruption and closed clinics broke opioid substitution therapy, so take-home dosing was widened to hold patients in treatment. Tobacco restriction produced both quit attempts and relapse. Outpatient and harm-reduction services closed or moved to teleconsultation, and relapse rose again as restriction lifted.
- **Behavioural addictions.** Screen use, gaming and online gambling rose with school closure and confinement, and betting platforms filled the space that closed venues left. ICD-11 already recognises gambling disorder and gaming disorder as disorders due to addictive behaviours, so the diagnosis was available where the behaviour disabled.

THEORY	THE MECHANISM IT PROPOSES	WHAT IT EXPLAINS BEST
Learning	Variable ratio reinforcement; a near miss reads as a win	Persistence through steady loss
Cognitive	Gambler's fallacy, illusion of control, recall biased to wins	Why able people keep playing
Neurobiological	Mesolimbic dopamine reward, shared with substance use	Its DSM-5 move beside substance use
Integrative	Pathways model: conditioned, emotionally vulnerable, impulsivist	Matching treatment to the route

WHAT EARNS THE MARKS

- **Supply, structure and treatment named as three losses,** before any substance.
- **The alcohol answer given both ways,** since direction followed local sale policy.
- **Gambling and gaming among the addictive behaviours,** not impulse-control.
- **The integrative model named last,** which lifts the limb above a list of labels.

SEE ALSO Slot IV - 05 electronic media in management Slot IV - 06 adolescent self-harm and suicide

IV - 10

What is a school mental health programme? What are its benefits? Discuss in brief any two Indian school mental health programmes. [2+2+3+3]

2 + 2 + 3 + 3 | COMMUNITY AND PUBLIC MENTAL HEALTH

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	Delivered through the school, not relocated into it
2 The three tiers	Universal, selective, indicated
3 The benefits	Reach, timing, access, stigma, transfer
3 Two Indian programmes	The table: who runs it, and what it does

THE DEFINITION

A school mental health programme is a planned set of activities delivered in and through the school, by teachers and school staff with specialist backup, to promote well-being, build life skills, identify difficulty early, and link the pupils who need care to a service. The delivery agent is the definition: a clinic relocated into a school building is not one.

- **The three tiers.** Universal: whole-school life skills teaching, a policy on bullying, and a staff room trained to notice. Selective: groups at raised risk, such as bereavement, learning difficulty or examination pressure. Indicated: named pupils identified, referred and followed up. Naming the tiers separates a programme from a talk.
- **The benefits.** Reach, because the age band is already assembled. Timing, because most disorders begin here and the school sees the change before the family names it. Access without a family having to seek a psychiatric service. Less stigma, because it arrives as education. Skills that transfer. And attendance and academic gain, which is what keeps a school engaged.

PROGRAMME	WHO RUNS IT	WHAT IT DOES	THE LIMIT
Rashtriya Kishor Swasthya Karyakram, 2014	Health and Family Welfare	Adolescent health with mental health as a strategic area; peer educators, and adolescent friendly health clinics	Health-system led, so school reach needs local linkage
Manodarpan, 2020	Education	Psychosocial support for students, teachers and families: a helpline, a counsellor directory, advisory handbooks	Helpline led, so thin at the universal tier
School component of the district programme	State health, district team	Life skills education and counselling in schools, with the district team as clinical backup	Needs a district team that is rarely complete

WHAT EARNS THE MARKS

- **The delivery agent named in the definition,** which is the distinction being tested.
- **The three tiers,** since the benefits differ by tier and a flat list cannot show it.
- **Two named programmes with the ministry behind each,** not two descriptions of life skills teaching.
- **A stated limit per programme,** which is what discuss asks for beyond naming.

SEE ALSO Slot IV - 06 adolescent self-harm and suicide Slot IV - 01 the District Mental Health Programme SPRINT Day 39, Community psychiatry and public health

IV - 11

What are the common myths and misconceptions about LGBTQIA+ identities such as intersex and Hijra? What are the basic tenets of LGBTQIA+ affirmative counselling? [4+6]

4 + 6 | COMMUNITY AND PUBLIC MENTAL HEALTH | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Three axes, kept apart	Orientation, identity, sex characteristics
2	The myths, corrected	The table, each against what is settled
3	The stance	Not a disorder, minority stress, who learns
3	The work, and the refusal	Disclosure, risk, access, conversion

THREE AXES, AND MOST MISCONCEPTIONS ARE ONE READ AS ANOTHER

Sexual orientation is who a person is drawn to. Gender identity is the gender a person knows themselves to be. Sex characteristics are anatomy, chromosomes and hormones, and intersex is variation in those, present from birth. The three vary independently.

THE MISCONCEPTION	WHAT IS SETTLED	WHERE THAT STANDS
These identities are mental disorders	Homosexuality left the classification in 1990; ICD-11 places gender incongruence outside the mental disorders chapter	Classification
Intersex and transgender are the same	Intersex is bodily variation from birth, not a claim about identity	Definition
Hijra is the Indian word for a trans woman	A South Asian community with its own kinship, not a translation	Social, not clinical
Upbringing causes it, so it can be changed	Conversion practice has no evidence of benefit and is associated with harm	Ruled professional misconduct

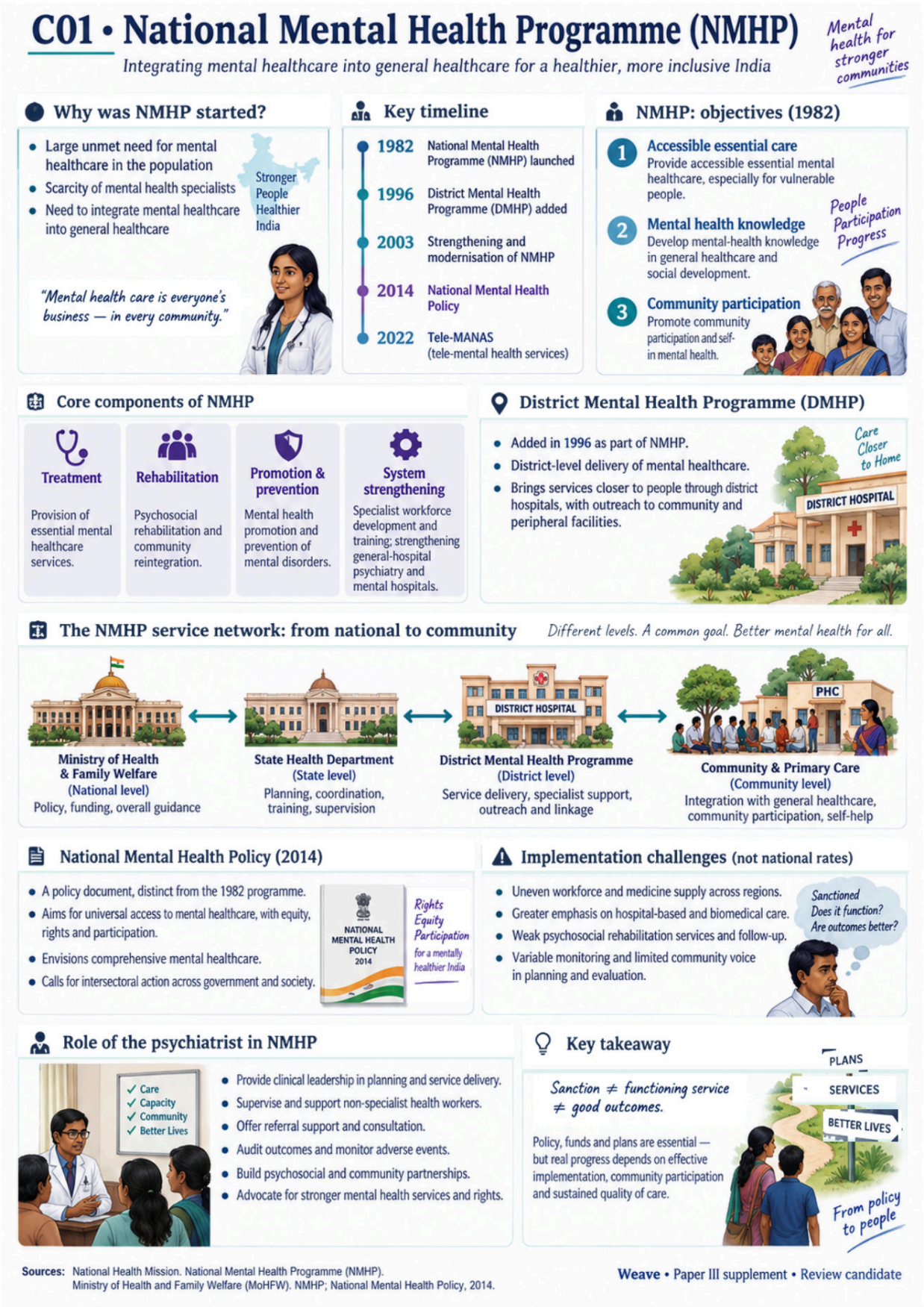
- **The stance.** The identity is not the presenting problem and is never a target of treatment. Minority stress is the working model: discrimination, rejection and violence from outside, and concealment, expected rejection and internalised stigma from within, together account for the raised rates of depression, anxiety, substance use and suicidality.
- **The conduct.** The therapist does the learning, not the client: know the terms, the local community structures and the law before the session, and use the person's own name and pronouns. Disclosure belongs to the client, so never out a person to family, school or employer, and let coming out be paced by them.
- **The work, and what is refused.** Screen for family coercion and forced marriage, violence, homelessness, unsafe hormone sources and suicide risk. Treat the presenting disorder on its own terms. Support access to gender-affirming care and to legal recognition, since identity is self-perceived in law and not certified by a psychiatrist. Refuse conversion practice plainly, including when a family asks for it.

WHAT EARNS THE MARKS

- **The three axes separated before any myth is answered,** since most of the myths are a category error.
- **Hijra treated as a social identity,** not as a synonym for a clinical term.
- **Minority stress named as the model,** which explains the raised rates without pathologising the identity.
- **Conversion practice refused in the answer,** and the refusal extended to the family who asks.

SEE ALSO Slot IV - 08 promotion of mental health Slot IV - 06 adolescent self-harm and suicide

WEAVE 100 · PAPER III · C01



National Mental Health Programme. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Community and public mental health.

Credits and references: retained within the illustration.

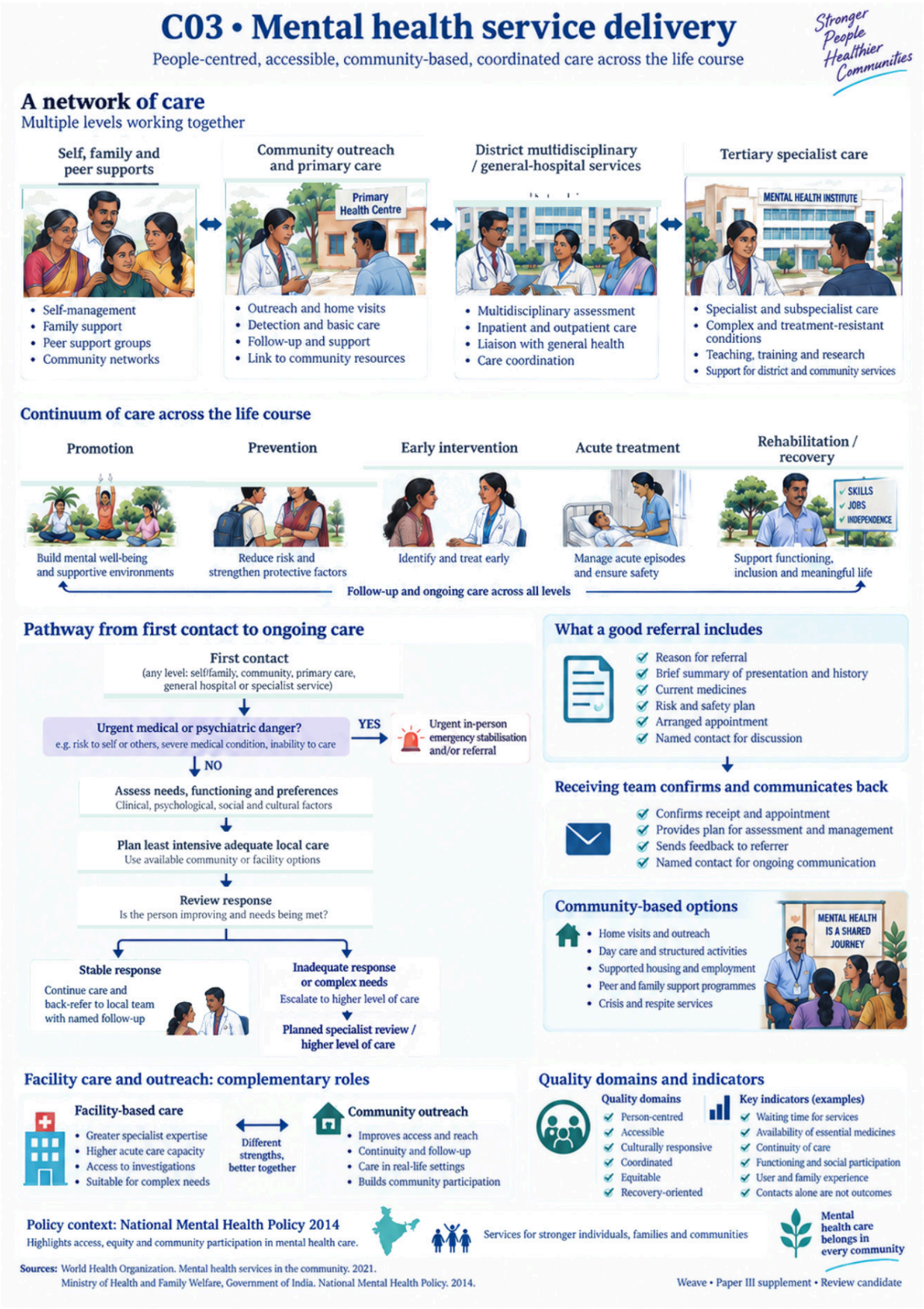
WEAVE 100 · PAPER III · C02



District Mental Health Programme. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Community and public mental health.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · C03



Community mental health service delivery. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Community and public mental health.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · C04

C04 • Telepsychiatry + Tele-MANAS

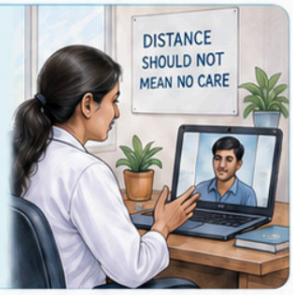
Bridging distances. Expanding access. People-centred mental health care.

Same care. More reach. Brighter lives.

What is telepsychiatry?

Provision of psychiatric care across a distance using communication technology.

- **Synchronous:** real-time audio/video interaction (e.g., video call, telephone).
- **Asynchronous:** store-and-forward exchange of information (e.g., messages, reports).



Tele-MANAS

National, **free, 24/7,** multilingual tele-mental-health service launched on **10 October 2022.**

Helpline: 14416
(Alternate: 1800-891-4416)

Listen
Support
Guide
Connect

Mental health support for every Indian, anytime, anywhere.

How Tele-MANAS works: call to care

A stepped, integrated model with back-linkages

1. Call



Person in need calls **14416** (alternate 1800-891-4416)

2. State/UT Tier 1



Trained counsellor provides initial support, assessment and guidance.
Tier 1 includes counsellors AND specialists.

3. Mental-health specialist (if needed)



Consultation with a mental-health specialist when indicated.

4. Tier 2 services



Linkage to DMHP, medical college or in-person service, or via eSanjeevani as appropriate.
Tier 2 is not simply all psychiatrists.

5. Documented follow-up

Advice, referrals and follow-up are documented, with back-linkage to ensure continuity of care.

Back-linkage for continuity of care

Clinical workflow (at each contact)

1. Verify identity, location and contact details.
2. Obtain consent and ensure privacy.
3. Assess suitability for tele-consultation and screen for immediate risk.
4. Provide remote assessment and care, or refer for in-person care if indicated.
5. Document advice, referrals and follow-up plan.

When NOT to rely on helpline-only management

In the following situations, arrange urgent local, in-person care (e.g., nearest health facility/emergency services):

- Emergencies or medical instability (e.g., risk to life or safety).
- When an adequate assessment cannot be done remotely; arrange in-person assessment, with urgency based on clinical need.

Safety first.



In-person care when needed.

Safeguards and key considerations

- Maintain privacy and confidentiality.
- Address digital access and language barriers (inclusive, multilingual support).
- Obtain valid consent.
- Keep appropriate records.
- Plan for local contingency if risk is identified.

Prescribing depends on consultation mode, assessment and applicable rules. Check current permitted and prohibited medicine categories.

Advantages

Care without borders

- Improves access to mental health care, especially in underserved areas.
- Supports continuity of care.
- Facilitates specialist reach and collaboration.
- Can reduce time, travel and other practical barriers.



Limitations

- Dependent on reliable connectivity and technology.
- Risk of digital exclusion for some populations.
- Incomplete physical and mental state examination in certain situations.
- Potential confidentiality challenges.

How to evaluate services

- Number of calls ≠ number of unique patients.
- Call volumes do not directly equal clinical outcomes.
- Evaluation should consider quality of care, appropriateness of referrals, follow-up and user experience — not just call counts.

Not the same as ...

Tele-MANAS and telepsychiatry are not the same as self-help mental health apps or standalone AI tools.
Apps/AI may support well-being, but are not substitutes for professional mental health assessment and care.



Tools can help. People care makes the difference.

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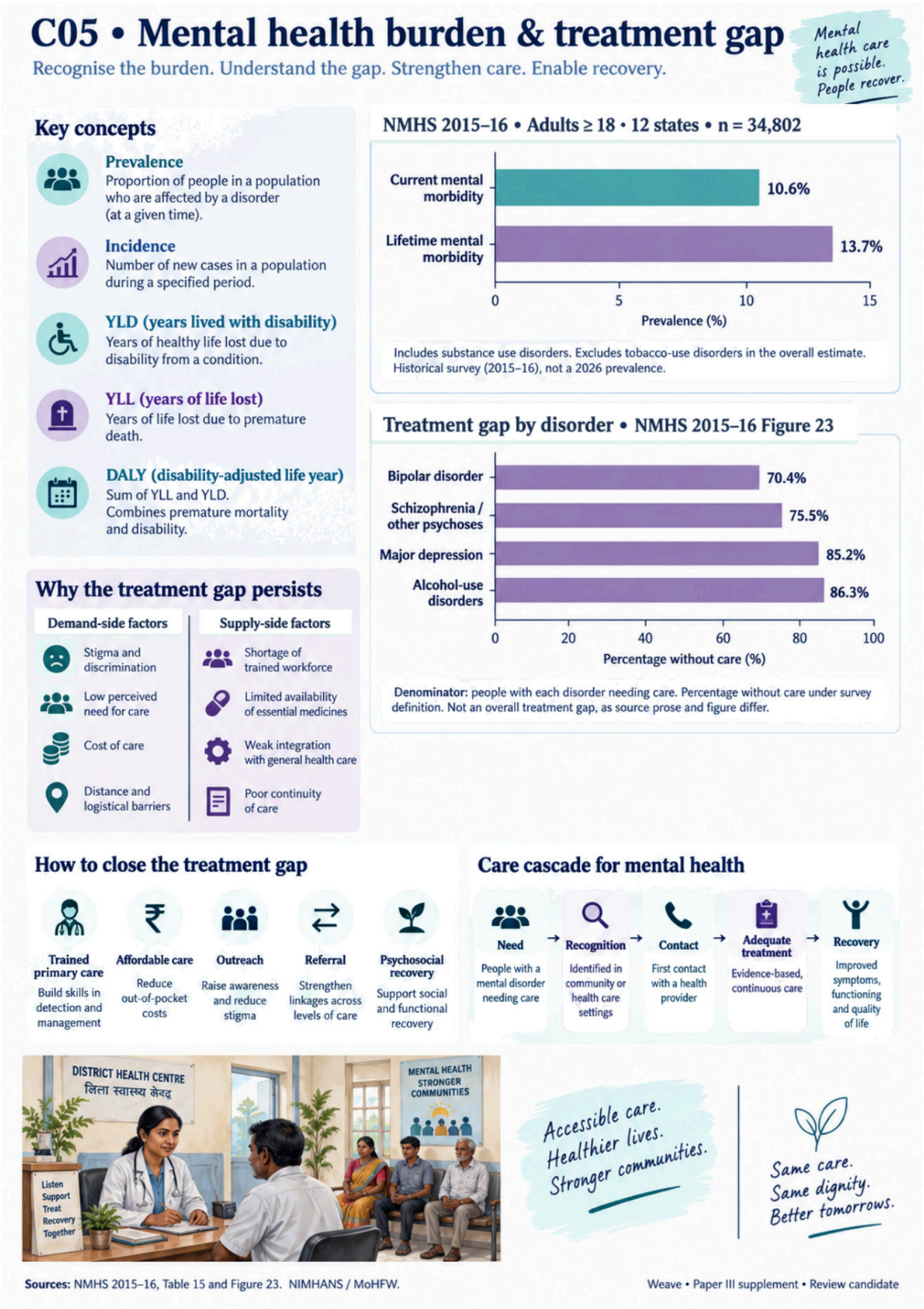
Mental health for a healthier India

Sources: PIB 2022/2024;
Telepsychiatry Operational Guidelines 2020 (check current prescribing rules).

Telepsychiatry and Tele-MANAS. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Community and public mental health.

Credits and references: retained within the illustration.

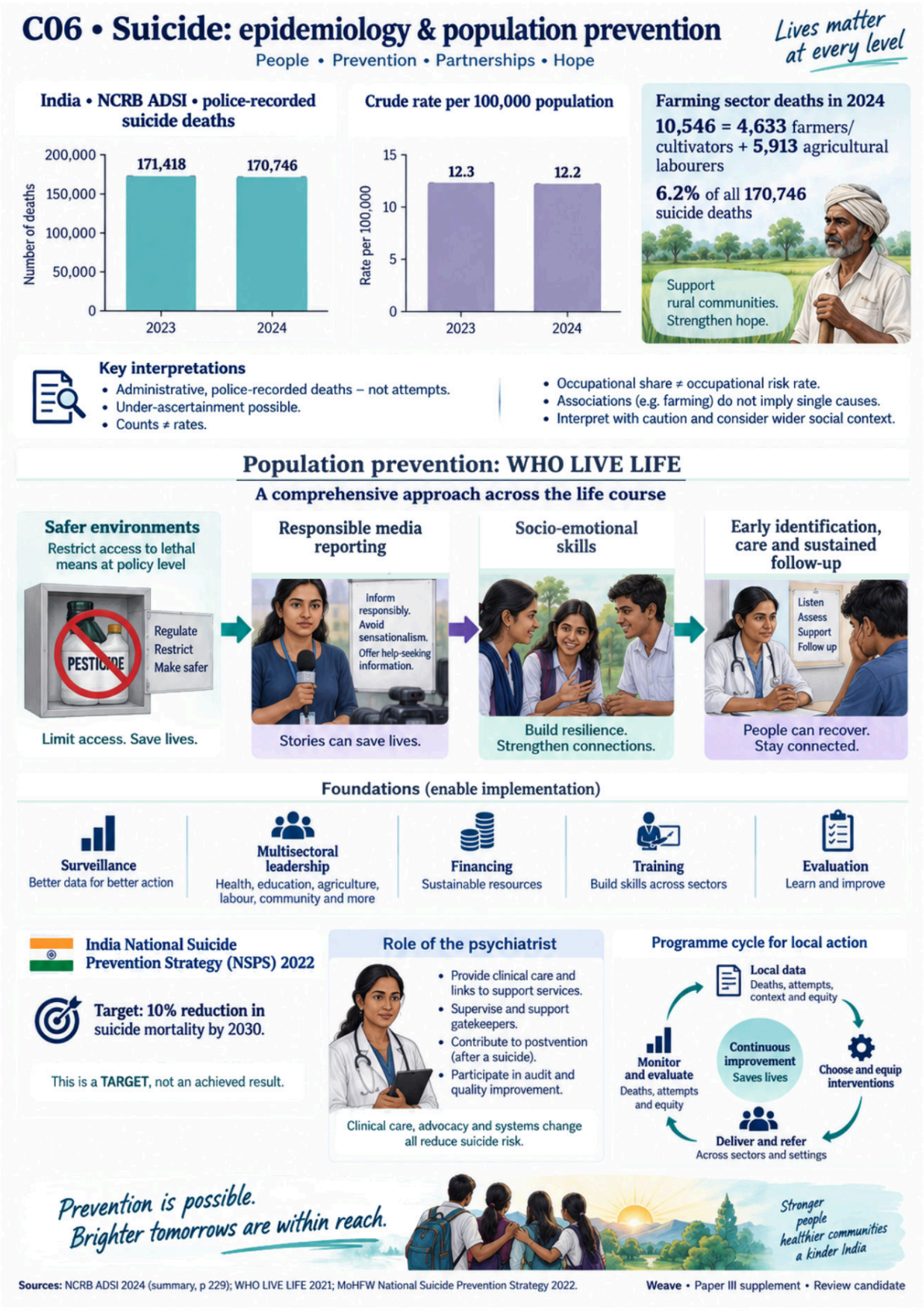
WEAVE 100 · PAPER III · C05



Mental health burden and treatment gap. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Community and public mental health.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · C06



Suicide epidemiology and population prevention. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Community and public mental health.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · C07

C07 • Student suicides & prevention

CONCERN • UNDERSTAND • RESPOND • PREVENT • SUPPORT

Students are more than their exams

Support today brighter tomorrows

The numbers (India, 2024)

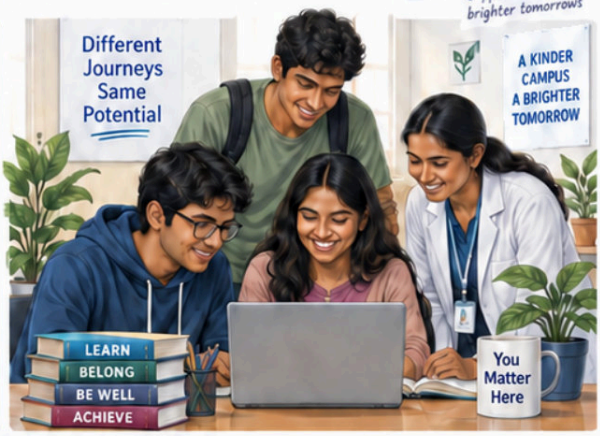
Share of all recorded suicides

14,488 recorded student suicide deaths

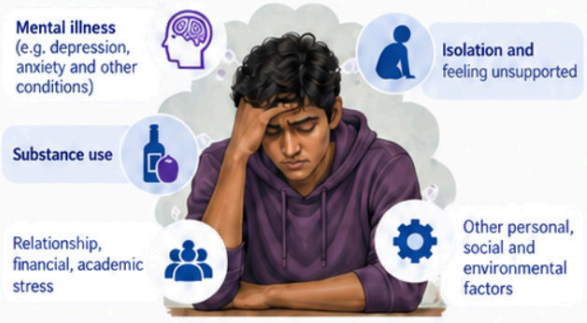
8.5% of all recorded suicide deaths

14,488 students out of **170,746** all recorded suicide deaths India, NCRB ADSI 2024.

Student is an occupational/status classification, NOT synonymous with youth, school pupils, college students, campus deaths or examination-related deaths. No student population rate available here.



Why it happens – multifactorial influences



Exam pressure alone does not explain student deaths.

Whole-institution prevention

Create environments where students can thrive

- Supportive learning climate
- Anti-bullying, ragging and discrimination
- Fair assessment and reasonable accommodations
- Accessible, confidential counselling
- Trained gatekeepers with clear referral pathways
- Means safety (without method details)
- Family and peer partnerships
- Continuity of support during transitions (e.g. joining, high-stakes periods, leave and return)

Safe Supportive Inclusive Learning for Life



What to do – action flow

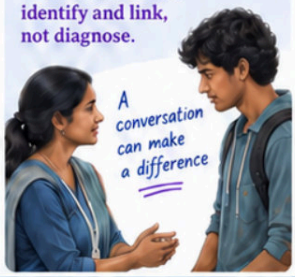
Practical steps when warning signs or distress are observed

Warning signs or distress observed (e.g. withdrawal, hopelessness, giving away valued items, expressions of self-harm) → **Private, compassionate enquiry** Include asking about suicide thoughts → **Immediate danger?**

YES → **Stay with the person** Urgent clinical/ emergency handover

NO → Timely assessment Collaborative safety plan Named follow-up and support

Peers and teachers identify and link, not diagnose.



Postvention – after a student's death

Care for the bereaved and reduce further risk



- Support bereaved students, family, staff and wider community
- Safe communication (accurate, sensitive, no glorification)
- Monitor and support vulnerable contacts
- Coordinated institutional response

Evaluation and monitoring

- Track referral completion, access to support and student wellbeing.
- Review and learn from relevant events.
- Counselling contacts are not proof of prevention.

"Ongoing, whole-institution efforts save lives."

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Sources: NCRB ADSI 2024 (p229); WHO LIVE LIFE 2021; Ministry of Education student wellbeing resources; Draft UMMEED 2023 (draft, not law).

Student suicide prevention. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Community and public mental health.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · C08

C08 • Stigma in mental health

People. Inclusion. Better mental health for all.

"Same potential. Same opportunities. A more inclusive tomorrow."

What is stigma?

Stigma involves negative beliefs, attitudes and actions towards people with mental health conditions. It is a major barrier to care, recovery and social inclusion.



Key distinctions *(all are examples of stigma)*

Stereotype	Prejudice	Discrimination
Generalised belief about a group.	Negative attitude or emotional judgement.	Unfair action or exclusion.
"People with mental illness are unreliable."	*"I feel uncomfortable around them."*	*"Not selected for the job."*

These are examples of stigma — not endorsed facts.

How stigma develops

A process, often within wider power structures.



Labelling	Stereotyping	Separation / status loss	Discrimination
"He has a mental illness."	*"They are unreliable."*	*"He is seen as different."*	*"He is excluded from opportunities."*

Occurs within social, cultural and institutional power structures

Types of stigma



Public stigma	Self-stigma (internalised)	Associative or courtesy stigma	Structural stigma
Negative beliefs and attitudes in the general community.	Applying negative beliefs to oneself (e.g. reduced self-esteem).	Stigma experienced by family members, carers or mental health staff.	Discriminatory rules, policies and unequal resources.

Consequences

Stigma can lead to:



Delayed help-seeking	Concealment of difficulties	Reduced opportunity	Disengagement from services and community

Important: these are common consequences, but not inevitable. Avoid causal determinism.

Interventions to reduce stigma

Different approaches target different levels.



Contact and accurate education	Peer support and empowerment	Inclusive workplaces and service policies	Family support
• Voluntary, supported and non-tokenistic contact with people with lived experience • Accurate, balanced education	• Peer-led support and empowerment • Builds self-acceptance and confidence	• Fair employment practices • Accessible, inclusive services • Address structural barriers	• Psychoeducation and support for families and carers • Reduces associative/courtesy stigma
↓ **Improves public attitudes**	↓ **Reduces self-stigma**	↓ **Tackles structural stigma**	↓ **Lessens associative effects**

WHO MOSAIC 2024 — three principles

Guides global action to end mental health-related stigma.

1 Lived-experience leadership
Meaningful involvement of people with lived experience at all levels.

2 Social contact
Voluntary, safe and supported opportunities for social contact.

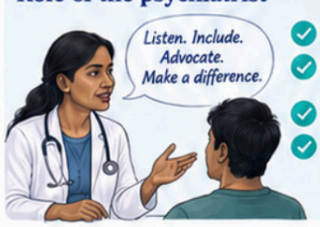
3 Inclusive partnerships
Collaborate across government, civil society and communities.

Measuring stigma

Use different tools for different aspects.

Knowledge	MAKS: Mental Health Knowledge Schedule
Clinician attitudes	MICA: Mental Illness: Clinicians' Attitudes
Reported/intended behaviour	RIBS: Reported and Intended Behaviour Scale
Experienced discrimination	DISC: Discrimination and Stigma Scale

Role of the psychiatrist



- Use respectful, person-first language.
- Share decisions and work collaboratively with patients.
- Examine and challenge your own biases.
- Advocate for equitable access to care, opportunities and rights.

From barriers to belonging



Mental health is a shared human experience. Stigma can change.

Weave • Paper III supplement • Review candidate

Sources: WHO MOSAIC 2024; WHO community mental-health services 2021.

Stigma prejudice and discrimination. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Community and public mental health.

Credits and references: retained within the illustration.

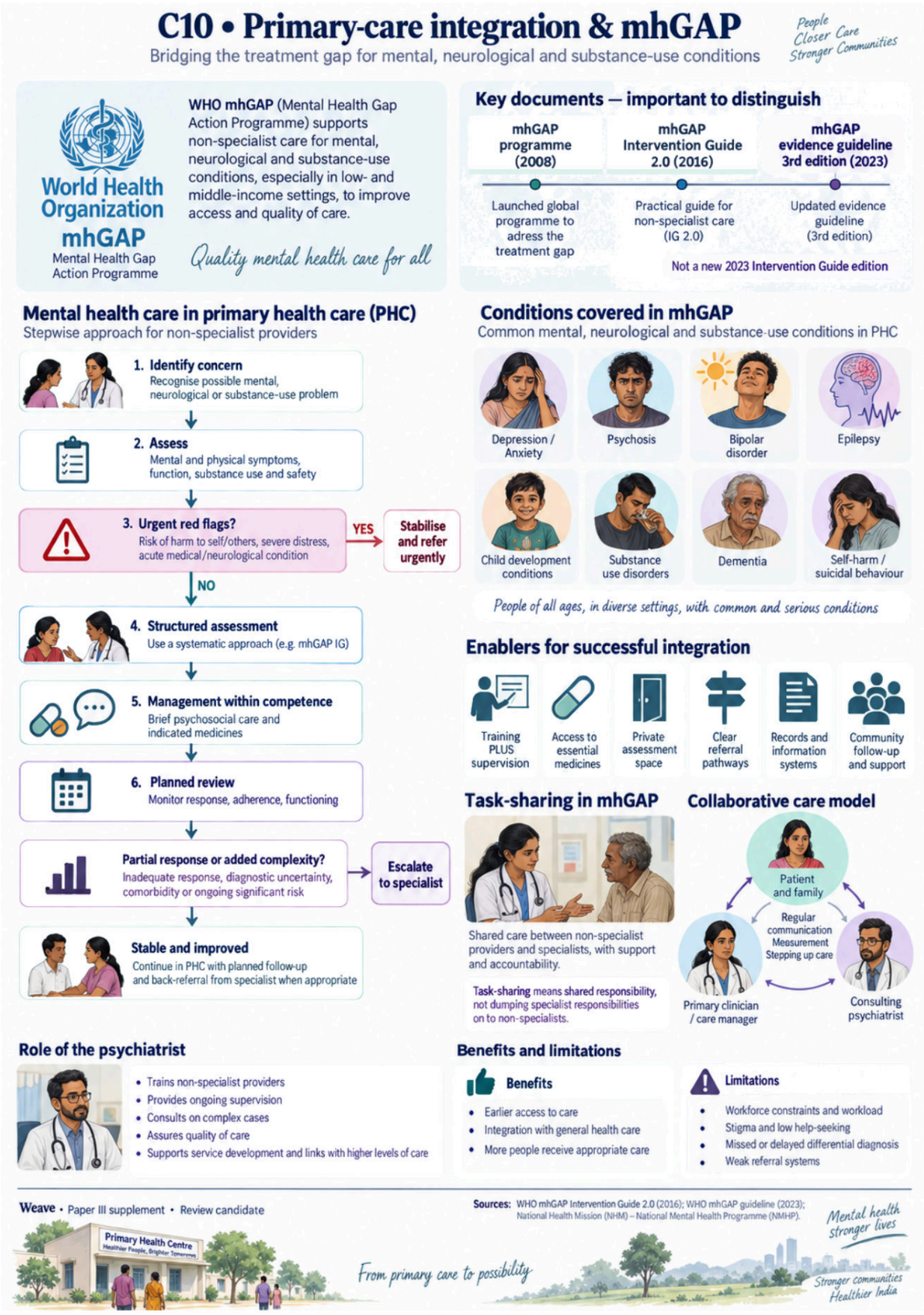
WEAVE 100 · PAPER III · C09



Psychiatric rehabilitation. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Community and public mental health.

Credits and references: retained within the illustration.

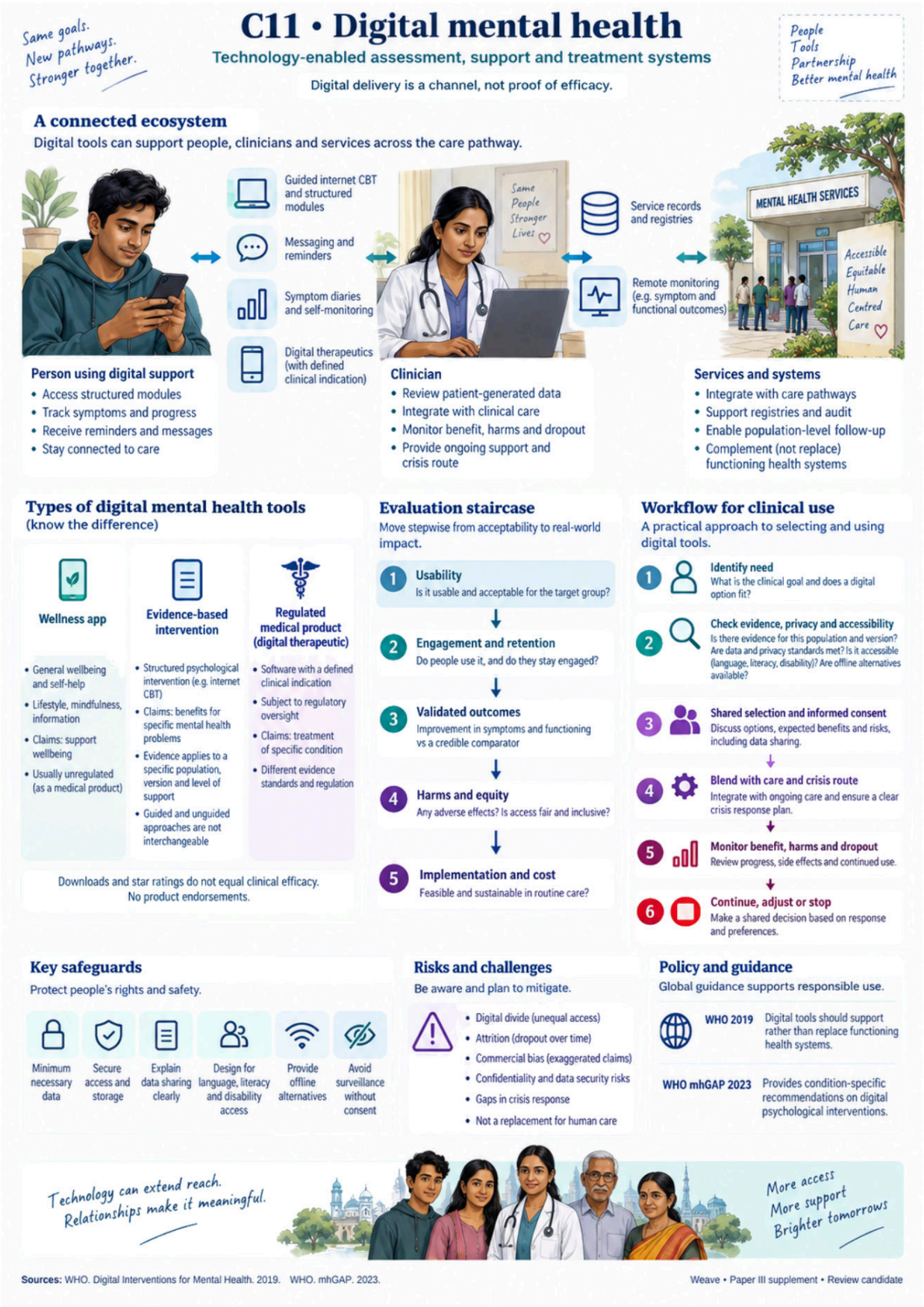
WEAVE 100 · PAPER III · C10



Primary care integration and mhGAP. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Community and public mental health.

Credits and references: retained within the illustration.

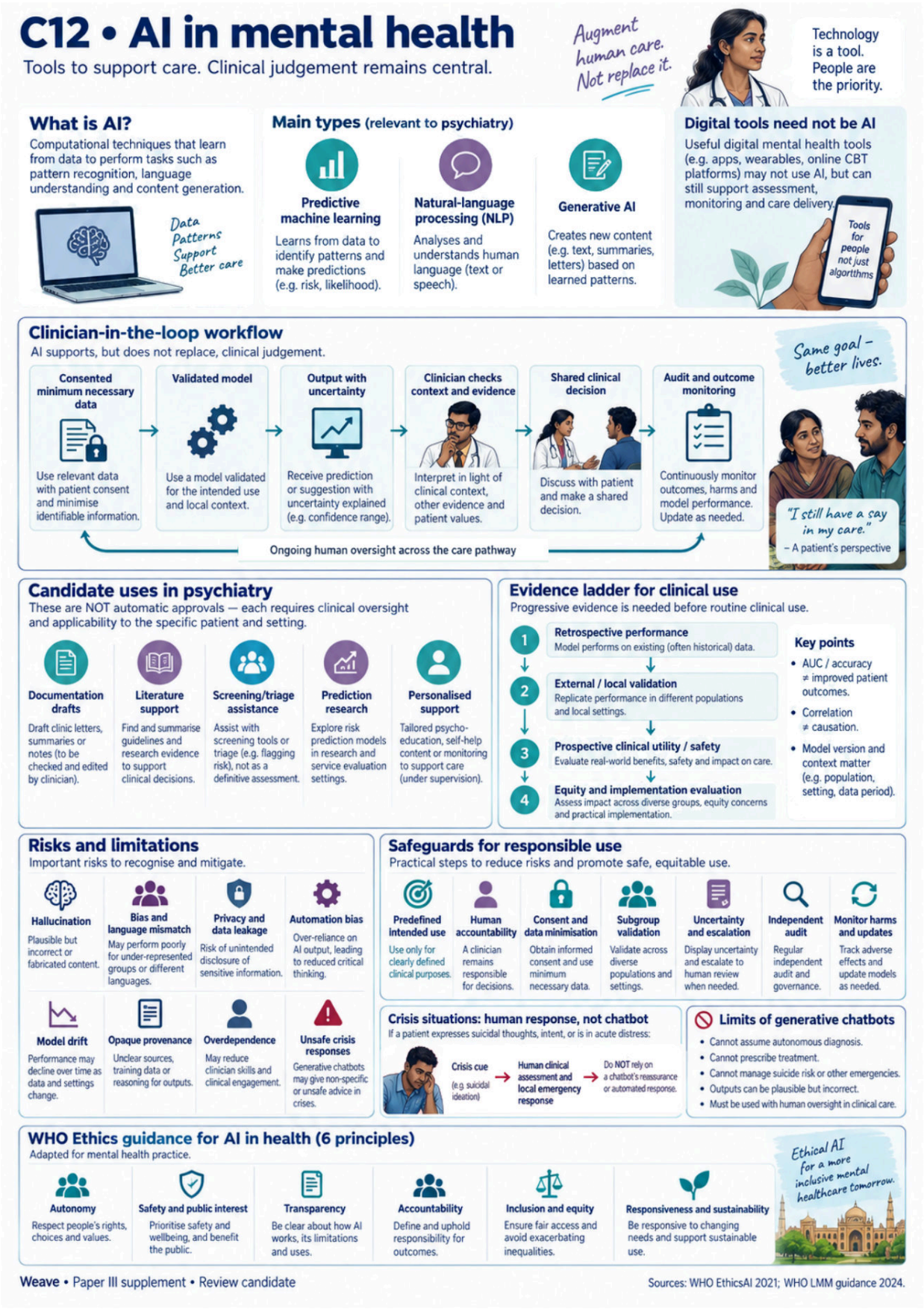
WEAVE 100 · PAPER III · C11



Digital mental health. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Community and public mental health.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · C12



AI in mental health. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Community and public mental health.

Credits and references: retained within the illustration.

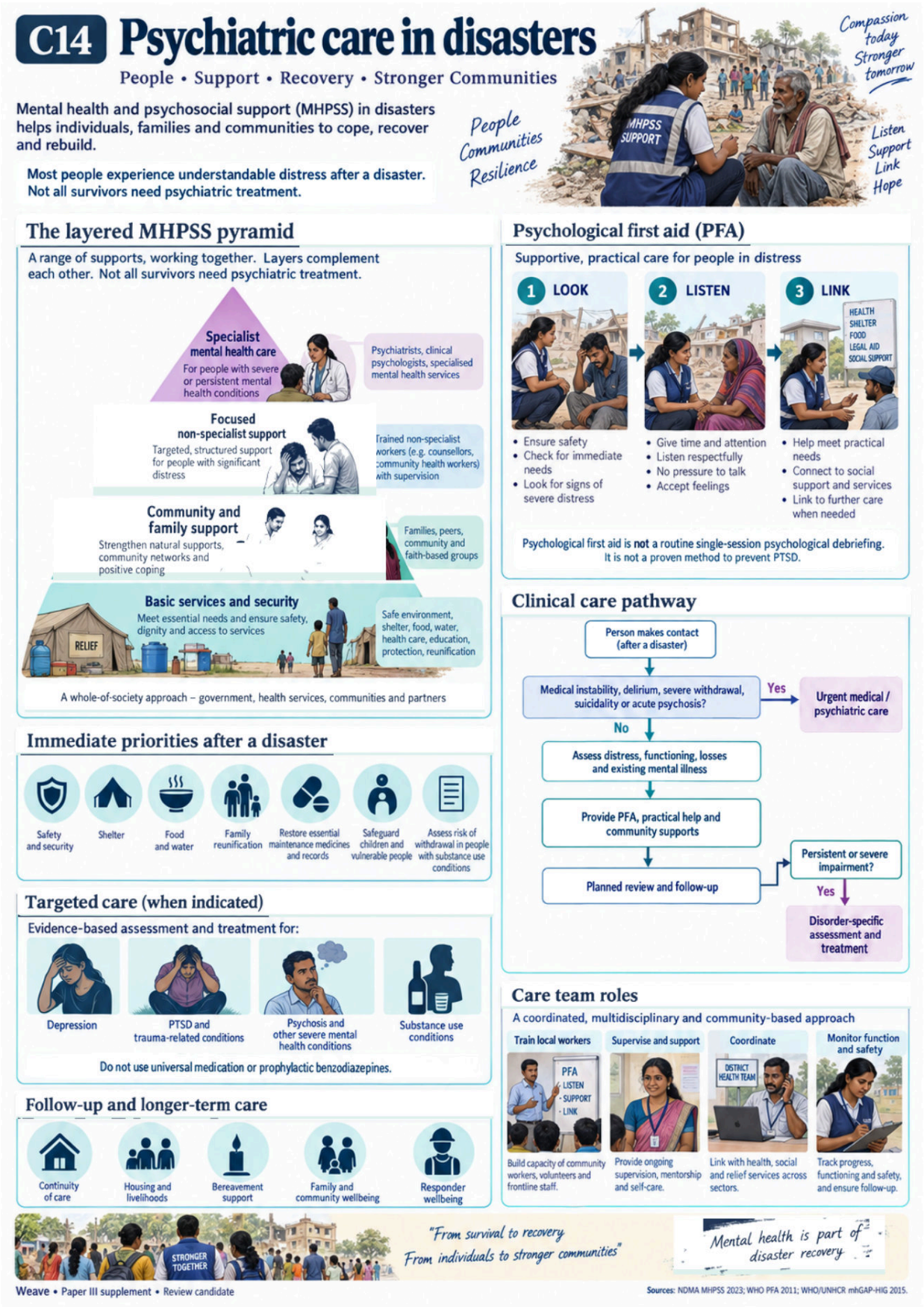
WEAVE 100 · PAPER III · C13



Disaster management overview and cycle. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Section companion: Community and public mental health.

Credits and references: retained within the illustration.

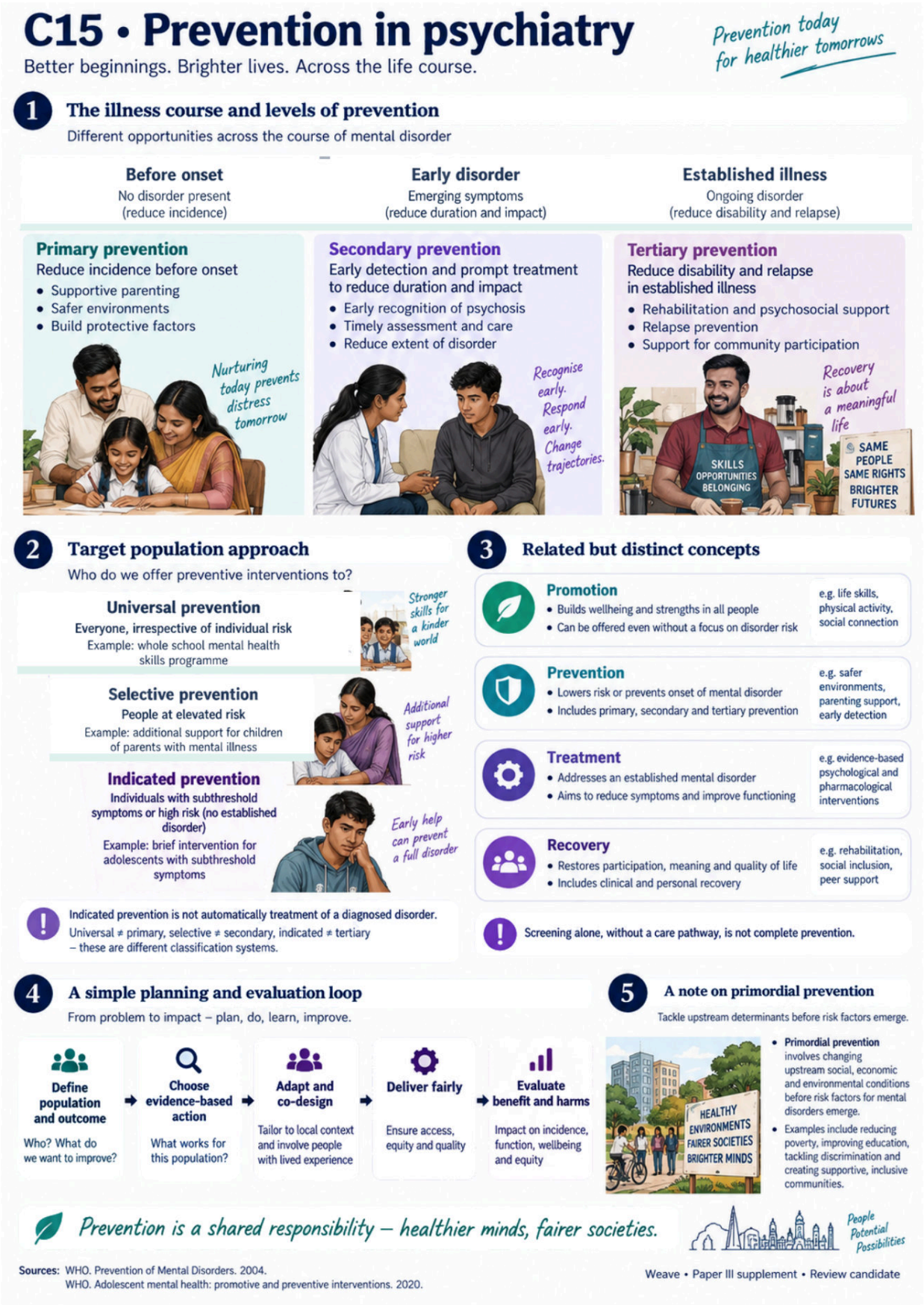
WEAVE 100 · PAPER III · C14



Psychiatric care in disasters. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Community and public mental health.

Credits and references: retained within the illustration.

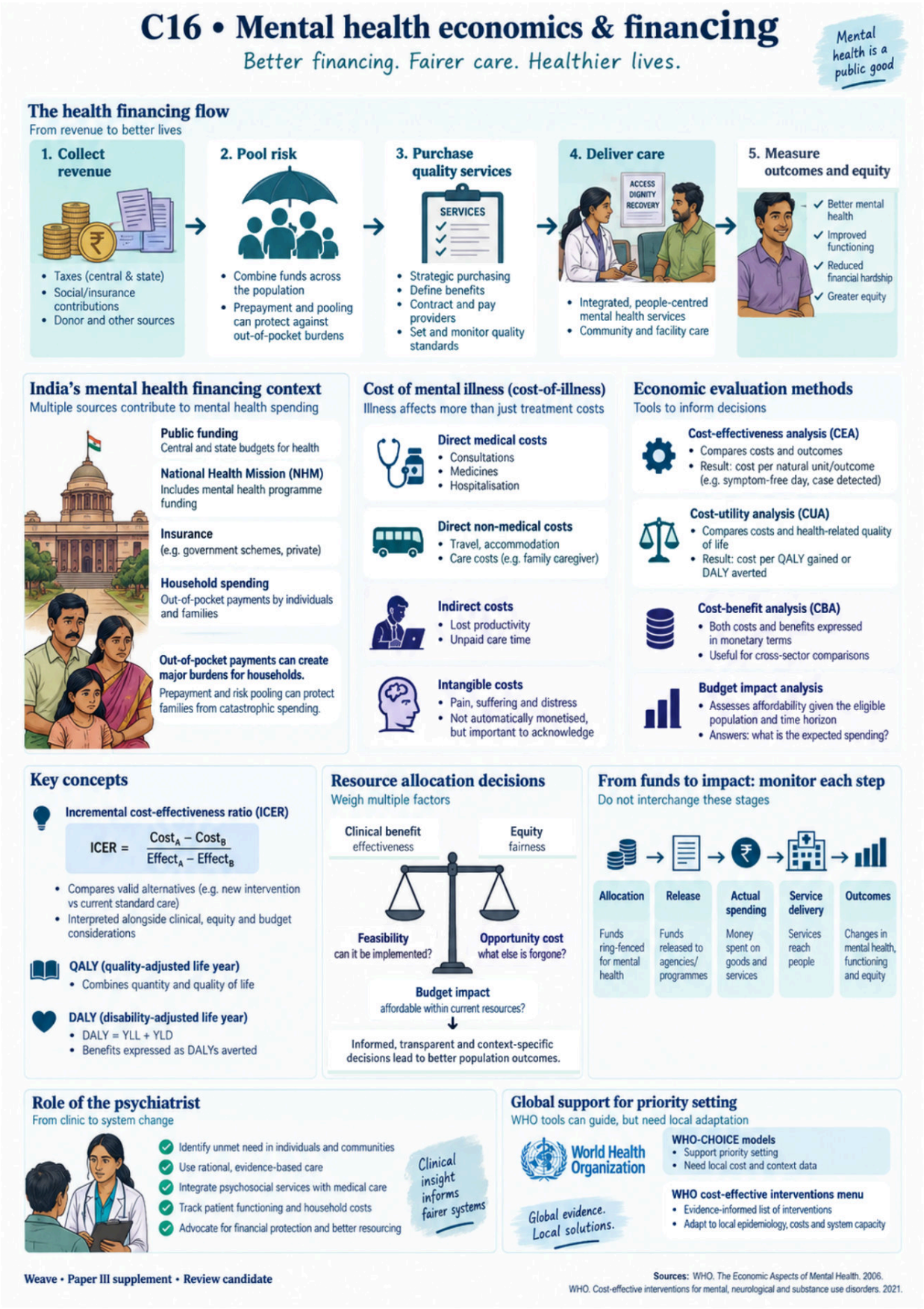
WEAVE 100 · PAPER III · C15



Basics of prevention. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Community and public mental health.

Credits and references: retained within the illustration.

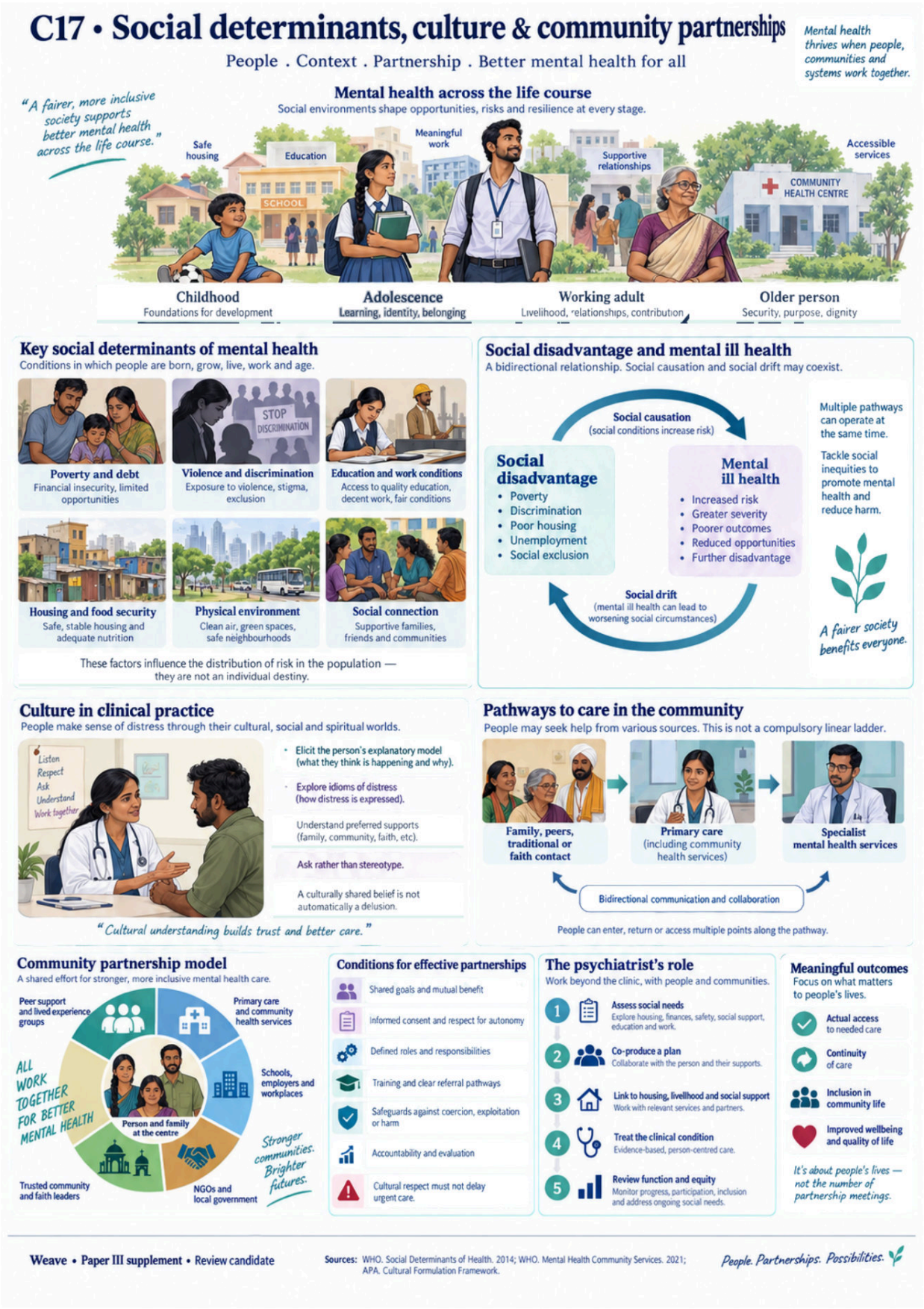
WEAVE 100 · PAPER III · C16



Mental health economics and financing. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Community and public mental health.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER III · C17



Social determinants culture and community partnerships. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Community and public mental health.

Credits and references: retained within the illustration.

AREA 02 OF 15 · P4-A03

Dementias: types and differential

10	11.4%	IV-12	IV-21
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 5 HIGH 2 SINGLE 3

REVISION ORDER

The two mild cognitive impairment slots together, they are one answer split and both fragments declare it. The two subcortical slots likewise, and the grid writes most of both. Frontotemporal and Lewy body dementia next as a pair, with pseudodementia directly on top of them because it is the differential they share. Prion disease, the Alzheimer prevention slot and the cholinesterase inhibitor slot are independent and each stands alone.

WHAT IS IN THIS AREA

Mild cognitive impairment in two slots, one asking for the definition, the subtypes and the assessment and one for the presentation, the course and the management. Frontotemporal dementia and Lewy body dementia. The subcortical dementias in two slots, one as a grid against the cortical picture and one as the concept, the circuit anatomy and the causes. Then pseudodementia, prion disease, the risk factors and prevention of Alzheimer disease, and the cholinesterase inhibitors with their indications and contraindications.

IV - 12

Mild cognitive impairment [10]

10 DEMENTIAS: TYPES AND DIFFERENTIAL

KNRUHS, KUHS, MUHS, NTRUHS, RGUHS - 8 APPEARANCES, 8 OF 78 SESSIONS

SKELETON

2 Definition	Decline beyond age, with independence still intact
2 Subtypes	Amnestic or not, one domain or several
3 Assessment	Informant, testing, and the reversible causes
3 Course and management	Three possible futures, and what is actually done

THE DEFINITION

Cognitive decline greater than expected for age and education, in one or more domains, reported by the patient or an informant and preferably shown on testing, with everyday independence essentially preserved. It is neither normal ageing nor dementia, and the same state is called mild neurocognitive disorder in current classification.

SUBTYPE	WHAT IS AFFECTED	MOST OFTEN PRECEDES	NOTE
Amnestic, single domain	Memory alone	Alzheimer disease	**Highest conversion risk**
Amnestic, multiple domain	Memory plus another domain	Alzheimer or vascular disease	Commonest in clinics
Non-amnestic, single	Language, executive or visuospatial	Frontotemporal or Lewy body disease	**Memory can be normal**
Non-amnestic, multiple	Two or more, memory spared	Lewy body or vascular disease	Screen for parkinsonism

Amber cells are the discriminators. Naming the subtype is what turns a screening result into a prediction the examiner can mark.

- **Assessment.** Informant history is essential. A cognitive screen sensitive to mild deficits is preferred to a basic one, and reversible contributors are sought: depression, thyroid disease, vitamin B12 deficiency, sleep apnoea, alcohol and anticholinergic load.
- **Testing in Indian practice.** Education and literacy shift every cut-off, so use an instrument validated in the local language rather than a translated score read against foreign norms.

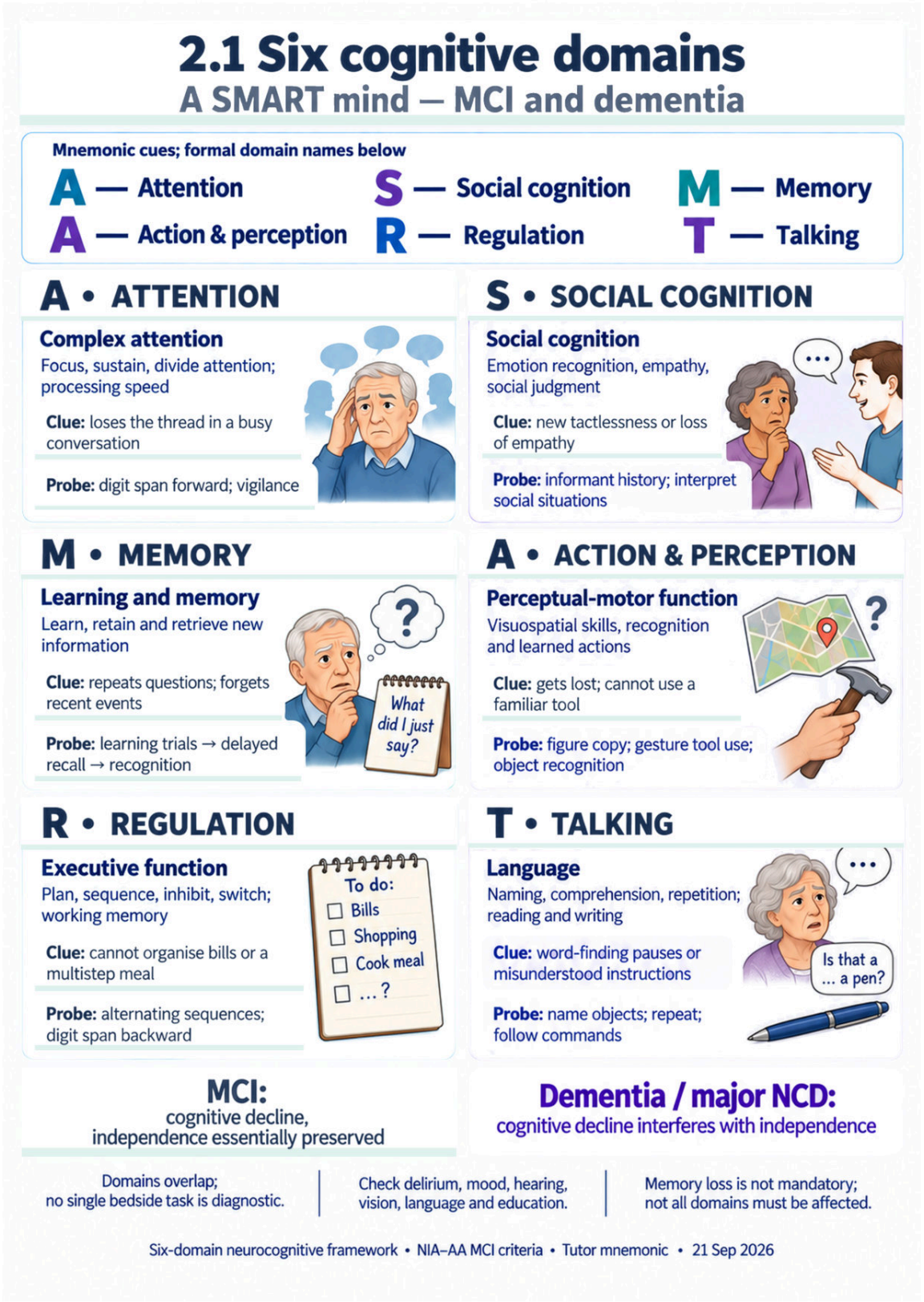
WHAT EARNS THE MARKS

- **Preserved independence written into the definition.** It is the single line that separates this from dementia.
- **Subtype named, with what it tends to precede.**
- **Reversible contributors listed,** because they are the part of the assessment that changes the outcome.

SEE ALSO Slot IV - 15 course and management of mild cognitive impairment Slot IV - 16 prevention of Alzheimer disease

SPRINT Day 32, Dementias & neurocognitive disorders

WEAVE 100 · PAPER IV · D2.1



Six cognitive domains - A SMART mind mnemonic. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-12.

Credits and references: retained within the illustration.

IV - 13

Etiology, Pathology, clinical features and management of Fronto-temporal Dementias. [10]

10 DEMENTIAS: TYPES AND DIFFERENTIAL

DNB, KNRUHS, KUHS, RGUHS, TN-MGRMU - 8 APPEARANCES, 8 OF 78 SESSIONS

SKELETON

2 **Aetiology**	About a third familial; three genes carry most of it
2 **Pathology**	Lobar degeneration, sorted by the protein inside
3 **Clinical features**	Behaviour and language first, memory relatively spared
3 **Management**	Nothing alters the course; name what genuinely helps

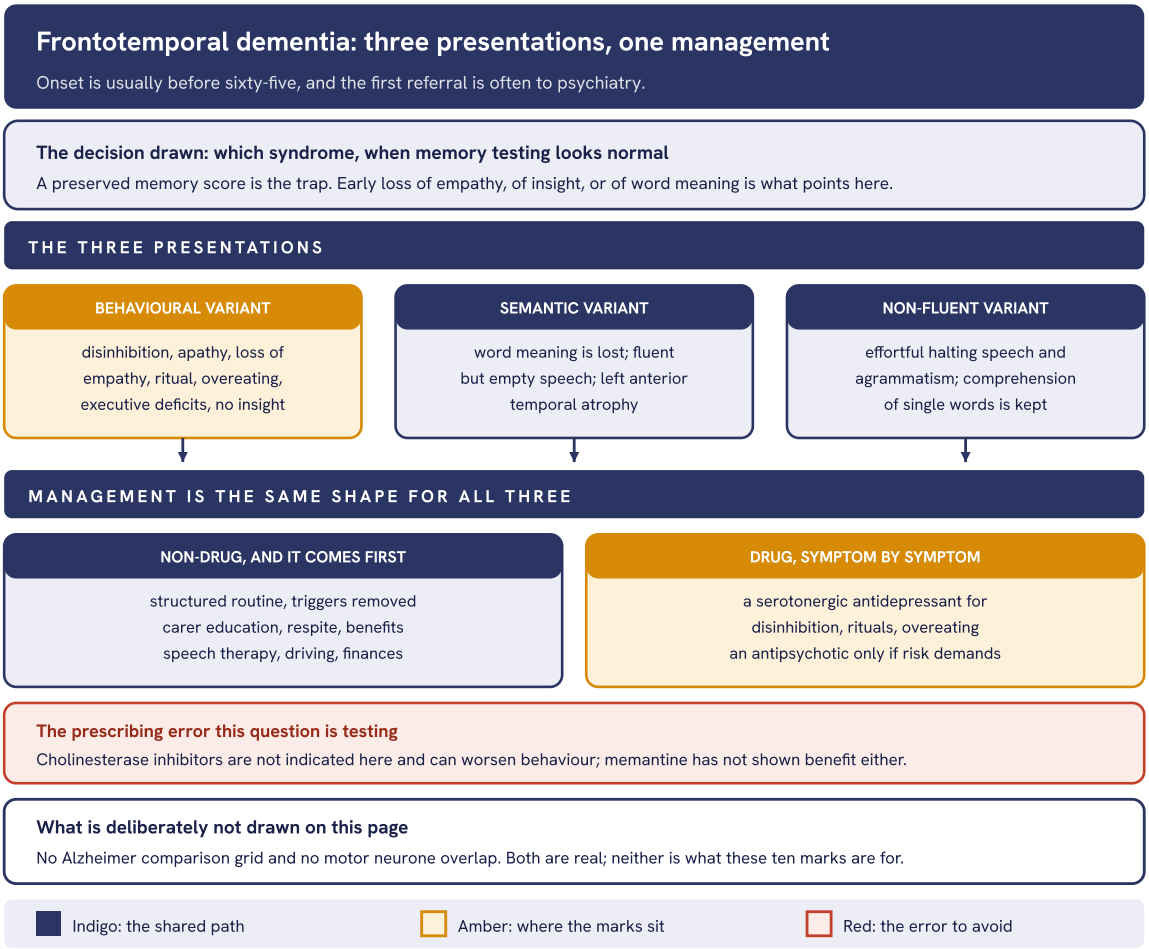


Fig 013.1 The three clinical presentations above, the single management shape below, and the prescribing error carried as a red strip because it is the line that separates this dementia from Alzheimer disease in practice.

- **Aetiology and pathology.** Around a third have a family history, most often through the MAPT, GRN or C9orf72 genes. The pathology is frontotemporal lobar degeneration, grouped by the protein in the inclusions: tau, TDP-43 or FUS.
- **Why it reaches psychiatry first.** Disinhibition, apathy and ritual look like mania, depression or obsessive compulsive disorder, and standard memory testing can be normal for years.

PITFALL

Waiting for a memory complaint. Episodic memory and orientation can test normally for years here, and insight is lost early, so the history that decides this diagnosis comes from the family rather than from the patient or the score.

WHAT EARNS THE MARKS

- **The three variants named separately,** not folded into one description of frontal behaviour.
- **Pathology given by inclusion protein,** which is what the word pathology in the stem is asking for.
- **Cholinesterase inhibitors explicitly excluded.** Most answers transplant Alzheimer treatment onto this page.
- **Carer support written as management,** because for this dementia it is most of it.

SEE ALSO Slot IV - 17 cholinesterase inhibitors Slot IV - 19 cortical and subcortical dementias **SPRINT** Day 32, Dementias & neurocognitive disorders

IV - 14

Clinical features of Lewy body dementia and its management. [10]

10 DEMENTIAS: TYPES AND DIFFERENTIAL

DNB, KNRUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

3	The core features	Four of them, and the biomarkers that support
2	Placing it	The one-year rule against Parkinson disease dementia
3	Drug management	Cholinesterase inhibitor first, and what is forbidden
2	Everything else	Sleep, falls, blood pressure, and the carer

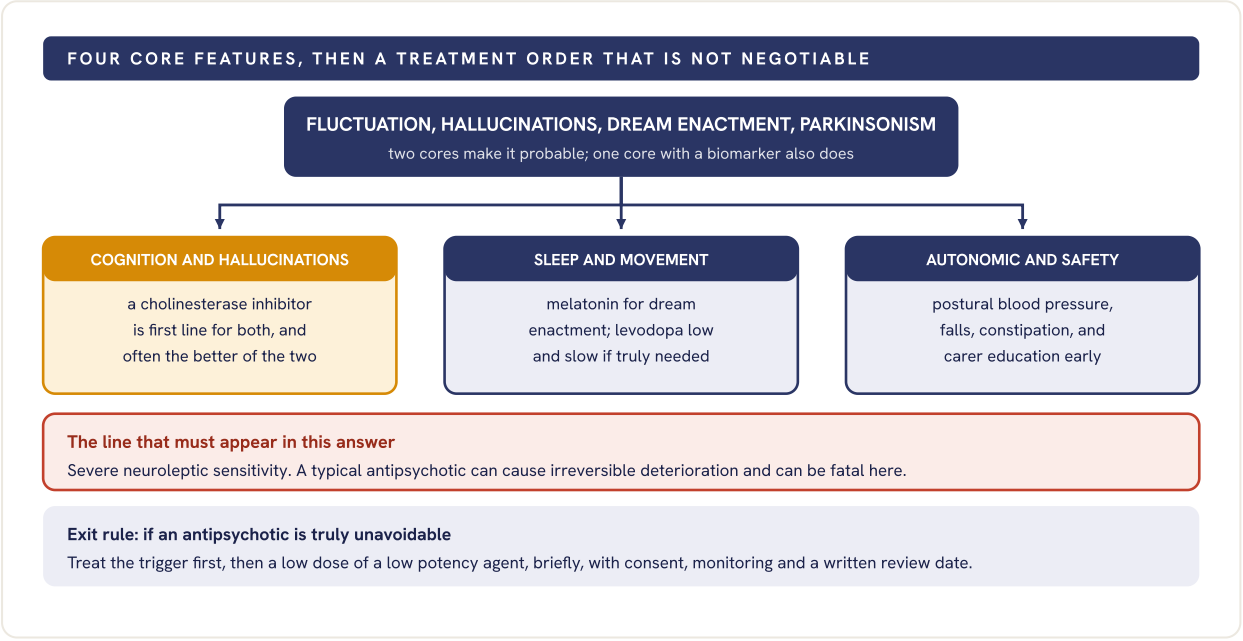


Fig 014.1 The four core features feeding three treatment strands, with neuroleptic sensitivity carried as a red strip because prescribing an antipsychotic here is the error that kills.

- **Supporting the diagnosis.** Reduced dopamine transporter uptake in the basal ganglia, abnormal cardiac MIBG scintigraphy, and sleep study confirmation of REM sleep without atonia.
- **The one-year rule.** Dementia before, or within a year of, parkinsonism is Lewy body dementia. Parkinsonism for longer than a year first is Parkinson disease dementia; the pathology is shared.

PITFALL

Visual hallucinations here are usually well formed and often non-threatening. Reaching for an antipsychotic rather than a cholinesterase inhibitor is the classic error.

WHAT EARNS THE MARKS

- **All four core features,** with dream enactment included; it is the one most often left out.
- **The one-year rule stated,** which shows the boundary is understood rather than memorised.
- **Neuroleptic sensitivity named as a danger,** not as a side effect.

SEE ALSO Slot IV - 17 cholinesterase inhibitors Slot IV - 19 cortical and subcortical dementias **SPRINT** Day 32, Dementias & neurocognitive disorders

4.1 Dementia comparison – Four major types

Feature	Alzheimer disease	Vascular dementia	Dementia with Lewy bodies	Frontotemporal dementia
Pathology	**Amyloid-beta plaques + tau tangles**	Cerebrovascular injury: infarcts, haemorrhage, small-vessel disease	**Alpha-synuclein:** Lewy bodies / neurites; AD co-pathology common	**FTLD:** tau or TDP-43; less often FUS. *MAPT, GRN, C9orf72* in genetic cases
Onset & course	Usually insidious, gradual progression	**Stroke-linked or stepwise;** small-vessel disease may be gradual	Progressive with **marked fluctuations in attention / alertness**	Insidious, progressive; often before 65, but older onset occurs
Early cognition	Typically **episodic memory:** impaired learning and retention	Often **processing speed, attention and executive function;** variable memory	**Attention, executive and visuoperceptual deficits;** memory may be less prominent	**Social cognition / executive function OR language;** memory may be relatively spared
Distinguishing clues	Repeated questions; forgotten recent events; later spatial and language problems	**Focal neurological signs, gait change,** temporal link to vascular events	**FOUR CORES:** fluctuations; recurrent formed visual **hallucinations; REM sleep behaviour disorder; spontaneous parkinsonism**	**Behavioural:** disinhibition, apathy, loss of empathy, rituals, hyperorality. **Semantic:** meaning lost. **Non-fluent:** grammar / speech production
Imaging / biomarkers	Medial temporal atrophy; amyloid / tau biomarkers when indicated	MRI vascular lesions consistent with clinical syndrome; burden and location matter	Indicative: reduced DAT uptake; reduced cardiac MIBG uptake; PSG-confirmed REM without atonia	Frontal / anterior temporal atrophy or hypometabolism; phenotype does not uniquely identify protein
Diagnostic anchor	Progressive decline with impaired independence; non-amnestic variants exist	**Cerebrovascular disease must plausibly explain the decline**	**Dementia + 2 core features OR 1 core + an indicative biomarker = probable DLB**	**Progressive behavioural or language syndrome +** functional decline; obtain informant history
Treatment focus	**Support + cholinesterase inhibitor** when indicated; memantine in appropriate stages. Selected early AD: specialist anti-amyloid eligibility assessment	**Vascular prevention + rehabilitation.** Cognitive drugs only with appropriate comorbid AD / PDD / DLB	**Donepezil or rivastigmine;** address sleep, autonomic symptoms and falls; cautious motor treatment	**Routine, carer support, speech therapy;** symptom-targeted SSRI / trazodone off-label
Exam trap	**Not all AD begins with memory loss**	**White-matter lesions alone do not establish vascular dementia**	**Severe antipsychotic sensitivity.** Dementia before / within 1 year of parkinsonism favours DLB	**Do not offer cholinesterase inhibitors or memantine for FTD**

Clinical clues are characteristic, not individually pathognomonic. Mixed pathology is common.

Source: Weave 100 IV + NIA; NICE NG97; DLB Consortium; AFTD • 21 Sep 2026

Dementia comparison - Alzheimer vascular Lewy body and frontotemporal. Read across the labelled contrasts to distinguish the related concepts. Read with IV-14.

Credits and references: retained within the illustration.

4.2 Dementia comparison — Four additional causes

Parkinson disease dementia • Huntington disease • Sporadic CJD • Normal-pressure hydrocephalus

Feature	Parkinson disease dementia	Huntington disease	Sporadic CJD	Normal-pressure hydrocephalus
Pathology / cause	Alpha-synuclein disease; degeneration of dopaminergic and other networks	HTT CAG-repeat expansion; autosomal dominant; striatal and cortical degeneration	Misfolded prion protein; spongiform degeneration	Disturbed CSF dynamics with ventriculomegaly; idiopathic or secondary
Typical course	**Cognitive decline after well-established Parkinson disease**	Progressive over years; cognitive / psychiatric changes may precede chorea	**Rapid progression over weeks to months**	Gradual gait and frontal-subcortical impairment
Cognitive profile	Executive, attention, visuospatial and retrieval deficits; fluctuations possible	Executive dysfunction, slowed processing, reduced flexibility and retrieval	Rapid multidomain decline; variable initial deficits	Slowed processing, attention / executive deficits and apathy
Clinical signature	**Parkinson motor syndrome precedes dementia by MORE THAN 1 YEAR**	**Chorea + behavioural / psychiatric change + cognitive decline**	Rapid dementia + myoclonus, ataxia or visual / pyramidal / extrapyramidal signs	**GAIT often first: short steps, initiation difficulty; cognitive decline + urinary urgency / incontinence**
Key tests	Cognitive and functional assessment; review medications and delirium; MRI if atypical	HTT genetic testing with counselling; MRI may show caudate atrophy	MRI DWI cortical / striatal restriction; CSF RT-QuIC; EEG may show periodic complexes	MRI ventriculomegaly with supportive morphology; objective gait testing before / after CSF drainage
Diagnostic nuance	**DLB and PDD share pathology; ONE-YEAR RULE is an operational clinical distinction**	Absent family history does not exclude HD; predictive testing requires a dedicated protocol	RT-QuIC is highly specific; MRI and EEG findings are not individually pathognomonic	Triad may be incomplete; ventriculomegaly alone is insufficient; negative tap test does not exclude
Management focus	Cholinesterase inhibitor, often rivastigmine; optimise motor drugs; support and safety	Multidisciplinary symptomatic care; treat chorea and psychiatric symptoms; swallowing / nutrition support	Urgent specialist evaluation; exclude treatable rapid dementias; supportive / palliative care	Specialist shunt assessment in selected patients; gait often improves more than cognition
Major caution	**Antipsychotic sensitivity; anticholinergic and dopaminergic drugs can worsen cognition / psychosis**	**Assess depression and suicide risk; genetic implications for family**	**Prion precautions for relevant procedures; do not miss autoimmune, infectious or metabolic mimics**	**Not every patient improves; weigh surgical risks and coexisting degeneration**

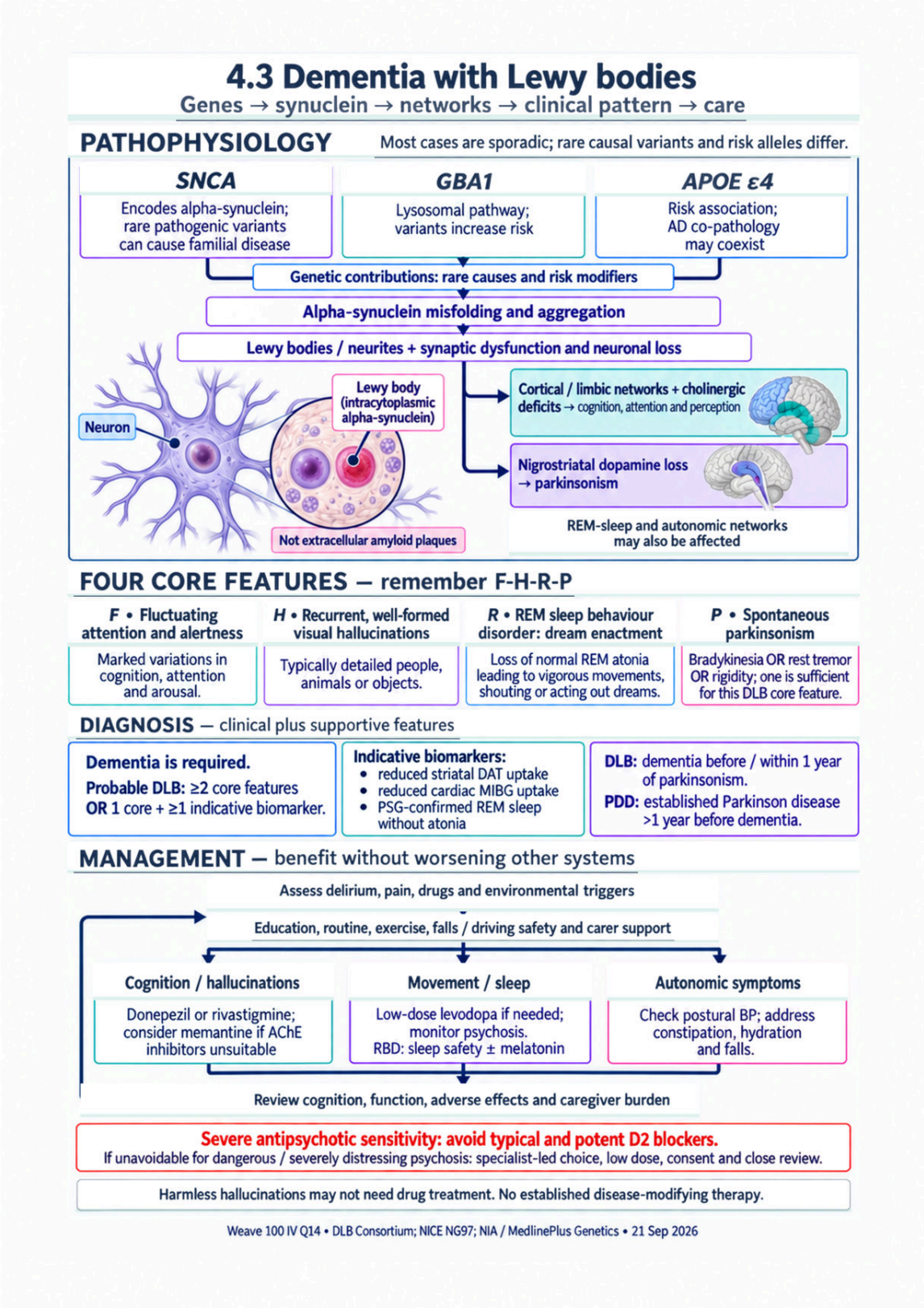
Clinical patterns guide investigation; none of these symptom clusters alone proves the cause.

MCI = preserved independence; dementia = cognitive decline interfering with independence.

Sources: NICE NG71; DLB Consortium; NINDS / GeneReviews; CJD and iNPH diagnostic guidance • 21 Sep 2026

Dementia comparison - Parkinson Huntington CJD and NPH. Read across the labelled contrasts to distinguish the related concepts. Read with IV-14.

Credits and references: retained within the illustration.



Dementia with Lewy bodies - Genes mechanisms diagnosis and care. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Read with IV-14.

Credits and references: retained within the illustration.

IV - 15

Definition, presentation, course and management of Mild Cognitive Impairment. [2+2+3+3]

2 + 2 + 3 + 3 | DEMENTIAS: TYPES AND DIFFERENTIAL

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

SKELETON

2	Definition	Decline present, everyday independence intact
2	Presentation	How it actually walks into the clinic
3	Course	Three futures, and what predicts which one
3	Management	Subtract, reduce, review; no drug is licensed

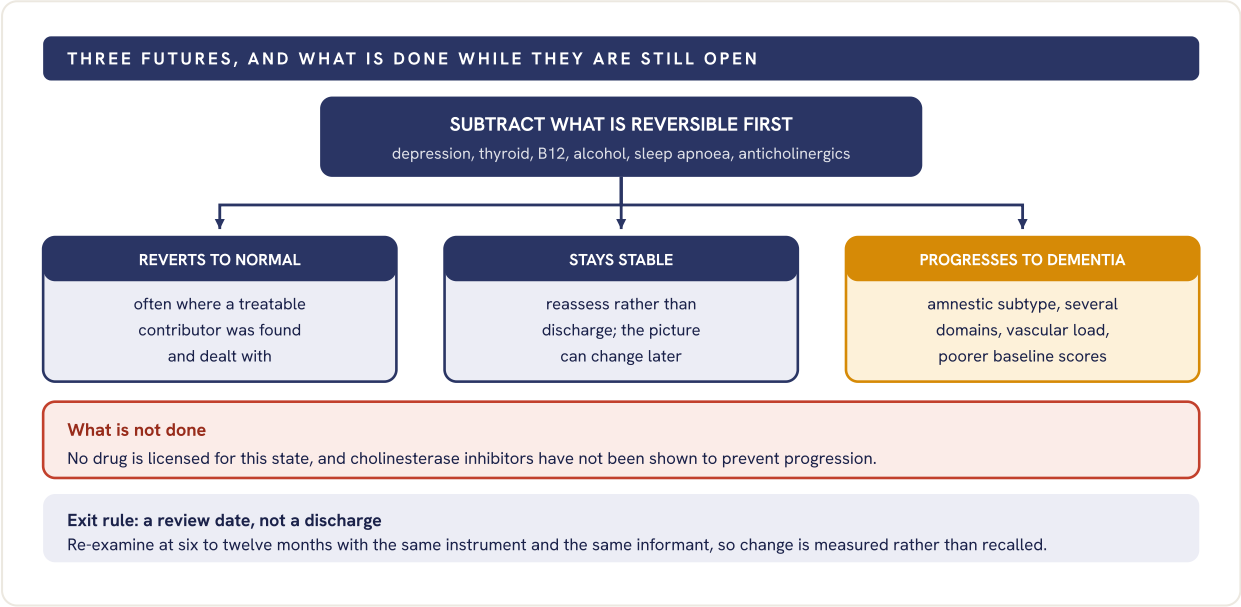


Fig 015.1 Reversible contributors are subtracted before the prognosis is discussed at all, and the three futures are then set against the features that predict each one.

- **Presentation.** A complaint of forgetting names, appointments or where things were put, corroborated by family, with daily tasks still managed, sometimes with lists.
- **Management, in three moves.** Treat what is reversible, reduce vascular risk, raise physical and social activity, then review. Hearing and blood pressure carry the best evidence.

PITFALL

Telling a family that this is early dementia. It is a state with three possible futures, and saying so accurately is both honest and clinically useful.

WHAT EARNS THE MARKS

- **Independence preserved, in the definition line.**
- **Three courses named,** with reversion included; most answers give only progression.
- **No licensed drug stated plainly,** and risk reduction given in its place.

SEE ALSO Slot IV - 12 mild cognitive impairment, subtypes and assessment Slot IV - 16 prevention of Alzheimer disease

SPRINT Day 32, Dementias & neurocognitive disorders

IV - 16

Discuss the risk factors and prevention of Alzheimer's disease. [10]

10 | DEMENTIAS: TYPES AND DIFFERENTIAL | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **The framing**	Risk accumulates across a life, so prevention has stages
3 **Not modifiable**	Age and genes, and what a genotype does not tell you
3 **Modifiable**	The named factors, placed at the stage they act
2 **Prevention in practice**	What has trial support, and what is only plausible

THE FRAMING THAT EARNS THE FIRST MARKS

Risk is not one list. It accumulates across early life, midlife and later life, and a prevention answer that mirrors those stages reads as understanding rather than recall. The Lancet Commission (2020) attributed roughly 40 per cent of dementia risk to 12 modifiable factors; the 2024 update raises this to about 45 per cent across 14.

STAGE	FACTORS	WHAT IS ACTUALLY DONE	NOTE
Early life	Less education	Schooling, literacy, cognitive reserve	Acts decades ahead
Midlife	Hearing loss, hypertension, obesity, head injury, excess alcohol	Hearing aids, blood pressure control, helmets, alcohol limits	**Largest single lever**
Later life	Smoking, depression, social isolation, inactivity, diabetes, air pollution	Cessation, treatment, activity, contact, glycaemic control	**Also good for the heart**
Not modifiable	Age, family history, APOE e4, trisomy 21, rare dominant mutations	Counselling, not screening	**Genotype is risk, not fate**

Amber cells are the discriminators. The stage of life a factor acts in is what turns a list into a prevention plan.

- **What has trial support.** Midlife blood pressure control has reduced later cognitive impairment in a randomised trial, and a multidomain programme of diet, exercise, cognitive training and vascular monitoring improved cognitive outcome in an older population at risk.
- **Treatment is not prevention.** The anti-amyloid antibodies slow decline modestly in early disease, carry imaging abnormality risk, and are not part of prevention or of routine Indian practice.

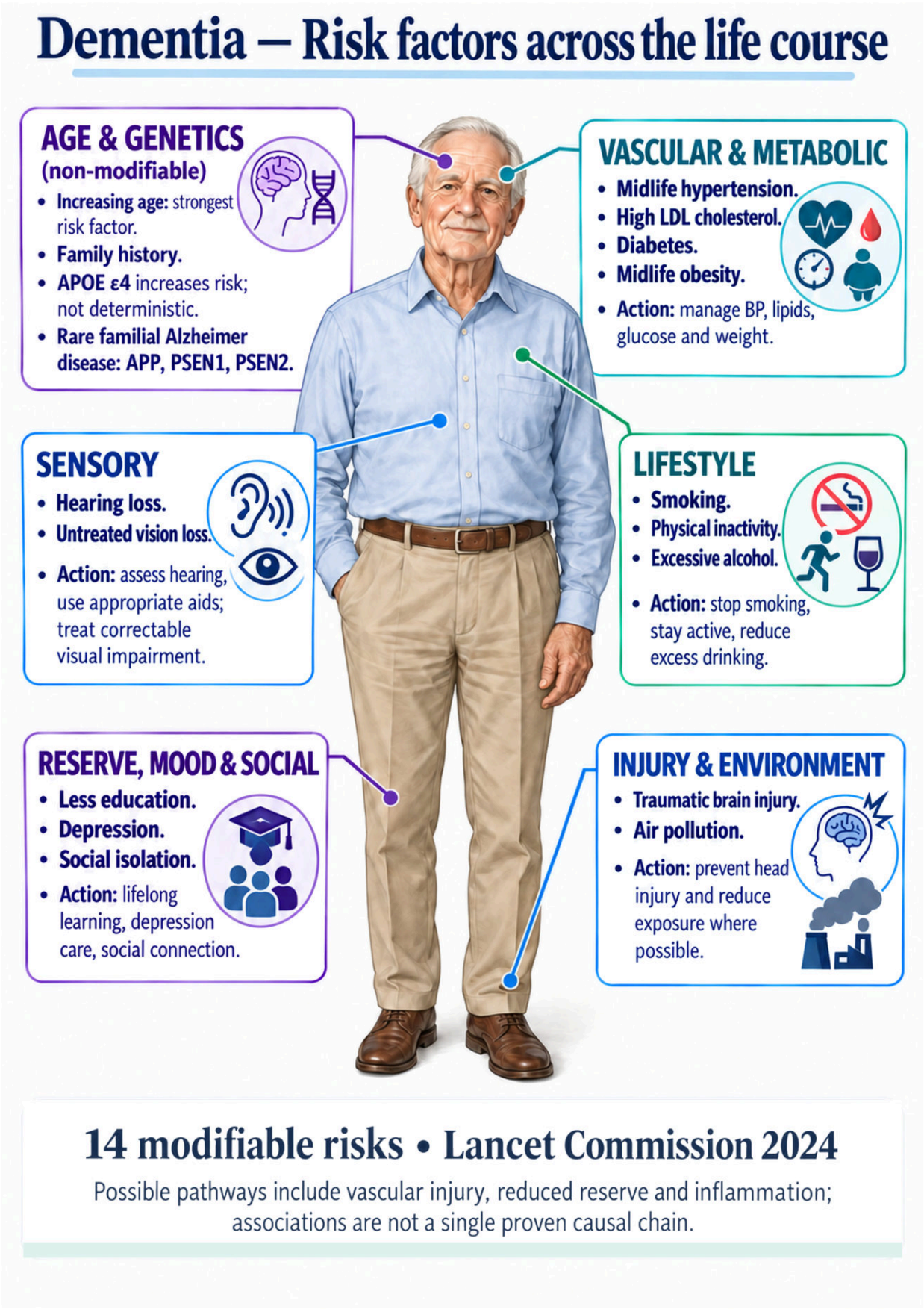
WHAT EARNS THE MARKS

- **Factors sorted by life stage,** which is the structure the marks are written around.
- **Genotype described as risk, not diagnosis,** with predictive testing not recommended.
- **Two named interventions with trial evidence,** separated from what is merely plausible.

SEE ALSO Slot IV - 12 mild cognitive impairment Slot IV - 15 course and management of mild cognitive impairment

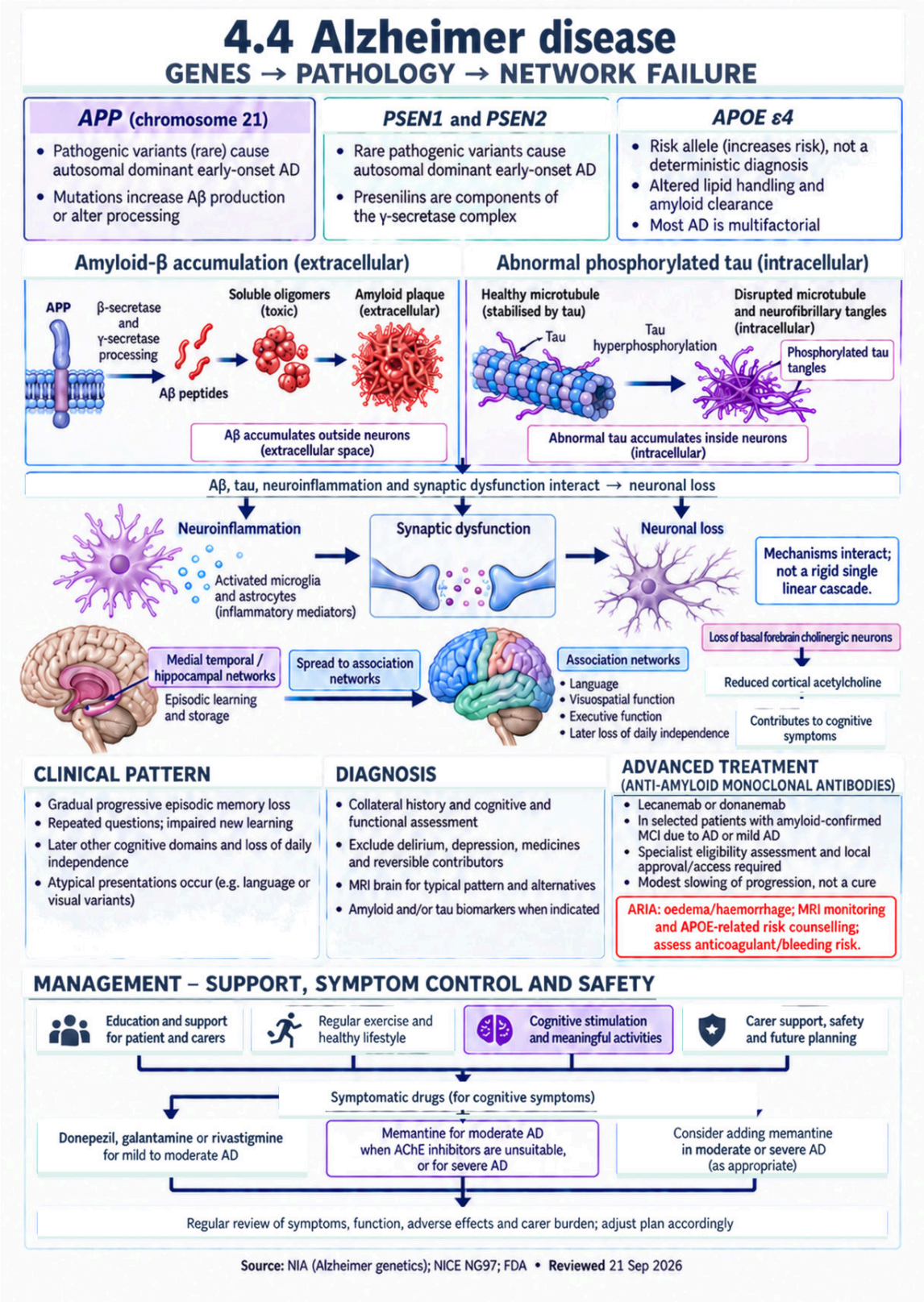
SPRINT Day 32, Dementias & neurocognitive disorders

WEAVE 100 · PAPER IV · D4.12



Dementia - Risk factors across the life course. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-16.

Credits and references: retained within the illustration.



Alzheimer disease - Genes pathology and care. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-16.

Credits and references: retained within the illustration.

IV - 17

Enumerate cholinesterase inhibitors and their indications and contraindications. [10]

10 | DEMENTIAS: TYPES AND DIFFERENTIAL | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The agents	Three in use, and how each one differs
3 Indications	Where they help, and the dementias where they do not
3 Contraindications	Heart, chest, gut, bladder, and the anaesthetic
2 Practical prescribing	Titrate, review, and know when to stop

AGENT	MECHANISM AND ROUTE	WHERE IT IS USED	ITS OWN NOTE
Donepezil	Reversible, selective for acetyl-cholinesterase; once daily, 5 mg then 10 mg	Alzheimer disease at every severity; Lewy body dementia	Long acting; vivid dreams if taken at night
Rivastigmine	Inhibits acetyl and butyryl cholinesterase; oral twice daily or a patch	Alzheimer disease; Parkinson disease dementia; Lewy body dementia	**Not metabolised through cytochrome P450, so fewer interactions**
Galantamine	Reversible inhibitor plus nicotinic receptor modulation; 8 to 24 mg daily	Alzheimer disease, mild to moderate	Avoid in severe hepatic or renal impairment
Where they do not belong	Not a class effect across all dementias	Frontotemporal dementia, mild cognitive impairment	**Behaviour can worsen in frontotemporal dementia**

Amber cells are the discriminators. The last row is the one that separates a drug list from a prescribing answer.

- **Contraindications and cautions.** Bradycardia, sick sinus syndrome and other conduction disease; syncope; active peptic ulcer or gastrointestinal bleeding; asthma and chronic obstructive disease; bladder outflow obstruction; a seizure history; and prolonged blockade with suxamethonium at anaesthesia.
- **Practical prescribing.** Start low, titrate at intervals of weeks with food, and set a review at about three months. Cholinergic effects are nausea, diarrhoea, appetite and weight loss, cramps and slow pulse.

PITFALL

Prescribing an anticholinergic for the urinary or gastrointestinal effects of the cholinergic. The two cancel each other, the cognition worsens, and the prescription that should have been reviewed is instead defended.

WHAT EARNS THE MARKS

- **Three agents named with a real difference each,** not three names in a row.
- **Indications and non-indications both given.** Frontotemporal dementia is the discriminating entry.
- **Cardiac and respiratory cautions first,** because they are the ones that cause harm.

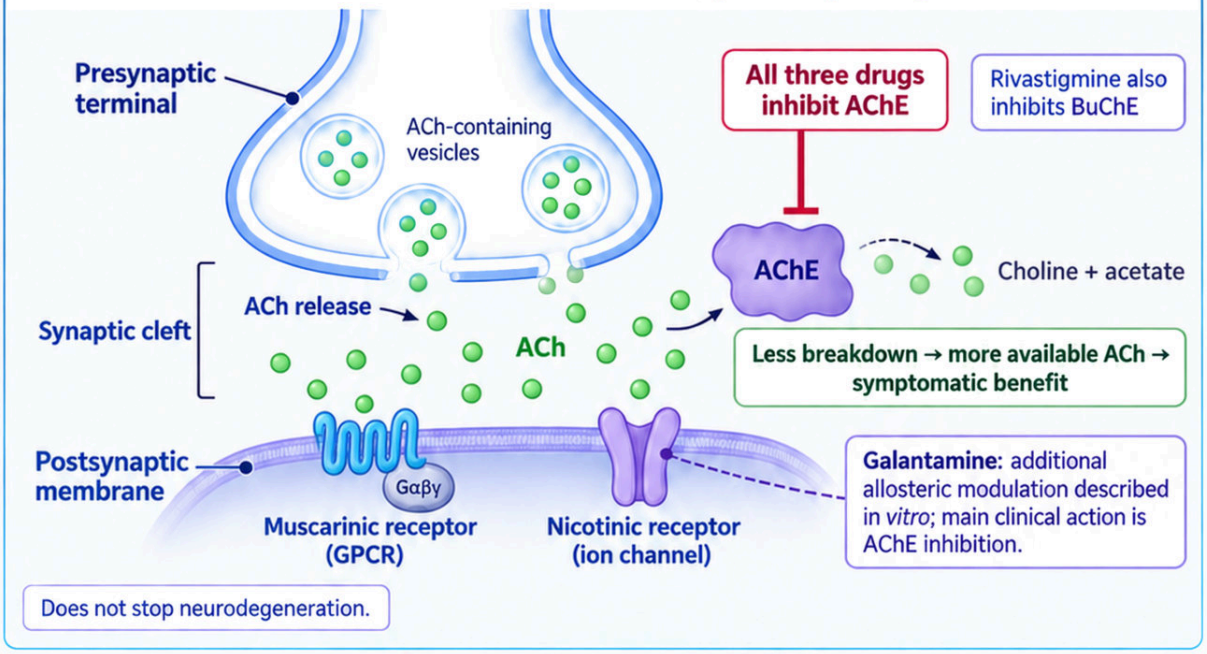
SEE ALSO Slot IV - 14 Lewy body dementia Slot IV - 13 frontotemporal dementia Slot IV - 100 memantine SPRINT Day 32, Dementias & neurocognitive disorders

WEAVE 100 · PAPER IV · D7.1

7.1 Cholinesterase inhibitors

Shared mechanism • Three drugs compared

Shared mechanism: inhibit breakdown, prolong acetylcholine action



DONEPEZIL

Target: Reversible, **selective AChE** inhibition.

Use: Alzheimer disease, all severities; DLB (guideline-supported).

Dose: Oral **5 mg once daily → 10 mg** after 4–6 weeks if tolerated.

Food: With or without food.

PK: Long acting; CYP2D6 / CYP3A4 interactions.

Distinctive: Insomnia / vivid dreams; consider morning dosing if troublesome.

RIVASTIGMINE

Target: AChE + BuChE inhibition.

Use: Alzheimer disease; **Parkinson disease dementia;** DLB (guideline-supported).

Forms: Oral twice daily with meals, or daily patch.

Patch: 4.6 → 9.5 → 13.3 mg/24 h; at least 4 weeks at each step if tolerated.

PK: Minimal CYP metabolism.

Patch safety: ONE patch only; remove old patch. If interrupted **>3 days**, restart 4.6 mg/24 h. Allergic contact dermatitis is a contraindication to patch reuse.

GALANTAMINE

Target: Reversible AChE inhibition.

Use: Mild–moderate Alzheimer disease.

Forms: IR twice daily; ER once daily.

ER dose: 8 → 16 → 24 mg each morning; at least 4 weeks at each step. Take with food.

PK: CYP2D6 / CYP3A4 interactions.

Limits: CrCl 9–59 mL/min: max 16 mg/day. Avoid **CrCl <9 or severe hepatic impairment;** moderate hepatic impairment: max 16 mg/day.

Shared cautions: bradycardia / heart block / syncope; GI effects and weight loss; ulcer / GI bleeding risk; asthma / COPD; bladder outflow obstruction; seizures; prolonged suxamethonium effect. These are cautions, not universal absolute contraindications. Drug hypersensitivity is a contraindication.

Monitor pulse, weight, tolerability and function. Avoid anticholinergic prescribing cascades.
Not routine treatment for MCI; do not offer for FTD. Follow formulation and local label.

Sources: Weave 100 IV Q17; DailyMed labels; NICE NG97 • 21 September 2026.

Cholinesterase inhibitors - Side by side comparison. Read across the labelled contrasts to distinguish the related concepts. Read with IV-17.

Credits and references: retained within the illustration.

IV - 18

Discuss the clinical features and management of Pseudodementia. [10]

10 | DEMENTIAS: TYPES AND DIFFERENTIAL | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **What the term means**	Cognitive impairment belonging to a psychiatric illness
3 **Distinguishing features**	History, answers, effort, and which came first
3 **Management**	Treat the depression properly, then test again
2 **The follow-up rule**	A good number later declare a true dementia

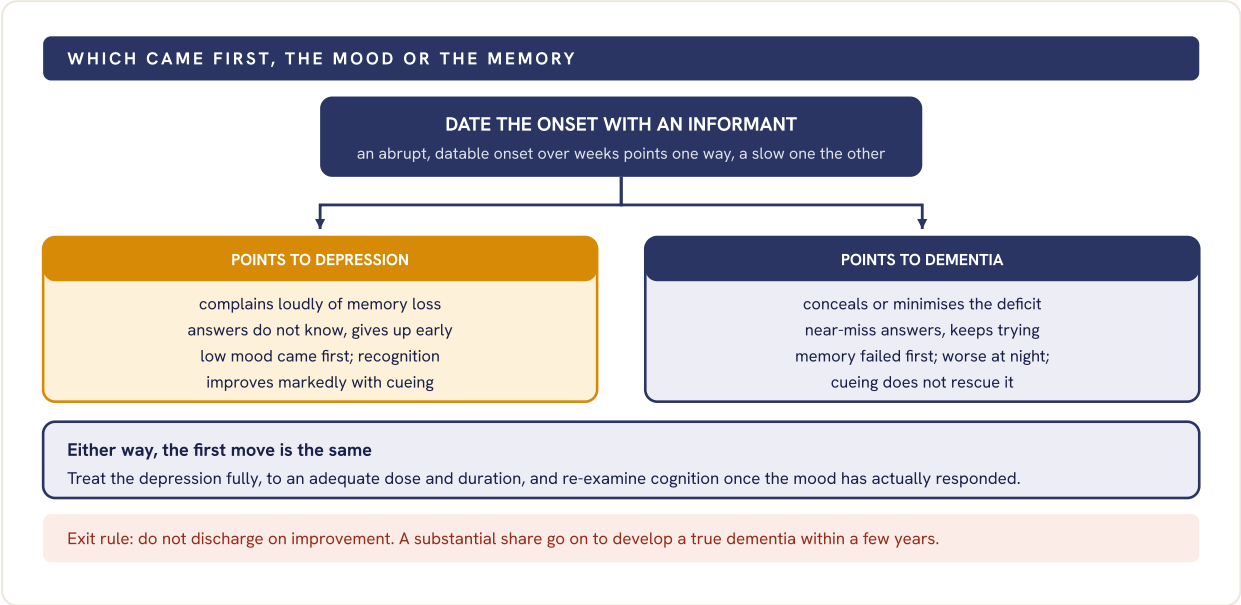


Fig 018.1 The two columns of the classical contrast, converging on one first move, with the follow-up rule carried along the foot because that is the mark most answers never reach.

- **What the term means.** Cognitive impairment produced by a psychiatric illness, most often depression in later life, which improves when that illness is treated. It is a description, not a diagnosis in current classification.
- **Treatment specifics.** An antidepressant at full dose for a full trial, with electroconvulsive therapy where the depression is severe, psychotic, food-refusing or carries high suicide risk. Do not start a cholinesterase inhibitor for the cognitive picture.

PITFALL

Treating the label rather than the patient. The features overlap, both conditions can coexist, and the safest position is to treat the depression well and to keep watching.

WHAT EARNS THE MARKS

- **Onset and sequence stated first.** Which came first, mood or memory, carries more weight than any single test.
- **The answer pattern named.** Do not know against near-miss is the classic discriminator.
- **Follow-up written into management,** not offered as an afterthought.

SEE ALSO Slot IV - 12 mild cognitive impairment Slot IV - 19 cortical and subcortical dementias **SPRINT** Day 32, Dementias & neurocognitive disorders

IV - 19

Enumerate subcortical Dementias. Describe the clinical features differentiating them from Cortical Dementias. [10]

10 DEMENTIAS: TYPES AND DIFFERENTIAL

DNB, RGUHS, TN-MGRMU - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2	The two lists	Named examples on each side, not a definition each
4	The grid	Domain by domain, with the discriminators marked
2	The three that settle it	Movement, cortical signs, and benefit from cueing
2	The honest limit	A clinical heuristic; most real cases are mixed

DOMAIN	CORTICAL	SUBCORTICAL	WHAT SETTLES IT
Named examples	Alzheimer disease, frontotemporal dementia	Huntington disease, progressive supranuclear palsy, Parkinson disease dementia, HIV dementia, Wilson disease	Enumerate before comparing
Memory	Storage fails; recognition is poor	Retrieval fails; recognition is better	**Cueing rescues the subcortical patient**
Language	Aphasia, and later anomia	Language intact; speech dysarthric and quiet	**Aphasia against dysarthria**
Speed of thought	Normal until late	Slowed throughout, called bradyphrenia	Timed tasks expose it
Higher cortical signs	Apraxia, agnosia, acalculia present	Absent, or explained by slowness	**Their presence is cortical**
Movement and gait	Normal until late in the illness	Abnormal early: rigidity, chorea, tremor, gait	**The strongest single pointer**
Mood and personality	Change later, less prominent early	Apathy and depression early and prominent	Referred to psychiatry first

Amber cells are the discriminators. Everything else in that column supports; saying which is which is itself worth marks.

- **The honest limit.** This is a clinical heuristic about where the pathology sits, not a pathological classification. Vascular disease and Lewy body disease straddle both columns, and mixed pictures are the rule in older patients.

PITFALL

Listing ten differences without ranking them. The examiner is looking for the three that would actually change a bedside opinion: a movement disorder, cortical signs, and whether cueing helps recall.

WHAT EARNS THE MARKS

- **Enumeration answered as enumeration.** The stem asks for named conditions before it asks for features.
- **A grid, not two paragraphs,** so each domain is readable across a row.
- **The discriminators identified as discriminators,** and the limit of the division stated once.

SEE ALSO Slot IV - 20 subcortical dementia Slot IV - 14 Lewy body dementia **SPRINT**

Day 32, Dementias & neurocognitive disorders

IV - 20

Subcortical dementia. [10]

10 DEMENTIAS: TYPES AND DIFFERENTIAL

KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 The concept	Where the term comes from and what it names
2 The anatomy	Frontal-subcortical circuits, not the cortex itself
3 The syndrome	Slowing, retrieval failure, apathy, and movement
3 Causes and reversibility	Grouped, with the treatable ones written first

THE CONCEPT

A dementia syndrome in which the burden of pathology sits in the basal ganglia, thalamus, brainstem and deep white matter rather than in the cortical mantle. The clinical signature is slowing of thought, retrieval-type forgetfulness, apathy and an early movement disorder, with language and praxis relatively preserved.

GROUP	EXAMPLES	HOW REVERSIBLE	FIRST STEP
Degenerative	Huntington disease, progressive supranuclear palsy, Parkinson disease dementia	Not reversible	Symptomatic and supportive
Vascular	Small vessel disease, lacunar and thalamic infarcts	Progression modifiable	**Control the vascular risk**
Infective	HIV-associated dementia, neurosyphilis	Substantially treatable	**Serology, then treat**
Metabolic and toxic	Wilson disease, chronic alcohol use, heavy metals	Treatable if caught	**Copper studies in the young**
Structural	Normal pressure hydrocephalus	Potentially reversible	Imaging, then referral

Amber cells are the discriminators. Sorting causes by what can be reversed is what makes this a clinical answer rather than a list.

- **The anatomy that explains the picture.** The frontal-subcortical circuits run from prefrontal cortex through striatum, pallidum and thalamus and back. Interrupting the loop below the cortex produces executive and motivational failure without aphasia or apraxia.
- **Management principle.** Treat the underlying disease where it is treatable, then treat depression and apathy on their own terms. Cholinesterase inhibitors are not routine here, the exception being Parkinson disease dementia.

WHAT EARNS THE MARKS

- **The circuit explanation given,** which turns a list of signs into a mechanism.
- **Causes grouped by mechanism,** with the reversible ones identified.
- **Retrieval failure distinguished from storage failure,** because that is the memory finding at the bedside.

SEE ALSO Slot IV - 19 cortical and subcortical dementias compared Slot IV - 14 Lewy body dementia **SPRINT** Day 32, Dementias & neurocognitive disorders

WEAVE 100 · PAPER IV · D10.1

10.1 Subcortical dementia

Six clinical portraits and memory hooks

Typical pattern: slowed thinking • retrieval difficulty • apathy • movement changes

Frontal–subcortical networks; language and praxis may be relatively spared. Mixed patterns occur.

HUNTINGTON DISEASE



Movements overflow, planning slows, mood changes

Chorea + executive decline + psychiatric change.

HTT CAG expansion; **autosomal dominant**.

Symptomatic care; assess swallowing and **suicide risk**.

PROGRESSIVE SUPRANUCLEAR PALSY (PSP)





Downward gaze blocked

Falls backward, looks down poorly, thinks slowly

Classic PSP: **early falls**, axial rigidity, **vertical gaze palsy**.

Tau-related degeneration; variants differ.

Falls and swallowing support; no established cure.

PARKINSON DISEASE DEMENTIA



PD → >1 year → dementia

Parkinsonism first, cognitive decline later

Established PD precedes dementia by **>1 year**.

Executive / visuospatial deficits; hallucinations may occur.

Rivastigmine may help; balance cognition and motor drugs.

HIV-ASSOCIATED DEMENTIA



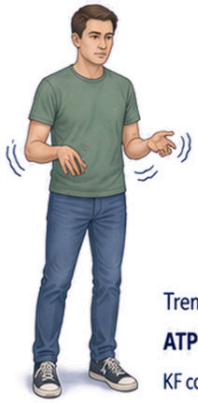
Thinking slows, initiative fades, walking slows

Marked cognitive decline impairs **daily function**.

HIV-related brain injury; **exclude other causes**.

ART and specialist assessment; improvement varies.

WILSON DISEASE





Liver involvement



Kayser–Fleischer corneal ring

Young patient: liver, movement, mind

Tremor / dystonia, liver and psychiatric findings.

ATP7B; **autosomal recessive**; copper accumulation.

KF corneal rings may occur; **treatable** with specialist care.

NORMAL-PRESSURE HYDROCEPHALUS





Ventriculomegaly



Urinary symptoms

Feet stick, bladder urges, thinking slows

Gait usually leads; urinary and cognitive symptoms.

Ventriculomegaly; assess response to CSF drainage.

Selected patients benefit from shunting; **no guaranteed reversal.**

Other causes: subcortical vascular disease • neurosyphilis • toxic / metabolic disorders

Six selected memory aids, not diagnostic criteria or an exhaustive list.

Source: Weave100 IV Q19–20; NINDS; NIH; NIDDK; iNPH guidelines • 21Sep2026

Subcortical dementia - Six clinical portraits and memory hooks. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-20.

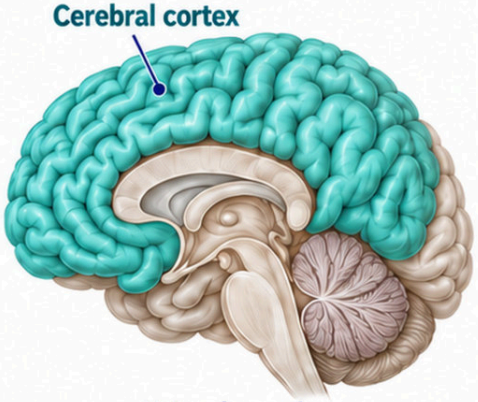
Credits and references: retained within the illustration.

7.2 Cortical vs Subcortical Dementia

Typical clinical patterns — a useful heuristic, not an absolute anatomical diagnosis

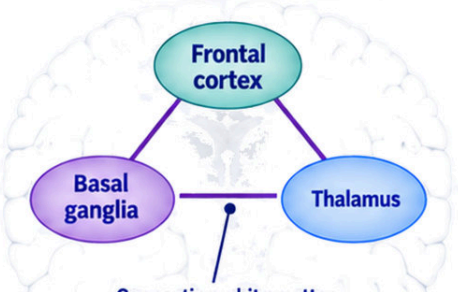
CORTICAL-PREDOMINANT

1. **MEMORY**
   Typical amnestic AD: **encoding / storage** impaired; rapid forgetting, limited cue benefit.
2. **PROCESSING SPEED**
   May be relatively preserved early; slowing can occur.
3. **EXECUTIVE FUNCTION**
   Can be impaired early — especially frontal syndromes / **FTD.**
4. **LANGUAGE**
   Aphasia / anomia or semantic loss may be prominent; syndrome-dependent.
5. **PRAXIS & GNOSIS**
   Apraxia, agnosia or acalculia suggest cortical network dysfunction.
6. **VISUOSPATIAL**
   May be marked with posterior cortical involvement.
7. **BEHAVIOUR & MOOD**
   Variable; **FTD** may cause early disinhibition, loss of empathy or apathy.
8. **MOTOR FEATURES**
   May be less prominent early in typical AD; important exceptions exist.
9. **EXAMPLES**
   Alzheimer disease; frontotemporal dementias / PPA — profiles differ.



Networks overlap.
Cortical and subcortical systems interact to produce cognition, behaviour and movement.

Subcortical circuits — schematic, not anatomical positions



SUBCORTICAL-PREDOMINANT

1. **MEMORY**
   Retrieval / strategy difficulty; **cueing** or recognition often helps, but not invariably.
2. **PROCESSING SPEED**
   Bradyphrenia: prominent slowed processing and responses.
3. **EXECUTIVE FUNCTION**
   Planning, initiation and set-shifting often prominently impaired.
4. **LANGUAGE**
   Primary **aphasia** less typical; reduced fluency, dysarthria or hypophonia may occur.
5. **PRAXIS & GNOSIS**
   Often relatively spared early; poor performance may reflect motor or executive deficits.
6. **VISUOSPATIAL**
   Can also be impaired, especially in Parkinson-related disease.
7. **BEHAVIOUR & MOOD**
   Apathy, reduced initiation and depression are common.
8. **MOTOR FEATURES**
   Movement clues: parkinsonism, chorea, gait and postural changes.
9. **EXAMPLES**
   Huntington disease, PSP, Parkinson disease dementia; subcortical small-vessel disease; HIV-associated dementia; Wilson disease.

Best bedside contrasts: cortical signs • movement pattern • recall versus recognition / cueing

Overlap is common: FTD can be dysexecutive; DLB has mixed cortical–subcortical features; vascular patterns depend on lesion location. No single feature proves a diagnosis.

Sources: Weave 100 IV Q19; Mack & Marsh, FOCUS 2017; FTD subcortical review 2021; MSD Manual • 21 September 2026

Cortical versus subcortical dementia - Brain circuits and clinical differences. Read across the labelled contrasts to distinguish the related concepts. Read with IV-20.

Credits and references: retained within the illustration.

IV - 21

Prion disease. [10]

10 DEMENTIAS: TYPES AND DIFFERENTIAL

KNRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 What a prion is	A misfolded protein that templates its own shape
3 The three routes	Sporadic, inherited, acquired, with named examples
3 Clinical picture	Rapid decline, myoclonus, and the psychiatric onset form
2 Diagnosis and care	The tests that support, and honest palliation

THE MECHANISM

The normal cellular prion protein, coded on chromosome 20, misfolds into a protease-resistant form that templates the same change in neighbouring molecules. The result is spongiform vacuolation, neuronal loss and astrocytic gliosis with no inflammatory response, which is why these illnesses are transmissible without being infections in the ordinary sense.

ROUTE	NAMED ENTITIES	DISTINGUISHING FEATURE	NOTE
Sporadic	Sporadic Creutzfeldt-Jakob disease	Around sixty, rapid dementia with myoclonus	**The great majority of cases**
Inherited	Familial Creutzfeldt-Jakob disease, Gerstmann-Straussler-Scheinker syndrome, fatal familial insomnia	Family history; insomnia and dysautonomia in the last	Genetic counselling needed
Acquired, medical	Iatrogenic disease from grafts, growth hormone, instruments	Long latency after the exposure	Instrument decontamination
Acquired, dietary	Variant Creutzfeldt-Jakob disease, kuru	**Young, psychiatric onset, painful sensory symptoms**	Comes to psychiatry first

Amber cells are the discriminators. The last row is the one a psychiatry examiner is testing for.

- **What supports the diagnosis.** Periodic sharp wave complexes on the record in the sporadic form, cortical ribboning and basal ganglia change on diffusion-weighted imaging, and cerebrospinal fluid assays including real-time quaking-induced conversion. Certainty comes only from tissue.
- **The red flag to state.** Rapidly progressive dementia over months rather than years, with myoclonus and ataxia. There is no curative treatment, so care is symptomatic, palliative and directed at the family.

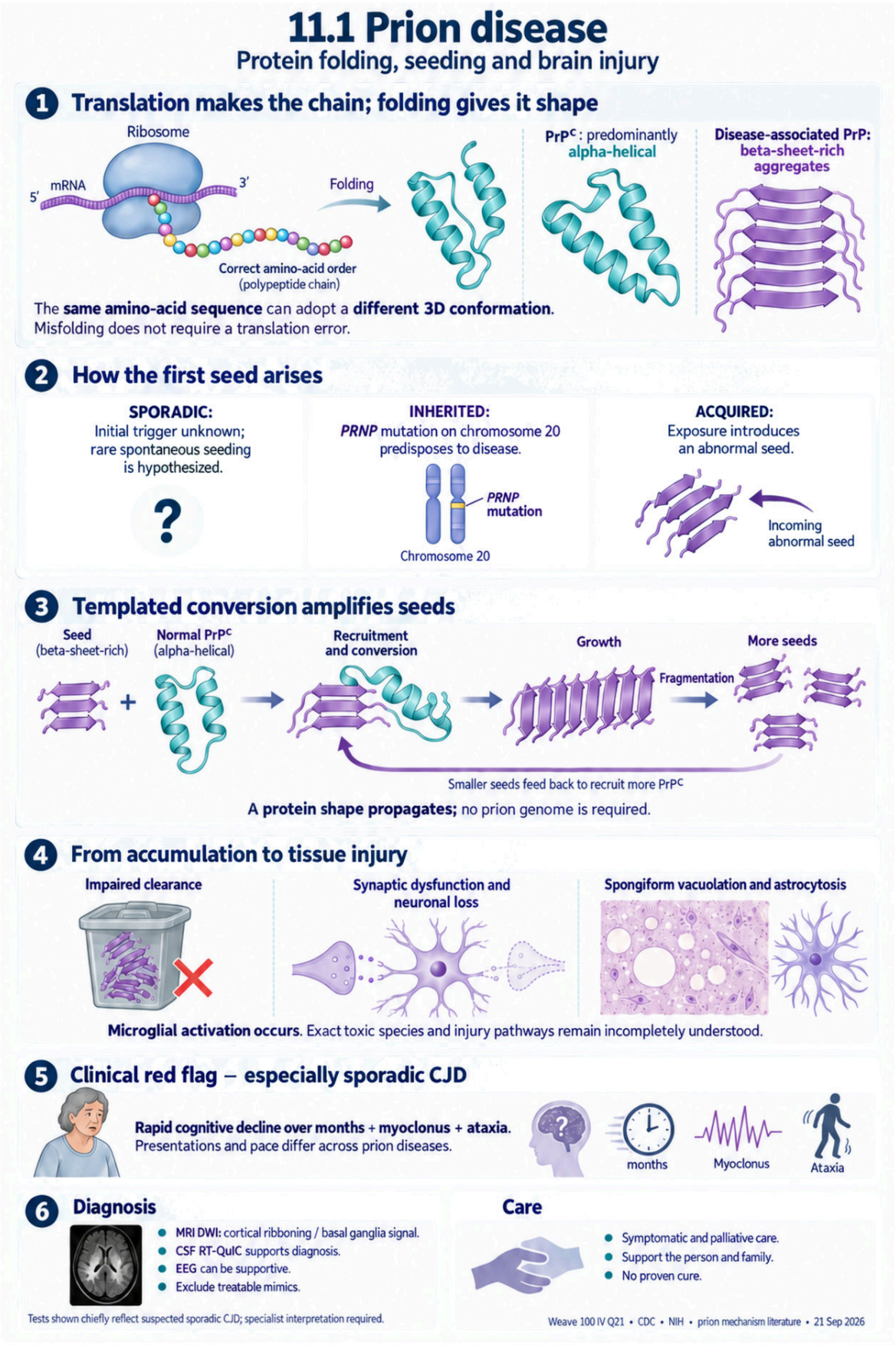
WHAT EARNS THE MARKS

- **Templated misfolding described,** not merely the words slow virus or infectious protein.
- **All three routes with a named entity in each.**
- **The variant form separated from the sporadic form** by age and by psychiatric onset.

SEE ALSO Slot IV - 19 cortical and subcortical dementias Slot IV - 18 pseudodementia **SPRINT**

Day 32, Dementias & neurocognitive disorders

WEAVE 100 · PAPER IV · D11.1



Prion disease - Protein folding seeding and brain injury. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-21.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · D11.2

Same protein. Different origins. Distinctive clues.

11.2 Prion diseases

Named entities and distinguishing clues

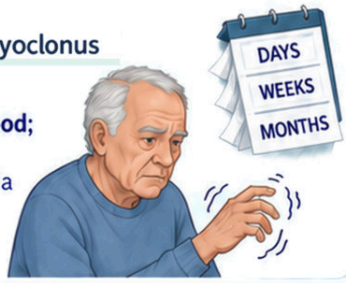
Route • Named entities • Distinguishing feature • Note

Misfolded proteins. Major consequences.

Named entity
Sporadic CJD
Route: SPORADIC

Distinguishing feature
Rapid decline + myoclonus

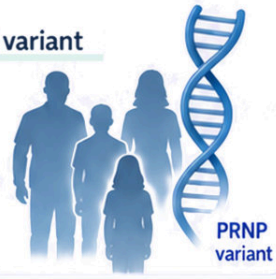
Note
Usually **later adulthood;** most CJD cases.
Rapid dementia, ataxia and myoclonus.
Initial trigger remains unknown.



Named entity
Genetic / familial CJD
Route: INHERITED

Distinguishing feature
CJD phenotype + PRNP variant

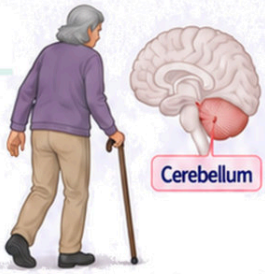
Note
Autosomal dominant ***PRNP*-related** disease.
Family history may be absent.
Offer specialist genetic counselling.



Named entity
Gerstmann–Sträussler-Scheinker syndrome (GSS)
Route: INHERITED

Distinguishing feature
Ataxia first; slower course

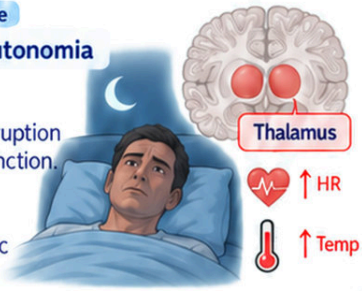
Note
Progressive **cerebellar** syndrome; dementia may follow. Often evolves **over years.**
PRNP-related; genetic counselling.



Named entity
Fatal familial insomnia (FFI)
Route: INHERITED

Distinguishing feature
Insomnia + dysautonomia

Note
Progressive sleep disruption and autonomic dysfunction.
Prominent **thalamic** degeneration.
***PRNP*-related**; genetic counselling.



Named entity
Iatrogenic CJD
Route: ACQUIRED — MEDICAL

Distinguishing feature
Medical exposure; long latency

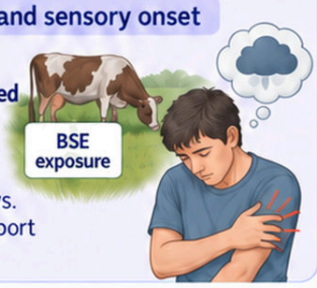
Note
Historical **cadaver-derived** growth hormone or dura mater grafts; **contaminated instruments.**
Requires specialist prion infection-control measures.



Named entity
Variant CJD (vCJD)
Route: ACQUIRED — BSE

Distinguishing feature
Younger; psychiatric and sensory onset

Note
Linked to **BSE-contaminated food.** Early **psychiatric symptoms** and **painful sensory symptoms;** neurological decline follows.
Pulvinar MRI sign can support diagnosis.



Named entity
Kuru
Route: ACQUIRED — HISTORICAL

Distinguishing feature
Historical exposure + cerebellar ataxia

Note
Associated with mortuary cannibalism among the Fore in Papua New Guinea. Tremor, gait ataxia and dysarthria; often prolonged incubation.

Kuru is distinct from vCJD; do not assign them the same phenotype.

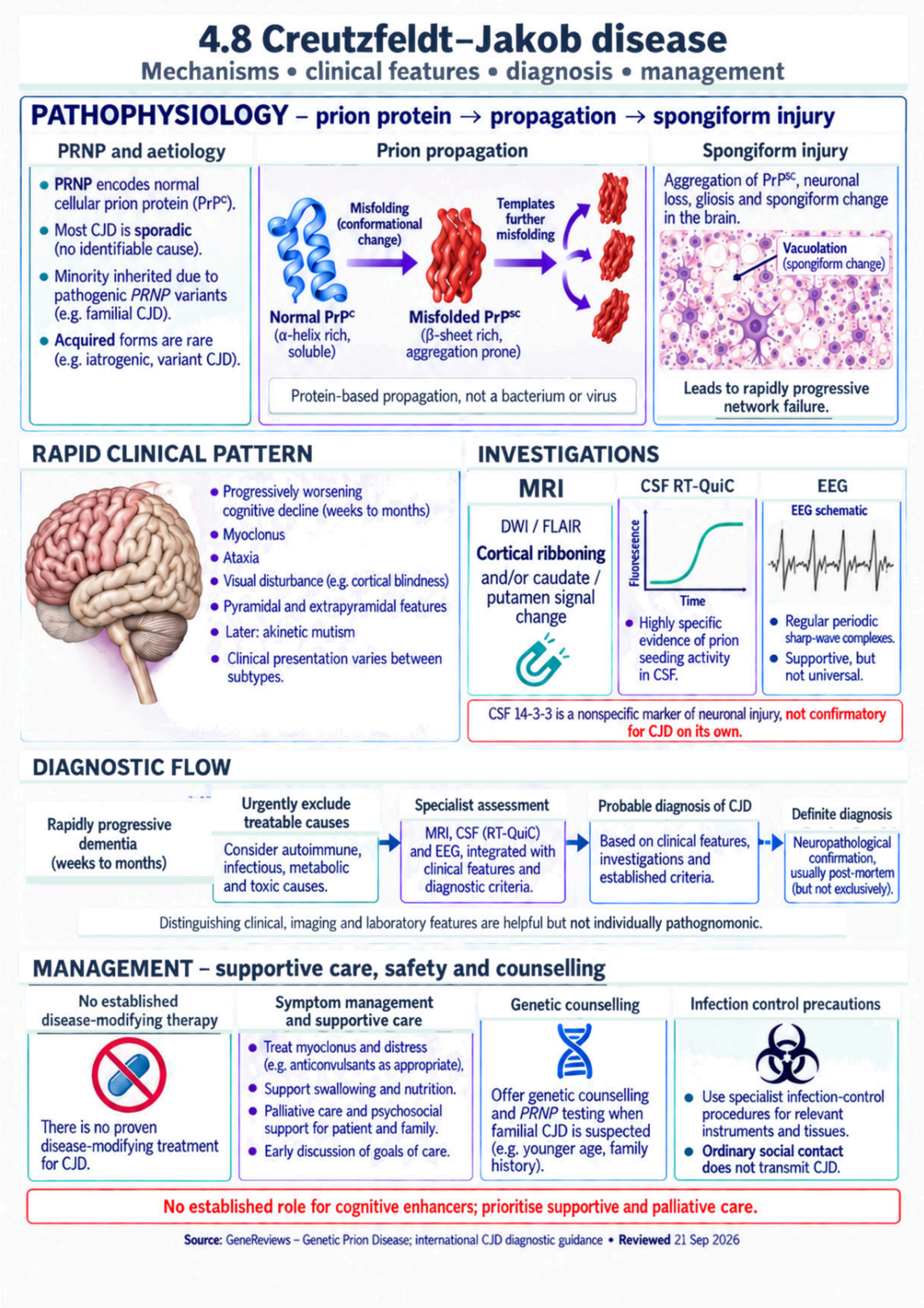


All are fatal prion disorders; no proven cure.
These are distinguishing clues, not stand-alone diagnostic criteria.

Weave 100 IV Q21, corrected and expanded
CDC • NINDS • GeneReviews • 21 Sep 2026

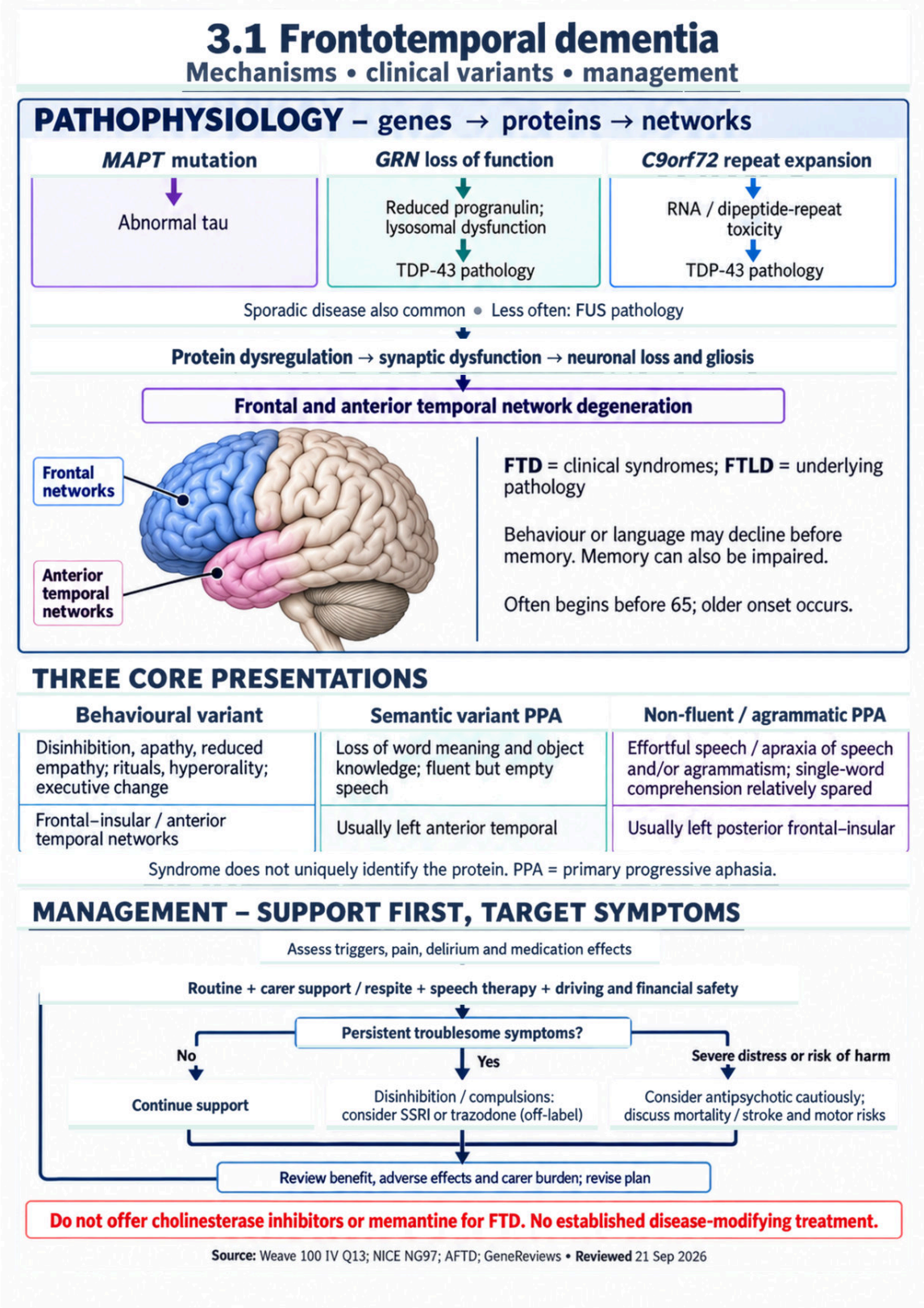
Prion diseases - Named entities and distinguishing clues. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-21.

Credits and references: retained within the illustration.



Creutzfeldt-Jakob disease - Prions and rapid decline. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-21.

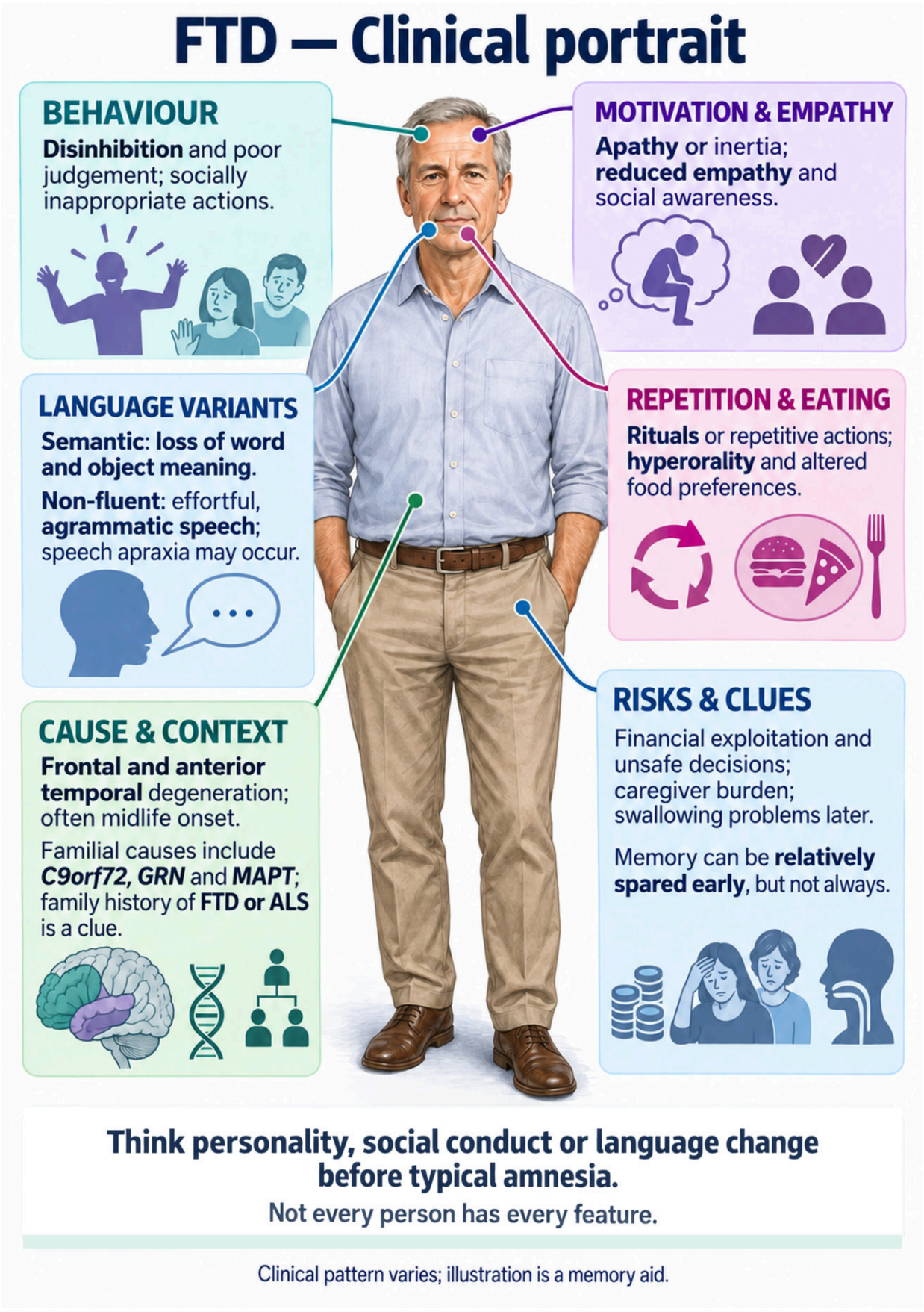
Credits and references: retained within the illustration.



Frontotemporal dementia - Mechanisms variants and management. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Section companion: Dementias: types and differential.

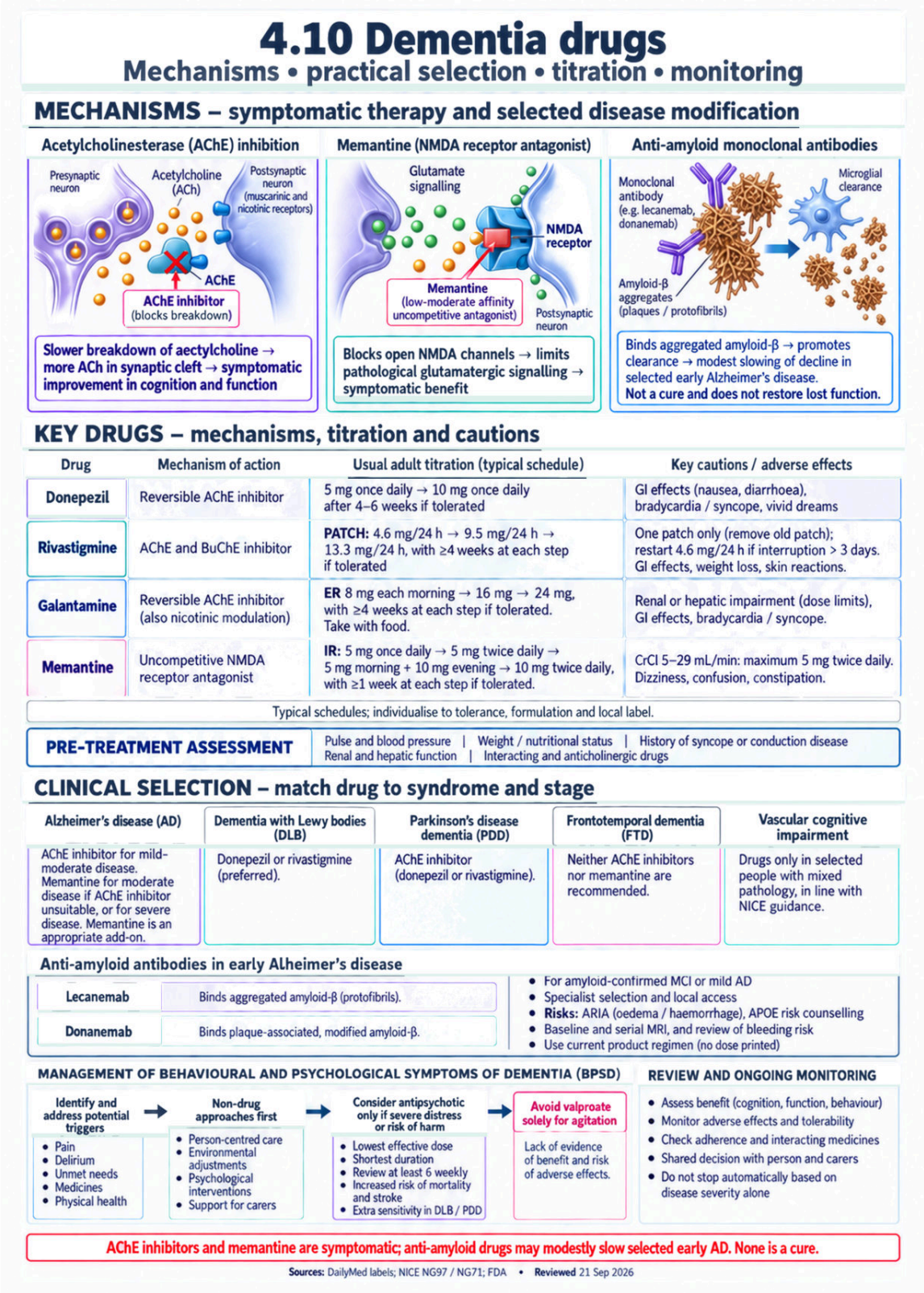
Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · D3.2



Frontotemporal dementia - Clinical portrait risks and features. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Dementias: types and differential.

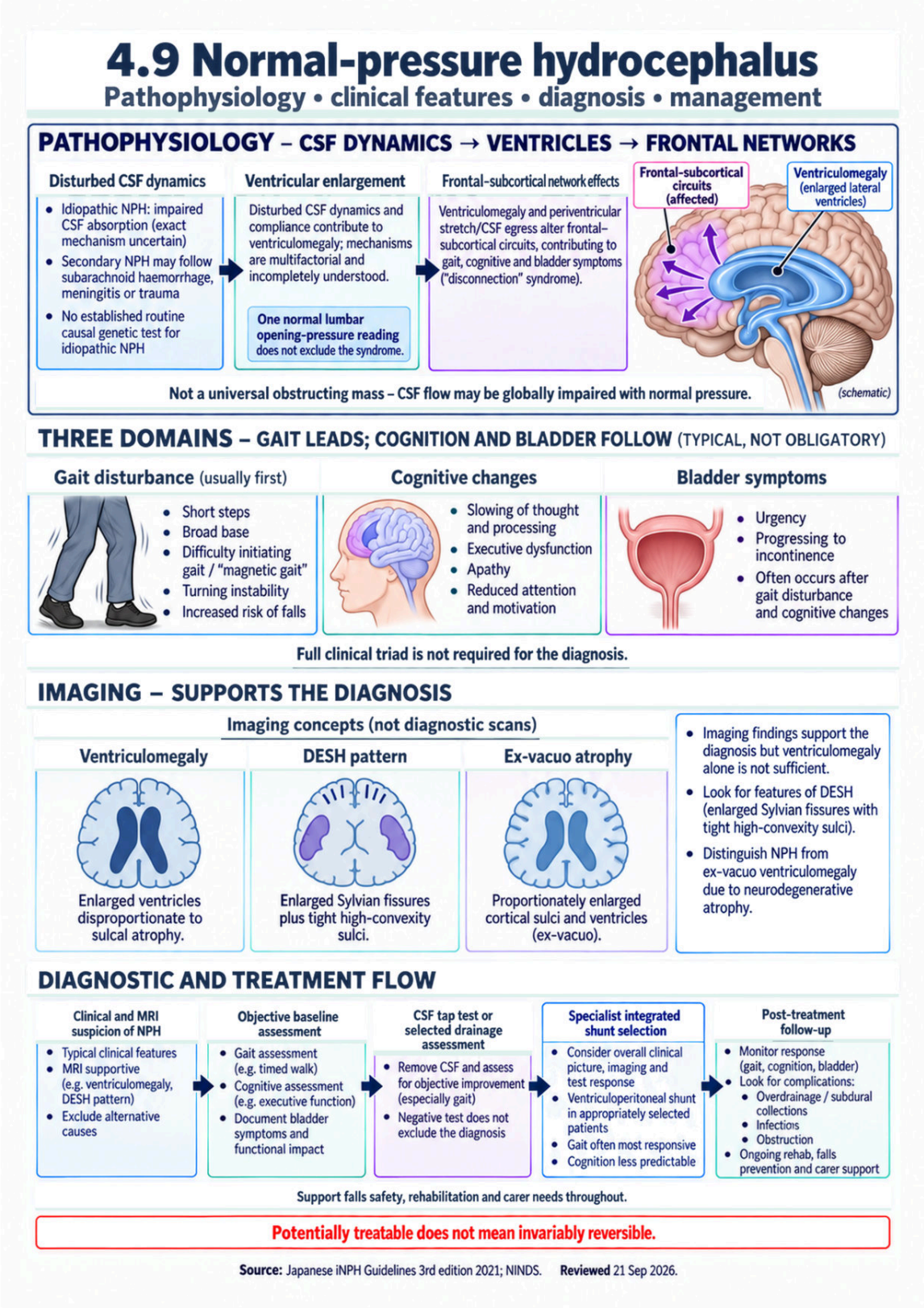
Credits and references: retained within the illustration.



Dementia drugs - Mechanisms selection and monitoring. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Section companion: Dementias: types and differential.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · D4.9



Normal-pressure hydrocephalus - CSF to shunt assessment. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Dementias: types and differential.

Credits and references: retained within the illustration.

AREA 03 OF 15 · P4-A15

Consultation-liaison and systemic medicine psychiatry

9	10.1%	IV-22	IV-30
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 2 · HIGH 2 · SINGLE 5

REVISION ORDER

The consultation-liaison slot first, it frames the other eight. The two transplant slots together, they share one timeline and are declared as a deliberate split in both fragments. The cardiac, gastrointestinal and respiratory slots next as a block, because the liaison shape is the same on all three. Fibromyalgia, the infertility and cancer slot and the pre-treatment evaluation slot last, and the last of those is three questions on one page.

WHAT IS IN THIS AREA

Consultation-liaison psychiatry itself, then the organ systems the boards actually set: transplantation in two slots, one across any solid organ and one specific to the kidney, depression with ischaemic heart disease, irritable bowel syndrome, bronchial asthma, and fibromyalgia. The last two are the mental health issues of infertility and of cancer, and a pre-treatment evaluation slot that puts hyperthyroidism, a generalised seizure disorder and intellectual impairment on one page.

IV - 22

Write a note on consultation liaison psychiatry. [10]

10 CONSULTATION-LIAISON AND SYSTEMIC MEDICINE PSYCHIATRY

KNRUHS, KUHS, WBUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	What it is	Consultation, liaison, and the difference between them
3	The models of service	The table, and what each one costs
3	Why patients are referred	The commonest reasons, in their real order
2	How it is actually done	Team first, question clarified, note usable

THE DEFINITION, AND THE DISTINCTION INSIDE IT

Consultation liaison psychiatry is the subspecialty that provides psychiatric assessment and treatment to patients in medical and surgical settings. Consultation is the episode of advice about one patient. Liaison is the standing attachment to a unit, with teaching, joint rounds and shared care. The name keeps both because the referral is only half the work.

MODEL	HOW THE SERVICE IS ORGANISED	WHAT IT BUYS AND WHAT IT COSTS
Consultation	Psychiatrist responds to written referrals from any ward	Cheap and wide; misses everyone not referred
Liaison	Psychiatrist attached to a named unit, attends its rounds	Finds cases early; covers few wards
Hybrid	Referral service plus attachment to high-yield units	The usual working compromise
Collaborative care	Case manager in primary care, psychiatrist supervising	Best evidence in chronic illness; needs staff

- **Why patients are referred.** Delirium, which is the commonest and is often referred as depression or as an uncooperative patient. Assessment after self-harm. Depression and adjustment in physical illness. Capacity and consent questions. Substance withdrawal on the ward. Agitation. Psychiatric effects of medical drugs. Medically unexplained symptoms. Pre-transplant and pre-surgical evaluation.
- **How the consultation is done.** Read the notes and speak to the team before seeing the patient, because the written question is often not the real one. Examine, including cognition. Review every drug, including those stopped. Then write a note the team can act on: a formulation in their language, one clear recommendation, and a named person for follow-up.

WHAT EARNS THE MARKS

- **Consultation and liaison defined apart,** which is the whole point of the compound name.
- **Models given as a table with their trade-offs,** not as four synonyms.
- **Delirium named first among referrals,** since it is the commonest and the commonest missed.
- **The note written for the referring team,** which is what makes the advice actually happen.

SEE ALSO Slot IV - 24 the psychiatrist in organ transplant Slot IV - 27 depression with ischaemic heart disease

SPRINT Day 40, Medical and systemic-disease psychiatry

WEAVE 100 · PAPER IV · 12.1

CONSULTATION–LIAISON PSYCHIATRY

Roles • principles • 10 common referral reasons

Paper IV • Area 3 • Rebuilt Q12 | Original Q22, p36 • Sheet 12.1

WHAT IT IS

Psychiatric assessment and care at the interface of mental and physical illness in medical and surgical settings.

CONSULTATION

Patient-specific assessment and advice after referral; may include repeated follow-up.

LIAISON

Sustained partnership with a unit; joint rounds, teaching, early detection and shared care.

They overlap. Several days of follow-up alone do not make a liaison service.

ROLES AT THREE LEVELS

PATIENT & FAMILY

Diagnose and treat; assess risk and capacity; support coping, adherence and caregivers.

TEAM

Explain the formulation; advise on safe prescribing; teach staff; resolve communication difficulties.

SYSTEM

Build referral pathways, prevention and continuity; audit quality; support research and service development.

PRACTICAL CONSULTATION: FIVE RULES

1 TRIAGE & CLARIFY — Immediate danger or instability → urgent medical care and safety measures. Clarify the referral question with the team; speak to nursing staff.

↓ next

2 REVIEW & ASSESS — Read records and collateral; examine mental state, attention and cognition. Check physical causes, investigations and all drugs, including recently stopped drugs.

↓ next

3 RESPECT RIGHTS — Explain your role and seek consent; protect privacy and confidentiality. Assess capacity for the particular decision at that time; refusal alone is not incapacity.

↓ next

4 FORMULATE & AGREE — Integrate biological, psychological and social factors. Consider illness effects, drug effects and primary psychiatric illness; avoid diagnostic overshadowing.

↓ next

5 COMMUNICATE & CLOSE THE LOOP — Hand over urgent findings directly. Give a written and verbal plan: priorities, treatment, interactions, monitoring and escalation. Name the responsible clinician, review time and discharge follow-up.

Principle: collaborate respectfully; referral does not transfer all clinical responsibility.

10 COMMON REASONS FOR REFERRAL

Illustrative list — not a universal rank order; frequency varies by service.

1 DELIRIUM / COGNITIVE CHANGE
Acute fluctuation and inattention; quiet withdrawal can mask hypoactive delirium.

2 SELF-HARM / SUICIDALITY
After poisoning or injury; assess intent, ongoing risk and a safe care plan.

3 DEPRESSION / ADJUSTMENT
Persistent low mood, anhedonia or difficulty coping with physical illness.

4 ANXIETY / PANIC
Distress, autonomic symptoms or procedure fear; assess medical mimics.

5 SUBSTANCE USE / WITHDRAWAL
Intoxication or interrupted alcohol/sedative use; withdrawal may be dangerous.

6 AGITATION / PSYCHOSIS / MANIA
Behavioural change or care disruption; look first for delirium and medical causes.

7 CAPACITY / REFUSAL / ADHERENCE
Clarify understanding and decision-making; explore communication and practical barriers.

8 FUNCTIONAL / SOMATIC SYMPTOMS
Persistent bodily symptoms; functional disorders need positive clinical features. Normal tests alone do not prove a psychiatric cause.

9 MEDICATION-RELATED PROBLEMS
New psychiatric symptoms, adverse effects or interactions; review the timing of drug changes.

10 PREOPERATIVE / TRANSPLANT ASSESSMENT
For selected procedures: assess psychiatric stability, substance use, understanding, supports and adherence needs.

SERVICE MODELS: ORGANISATION → TRADE-OFF

Consultation: referral response → broad reach, misses unreferred need.

Liaison: embedded unit work → earlier detection, staff intensive.

Hybrid: referrals + selected unit attachments → balances coverage and depth.

Collaborative care: care manager + treating clinician + psychiatric supervision → structured follow-up, needs infrastructure.

Sources: Weave 100 IV Q22; IPS/IJP consultation-liaison guidelines (2022): Gautam et al., S201–210; Ghosal & Ray, S211–227; Grover & Singh, S228–235.

Exam synthesis • 21 Sep 2026 • v1

Consultation liaison psychiatry. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-22.

Credits and references: retained within the illustration.

IV - 23

Write a note on fibromyalgia. [10]

10 CONSULTATION-LIAISON AND SYSTEMIC MEDICINE PSYCHIATRY

KNRUHS, KUHS, RGUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2 **The definition**	Widespread pain, with the mechanism named
2 **How it is diagnosed**	From tender points to a symptom score
3 **What travels with it**	Sleep, mood, adversity, without reducing it to them
3 **Management**	The table: what works, what helps, what does not

THE DEFINITION

Fibromyalgia is chronic widespread musculoskeletal pain with tenderness, accompanied by fatigue, unrefreshing sleep and cognitive difficulty, without an inflammatory or structural cause sufficient to explain it. The mechanism is central: altered central pain processing, or nociplastic pain, rather than peripheral tissue damage. Saying that sentence is what stops the answer becoming a debate about whether the condition is real.

- **How it is diagnosed.** The 1990 criteria required widespread pain for at least three months and tenderness at eleven of eighteen defined points. The 2010 and 2016 revisions dropped the tender point count for a widespread pain index and a symptom severity scale, so the diagnosis became clinical and symptom-based, and may now be made alongside another disorder rather than only after excluding one.
- **What travels with it, and what that does not mean.** Sleep disturbance is close to universal. Depression, anxiety, post-traumatic disorder and childhood adversity are over-represented. None of that makes this a psychiatric disorder, and treating it as one is the commonest error. Give the diagnosis positively and early, because a label withheld for years drives further investigation.

TIER	WHAT IT IS	WHERE IT STANDS
First line, non-drug	Graded aerobic exercise, education, cognitive behavioural therapy, sleep treated directly	**The strongest evidence in this condition**
Drugs	Low-dose amitriptyline at night; duloxetine or milnacipran; pregabalin or gabapentin	Useful, modest, and often not tolerated
Not helpful	Opioids; corticosteroids and anti-inflammatories used alone	**Opioids worsen the central mechanism**

WHAT EARNS THE MARKS

- **Central sensitisation named as the mechanism,** not an apology for the absence of pathology.
- **Both criteria sets, and what changed between them.**
- **Exercise and therapy placed above the drugs,** which is the order the evidence gives.
- **Opioids named as harmful,** not merely as unhelpful.

SEE ALSO Slot IV - 30 irritable bowel syndrome Slot IV - 22 consultation liaison psychiatry **SPRINT**

Day 40, Medical and systemic-disease psychiatry

WEAVE 100 · PAPER IV · 13.1

FIBROMYALGIA

Pain processing • complete 2016 criteria • tailored management

Paper IV • Rebuilt Question 13 | Original Question 23, page 37 • Sheet 13.1

DEFINE THE TERMS

Fibromyalgia: persistent widespread pain with tenderness, fatigue, unrefreshing sleep and cognitive difficulty; a clinical syndrome, often with nociplastic pain.

Nociceptive pain: non-neural tissue damage or threatened damage activates pain-sensing receptors.

Neuropathic pain: caused by disease or a lesion of the somatosensory nervous system.

Nociplastic pain: altered nociception without clear tissue damage or somatosensory lesion sufficient to explain the pain; mechanisms can coexist.

Central sensitisation: increased responsiveness of central pain-processing neurons to normal or normally subthreshold input. A mechanism, not a synonym or diagnostic test.

Hyperalgesia: increased pain from a normally painful stimulus.

Allodynia: pain from a normally non-painful stimulus; neither finding is specific to fibromyalgia.

RECOGNISE & EVALUATE

Fatigue = reduced physical or mental energy. **Unrefreshing sleep** = waking without restoration. **Cognitive difficulty** = impaired attention, memory or mental processing.

History, pain distribution, functional impact, medication review and physical examination guide testing. No confirmatory blood test or scan.

Possible mimics: inflammatory rheumatic disease, hypothyroidism, myopathy, anaemia, medication effects. Synovitis, fever, weight loss, objective weakness or focal neurological signs require targeted assessment.

Comorbidities: depression, anxiety, migraine, irritable bowel syndrome and sleep apnoea; treat independently. They do not make pain imaginary.

Overlap: chronic fatigue syndrome requires post-exertional malaise; neuropathic pain requires a relevant lesion/disease and plausible distribution. Either can coexist.

Somatic symptom disorder requires disproportionate symptom-related thoughts, anxiety or behaviour; pain or normal tests alone do not establish it.

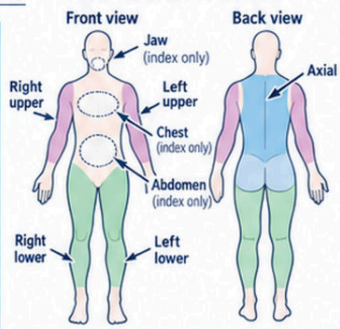
2016 DIAGNOSTIC CRITERIA — ALL CONDITIONS

1. **Widespread Pain Index ≥7 AND Symptom Severity Scale ≥5**
   OR Widespread Pain Index 4–6 AND Symptom Severity Scale ≥9.
2. **Generalised pain in at least 4 of 5 regions.**
3. **Symptoms generally present for at least 3 months.**

Diagnosis remains valid with other illnesses; continue assessment for coexisting disease.

Tender-point counting is **NOT** required.

FIVE-REGION PAIN MAP



Five-region map labels:
Left upper • Right upper • Left lower • Right lower • Axial.
Jaw, chest and abdomen are index-only sites.

WIDESPREAD PAIN INDEX: 0–19

Past week: 1 point for pain at each site.

- **Left upper:** shoulder girdle, upper arm, lower arm.
- **Right upper:** shoulder girdle, upper arm, lower arm.
- **Left lower:** hip/buttock/trochanter, upper leg, lower leg.
- **Right lower:** hip/buttock/trochanter, upper leg, lower leg.
- **Axial:** neck, upper back, lower back.
- **Index-only sites:** left jaw, right jaw, chest, abdomen.

Total = 15 regional sites + 4 index-only sites = 19.
Jaw, chest and abdomen **COUNT** in the index but **NOT** in the 4-of-5 region requirement.

SYMPTOM SEVERITY SCALE: 0–12

Past week: rate EACH of fatigue, waking unrefreshed and cognitive symptoms from 0–3:

- **0** none; **1** mild/intermittent; **2** moderate/considerable, often present; **3** severe, pervasive, continuous, life-disturbing.

Three ratings give **0–9** points.

Past 6 months: add **1** each for headaches, lower abdominal pain/cramps and depression (absent = 0).
Three yes/no items give **0–3** points.

TOTAL = 0–9 + 0–3 = 0–12.
Do not substitute the older "general somatic symptoms" rating.

MANAGEMENT — AGREE FUNCTIONAL GOALS, THEN REVIEW

Confirm diagnosis + explain pain processing → **Choose meaningful function, sleep and pain goals** → **Begin combined non-drug care** → **Persistent disabling symptoms?** Consider **ONE** medicine matched to target and risk; benefits are usually modest.

ACTIVITY	Choose enjoyable aerobic, resistance or water-based activity. Start below current tolerance in short bouts; pace tasks and rest to avoid boom–bust cycles.	Tolerated without sustained worsening? → increase gently, one variable at a time. Flare or prolonged worsening? → reduce intensity/duration, stabilise and reassess. Delayed multisystem deterioration? → assess chronic fatigue syndrome overlap; do not push fixed increases.
SLEEP	Regular schedule; assess sleep apnoea/restless legs and medication effects.	
PSYCHOLOGICAL CARE	Cognitive behavioural therapy supports pain coping and function; treat mood, trauma and anxiety when present. It does not imply a psychological cause.	

MEDICINE	WHY CHOOSE IT	LIMITS & MONITORING
Duloxetine	Pain with depression/anxiety; enhances descending pain inhibition	Nausea, sweating, blood pressure, withdrawal. Avoid significant liver disease or severe renal impairment.
Milnacipran	Alternative serotonin–noradrenaline reuptake inhibitor for pain	Nausea, raised pulse/blood pressure; adjust for renal impairment. Verify local availability.
Pregabalin	Pain with disturbed sleep; reduces excitatory transmitter release	Dizziness, sedation, oedema, weight gain; renal dose adjustment and dependence risk.
Low-dose amitriptyline	Night pain/sleep; off-label option, limited-quality evidence	Anticholinergic effects, orthostasis, sedation, cardiac conduction and overdose risk.

Start low, titrate cautiously; check interactions and mood-switch risk with antidepressants. Reassess tolerability early and pain/function after an adequate trial. Continue only meaningful benefit; taper ineffective or harmful treatment.

Gabapentin: insufficient fibromyalgia evidence; not equivalent to pregabalin.

Strong opioids and corticosteroids: avoid for fibromyalgia. Anti-inflammatory drugs do not treat its core mechanism; use only for a separate indication.

Tramadol: weak support in European recommendations for selected severe pain, not routine; dependence, seizures and serotonin toxicity limit use.

Severe disability despite care → multidisciplinary rehabilitation; reassess diagnosis, comorbidity and goals.

Sources: Wolfe et al., 2016 criteria; International Association for the Study of Pain terminology; European League Against Rheumatism, 2017; Cochrane reviews; medicine prescribing information.

Modern evidence synthesis, not verbatim source • 21 September 2026 • v1

Fibromyalgia. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-23.

Credits and references: retained within the illustration.

IV - 24

Discuss the role of the psychiatrist in organ transplant. [10]

10 CONSULTATION-LIAISON AND SYSTEMIC MEDICINE PSYCHIATRY

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

SKELETON

2 **Four points on one timeline**	Not one assessment, a continuing role
3 **Evaluating the recipient**	Capacity, adherence, substance, support
2 **Evaluating the living donor**	Voluntariness, and what the law requires
3 **After the operation**	Delirium, the drugs, and the long adherence

STAGE	WHAT THE PSYCHIATRIST DOES	WHAT THE FINDING DECIDES
Pre-transplant, recipient	Capacity, psychiatric history and its treatment, substance use, cognition, social support, adherence record	**Listing, deferral, or conditions attached**
Pre-transplant, living donor	Voluntariness in a private interview, capacity, absence of coercion or payment, understanding of risk	**Whether the donation may proceed**
Peri-operative	Delirium, steroid-induced mood or psychosis, calcineurin inhibitor neurotoxicity	Treatment on the unit, not a new diagnosis
Long term	Adherence, depression, quality of life, the donor's own outcome	Graft survival and who is followed

- **The recipient assessment.** Adherence history is the single best predictor and should be sought from records, not from the interview alone. Structured instruments exist for this judgement, SIPAT and PACT among them, and using one makes the reasoning auditable. Active dependence, untreated psychosis and severe cognitive impairment are usually grounds for deferral with a treatment plan, not for permanent exclusion, and writing it that way is what distinguishes an assessment from a gatekeeping exercise.
- **The donor, and the law.** The living donor is interviewed alone. Donation in India is governed by the Transplantation of Human Organs and Tissues Act 1994 as amended in 2011, under which an authorisation committee scrutinises donation by anyone who is not a near relative, to exclude commercial dealing. The psychiatrist tests voluntariness; the committee tests the relationship.
- **Prescribing across the graft.** Immunosuppressants are metabolised through CYP3A4, so fluvoxamine, fluoxetine and nefazodone raise calcineurin inhibitor levels. Steroids raise mood, then lower it on withdrawal. Lithium needs particular care where renal function carries the graft.

WHAT EARNS THE MARKS

- **A timeline, not a single pre-operative assessment.**
- **Adherence named as the best predictor,** and sought from the record.
- **The donor interviewed alone, and the Act named,** which is the Indian limb of this question.
- **Deferral written as deferral,** rather than an exclusion list.

SEE ALSO Slot IV - 28 the psychiatrist in kidney transplant Slot IV - 22 consultation liaison psychiatry SPRINT Day 40, Medical and systemic-disease psychiatry

WEAVE 100 · PAPER IV · 14.1



Organ transplant psychiatry. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-24.

Credits and references: retained within the illustration.

IV - 25

Describe the pre-treatment evaluation of a psychiatry patient suffering from hyperthyroidism, generalised seizure disorder, and intellectual impairment. [3+3+4]

3 + 3 + 4 | CONSULTATION-LIAISON AND SYSTEMIC MEDICINE PSYCHIATRY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**Hyperthyroidism**	Confirm the thyroid state before naming a disorder
3	**Generalised seizure disorder**	The drug already there, and the threshold
4	**Intellectual impairment**	Baseline, communication, pain, and consent

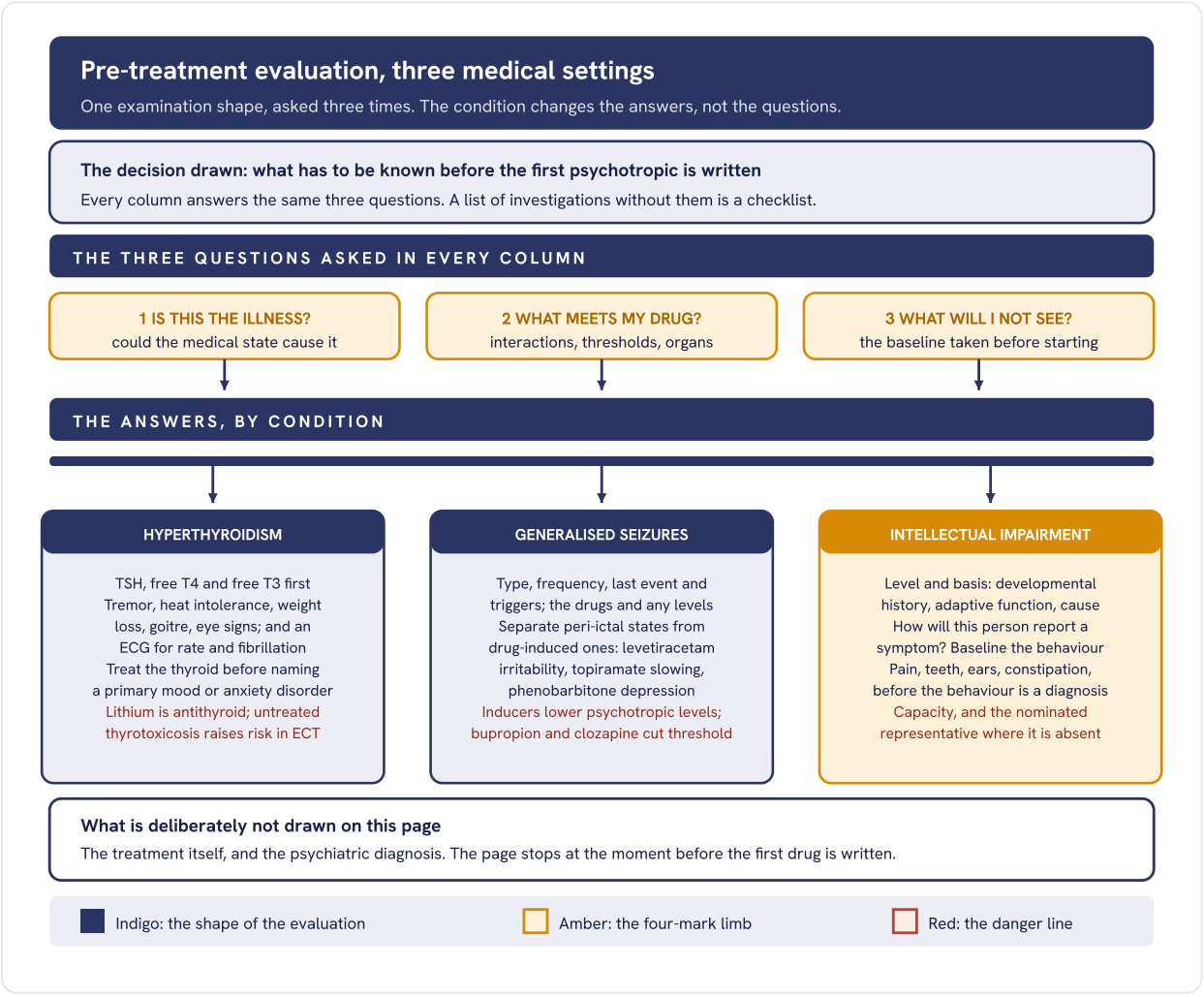


Fig 025.1 The same three questions run along one spine into all three columns, so the answer is one method applied three times, not three checklists.

- **The seizure limb, completed.** Carbamazepine, phenytoin and phenobarbitone induce enzymes and lower many psychotropic levels; valproate inhibits and raises lamotrigine. Then by seizure threshold: bupropion and clozapine lower it most, chlorpromazine more than haloperidol, and the serotonin reuptake inhibitors are safer. Electroconvulsive therapy is not contraindicated in epilepsy.

- **The impairment limb, completed.** Behaviour that challenges is communication or pain until proved otherwise, so the physical search comes first. Record a side-effect baseline the person cannot report, then start low, review on a date, and stop what has not worked.

WHAT EARNS THE MARKS

- **One method stated before the columns,** not three unconnected lists.
- **The thyroid treated as a possible cause,** which is the first limb's whole point.
- **Enzyme induction and seizure threshold both named,** as different problems.
- **A behaviour baseline and a pain search** in the four-mark limb.

SEE ALSO Slot IV - 22 consultation liaison psychiatry **SPRINT** Day 33, Endocrine & metabolic psychiatry Day 30, Epilepsy & psychiatry

IV - 26

What is mental health? Discuss the mental health issues related to infertility and to cancer. [2+4+4]

2 + 4 + 4 | CONSULTATION-LIAISON AND SYSTEMIC MEDICINE PSYCHIATRY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Mental health, defined**	Well-being, and not merely the absence of illness
4	**Infertility**	Grief, the couple, and where the blame lands
4	**Cancer**	The sequence, the scoring problem, the interactions

THE DEFINITION

Mental health is a state of well-being in which the individual realises his or her own abilities, can cope with the normal stresses of life, can work productively, and is able to contribute to the community. It is not the absence of illness, which matters here because both these populations are ill and most of them are not mentally ill.

- **Infertility.** The evidence that distress causes infertility is weak; the evidence that infertility causes distress is strong, and saying so in that order protects the patient from being blamed for it. What is seen: grief that has no ritual and recurs every month; anxiety and depression that peak at a failed cycle; sex that becomes scheduled and instrumental, with sexual dysfunction following; and a social burden that in Indian practice falls disproportionately on the woman, with in-law pressure, threat of separation and sometimes violence.
- **Infertility, what the psychiatrist does.** Name the grief. Treat depression and anxiety on their own terms. Work with the couple, not only the woman who was sent. Counsel the decision to stop treatment, which nobody else offers. And know that clomifene and the gonadotropin releasing hormone agonists carry mood effects of their own.
- **Cancer.** The sequence runs from distress at diagnosis, through adjustment disorder, depression and anxiety, to delirium, which is the commonest psychiatric diagnosis in advanced disease, and on to demoralisation and desire for hastened death, with fear of recurrence and body image difficulty in survivors. Somatic items score falsely, so weight hopelessness, worthlessness, guilt and anhedonia. Exclude pain, hypercalcaemia, brain secondaries, steroids and opioid toxicity before diagnosing depression. Avoid strong CYP2D6 inhibitors such as fluoxetine and paroxetine alongside tamoxifen; mirtazapine buys appetite and sleep as well as mood.

WHAT EARNS THE MARKS

- **The direction of causation stated for infertility,** which is the examinable claim in that limb.
- **The social and gendered burden named,** since that is where Indian practice differs.
- **Delirium named in advanced cancer,** not depression alone.
- **The somatic-item problem, and one named drug interaction,** which lift this above a sad list.

SEE ALSO Slot IV - 08 promotion of mental health Slot IV - 22 consultation liaison psychiatry **SPRINT** Day 40, Medical and systemic-disease psychiatry

WEAVE 100 · PAPER IV · F02

CANCER: WHEN CELL CONTROL FAILS

Foundation 02 • Biology → local damage → systemic effects → treatment logic

NORMAL CELL CONTROL

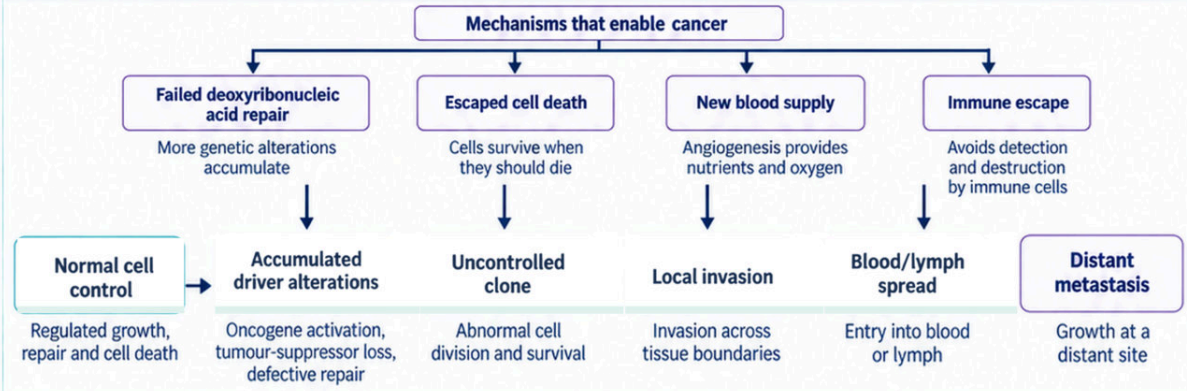
Healthy tissues balance cell division, differentiation and programmed cell death. Growth signals tell cells when to divide; **tumour-suppressor systems** restrain inappropriate growth; **deoxyribonucleic acid-repair systems** correct damage. Immune surveillance can recognise abnormal cells. A tissue maintains its structure because cells respond to their neighbours and remain in their normal compartment.

HOW CANCER DEVELOPS

Cancer results from **acquired** genetic and epigenetic changes that allow a clone of cells to grow abnormally. Some changes are inherited, but most arise during life through replication errors or exposures. **Oncogene activation** accelerates growth; **tumour-suppressor loss** removes restraint; **defective repair** permits further alterations. Cells may **evade death** and immune attack and recruit a blood supply. These changes accumulate; a single mutation does not automatically mean cancer.

Benign tumours remain local and do not metastasise, although compression can still be dangerous. **Malignant tumours** invade tissue and may spread. **Carcinomas** arise from **epithelium**; **sarcomas** from supporting tissues; **leukaemias and lymphomas** from blood-forming or lymphoid cells. Some cancers do not form a discrete solid mass.

LABELLED DISEASE PATHWAY



Normal control → driver alterations → clonal expansion → invasion across tissue boundaries → entry into blood or lymph → growth at a distant site.

A breast cancer deposit in bone remains metastatic breast cancer, not a new primary bone cancer. Spread and tumour biology influence prognosis and treatment.

WHY SYMPTOMS OCCUR

- **Local growth** → lump, obstruction or impaired organ function.
- **Ulceration and fragile vessels** → bleeding; chronic blood loss can cause anaemia and fatigue.
- **Compression or invasion of nerves and bone** → pain; bone destruction can cause fracture.
- **Inflammation plus altered metabolism and reduced intake** → fatigue, weight loss and cachexia. Cachexia includes muscle loss and is not simply starvation.
- **Bone-marrow infiltration or treatment injury** → low red cells, platelets or neutrophils: fatigue, bleeding or infection.
- **Ectopic hormone production or immune effects** → **paraneoplastic syndromes**; these are effects away from direct tumour invasion.

Symptoms vary by site. Neither pain nor weight loss is required, and many suspicious symptoms have non-cancer causes.

DIAGNOSE, THEN CHARACTERISE

History and examination guide imaging and blood tests. Tissue sampling usually establishes cancer type; selected blood cancers need blood or marrow studies. Pathology examines cell appearance and may test receptors or molecular alterations that guide therapy.

Stage describes extent: the primary tumour, regional nodes and distant spread. The tumour–node–metastasis system applies to many solid cancers, not all malignancies. **Grade** describes microscopic differentiation/aggressiveness; **stage and grade are different.**

Complete blood count, renal and liver tests assess complications and treatment fitness. **Tumour markers** have selected diagnostic or monitoring roles; they do not provide a universal cancer screen. A normal marker cannot exclude cancer. There is no single numerical threshold that diagnoses all cancers: use disease-specific criteria, units and laboratory ranges.

TREATMENT FOLLOWS THE TARGET

Surgery removes local disease; **radiotherapy** damages deoxyribonucleic acid in a planned treatment field. **Chemotherapy** acts systemically against susceptible cells. **Endocrine treatment** blocks hormone-dependent growth; **targeted therapy** acts on a relevant molecular pathway; **immunotherapy** helps immune control.

Many patients need combinations. Choice depends on cancer type, extent, biomarkers, organ function and patient goals—not stage alone.

Supportive and palliative care treat pain, nausea, nutrition problems and other burdens throughout care, including during curative treatment.

Prevention includes tobacco avoidance, appropriate vaccination and evidence-based screening; screening recommendations depend on cancer type and individual risk.

URGENT COMPLICATIONS

Fever during neutropenia; new limb weakness or bladder disturbance suggesting spinal compression; severe breathlessness; major bleeding; or rapidly developing metabolic illness requires **urgent assessment**. Do not attribute every new symptom to tumour progression: treatment toxicity and infection can be reversible.

MEMORY AID

Stage = where it has spread; **grade** = how cells look.

Sources: National Cancer Institute—What is cancer, diagnosis, staging, treatment types and side effects.
Supplementary foundation linked to original Paper IV Question 26, page 41. Reviewed September 2026. Foundation 02.

Cancer biology. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-26.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · F14

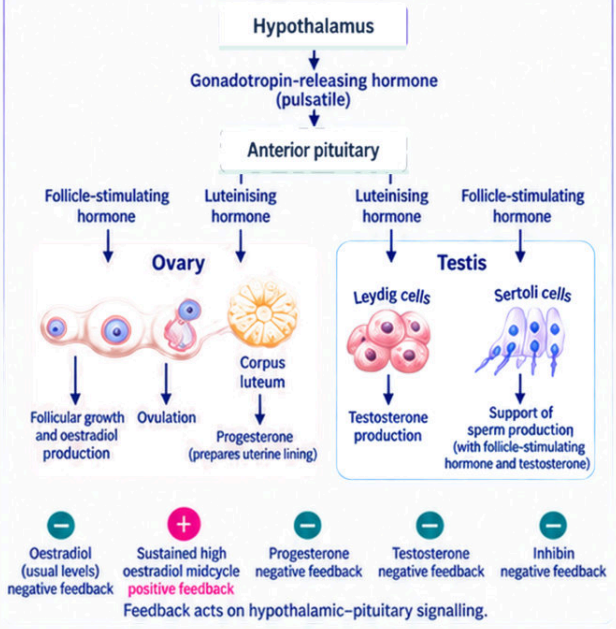
REPRODUCTIVE PHYSIOLOGY AND INFERTILITY

Foundation 14

NORMAL REPRODUCTION

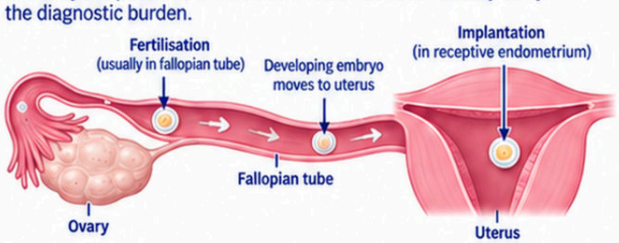
The hypothalamus releases pulsatile gonadotropin-releasing hormone. The pituitary responds with follicle-stimulating hormone and luteinising hormone. In ovaries, follicle-stimulating hormone supports follicular growth and oestradiol production. Sustained high oestradiol switches to positive feedback, generating the luteinising-hormone surge and ovulation. After ovulation, the corpus luteum produces progesterone, preparing the uterine lining. If implantation does not occur, hormone levels fall and menstruation begins. In testes, luteinising hormone stimulates Leydig-cell testosterone production; follicle-stimulating hormone and testosterone support Sertoli-cell sperm production. Testosterone and inhibin provide negative feedback.

HYPOTHALAMIC–PITUITARY–GONADAL AXES



FROM OVULATION TO IMPLANTATION

Sperm deposited in the reproductive tract travel towards the fallopian tube. Fertilisation usually occurs there; the developing embryo moves into the uterus and implants in a receptive endometrium. Pregnancy therefore requires sufficiently functional sperm, ovulation, accessible tubes, fertilisation and successful implantation. Female age affects oocyte number and quality, particularly chromosomal competence. Male factors, female factors, combined factors or unexplained infertility are possible; the woman should not automatically carry the diagnostic burden.



WHAT INFERTILITY MEANS

The World Health Organization defines infertility as failure to achieve pregnancy after at least twelve months of regular unprotected sexual intercourse. Primary infertility means no previous pregnancy; secondary infertility follows a previous pregnancy. Evaluation should begin earlier when a known risk exists. Common specialist practice begins evaluation after six months when the woman is at least 35 years, and more promptly over 40 years or with clear pathology. This evaluation threshold is not a different biological definition.

MECHANISM → CLUE

- **Absent or irregular ovulation** → irregular or absent periods; consider polycystic ovary syndrome, thyroid dysfunction, hyperprolactinaemia, hypothalamic suppression or ovarian insufficiency.
- **Tubal damage** from infection or surgery → impaired gamete transport.
- **Endometriosis** can cause pain, inflammation and adhesions.
- **Uterine abnormalities** may impair implantation.
- **Reduced sperm production**, obstruction, erectile or ejaculatory dysfunction may prevent conception.
- **Medicines**, gonadotoxic chemotherapy, exogenous testosterone and anabolic steroids matter; external testosterone can suppress sperm production.
- **Distress** is often a consequence and does not establish the cause.

ASSESS BOTH PARTNERS TOGETHER

Take reproductive, menstrual, sexual, medical, medication and exposure histories; examine as indicated. Perform semen analysis and assess ovulation and reproductive anatomy. Regular cycles of 21–35 days often indicate ovulation. When needed, progesterone is measured about one week before the expected next period, not automatically on day 21; a value above 3 nanograms/millilitre supports recent ovulation but does not grade luteal quality. Semen parameters vary substantially; repeat abnormal results and interpret with laboratory reference limits. A result above or below a lower reference limit does not alone prove fertility or infertility. Ultrasound assesses ovaries and uterus; tubal patency testing is selected according to history. Anti-Müllerian hormone and antral follicle count estimate ovarian response to stimulation, not a guaranteed chance of spontaneous pregnancy. There is no single universal normal fertility value.

CAUSE-DIRECTED CARE

Clarify goals and prognosis → address smoking, relevant exposures, nutrition, chronic disease and intercourse difficulties → treat identified causes. Ovulation induction may help selected anovulatory disorders; tubal, uterine or male disease may need specialist treatment. Intrauterine insemination places prepared sperm in the uterus. In-vitro fertilisation brings eggs and sperm together outside the body, followed by embryo transfer; intracytoplasmic sperm injection injects one sperm into an egg in selected cases. Discuss treatment burden, multiple pregnancy, ovarian hyperstimulation, costs and realistic success by age and diagnosis. Support informed choices, including pausing or stopping treatment, without blame or promises of success.

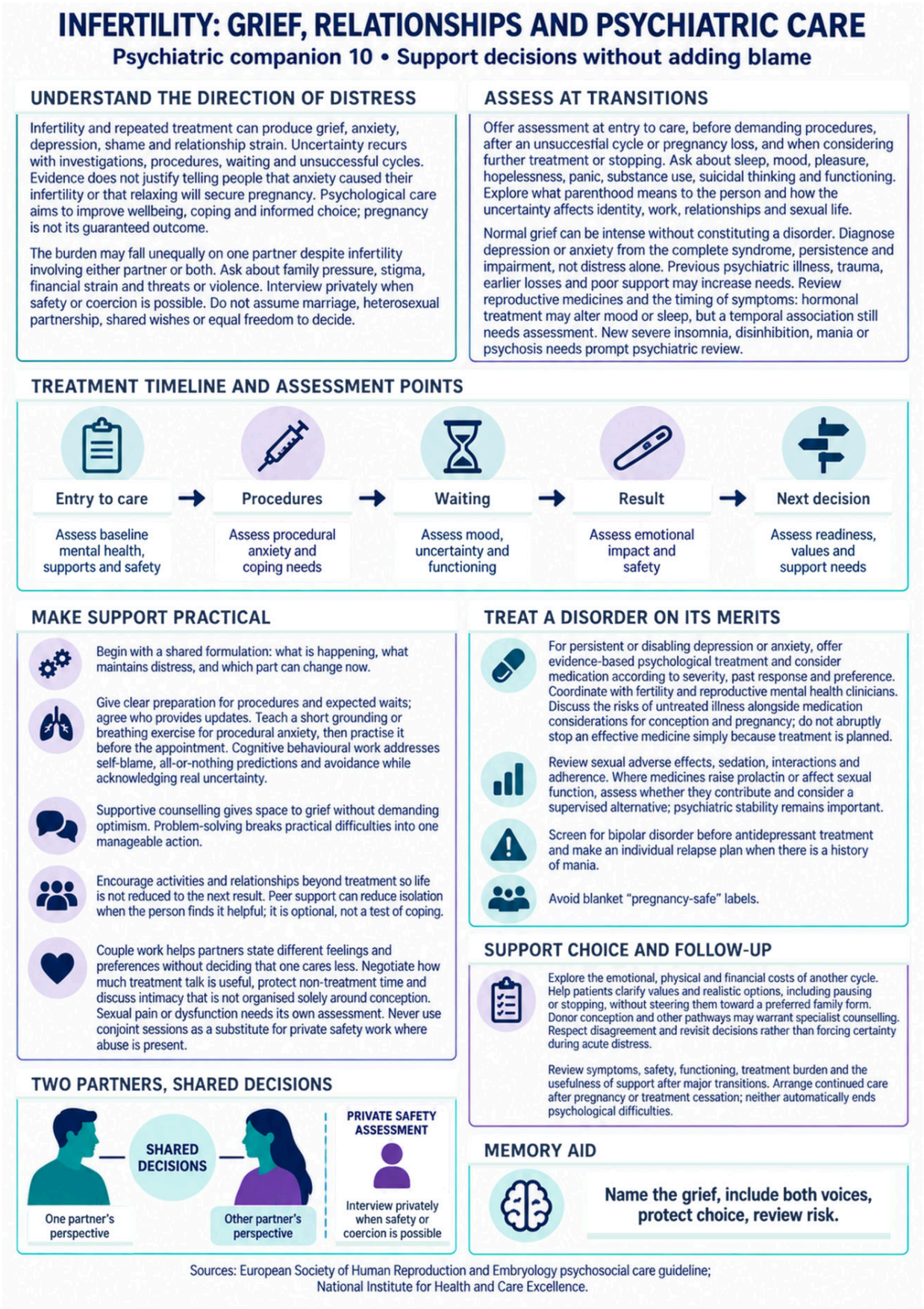
Signals, sperm, ovulation, tubes and implantation: assess together.

Sources: World Health Organization; American Society for Reproductive Medicine.

Reproductive physiology and infertility. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-26.

Credits and references: retained within the illustration.

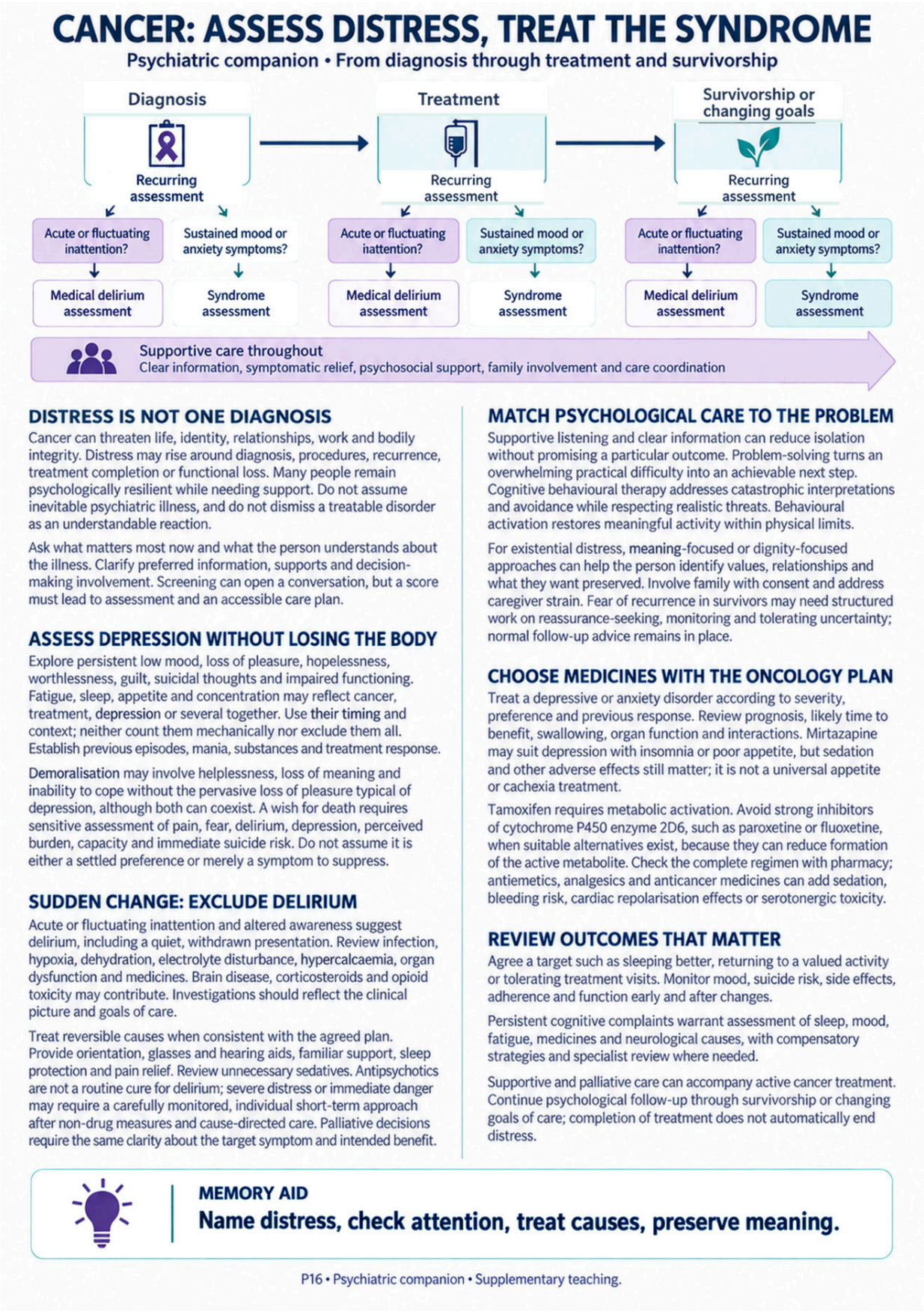
WEAVE 100 · PAPER IV · P10



Infertility psychiatry. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-26.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P16



Cancer psychiatry. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-26.

Credits and references: retained within the illustration.

IV - 27

Write a note on depression with ischaemic heart disease. [10]

10 CONSULTATION-LIAISON AND SYSTEMIC MEDICINE PSYCHIATRY

KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	The association	Common, and independently prognostic
3	Why they travel together	Behaviour and biology, in both directions
2	Making the diagnosis	The overlap, and which items to weight
3	Treatment	The table, rehabilitation, and the honest limit

THE CLAIM WORTH WRITING FIRST

Depression is common after myocardial infarction and in stable coronary disease, and it predicts a worse cardiac outcome independently of the severity of the heart disease. That independence is why this is a question at all: a mere reaction to illness would carry no separate prognosis.

- **Why they travel together.** Behaviourally: smoking, inactivity, poor diet, and poor adherence to cardiac drugs and to rehabilitation. Biologically: reduced heart rate variability from autonomic imbalance, platelet activation, inflammation, and activation of the hypothalamic pituitary adrenal axis. The traffic runs both ways, which is why the answer should not be written as one causing the other.
- **Making the diagnosis.** Fatigue, poor sleep and poor appetite belong to both illnesses, so weight the cognitive and affective items: low mood, anhedonia, hopelessness, guilt. Screen rather than wait for the patient to volunteer it. Then exclude the impostors: adjustment disorder, delirium after bypass surgery, hypoxia, and the effects of drugs.

TIER	THE AGENT OR THE MEASURE	WHY IT SITS THERE
First choice	Sertraline, with citalopram and escitalopram acceptable	**Best cardiac safety evidence after infarction**
Non-drug	Cardiac rehabilitation with an exercise component; cognitive behavioural therapy	Improves mood, and the exercise limb helps the heart
Watch	Citalopram at higher dose, and other agents that prolong the QT interval	Get an electrocardiogram before and after
Avoid	Tricyclic antidepressants	**Conduction delay, tachycardia, postural drop**

- **The honest limit, and the interactions.** Treating the depression reliably improves mood, function and adherence; that it reduces cardiac events is not established, and trials of therapy after infarction have shown the mood gain without a survival gain. Serotonergic drugs with antiplatelets raise bleeding risk, and strong inhibitors of CYP2C19 may blunt clopidogrel, which is a prodrug.

WHAT EARNS THE MARKS

- **Independence of the prognosis stated,** not just that the two occur together.
- **Both mechanisms, behavioural and biological.**
- **Sertraline named, and tricyclics named as the thing to avoid.**
- **The limit admitted,** since claiming a survival benefit is the error this question invites.

SEE ALSO Slot IV - 22 consultation liaison psychiatry Slot IV - 29 psychological intervention in bronchial asthma

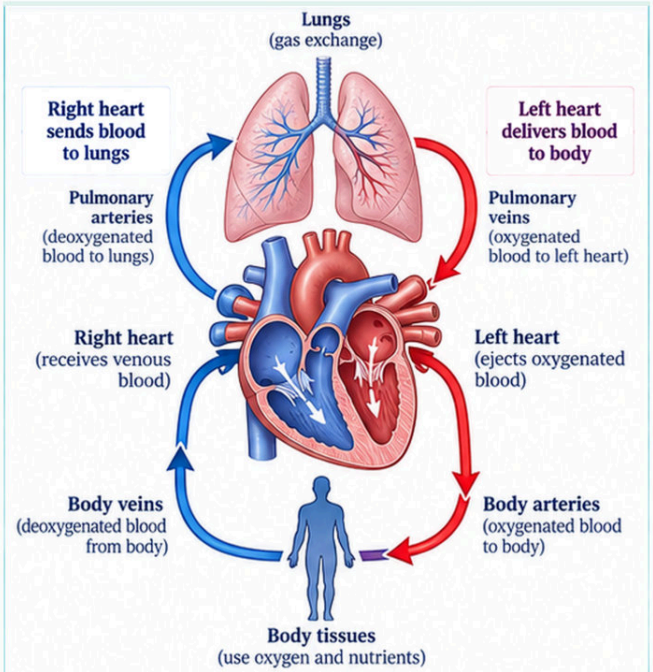
SPRINT Day 40, Medical and systemic-disease psychiatry

WEAVE 100 · PAPER IV · F06

HEART FAILURE: PUMPING, FILLING AND CONGESTION

Foundation 06 • Why breathlessness and oedema occur

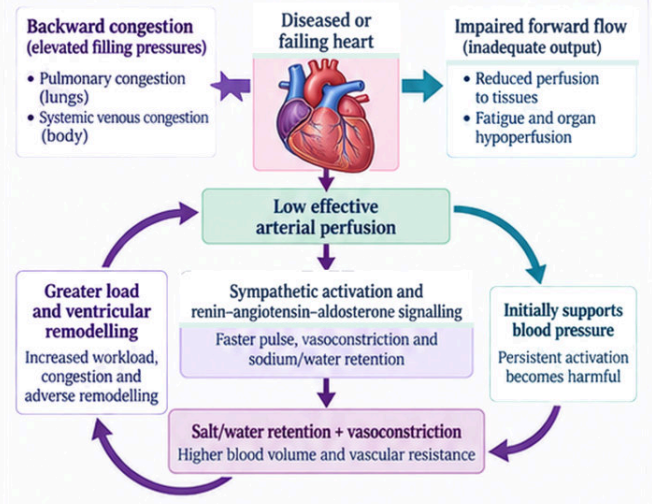
NORMAL CIRCULATION



The right heart receives systemic venous blood and sends it through the lungs; the left heart delivers oxygenated blood to the body. Valves maintain one-way flow. Relaxation allows filling; contraction ejects blood. Cardiac output equals heart rate multiplied by stroke volume. Output depends on filling, contractility and resistance to ejection, not heart rate alone.

Coronary arteries supply the myocardium itself. Electrical activation coordinates contraction. A normal ejection fraction describes the proportion of ventricular blood ejected per beat; it is not the percentage of the entire body's blood pumped or a complete measure of cardiac performance.

FORWARD FLOW, BACKWARD CONGESTION AND COMPENSATION



WHAT HEART FAILURE MEANS

Heart failure is a clinical syndrome from structural or functional cardiac abnormality, producing symptoms/signs through elevated filling pressures and/or inadequate output. It can arise from impaired contraction, impaired filling or both. 'Congestive' highlights fluid accumulation; not every stable patient is visibly congested. The heart has not necessarily stopped.

Common causes include coronary disease/infarction, prolonged hypertension, cardiomyopathy, valve disease and arrhythmia. Acute deterioration may be triggered by ischaemia, infection, arrhythmia, uncontrolled pressure, kidney dysfunction or interruption of treatment.

THE COMPENSATION THAT BECOMES A PROBLEM

Reduced effective arterial perfusion → sympathetic activation and renin–angiotensin–aldosterone signalling → faster pulse, vasoconstriction and sodium/water retention.

Initially this supports pressure; persistent activation increases workload, congestion and adverse ventricular remodelling. Natriuretic peptides released with cardiac wall stress promote salt exceetion and counter-regulation, but may not overcome the disease.

MECHANISM → SYMPTOM

Raised left-sided filling pressures → pulmonary venous congestion → exertional breathlessness, orthopnoea and night-time breathlessness. Severe fluid movement into alveoli causes pulmonary oedema.

Systemic venous congestion → raised jugular venous pressure, workloadt oedema, liver congestion or ascites.

Reduced forward perfusion and skeletal-muscle changes → fatigue and poor exercise tolerance; severe hypoperfusion causes cold extremities, reduced urine and altered consciousness.

Fluid retention → rapid weight increase. Congestion can impair kidney function, while renal sodium retention worsens cardiac congestion: a two-way interaction.

EJECTION FRACTION: CLASSIFY, DO NOT DIAGNOSE ALONE

Left ventricular ejection fraction ≤40%: reduced.
41–49%: mildly reduced.
≥50%: preserved; heart failure still requires clinical and objective evidence, often raised filling pressures.

A preserved fraction does not prove normal filling. Echocardiography also assesses valves, chamber structure and other functional abnormalities.

INVESTIGATIONS AND NUMBERS

History/examination, electrocardiogram, natriuretic peptide testing and echocardiography establish the syndrome and cause. Check blood count, kidney function, electrolytes, liver/thyroid function and other tests as indicated; chest radiography helps assess congestion and alternatives.

National Institute for Health and Care Excellence chronic suspected-heart-failure pathway: N-terminal pro-B-type natriuretic peptide >2,000 ng/L → specialist assessment/echocardiography within 2 weeks; 400–2,000 ng/L → within 6 weeks; <400 ng/L in an untreated patient makes failure less likely, not impossible.

These are referral thresholds, not universal normal ranges or acute-care cutoffs. Obesity and treatment can lower levels; renal dysfunction, atrial fibrillation, age and other illnesses can raise them.

BASIC MANAGEMENT

Treat the cause and precipitant. Diuretics relieve congestion. Reduced-ejection-fraction disease generally needs disease-modifying therapy across four groups: renin–angiotensin-system/neprilysin therapy, evidence-based beta-blockade, mineralocorticoid-receptor antagonism and sodium–glucose cotransporter 2 inhibition, with individual eligibility and monitoring. Preserved-fraction care treats congestion and comorbidity; selected therapies also improve outcomes.

Monitor symptoms, weight, blood pressure, renal function and potassium. Offer rehabilitation, vaccination, self-management support and selected devices/procedures. Avoid indiscriminate fluid restriction or harmful medicines. Severe breathlessness at rest, chest pain, syncope or shock signs needs urgent assessment rather than routine clinic triage.

Filling pressure congests; poor perfusion fatigues.

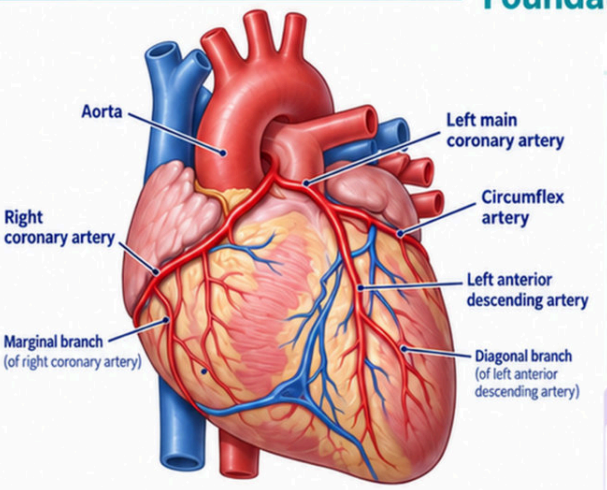
Heart physiology and congestive heart failure. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-27.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · F11

CORONARY CIRCULATION AND ISCHAEMIC HEART DISEASE

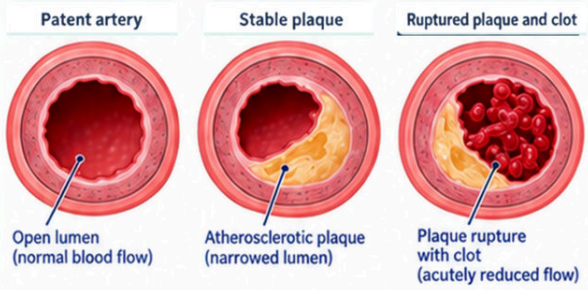
Foundation 11



Anterior schematic; posterior venous drainage not shown.

Venous blood returns mainly through the coronary sinus.

CORONARY ARTERY CROSS-SECTIONS

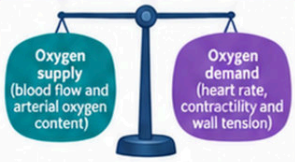


DIASTOLIC PERFUSION

Left ventricular perfusion occurs predominantly during diastole, when the muscle relaxes.



SUPPLY–DEMAND BALANCE



Balance maintains myocardial function. Imbalance can cause ischaemia.

NORMAL SUPPLY MEETS DEMAND

The coronary arteries arise from the aortic root and deliver oxygenated blood to the myocardium, the heart muscle. The left main divides into the left anterior descending and circumflex arteries; the right coronary supplies much of the right heart and, in most people, the inferior left ventricle. Distribution varies. Venous blood returns mainly through the coronary sinus. Left ventricular perfusion occurs predominantly during diastole, when the muscle relaxes. Faster heart rate both raises oxygen demand and shortens filling and perfusion time. Oxygen supply depends on blood flow and arterial oxygen content; demand rises with heart rate, contractility and wall tension.

WHAT GOES WRONG

Ischaemia means oxygen supply is insufficient for tissue demand. **Atherosclerosis** is an inflammatory plaque process within an arterial wall: retained cholesterol → inflammation → fibrous plaque. A fixed narrowing limits flow reserve, producing reproducible exertional angina. Plaque rupture or erosion can trigger platelet activation and a clot, abruptly reducing flow: an acute coronary syndrome. Myocardial infarction means myocardial injury caused by ischaemia, with death of heart muscle. Not every raised troponin is infarction. Severe anaemia, hypoxaemia or tachyarrhythmia can cause supply–demand mismatch without acute plaque rupture. Coronary spasm and small-vessel dysfunction can cause ischaemia even without a major obstructive plaque.

MECHANISM → CLINICAL FINDING

Transient ischaemia → pressure, heaviness or constriction in the chest, sometimes spreading to arm, jaw or back. Exertion or emotional stress raises demand; rest reduces it. Breathlessness may be an anginal equivalent, especially in older adults or diabetes. **Persistent ischaemia** → myocardial necrosis, reduced pumping, arrhythmia or shock. Sweating, nausea, collapse, new rest pain or prolonged chest pressure require emergency assessment. Neither a normal initial electrocardiogram nor atypical pain excludes an acute coronary syndrome.

INVESTIGATE THE QUESTION

Acute pain → immediate clinical assessment, twelve-lead electrocardiogram and serial high-sensitivity cardiac troponin using a validated local pathway. The troponin threshold is the assay-specific ninety-ninth percentile; there is no universal normal number. A rise or fall plus evidence of ischaemia supports infarction. Repeat electrocardiography when symptoms continue. Echocardiography assesses regional movement, valves and ventricular function. For stable suspected coronary disease, coronary computed tomography angiography or functional stress imaging is selected according to probability, previous disease and local guidance. Check blood count, kidney function, glucose, glycated haemoglobin and lipids. Resting adult pulse commonly ranges from 60–100 beats/minute; this is a reference, not a treatment target. Risk-factor targets are individualised; laboratory reference intervals are not equivalent to secondary-prevention goals.

BASIC MANAGEMENT FLOW

Possible acute coronary syndrome → emergency cardiac pathway → distinguish occlusive infarction, other infarction and unstable angina → antithrombotic treatment and reperfusion when indicated. Do not delay emergency care for an outpatient test.

Stable disease → stop smoking, tailored activity and cardiac rehabilitation, dietary support, blood-pressure and diabetes management. A statin reduces future vascular risk; antiplatelet therapy is prescribed when indicated after assessing bleeding risk. A short-acting nitrate relieves angina; avoid combining nitrates with phosphodiesterase-five inhibitors because severe hypotension may result. A beta blocker or calcium-channel blocker is selected for symptom prevention; consider pulse, conduction, heart failure and airway disease. Persistent symptoms or high-risk anatomy → specialist review for coronary intervention or bypass surgery. Review symptoms, adherence, adverse effects and function; symptom relief and prevention of future events are separate goals.

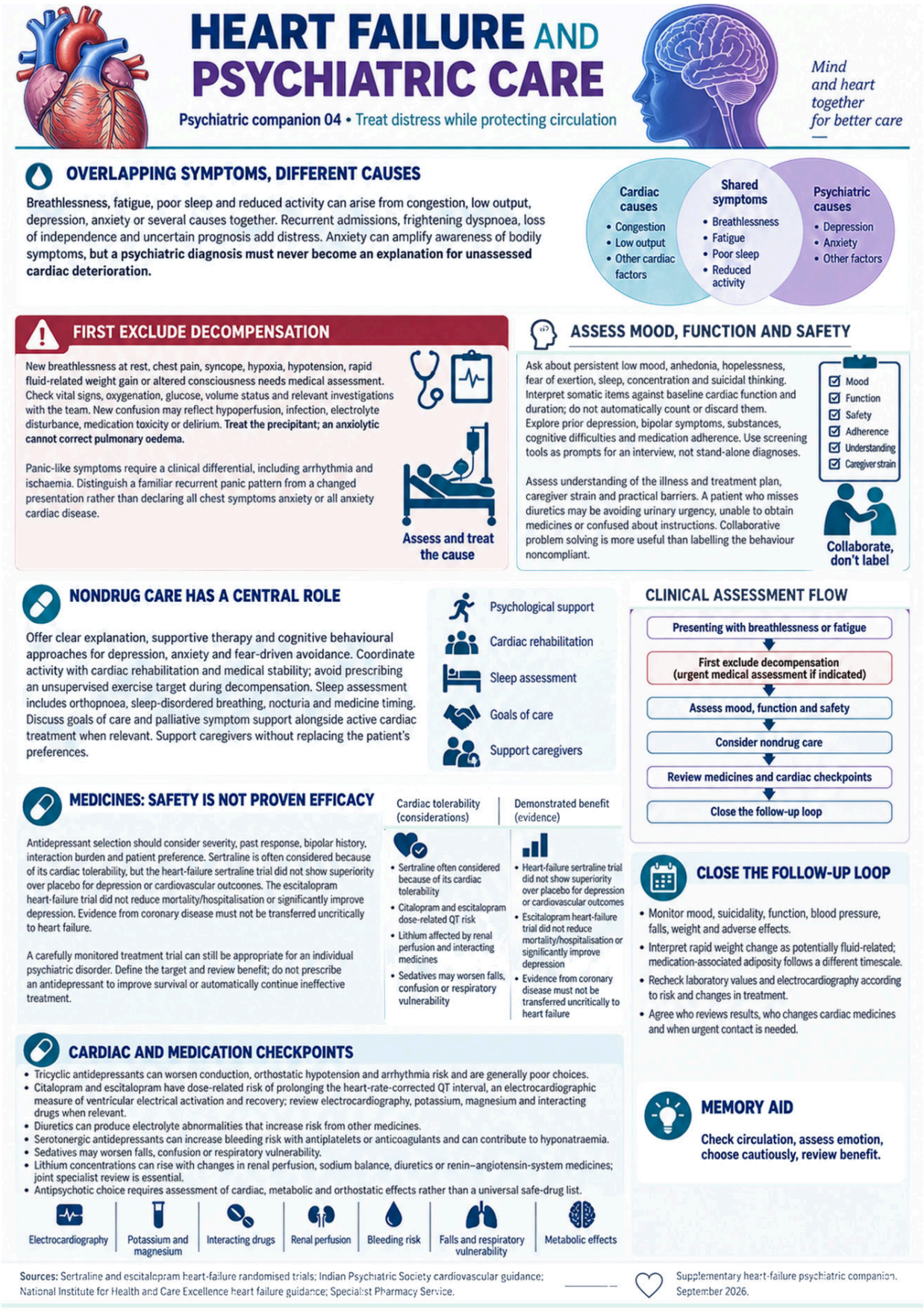
Match supply to demand; distinguish injury from infarction.

Sources: National Heart, Lung, and Blood Institute; National Institute for Health and Care Excellence.

Coronary circulation and ischaemic heart disease. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-27.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P04



Heart failure psychiatry. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-27.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P09

ISCHAEMIC HEART DISEASE: DEPRESSION AND *RECOVERY*

Psychiatric companion 09 • Identify the syndrome → protect the heart → restore participation

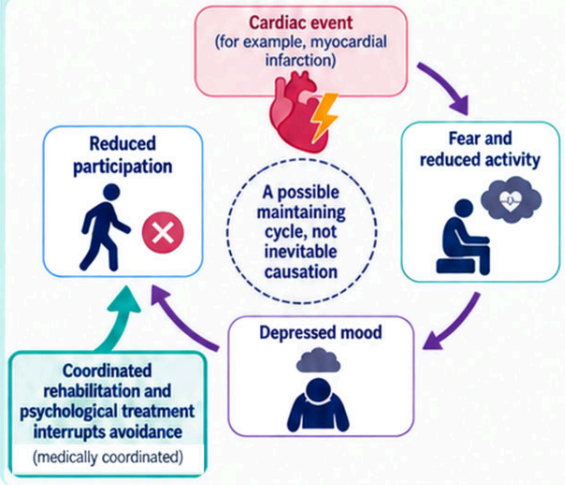
WHY THE ASSOCIATION MATTERS

- Depression and coronary disease influence each other through behaviour, physiology and the burden of illness.
- Smoking, inactivity and disrupted treatment routines can increase cardiovascular risk; infarction, disability and fear can precipitate depression.
- Autonomic imbalance, inflammatory pathways and platelet activity may contribute.
- An association with worse cardiac prognosis does not prove that depression is the sole cause or that antidepressant treatment prevents another infarction.

ASSESS THE PERSON, NOT THE SOMATIC SCORE

- Ask directly about persistent low mood, loss of pleasure, hopelessness, guilt, suicidal thoughts and withdrawal from valued activities.
- Fatigue, poor sleep, appetite change and slowed activity may arise from cardiac illness, depression or both; do not automatically discard them, but interpret their timing and context.
- A questionnaire identifies people needing assessment, not the diagnosis by itself.
- Establish onset relative to the cardiac event, duration, functional impairment, past depression, mania, substance use and treatment response.
- Seek collateral history with consent.
- Distinguish understandable distress, adjustment disorder, major depression and delirium after surgery.
- Review hypoxia, anaemia, thyroid disease, sleep problems and medicines when clinically indicated.
- New chest discomfort, breathlessness, syncope or unstable observations needs medical assessment before an anxiety explanation.

A POSSIBLE MAINTAINING CYCLE (NOT INEVITABLE)



MEDICATION-SAFETY GATE

Bleeding
Higher risk with selective serotonin reuptake inhibitors and antiplatelets/ anticoagulants

Rhythm
Caution with citalopram and escitalopram (dose-related prolongation of ventricular repolarisation); avoid tricyclic antidepressants

Interactions
Check for interactions with cardiovascular medicines (including enzyme inhibitors)

BUILD A JOINED TREATMENT PLAN

- Explain the link without blaming the patient. Set one achievable rehabilitation goal, identify the barrier and agree how progress will be reviewed.
- Coordinate psychiatry, cardiology, primary care and rehabilitation so advice on exercise, medicines and follow-up is consistent.
- Support tobacco cessation, sleep regularity and attendance at medically supervised cardiac rehabilitation.
- Help the patient plan transport, reminders and manageable daily activity.
- Cognitive behavioural therapy examines catastrophic interpretations such as "any faster heartbeat means another heart attack", tests these against the cardiac plan and rebuilds safe activity gradually.
- Behavioural activation schedules rewarding tasks within physical limits.
- Problem-solving therapy addresses practical obstacles rather than giving generic reassurance.
- Include family when the patient wants this; excessive protective restriction can reinforce avoidance.

CHOOSE MEDICINES WITH CARDIAC RISK IN VIEW

- For depression requiring medication, sertraline is commonly preferred because of its established cardiac safety experience. Choice also depends on previous response, interactions, bleeding risk and patient preference.
- Start conservatively and titrate against benefit and tolerability; cardiac safety does not remove ordinary monitoring for activation, suicidality or possible bipolar disorder.
- Selective serotonin reuptake inhibitors can increase bleeding risk when combined with aspirin, clopidogrel or anticoagulants. Review bruising, gastrointestinal bleeding history and the need for gastroprotection with the treating team.
- Strong enzyme inhibitors may interact with cardiovascular medicines, so reconcile the complete prescription and non-prescription list.
- Citalopram and escitalopram require particular caution because of dose-related prolongation of ventricular repolarisation; they are not interchangeable default first choices in coronary disease. Consider an electrocardiogram, electrolyte abnormalities and interacting medicines when their use is justified.
- Tricyclic antidepressants are generally avoided because of conduction effects, arrhythmia, postural hypotension and toxicity in overdose.
- Mirtazapine may be an alternative, with weight gain and sedation considered.

MONITOR WHAT RECOVERY MEANS

- Review mood, suicide risk, adherence, adverse effects and meaningful function early after starting treatment and after dose changes.
- Track participation in rehabilitation, activity and relationships, not only a questionnaire total.
- Persistent symptoms require reassessment of diagnosis, adequate treatment exposure, medical contributors and barriers before changing treatment.
- Continue effective care through recovery and plan gradual withdrawal when appropriate.
- Treating depression can improve symptoms and quality of life. Do not promise fewer cardiac events or a survival benefit from antidepressants alone.

MEMORY AID

Assess mood, check the heart, restore activity, review safety,

Sources: Specialist Pharmacy Service; National Institute for Health and Care Excellence.

Ischaemic heart disease psychiatry. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-27.

Credits and references: retained within the illustration.

IV - 28

Discuss the role of the psychiatrist in kidney transplant. [10]

10 CONSULTATION-LIAISON AND SYSTEMIC MEDICINE PSYCHIATRY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Why kidney is different**	A living-donor programme, and a dialysis population
3	**The recipient**	Depression on dialysis, cognition, adherence
3	**The living donor**	Voluntariness, the committee, coercion at home
2	**Prescribing and after**	The table, and what follows graft failure

WHY THIS QUESTION IS NOT THE GENERIC TRANSPLANT QUESTION

Kidney is the transplant with a large living-donor programme, so the donor assessment is a routine part of the work rather than an occasional one. And the recipient has usually spent years on dialysis, which supplies both the commonest psychiatric problem in this group and the best available test of adherence.

- **The recipient.** Depression is the commonest psychiatric disorder in end-stage renal disease, is under-recognised, and is associated with poorer outcome and with withdrawal from dialysis. Score it on the cognitive items, because fatigue, poor sleep and poor appetite belong to uraemia and to dialysis. Assess cognition, which is often impaired. Then read the dialysis record: attendance, fluid restriction and diet are the behavioural rehearsal for post-transplant immunosuppression.
- **The living donor.** Interview the donor alone, without the family in the room. Test voluntariness, capacity, understanding of the risk, and the absence of payment or pressure. Donation is governed by the Transplantation of Human Organs and Tissues Act 1994 as amended in 2011, under which an authorisation committee scrutinises donation by anyone who is not a near relative. Coercion inside a family is the harder case, and in Indian practice it falls most often on wives, daughters-in-law and unmarried sisters.

PRESCRIBING	WHAT TO DO	WHY
Usual first choices	Sertraline or citalopram, started low	Little renal handling, few graft interactions
Reduce or avoid	Renally cleared agents, including amisulpride, sulpiride, paliperidone, gabapentin, pregabalin	**They accumulate as clearance falls**
Special care	Lithium	**The graft is the clearance route now**
Expect from the graft drugs	Steroid mood change; calcineurin inhibitor tremor, insomnia, confusion	Treat the cause, not a new diagnosis

WHAT EARNS THE MARKS

- **Depression on dialysis named as the commonest problem,** and scored on cognitive items.
- **The donor interviewed alone, and the Act and committee named.**
- **Coercion inside the family raised,** which is the part a generic answer never reaches.
- **Renal clearance driving the prescribing,** not a list of favourite antidepressants.

SEE ALSO Slot IV - 24 the psychiatrist in organ transplant Slot IV - 22 consultation liaison psychiatry **SPRINT** Day 40, Medical and systemic-disease psychiatry

WEAVE 100 · PAPER IV · F05

KIDNEYS: FILTER, BALANCE AND SIGNAL

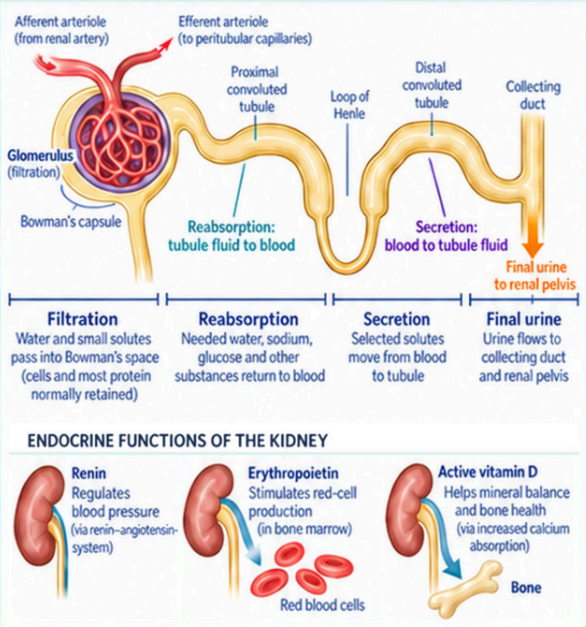
Foundation 05 • Normal function → chronic injury → kidney failure

NORMAL PHYSIOLOGY

Each kidney contains nephrons. Glomeruli filter water and small solutes from blood while normally retaining cells and most protein. Tubules reclaim needed water, sodium, glucose and other substances, secrete selected solutes, and determine the final urine. The kidneys regulate extracellular volume, potassium, acid–base balance and removal of metabolic waste and many medicines.

They also release renin for blood-pressure regulation, make erythropoietin to support red-cell production and activate vitamin D for mineral balance. Urine volume is not the same as filtration: someone can pass urine despite major kidney impairment.

THE NEPHRON: FILTRATION, REABSORPTION, SECRETION AND ENDOCRINE FUNCTIONS



ENDOCRINE FUNCTIONS OF THE KIDNEY

HOW CHRONIC DISEASE DEVELOPS

Diabetes, hypertension, glomerular inflammation, inherited disorders or obstruction can injure nephrons. Surviving nephrons compensate, but persistent pressure, protein leakage, inflammation and scarring may drive further loss. Early disease is often silent.

Chronic kidney disease means abnormal kidney structure or function with health implications for at least 3 months. Either reduced filtration or a marker of damage can establish it. One abnormal creatinine result does not establish chronicity: exclude acute kidney injury and review previous results.

FUNCTION LOST → CLINICAL CONSEQUENCE

Function lost	Clinical consequence
Sodium/water excretion falls	oedema, hypertension or pulmonary congestion.
Potassium excretion falls	hyperkalaemia and potentially dangerous cardiac arrhythmia, sometimes without warning symptoms.
Acid excretion falls	metabolic acidosis and increased breathing effort when severe.
Waste products accumulate	uraemic nausea, reduced appetite, itching, impaired cognition or pericarditis in advanced disease.
Erythropoietin production fails	anaemia and fatigue; iron deficiency and other causes may coexist.
Phosphate retention and reduced active vitamin D	secondary hyperparathyroidism, disturbed calcium balance and bone disease.
Drug clearance falls	accumulation and toxicity unless doses are reviewed. Cardiovascular risk also rises.

TWO AXES OF RISK

Estimated glomerular filtration rate is reported in mL/min/1.73 m²; it is not a percentage. Categories: G1 ≥90; G2 60–89; G3a 45–59; G3b 30–44; G4 15–29; G5 <15. G1/G2 alone do not mean chronic disease without another damage marker.

Urine albumin:creatinine ratio: A1 <30 mg/g (<3 mg/mmol); A2 30–300 mg/g (3–30 mg/mmol); A3 >300 mg/g (>30 mg/mmol). Higher albuminuria can confer risk even with preserved filtration.

Classify by cause, filtration and albuminuria. Confirm persistent abnormalities; exercise, infection and other temporary factors can alter urine results.

Kidney failure is G5; dialysis is not started from that number alone.

GLOMERULAR FILTRATION RATE CATEGORIES

Category	Estimated glomerular filtration rate (mL/min/1.73 m²)	Description
G1	≥90	Normal or high
G2	60–89	Mildly reduced
G3a	45–59	Mildly to moderately reduced
G3b	30–44	Moderately to severely reduced
G4	15–29	Severely reduced
G5	<15	Kidney failure

ALBUMINURIA CATEGORIES

Category	Urine albumin:creatinine ratio	
A1	<30 mg/g (<3 mg/mmol)	Normal to mildly increased
A2	30–300 mg/g (3–30 mg/mmol)	Moderately increased
A3	>300 mg/g (>30 mg/mmol)	Severely increased

FROM NEPHRON LOSS TO SYMPTOMS

Nephron loss
- Diabetes
- Hypertension
- Glomerular inflammation
- Inherited disorders
- Obstruction

→ **Reduced function**
- Lower filtration
- Impaired excretion
- Endocrine failure

→ **Clinical consequences (symptoms and signs)**
- Oedema, hypertension, pulmonary congestion
- Hyperkalaemia and arrhythmia
- Metabolic acidosis
- Uraemic symptoms (nausea, itching, cognition)
- Anaemia and fatigue
- Bone disease
- Drug accumulation and toxicity
- Increased cardiovascular risk

INVESTIGATIONS

Trend serum creatinine and estimated filtration; creatinine is affected by muscle mass, diet and medicines. Cystatin C can improve estimation when creatinine is misleading or precision changes decisions. Check urine albumin, urinalysis, electrolytes, bicarbonate, haemoglobin and relevant mineral measures. Ultrasound may identify obstruction or structural disease. Cause-specific testing or biopsy is selective.

BASIC MANAGEMENT

Treat the cause and reduce cardiovascular risk. Manage blood pressure and diabetes, support smoking cessation, appropriate exercise and individualised nutrition, and avoid unnecessary nephrotoxins. Renin–angiotensin-system blockade and sodium–glucose cotransporter 2 inhibitors protect kidneys in eligible patients; eligibility and laboratory monitoring matter. Review all medicine doses and potassium/creatinine after relevant changes.

Treat fluid overload, anaemia, acidosis and mineral complications according to findings. Plan kidney-replacement options early when progression warrants: haemodialysis, peritoneal dialysis or transplantation; comprehensive conservative care is also an active care pathway.

Start dialysis based on symptoms, refractory complications, laboratory findings, preferences and overall assessment. Urgent problems include refractory hyperkalaemia/acidosis, pulmonary oedema and uraemic complications. Dialysis replaces selected functions, not every hormonal function or all normal kidney physiology.

Filter, balance, signal: assess all three.

Kidney physiology chronic and end-stage kidney disease. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-28.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P03

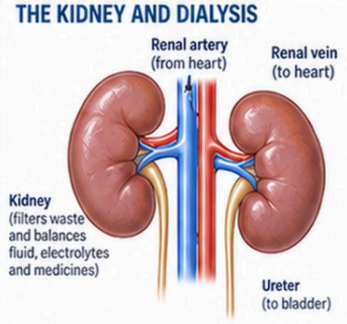
KIDNEY DISEASE, DIALYSIS AND PSYCHIATRIC CARE

Psychiatric companion 03 • Symptoms, clearance and treatment burden

WHY THE PRESENTATION IS COMPLEX

Chronic kidney disease can alter mood, sleep, cognition and medication handling. Uraemia, anaemia, vascular disease, inflammation, pain, itching and disrupted sleep overlap with depression. Dialysis schedules, dietary restrictions, dependence and uncertainty add psychological burden. Renal clearance is only one issue: protein binding, active metabolites, electrolyte shifts and dialysis removal can also change drug effects.

THE KIDNEY AND DIALYSIS





Dialysis removal is drug-dependent

Not all psychotropics are removed. Dialysability depends on the molecule, binding, distribution and dialysis technique.

ACUTE CHANGE IS A MEDICAL SIGNAL

New inattention, fluctuation, altered arousal, hallucinations or agitation requires delirium assessment. Review infection, hypoxia, glucose, sodium, calcium, uraemia, drug accumulation and withdrawal. A close relation to dialysis raises additional questions about haemodynamic or metabolic change: do not assume every post-dialysis symptom is psychiatric. Seizures, focal deficits or marked deterioration require urgent medical review. Stabilise physiology and treat the cause before relying on sedating medication.

Common medical precipitants of delirium in kidney disease

- Infection
- Hypoxia
- Glucose disturbance
- Sodium or calcium disturbance
- Uraemia
- Drug accumulation or withdrawal
- Haemodynamic or metabolic change related to dialysis

MEDICATION CLEARANCE CHECKPOINT

Consider more than renal clearance

Renal clearance
Reduced kidney function can increase exposure.

Protein binding
Highly bound drugs are less likely to be removed by dialysis.

Active metabolites
Some metabolites accumulate and may be active or toxic.

Electrolyte shifts
Changes in sodium, calcium and other electrolytes can atug effects.

Dialysis removal
Drug-dependent. Do not assume haemodialysis removes every psychotropic or automatically prescribe a replacement dose after each session.

DIAGNOSE DEPRESSION CAREFULLY

Ask about persistent low mood, loss of interest, hopelessness, guilt, suicidal thinking and functional change alongside energy, sleep and appetite. A screening questionnaire identifies possible illness; clinical assessment establishes the diagnosis. Somatic symptoms are not automatically excluded, but renal explanations and temporal patterns matter. Assess mania history, anxiety, substances use and previous treatment response. Distinguish distress about treatment from a depressive syndrome without minimising either.

Cognitive complaints require baseline and collateral history, attention testing and functional assessment. Consider delirium, vascular injury, sleep disorder and depression before assigning progressive dementia. Use interpretable cognitive tests and repeat assessment after reversible contributors improve.

PRESCRIBING: CHECK THE DRUG, NOT JUST THE CLASS

Sertraline
- Limited renal clearance
- Can be a practical option
- Consider bleeding, sodium, gastrointeistnal and interaction risks

Duloxetine
- United States labelling advises avoidance in severe renal impairment, below approximately 30 millilitres/minute
- Do not generalise this threshold to every antidepressant

Gabapentin, pregabalin, amisulpride and paliperidone
- Accumulate in renal impairment
- Require renal-specific review

Lithium
- Requires particular caution
- Kidney function, fluid/sodium balance and interacting medicines strongly affect concentrations
- Advanced kidney disease or dialysis use is a specialist decision with a specific monitoring plan, not routine prescribing

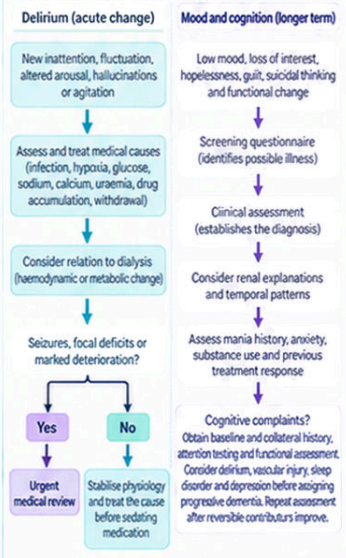
General prescribing principles
- Many agents need slower titration or adjusted exposure.
- Use the actual product guidance and renal measure it specifies.
- Do not assume haemodialysis removes every psychotropic or automatically prescribe a replacement dose after each session.
- Dialysability depends on the molecule, binding, distribution and dialysis technique.

TREATMENT MUST FIT THE DIALYSIS DAY



- Provide supportive care, problem solving and cognitive behavioural therapy.
- with appointments coordinated around fatigue and dialysis schedules.
- Address pain, itching, restless legs and sleep problems with the renal team.
- Review practical barriers such as transport, costs, health literacy and medication complexity.
- Family involvement requires agreement, not automatic disclosure.
- Set functional goals that matter to the patient and monitor them.
- Antidepressants can be considered for a diagnosed disorder, but do not promise a renal-outcome benefit.
- The placebo-controlled sertraline trial in nondialysis chronic kidney disease did not demonstrate superior depressive-symptom improvement. This finding supports careful individual trials and review, not withholding all depression care or assuming identical results in every dialysis population.

SYMPTOM PATHWAYS



REFUSAL, SAFETY AND MONITORING

Refusal and decision-specific capacity



A decision to decline dialysis is not by itself evidence of incapacity. Distinguish informed treatment preference from suicidal intent; both deserve direct assessment.

Monitoring

Monitor symptoms, function, sedation, falls, movement effects, blood pressure and adherence.

Review renal trends, electrolytes and electrocardiography, and drug concentrations where relevant, especially after acute illness or medication changes.

Name follow-up responsibility across psychiatry and nephrology.

MEMORY AID
Check the cause, check clearance, fit care to life.

Sources: Indian Psychiatric Society renal guidance 2022; sertraline kidney-disease trial; duloxetine prescribing information; Specialist Pharmacy Service lithium monitoring; National Institute for Health and Care Excellence capacity guidance. Original Paper IV Question 28, page 43 plus verified expansion. September 2026.

Kidney disease and dialysis psychiatry. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-28.

Credits and references: retained within the illustration.

IV - 29

Discuss psychological intervention in bronchial asthma. [10]

10 CONSULTATION-LIAISON AND SYSTEMIC MEDICINE PSYCHIATRY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	How the two interact	Perception, adherence, and the panic overlap
2	Assess before intervening	Objective control set against reported symptoms
4	The interventions	The flowchart: three mismatches, three answers
2	The drugs, both ways	What the asthma drugs do, and what to avoid

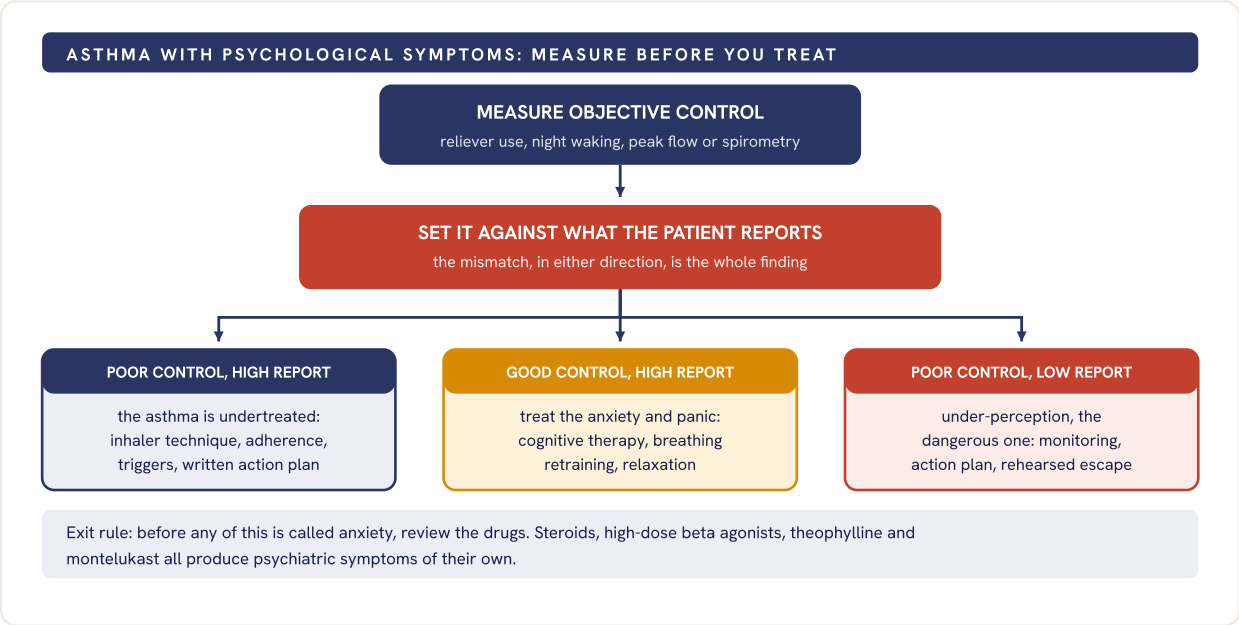


Fig 029.1 The intervention is chosen by the direction of the mismatch between measured control and reported symptoms, which is why nothing is prescribed before the measurement. Indigo optimises the asthma, amber treats the anxiety, red is danger.

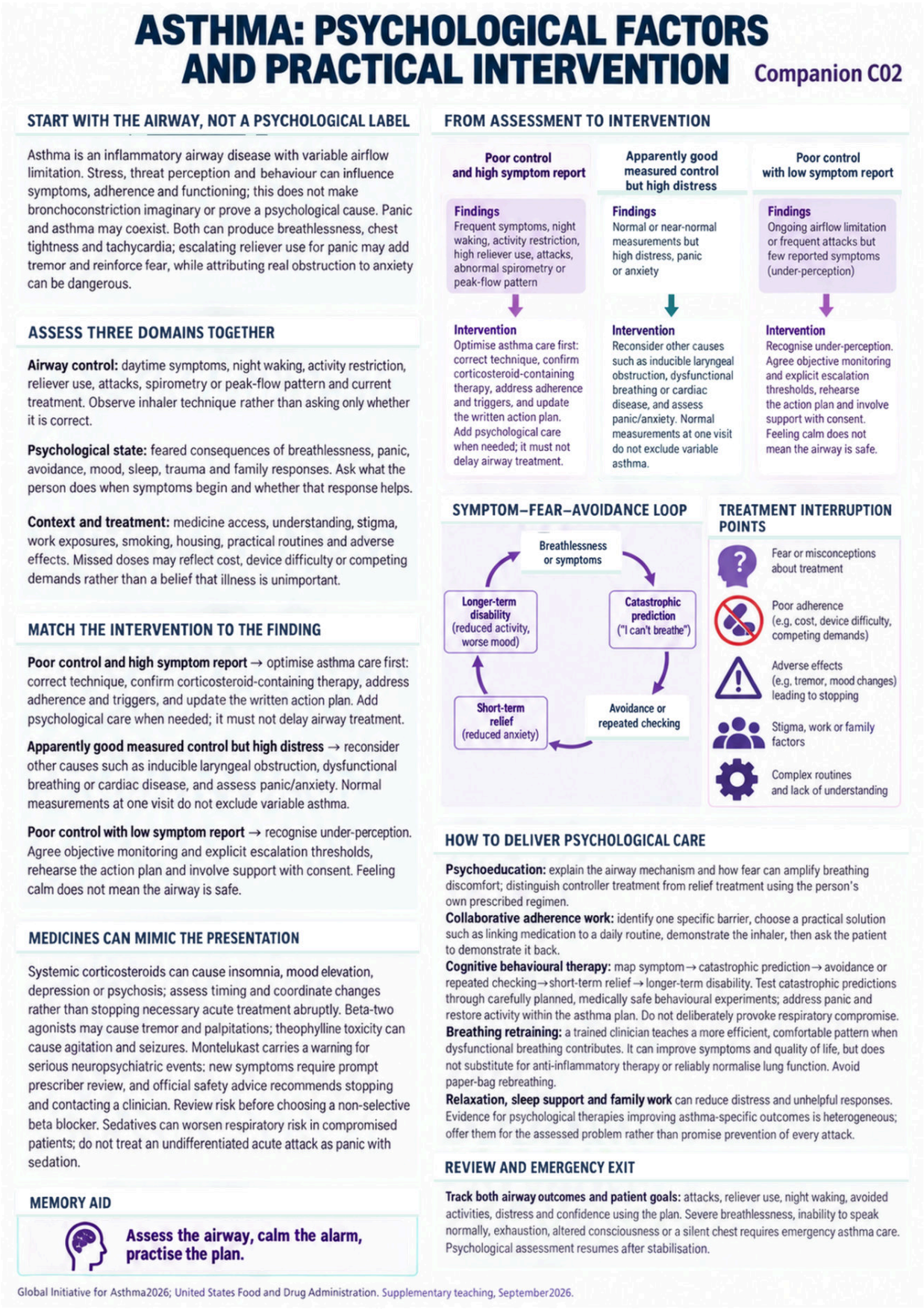
- **What the evidence supports.** Self-management education with a written action plan and regular review is the best-evidenced behavioural intervention in asthma. Breathing retraining improves symptoms and quality of life without changing airway measures. Cognitive therapy helps comorbid anxiety and depression; its effect on asthma outcome is inconsistent. Family work matters in children.
- **The overlap, and the prescribing.** Panic and asthma share breathlessness, chest tightness and tachycardia, and each is mistaken for the other; a reliever taken for panic adds tremor and closes the loop. Serotonin reuptake inhibitors are usually safe; use benzodiazepines briefly if at all where there is respiratory compromise.

WHAT EARNS THE MARKS

- **Objective control measured before anything is called psychological.**
- **Under-perception named as the dangerous mismatch,** which almost no answer includes.
- **Breathing retraining described accurately:** symptoms and quality of life, not lung function.
- **The asthma drugs reviewed as a psychiatric cause,** before a psychiatric diagnosis is made.

SEE ALSO Slot IV - 27 depression with ischaemic heart disease Slot IV - 22 consultation liaison psychiatry **SPRINT** Day 40, Medical and systemic-disease psychiatry

WEAVE 100 · PAPER IV · C02



Asthma psychological and psychosomatic care. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-29.

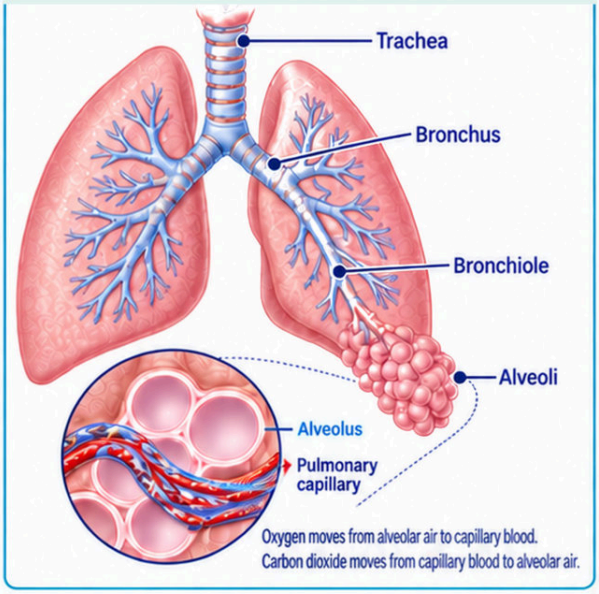
Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · F12

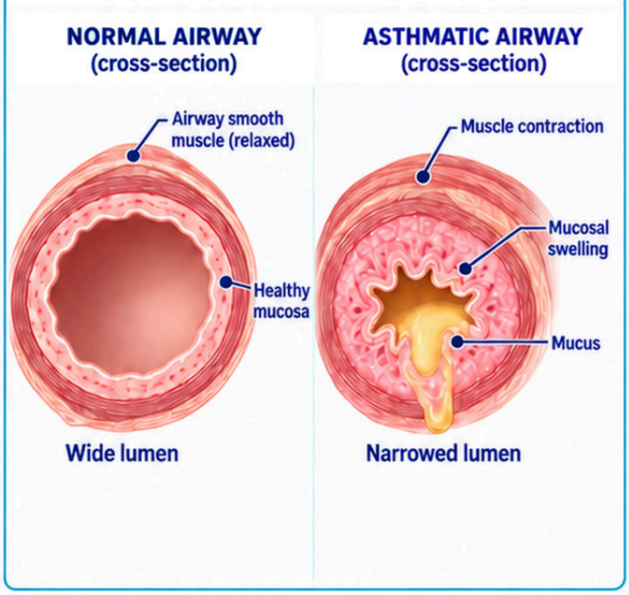
AIRWAY PHYSIOLOGY AND BRONCHIAL ASTHMA

Foundation 12

THE RESPIRATORY AIRWAYS



NORMAL VERSUS ASTHMATIC AIRWAY



NORMAL AIRFLOW

Air passes through the trachea and branching bronchi to alveoli, where oxygen enters pulmonary capillary blood and carbon dioxide leaves it. Cilia move mucus and trapped particles towards the throat. Airway smooth muscle adjusts calibre; the lining acts as a barrier. During quiet expiration, elastic recoil drives air out. Narrower airways greatly increase resistance, especially during expiration. Ventilation is movement of air; oxygenation is transfer of oxygen into blood. These are related but different functions.

WHAT ASTHMA MEANS

Asthma is a heterogeneous disease with chronic airway inflammation, variable respiratory symptoms and variable expiratory airflow limitation. Hyperresponsiveness means excessive airway narrowing to triggers. Allergen exposure, viral infection, smoke, exercise or occupational agents may activate inflammation and smooth-muscle contraction. Swollen mucosa, excess mucus and bronchoconstriction reduce the airway lumen. Type-two inflammation involving eosinophils and related immune pathways is common but does not explain every phenotype. Long-standing disease may cause airway remodelling and persistent limitation. Asthma is not simply anxiety, and normal examination between attacks does not exclude it.

MECHANISM → SYMPTOM

Narrowed airways → wheeze, breathlessness and chest tightness. Airway irritation → cough, sometimes the main symptom. Symptoms often vary over time and worsen at night or early morning. Expiratory obstruction → air trapping and increased breathing effort. Severe obstruction may produce little audible wheeze because airflow is critically low: a silent chest is dangerous. Exhaustion, inability to speak normally, confusion, cyanosis or falling oxygen saturation require urgent emergency treatment. A normal or rising carbon dioxide level during a severe attack can signal failing ventilation rather than recovery.

CONFIRM AND CHARACTERISE

Measure spirometry when possible before long-term treatment. Forced expiratory volume in one second measures the volume blown out in the first second; forced vital capacity is the full forced exhaled volume. Their ratio detects obstruction using age-appropriate lower limits rather than one universal cutoff. In adults, an increase in forced expiratory volume in one second of at least 12% and 200 millilitres after bronchodilator supports variable airflow limitation. Repeat testing during symptoms if initially normal. Peak expiratory flow diaries or challenge tests may help. Normal adult resting respiratory rate is commonly 12–20 breaths/minute; saturation is usually 95–100% at sea level, modified by altitude and disease. Blood eosinophils or exhaled nitric oxide may support inflammatory phenotyping; neither establishes asthma alone. Consider chronic obstructive pulmonary disease, heart failure, inducible laryngeal obstruction and panic as alternatives or comorbidities.

CONTROL AND PREVENT ATTACKS

Confirm diagnosis → assess symptom control and future attack risk → check inhaler technique, adherence, exposures and comorbidities → select inhaled corticosteroid-containing treatment. Adults and adolescents should not receive a short-acting bronchodilator alone as the whole treatment strategy. In this preferred pathway, low-dose inhaled corticosteroid–formoterol is the reliever, at higher steps it also provides scheduled maintenance treatment. Only suitable formoterol combinations are used in this way. Alternative regimens require reliable corticosteroid use. Provide a written action plan and demonstrate the device. Step up only after correcting technique and adherence: step down cautiously after sustained control.

Attack → rapid assessment, repeated inhaled bronchodilator, controlled oxygen and systemic corticosteroid when indicated; severe attacks need emergency transfer and additional treatment. Long-term oral corticosteroids are a last resort because cumulative harm is substantial. Persistent difficult disease → specialist reassessment, phenotype testing and possible biological treatment. Track exacerbations, night waking, activity limits and lung function; review after attacks. Breathing exercises can support symptoms but do not replace anti-inflammatory treatment.

Variable airflow, inflamed airways: confirm, control, prevent.

Sources: Global Initiative for Asthma; National Institute for Health and Care Excellence.

Airway physiology and bronchial asthma. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-29.

Credits and references: retained within the illustration.

IV - 30

What are the clinical features of irritable bowel syndrome? Discuss its pharmacological and non-pharmacological management. [4+6]

4 + 6 | CONSULTATION-LIAISON AND SYSTEMIC MEDICINE PSYCHIATRY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4	Clinical features	The criteria, the subtypes, and the alarm features
3	Non-pharmacological	A confident diagnosis, diet, and the therapies
3	Pharmacological	The flowchart: by subtype, then the neuromodulator

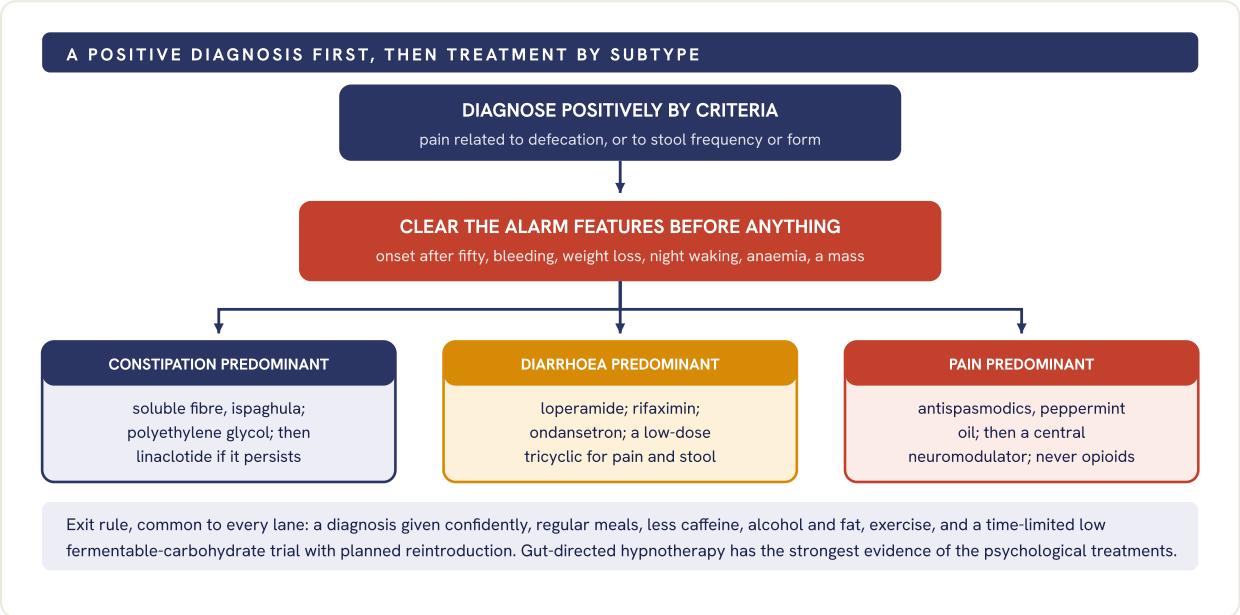


Fig 030.1 The alarm features sit between the diagnosis and every treatment lane, because they are the one thing that removes this diagnosis rather than modifying it. Indigo is structure, amber the fullest lane, red exclusion and pain.

- **The clinical features.** Recurrent abdominal pain, on average at least one day a week over three months, related to defecation or to a change in stool frequency or form, with onset at least six months earlier. Bloating, urgency, mucus and a sense of incomplete evacuation travel with it, symptoms worsen with meals and with stress, and examination is normal. Subtypes follow the predominant stool form: constipation, diarrhoea, mixed, or unclassified.
- **The mechanism, and the sentence patients need.** A disorder of gut-brain interaction: visceral hypersensitivity, altered motility and altered central pain processing, with anxiety, depression and early adversity over-represented. Say that a tricyclic is being used at a low dose for the gut and not as an antidepressant, or the patient reads the prescription as a verdict and stops it.

WHAT EARNS THE MARKS

- **The criteria given with their time frame,** not a vague description of a sensitive bowel.
- **The alarm features listed,** since they are what makes the positive diagnosis safe.
- **Treatment organised by subtype,** rather than one list of every drug ever used.
- **The neuromodulator explained to the patient,** which is the psychiatrist's specific contribution.

SEE ALSO Slot IV - 23 fibromyalgia Slot IV - 22 consultation liaison psychiatry **SPRINT**

Day 40, Medical and systemic-disease psychiatry

WEAVE 100 · PAPER IV · C03

IRRITABLE BOWEL SYNDROME: ANXIETY, GUT–BRAIN CARE AND MEDICINES

Companion C03

A POSITIVE GUT–BRAIN DIAGNOSIS

Irritable bowel syndrome involves recurrent abdominal pain with altered bowel habits, without another explanation that better accounts for the pattern. Altered motility, visceral sensitivity and gut–brain signalling contribute, mechanisms differ between patients. Anxiety can amplify vigilance, threat interpretation and avoidance, while unpredictable pain or urgency creates anxiety. This is a two-way relationship, not evidence that distress caused the disease. Normal routine tests do not mean invented symptoms, and symptom severity alone does not diagnose somatic symptom disorder.

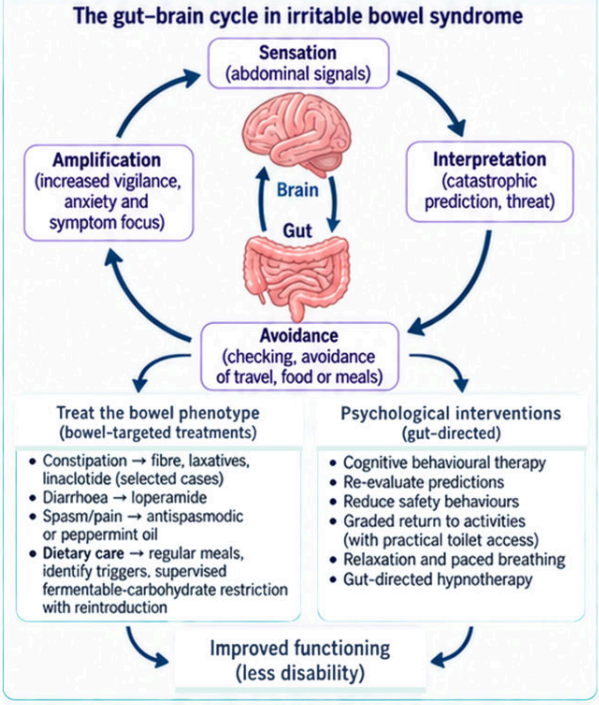
ASSESS WHAT MAINTAINS DISABILITY

Confirm symptom pattern and predominant stool type, previous investigations, warning features and treatment response. Ask about pain, urgency, fear of accidents, food restriction, toilet checking, avoidance of travel or meals and consequences at work. Identify panic, depression, trauma-related symptoms and health anxiety where present. Examine medicine effects and dietary practices; repeated antibiotics, unnecessary exclusion diets or escalating laxatives may worsen problems. Bleeding, anaemia, weight loss, a mass, persistent nocturnal diarrhoea or new concerning change requires medical reassessment, not more reassurance alone.

EXPLAIN AND AGREE A PLAN

Offer a clear formulation: "Your bowel is more sensitive and its signalling and movement are altered; we can treat the bowel pattern and the alarm response together." Explain which findings support the diagnosis and why further tests are or are not needed. Agree measurable goals such as commuting, eating a wider range of foods or fewer days disrupted by pain. Plan follow-up; repeated unstructured reassurance can unintentionally maintain checking. Treat established anxiety or depression as a comorbidity with its own goals.

GUT–BRAIN LOOP AND TREATMENT APPROACH



PSYCHOLOGICAL TREATMENT: HOW IT WORKS

Gut-directed cognitive behavioural therapy maps sensation→ catastrophic prediction→checking/avoidance→increased vigilance. Use symptom and behaviour records to identify the cycle without turning monitoring into constant checking. Re-evaluate predictions, reduce excessive safety behaviours and plan graded return to avoided situations with practical access to toilets. Exposure is collaborative and does not mean ignoring genuine diarrhoea or medical warning signs. Relaxation and paced breathing can reduce autonomic arousal. Gut-directed hypnotherapy uses structured suggestions and imagery to modify perceived gut sensations and regulation, delivered by a trained practitioner. These approaches can improve global symptoms in selected patients; they are not restricted to people with a psychiatric diagnosis.

TREAT THE BOWEL PHENOTYPE

- **Constipation** → soluble fibre and an appropriate osmotic laxative; selected persistent cases may benefit from linaclotide, watching for diarrhoea.
- **Diarrhoea** → loperamide can improve stool consistency and urgency but often leaves pain unchanged; consider alternative causes when response is poor.
- **Spasm/pain** → a selected antispasmodic or peppermint oil, considering anticholinergic effects or reflux respectively.
- **Dietary care** starts with regular meals and individual triggers; a supervised fermentable-carbohydrate restriction trial should be followed by reintroduction, not indefinite broad avoidance.

WHEN A NEUROMODULATOR HELPS

Persistent pain/global symptoms despite first measures → discuss low-dose amitriptyline as a gut–brain pain treatment, often below antidepressant doses. Explain that the indication is not proof of depression. Start cautiously, titrate to benefit and tolerability, and review early for dry mouth, sedation, constipation, urinary difficulty, orthostatic symptoms and cardiac or overdose risk. Its tendency to slow transit can matter when constipation predominates. Randomised trial evidence supports second-line low-dose use, but not every patient benefits.

Selective serotonin reuptake inhibitors may be chosen for a clear comorbid anxiety/depressive disorder; evidence for core bowel symptoms is less consistent than for tricyclic agents. They can cause nausea or diarrhoea. Use an adequate psychiatric regimen when treating a psychiatric disorder rather than assuming a low pain dose covers both. Check interactions and bipolar-spectrum history where relevant.

Amitriptyline: two different indications

Feature	Gut–brain pain treatment (in irritable bowel syndrome)	Antidepressant treatment (for depressive disorder)
Indication	Persistent pain/global symptoms in irritable bowel syndrome	Depressive disorder
Typical dose range	Low dose for pain, individually titrated	Adequate antidepressant regimen, individually titrated
Purpose	Modulate gut–brain pain signalling	Treat mood disorder
Dose and review	Start low, titrate to benefit and tolerability	Use standard antidepressant regimen, titrate
Key adverse effects to review	Dry mouth, sedation, constipation, urinary difficulty, orthostatic symptoms, cardiac or overdose risk	As for pain indication (plus other antidepressant-related effects)
Note	Indication is not proof of depression	Not used to treat bowel symptoms alone

REVIEW BOTH OUTCOMES

Track pain, stool pattern, functioning and mood/anxiety separately. Continue useful treatment, adjust adverse effects, and taper ineffective medicines appropriately. Re-open the medical differential if the pattern changes. Avoid opioids for chronic functional abdominal pain because dependence, constipation and pain amplification can worsen disability.

MEMORY AID

Validate symptoms, explain the gut–brain link, target the maintaining loop.

Sources: National Institute for Health and Care Excellence; National Institute of Diabetes and Digestive and Kidney Diseases; ATLANTIS trial (Amitriptyline at Low-Dose and Titrated for Irritable Bowel Syndrome as Second-Line Treatment). Supplementary teaching, September 2026.

Irritable bowel syndrome anxiety and pharmacology. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-30.

Credits and references: retained within the illustration.

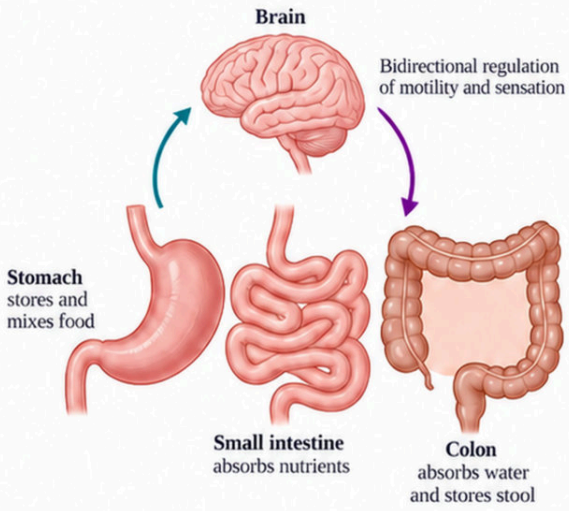
WEAVE 100 · PAPER IV · F13

GUT PHYSIOLOGY AND IRRITABLE BOWEL SYNDROME

Foundation 13

NORMAL DIGESTION AND SENSATION

The stomach stores and mixes food; the small intestine digests nutrients and absorbs most of them. The colon absorbs water and electrolytes and stores stool. Coordinated muscle contractions propel contents. The enteric nervous system organises local motility and secretion, while autonomic nerves and the brain modulate these functions. Sensory nerves report stretching and chemical signals. The epithelial barrier and immune system interact with the intestinal microbiota. Gut–brain signalling is bidirectional: symptoms are biologically real even when routine structural tests are normal.



WHAT GOES WRONG

Irritable bowel syndrome is a disorder of gut–brain interaction characterised by recurrent abdominal pain or discomfort with altered bowel habits; the chosen diagnostic framework determines the formal symptom threshold. Mechanisms vary: visceral hypersensitivity amplifies pain from ordinary distension; altered motility changes transit; microbial, immune and barrier changes may contribute in some people. Symptoms can begin after gastroenteritis. Stress can modify perception and motility but is neither required nor proof of a psychological cause. It is distinct from inflammatory bowel disease, which involves demonstrable intestinal inflammation. The syndrome does not itself cause intestinal cancer or destructive bowel inflammation, but other diseases can coexist.

RECOGNISE AND DEFINE

Pain related to defecation may improve or worsen after passing stool. Bloating, altered stool frequency or form, urgency, straining, mucus and incomplete evacuation are common. Older exam framework — Rome IV (2016): abdominal pain averaging at least one day weekly during the last three months, associated with at least two of the following: relation to defecation, change in stool frequency, change in stool form. Symptoms began at least six months before diagnosis. Current update — Rome V (2026): the Rome Foundation reintroduced discomfort alongside pain and lowered the published symptom-frequency threshold to three days per month. Rome V distinguishes research criteria from clinical criteria, which allow contextual clinical judgement. Do not silently combine the old weekly pain rule with the new framework or assume all old duration requirements carry over; use the full relevant current criteria when diagnosing. Constipation-predominant, diarrhoea-predominant, mixed and unclassified subtypes are based on abnormal stool form. The Bristol stool scale runs from type 1, hard separate lumps, to type 7, watery stool; types 3–4 are commonly well-formed. Stool frequency varies: roughly three daily to three weekly can be normal when comfortable and stable.

POSITIVE DIAGNOSIS, TARGETED EXCLUSION

History, examination and a consistent symptom pattern guide diagnosis. Check full blood count, inflammatory markers and coeliac serology when appropriate. Faecal calprotectin can help distinguish inflammatory disease in selected patients with diarrhoea; interpret using the laboratory assay and clinical context. There is no diagnostic blood biomarker for irritable bowel syndrome. Bleeding, iron-deficiency anaemia, unexplained weight loss, a mass, persistent nocturnal diarrhoea, fever or relevant family history require further assessment. New symptoms in an older person deserve reconsideration. Investigate suspected cancer, inflammatory bowel disease, coeliac disease, infection or bile-acid diarrhoea rather than assuming the label explains everything. Colonoscopy is not automatically necessary for every typical presentation without warning features.

MANAGEMENT BY DOMINANT PROBLEM

Explain the positive diagnosis → agree symptom and function goals → regular meals, hydration, activity and sleep → review individual dietary triggers. Soluble fibre such as ispaghula is preferable to routinely increasing coarse insoluble bran. Persistent symptoms may justify a dietitian-supervised trial reducing fermentable carbohydrates, followed by reintroduction and personalisation; avoid unnecessary permanent restriction. Constipation → soluble fibre, then an osmotic laxative; persistent selected cases may need a secretagogue such as linaclotide. Diarrhoea → loperamide helps stool frequency and consistency but may not relieve pain; investigate alternative causes when treatment fails. Pain or spasm → selected antispasmodic or peppermint oil, considering adverse effects. Persistent global symptoms → consider low-dose tricyclic neuromodulation or gut-directed psychological treatment after shared discussion. These treatments target pain signalling and coping, not an assumption that symptoms are imagined. Cognitive behavioural therapy and gut-directed hypnotherapy can help selected patients. Reassess benefit and adverse effects, subtype changes, nutritional restriction and any emerging alarm signs; do not endlessly repeat low-yield investigations.

Pattern, warning signs, dominant problem: guide the plan.

Sources: Rome Foundation 2026; National Institute for Health and Care Excellence; National Institute of Diabetes and Digestive and Kidney Diseases.

Gut physiology and irritable bowel syndrome. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-30.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · 13.2

MYALGIC ENCEPHALOMYELITIS / CHRONIC FATIGUE SYNDROME

Recognise post-exertional malaise • distinguish diagnostic frameworks • protect energy limits

Paper IV • Supplement to rebuilt Question 13 • Sheet 13.2

WHAT THE TERMS MEAN

A disabling multisystem illness with reduced activity tolerance, post-exertional malaise and disturbed sleep; ordinary persistent tiredness alone is insufficient.

Fatigue: loss of physical or mental energy, not simply sleepiness.

Post-exertional malaise: disproportionate worsening of multiple symptoms after physical, cognitive, emotional or sensory effort previously tolerated.

Unrefreshing sleep: sleep does not restore wellbeing.

Cognitive impairment: problems with concentration, memory or processing.

Orthostatic intolerance: symptoms worsen upright and improve on lying down; assess pulse and blood pressure, not symptoms alone.

POST-EXERTIONAL MALAISE: ASK ABOUT THE NEXT TWO DAYS

Activity or sensory load → often delayed 12–48 hours → worse fatigue, cognition, pain, sleep or flu-like symptoms → recovery may take days, weeks or longer.



Timing varies; an apparently successful activity can still trigger delayed deterioration.

Mechanisms remain uncertain. Immune, autonomic, metabolic and nervous-system abnormalities are under study; no single causal pathway or diagnostic biomarker is established. Do not equate the illness with proven mitochondrial failure, deconditioning or depression.

TWO FRAMEWORKS — DO NOT MERGE THEIR RULES

INSTITUTE OF MEDICINE, 2015

All THREE:

1. Substantial reduction in previous occupational, educational, social or personal activity for **MORE THAN 6 months**, with new, often profound fatigue; not lifelong, not due to excessive ongoing exertion and not substantially relieved by rest.
2. Post-exertional malaise.
3. Unrefreshing sleep.

PLUS at least ONE:

4. Cognitive impairment **OR** orthostatic intolerance.

Evaluate frequency/severity: core symptoms should occur at least half the time, with at least moderate intensity.

Clinical assessment excludes another explanation; comorbid conditions may coexist.

NATIONAL INSTITUTE FOR HEALTH AND CARE EXCELLENCE, 2021

All FOUR:

1. Debilitating fatigue worsened by activity, not due to excessive exertion, not significantly relieved by rest.
2. Post-exertional malaise: often delayed, disproportionate, with prolonged recovery.
3. Unrefreshing sleep **OR** sleep disturbance.
4. Cognitive difficulty.

Also require significant reduction from pre-illness activity and no better explanation.

Suspect after 6 weeks in adults or 4 weeks in children/young people.

Diagnose after symptoms persist for 3 months; specialist confirmation and care planning.

Cognitive difficulty is **REQUIRED**; orthostatic intolerance is associated, not an alternative criterion.

EVALUATE WITHOUT DISMISSING OR OVERTESTING

- **Establish** onset, preceding infection, delayed exertional response, sleep, medicines, substance use, nutrition, mental health and functional loss.
- Examine generally and neurologically; assess orthostatic vital signs when indicated.
- **Initial tests:** full blood count, kidney function/electrolytes, liver and thyroid function, inflammatory markers, calcium/phosphate, glycated haemoglobin, ferritin, coeliac screen, creatine kinase and urinalysis.
- **Select further** tests from findings: vitamin deficiencies, infection studies or morning cortisol; no indiscriminate immune panels.
- **Mimics to identify:** anaemia, hypothyroidism, adrenal insufficiency, sleep apnoea, inflammatory/neurological disease, infection, medication effects and major depression.
- **Comorbidities to treat:** fibromyalgia, migraine, irritable bowel syndrome, orthostatic disorders, sleep disorders, anxiety or depression.
- **Pain** may be prominent and fibromyalgia can coexist. Neither normal tests nor unexplained symptoms establish somatic symptom disorder; disproportionate symptom-related thoughts, feelings or behaviour must be assessed separately.
- **New** focal signs, weight loss, fever, progressive weakness or other red flags → reassess urgently; do not attribute everything to the established diagnosis.

MANAGEMENT FLOW — INDIVIDUALISE, STABILISE, REVIEW

Confirm/support diagnosis → **Agree priorities and practical support** → **Establish a sustainable energy baseline**

ENERGY MANAGEMENT

- Count physical, cognitive, social and emotional activity. Plan rest, break tasks into short portions, alternate demands, use aids and a symptom/activity diary.
- **Stable within limits?** → maintain; any chosen change must be flexible and symptom-contingent.
- **Flare or relapse?** → reduce demands to current limits, reassess causes, stabilise, then establish a new baseline.
- **Do NOT prescribe** fixed-increment graded exercise therapy or exercise as a cure.
- If the person chooses an activity programme, use a specialist-trained physiotherapist, begin below a symptom-worsening baseline and adjust up OR down.

SYMPTOM-TARGETED CARE

- **Sleep:** personalise rest/sleep routine and assess a primary sleep disorder.
- **Pain:** identify its type; cautious individual treatment and side-effect review.
- **Orthostatic symptoms:** assess causes; consider hydration, salt and compression only when appropriate to heart/kidney status; specialist treatment if needed.
- **Nutrition:** maintain intake; support severe illness, swallowing difficulty and malnutrition.

PSYCHOLOGICAL SUPPORT

- **Cognitive behavioural therapy:** optional support for coping and distress, not a cure and not based on presumed abnormal illness beliefs.

GENERAL PRINCIPLES

- **Medicines** do not cure this syndrome. Review medication sensitivity; start cautiously.
- Do not routinely prescribe stimulants, steroids, antivirals or immune therapies as disease treatment.
- Arrange accessible follow-up, disability/work or education adjustments and caregiver support.
- Severe illness may need home-based care and reduced sensory load.

Sources: Institute of Medicine, 2015, as presented by Centers for Disease Control and Prevention; National Institute for Health and Care Excellence guideline 206, 2021; American Psychiatric Association.

Modern synthesis corrects older local notes • No established cure • 21 September 2026 • v1

Chronic fatigue syndrome. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Consultation-liaison and systemic medicine psychiatry.

Credits and references: retained within the illustration.

FIBROMYALGIA AND CHRONIC FATIGUE SYNDROME

Shared symptoms • different diagnostic anchors • different activity decisions

Paper IV • Supplement to rebuilt Question 13 • Sheet 13.3



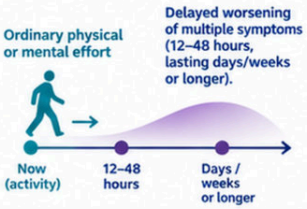
Heightened pain signalling in the nervous system (amplification, not tissue damage).

FIBROMYALGIA

Dominant anchor: widespread pain and symptom burden.

MYALGIC ENCEPHALOMYELITIS / CHRONIC FATIGUE SYNDROME

Dominant anchor: post-exertional malaise with reduced activity tolerance.



COMPARE THE PATTERN		BOTH CAN HAVE (OVERLAP)	COMPARE THE PATTERN
PAIN	Widespread musculoskeletal pain and tenderness; nociplastic pain is common.	Pain may occur in either.	Pain may occur, but widespread pain is not essential.
EXERTION	Activity can transiently aggravate pain; individually adjusted exercise can improve function.	Activity can worsen symptoms in either.	Disproportionate multisystem worsening, often delayed 12–48 hours, lasting days/weeks or longer; ask beyond immediate tolerance.
SLEEP & COGNITION	Unrefreshing sleep, fatigue and difficulty concentrating or remembering.	Unrefreshing sleep, fatigue and cognitive symptoms.	These contribute to diagnostic criteria; orthostatic intolerance may also be prominent.
MECHANISM	Altered nociception and central pain amplification are important; not necessarily peripheral tissue injury.	Mechanisms remain incompletely understood.	Mechanisms remain uncertain; immune, autonomic, metabolic and neural findings do not establish one proven cause.

Neither diagnosis means symptoms are imagined or simply depression.

DIAGNOSTIC ANCHORS — APPLY THE COMPLETE FRAMEWORK

FIBROMYALGIA: WOLFE AND COLLEAGUES, 2016

- Widespread Pain Index ≥7 with Symptom Severity Scale ≥5 **OR** Widespread Pain Index 4–6 with Symptom Severity Scale ≥9.
- **PLUS** pain in at least 4 of 5 regions and symptoms for at least 3 months.
- Jaw/chest/abdomen count in the index, not the region criterion.
- Other diagnoses may coexist; tender-point counting is not required.
- Full 19-site index and 0–12 symptom scoring: companion fibromyalgia sheet 13.1.

DIAGNOSTIC ANCHORS — KEEP TWO FRAMEWORKS SEPARATE

CHRONIC FATIGUE SYNDROME

- **Institute of Medicine 2015:** substantial activity reduction with new fatigue for **MORE THAN 6 months;** post-exertional malaise; unrefreshing sleep; **PLUS** cognitive impairment **OR** orthostatic intolerance. Core symptoms at least half the time, at least moderate.
- **National Institute for Health and Care Excellence 2021:** debilitating fatigue, post-exertional malaise, unrefreshing/disturbed sleep **AND** cognitive difficulty, significant functional reduction, no better explanation; diagnose at 3 months.
- Orthostatic intolerance does not substitute for cognitive difficulty in the 2021 framework.
- Full definitions/evaluation: companion chronic fatigue syndrome sheet 13.2.

WHAT CHANGES MANAGEMENT?

ACTIVITY	Explain benefits; choose aerobic, resistance or water-based activity, start gently and pace tasks. Adjust intensity/duration to tolerance; sustained flare → reduce and reassess.
MEDICINES	Selected symptom-targeted trials of duloxetine, milnacipran, pregabalin or low-dose amitriptyline; modest benefit, adverse effects and interactions matter. Gabapentin evidence is insufficient.
PSYCHOLOGICAL CARE	Cognitive behavioural therapy may help pain coping and function; treat coexisting mood/anxiety disorders.
FOLLOW-UP	Agree patient-valued functional goals; review sleep, daily function, adverse effects, access and support. Reassess new symptoms and red flags.

WHAT CHANGES MANAGEMENT?

ACTIVITY	Manage ALL activity within current energy limits, with planned rest. No fixed-increment graded exercise therapy; no instruction to push through symptoms. Optional specialist activity plan adjusts up OR down.
MEDICINES	No medicine is established as a cure. Treat identified pain, sleep, orthostatic and other comorbid problems cautiously; avoid unsupported disease-modifying claims.
PSYCHOLOGICAL CARE	Cognitive behavioural therapy is optional coping support, not a cure or correction of presumed false illness beliefs.
FOLLOW-UP	Agree patient-valued functional goals; review sleep, daily function, adverse effects, access and support. Reassess new symptoms and red flags.

OVERLAP IS POSSIBLE – DO NOT FORCE A SINGLE LABEL

- Shared fatigue, pain, sleep disturbance, cognitive symptoms and bowel/mood comorbidity are not enough to distinguish them.
- Both criteria fulfilled? → diagnose and manage both → post-exertional malaise sets the activity safety limit.
- Relevant medical mimics include anaemia, thyroid disease, inflammatory disease, sleep disorders and medicine effects.
- Neuropathic pain requires a somatosensory lesion/disease with a plausible distribution; it can coexist.
- Somatic symptom disorder requires disproportionate symptom-related thoughts, feelings or behaviour. Normal tests, severe symptoms or either physical diagnosis alone do not establish it.

PRACTICAL DISCRIMINATOR

Widespread pain?	Delayed multisystem deterioration after ordinary effort?	Both?
↓	↓	↓
Score fibromyalgia criteria.	Assess chronic fatigue syndrome criteria.	Combine symptom treatment with an energy-limited activity plan.

SOURCES

- Wolfe et al. 2016
- European League Against Rheumatism 2017
- Institute of Medicine 2015
- National Institute for Health and Care Excellence 2021
- Centers for Disease Control and Prevention
- American Psychiatric Association

Clinical synthesis • 21 September 2026 • v1

Fibromyalgia versus chronic fatigue syndrome. Read across the labelled contrasts to distinguish the related concepts.
Section companion: Consultation-liaison and systemic medicine psychiatry.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · F16

TRANSPLANTATION, GRAFT REJECTION AND IMMUNOSUPPRESSIION

Foundation 16 • Normal function → mechanism → clinical recognition → management

NORMAL IMMUNE RECOGNITION

The immune system distinguishes threats through coordinated innate and adaptive responses. Antigen-presenting cells display peptides to T lymphocytes using human leucocyte antigen molecules. Activated helper T lymphocytes support other immune cells; cytotoxic T lymphocytes can destroy target cells. B lymphocytes produce antibodies. These responses protect against infection but can recognise a transplanted organ as foreign. A graft is transferred tissue or an organ; an allograft comes from a genetically different person of the same species. Immunosuppression reduces immune activity enough to protect the graft while trying to preserve defence against infection and cancer.

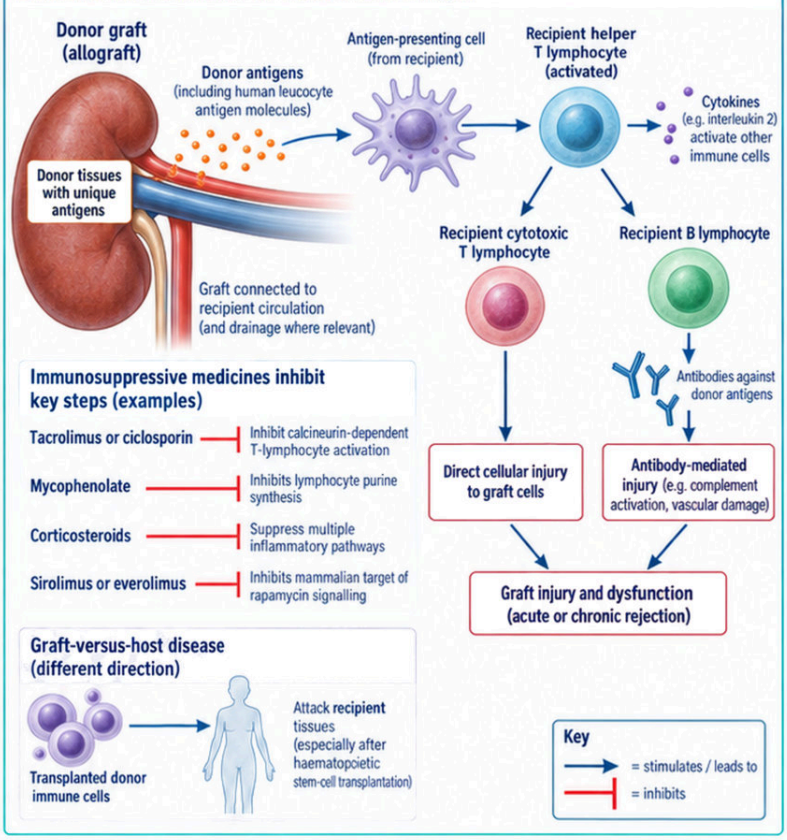
WHY REJECTION OCCURS

Donor antigens activate recipient T lymphocytes and antibodies. Hyperacute rejection is rapid vascular injury from pre-existing antibodies, now uncommon with appropriate compatibility testing. Acute rejection can be T-lymphocyte-mediated or antibody-mediated and may occur early or later, especially with inadequate immunosuppression. Chronic graft dysfunction involves cumulative immune and non-immune injury, including vascular disease, fibrosis, recurrent original disease and medicine toxicity. Timing alone does not identify the mechanism. Rejection differs from graft-versus-host disease: in the latter, transplanted donor immune cells attack recipient tissues, especially after haematopoietic stem-cell transplantation.

MATCHING AND PREPARATION

Assess organ-specific disease severity and alternatives; optimise infection, cardiovascular risk and nutrition. Blood-group compatibility, human leucocyte antigen typing, donor-specific antibody testing and crossmatching reduce immunological risk. A positive crossmatch may indicate antibodies capable of attacking donor cells; specialised protocols exist for selected incompatible transplants. Donor and recipient are independently assessed. Consent, voluntariness and lawful donation are essential; medical suitability does not itself establish consent. The operation connects the graft to recipient circulation and, where relevant, drainage pathways. Postoperative dysfunction can reflect vascular thrombosis, obstruction, ischaemic injury, infection or rejection: do not assume every abnormal test is rejection.

IMMUNE MECHANISMS IN TRANSPLANTATION



THE MEDICINE BALANCE

Induction treatment provides stronger immune suppression around transplantation; maintenance usually combines agents with complementary mechanisms. **Tacrolimus or ciclosporin** inhibit calcineurin-dependent T-lymphocyte activation; monitor concentrations, kidney function, potassium, blood pressure and neurological toxicity. **Mycophenolate** inhibits lymphocyte purine synthesis; monitor blood counts and gastrointestinal toxicity and address its major pregnancy risks. **Corticosteroids** suppress multiple inflammatory pathways; monitor infection, glucose, blood pressure and bone health. **Sirolimus or everolimus** inhibit mammalian target of rapamycin signalling; selected regimens require monitoring for dyslipidaemia, cytopenias, proteinuria and impaired wound healing. Regimens and targets depend on organ, time since transplant, immunological risk and toxicity. There is no universal normal tacrolimus concentration applicable to every recipient.

RECOGNISE AND INVESTIGATE DYSFUNCTION

Kidney graft: rising creatinine, reduced urine or proteinuria. **Liver graft**: changing bilirubin, enzymes or coagulation. **Heart graft**: reduced pumping, rhythm disturbance or congestion. Fever or malaise may reflect infection; rejection can be initially asymptomatic and found on surveillance. Assess adherence, new medicines, vomiting and diarrhoea; measure relevant drug concentrations and organ function, use imaging, and consider biopsy. A concentration in target range does not alone exclude rejection. Compare results with the recipient's baseline, not merely population reference limits.

LIFELONG CARE FLOW

Stable graft → take medicines consistently → scheduled organ and drug monitoring → infection prevention, vaccination planning, skin and cancer surveillance and cardiovascular risk reduction. Suspected rejection or infection → prompt transplant-team assessment → establish cause → mechanism-directed treatment. Do not independently stop immunosuppressants. Medicines that inhibit cytochrome P 4503A can raise tacrolimus concentrations and toxicity; inducers can lower them and increase rejection risk. Check every new prescription and herbal product with the transplant team. Severe diarrhoea can alter exposure. Long-term success requires balancing rejection prevention against infection, malignancy and treatment toxicity; transplantation treats organ failure but does not remove the need for continuing care.

MEMORY AID

Protect the graft, prevent infection, check every interaction.

Foundation 16 • Medical foundations • Source-checked supplementary teaching • September 2026. Laboratory ranges and treatment protocols require local context.

Transplantation graft rejection and immunosuppression. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Consultation-liaison and systemic medicine psychiatry.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P22

P22 — Fibromyalgia: pain, mood and psychological care

Fibromyalgia involves persistent widespread pain with symptoms such as fatigue, unrefreshing sleep and cognitive difficulty. Altered pain processing is relevant, but no single mechanism explains every person's experience. Depression and anxiety may coexist and worsen disability; they are not required to make the diagnosis. Psychological treatment can improve coping and function without implying that the pain is imaginary or that emotional conflict caused the illness.

Separate diagnosis from distress.

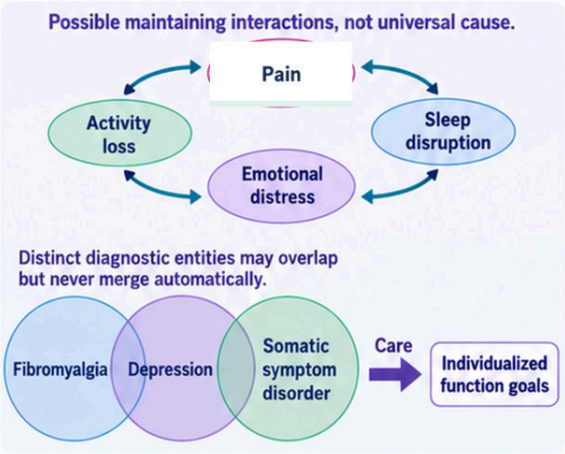
Establish the distribution and duration of pain, associated symptoms and effect on work, sleep, relationships and activity. Review the examination and targeted investigations for plausible alternatives or coexisting disease. Fibromyalgia can coexist with inflammatory arthritis or other medical conditions. New focal weakness, objective joint inflammation, fever, unexplained weight loss or a substantial change in pattern warrants reassessment. Normal routine tests do not by themselves establish fibromyalgia or a psychiatric disorder.

Assess mood directly.

Ask about persistent low mood, loss of interest, hopelessness, guilt and suicidal thoughts. Fatigue, poor sleep and concentration difficulty overlap with pain disorders, so interpret the full syndrome and timeline. Assess anxiety, trauma symptoms when relevant, substance use and medication effects without assuming a trauma history. Clarify whether the person avoids activity because of anticipated pain, exhaustion, previous symptom flares or another concern. Ask about strengths, meaningful activities and what improvement would matter most to them.

Do not equate fibromyalgia with somatic symptom disorder.

Somatic symptom disorder requires distressing bodily symptoms together with excessive symptom-related thoughts, anxiety or behaviour. The judgement must account for actual illness, disability and context. Repeated care-seeking can reflect inadequate treatment or uncertainty rather than psychopathology. A medically explained symptom does not rule the disorder out, but unexplained pain does not rule it in. Diagnose both only when each set of criteria is independently met, and explain this without invalidating the physical condition.



Create a shared formulation.

Pain can disrupt sleep; poor sleep can increase sensitivity, fatigue and emotional strain. Worry and sustained attention to threat can intensify the burden, while disability can reduce rewarding activity and social contact. These are interacting, potentially modifiable processes, not proof of a single psychological origin. Identify which processes actually occur for this patient. Set goals such as improved sleep consistency, participation or reduced distress rather than promising complete pain elimination.

Psychological and physical care.

Explain the condition clearly and offer individualized education. Cognitive behavioural approaches can address catastrophic predictions, unhelpful avoidance, sleep patterns and problem solving. Acceptance-based approaches can support valued activity while acknowledging symptoms. Relaxation and stress-management skills may help selected patients. Physical activity has guideline support in fibromyalgia and should be tailored to tolerance, preference and comorbidity; it is not a punishment or a test of motivation. If delayed, prolonged post-exertional worsening suggests coexisting myalgic encephalomyelitis/chronic fatigue syndrome, reassess before applying standard exercise progression.

Medication and follow-up.

Selected medicines may target pain, sleep or a comorbid depressive disorder; discuss expected benefit and adverse effects rather than adding drugs indefinitely. Antidepressant use for pain does not prove depression. Review sedation, cognitive effects, falls, weight change and interactions. Avoid reflex escalation to strong opioids for fibromyalgia. Coordinate one care plan, review treatments that are ineffective and support occupational accommodations. Monitor pain interference, sleep, mood, safety and chosen functional goals, not pain intensity alone. A flare calls for reassessment and adjustment, not blame.

Memory aid: "Validate pain, assess mood, build function."
Each part is necessary; none substitutes for the others.

Supplementary psychiatric teaching • September2026

Fibromyalgia psychiatric care. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Consultation-liaison and systemic medicine psychiatry.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P23

P23 — Chronic fatigue: psychiatric differential and supportive care

Chronic fatigue is a symptom with many causes. Myalgic encephalomyelitis/chronic fatigue syndrome is a specific clinical illness involving substantial loss of function and characteristic symptoms, especially post-exertional malaise. It is not synonymous with depression, lack of motivation or ordinary tiredness. Psychiatric assessment can identify treatable comorbidity and support adaptation while preserving appropriate medical evaluation.

Clarify the experience.

Ask what "fatigue" means: sleepiness, low energy, muscular weakness, breathlessness, reduced motivation or worsening after activity. Establish onset, infections, medicines, substances, sleep, pain and previous function. Post-exertional malaise is a disproportionate exacerbation of symptoms after physical, cognitive, emotional or social exertion, often delayed and prolonged. Ask what happens later that day and over the following days, not only during a clinic task. A person may complete an activity yet experience substantial subsequent deterioration.

Use a named diagnostic framework.

Diagnostic systems differ in required duration and symptom combinations. The 2015 United States framework requires more than six months of substantial impairment with fatigue, post-exertional malaise and unrefreshing sleep, plus cognitive impairment or orthostatic intolerance. The 2021 National Institute for Health and Care Excellence framework permits diagnosis after three months when its required symptoms persist and alternatives have been considered. Do not combine the duration from one framework with the symptoms of another without explanation. There is no confirmatory routine blood test.

Consider medical and psychiatric alternatives.

Review anaemia, thyroid disease, sleep apnoea, medication effects, infection, inflammatory disease and other causes according to the history and examination. Assess orthostatic symptoms, nutrition and pain. Investigations should be targeted and revisited if new signs appear. For depression, ask directly about persistent low mood, loss of pleasure, guilt, hopelessness and suicidal thinking. Fatigue and poor concentration alone are insufficient. A wish to be active does not exclude depression, and reduced activity does not establish it. Anxiety, trauma-related symptoms and substance use can coexist rather than explain everything.

Offer psychological support with an honest rationale.

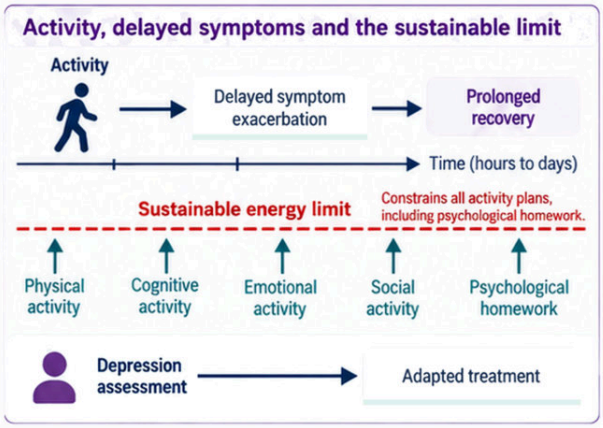
Validate the illness and acknowledge losses in work, education, relationships and identity. Cognitive behavioural therapy may be chosen to help cope with symptoms, distress and practical problems; it is not a cure and should not be based on the assumption that false beliefs cause the illness. Adapt session length, sensory load, homework and communication to cognitive and energy limitations. Treat established depression or anxiety using evidence-based approaches adjusted to the person's physical limits. Monitor suicide risk without treating understandable distress as automatic evidence of major depression.

Protect the activity boundary.

Energy management aims to stay within a sustainable individual limit across all forms of activity. Plan rest and prioritize meaningful tasks; activity may need to decrease during relapse. Do not prescribe fixed incremental exercise increases or insist that the patient push through post-exertional malaise. If a personalized physical-activity programme is considered, it should be overseen by appropriately experienced professionals and adjusted flexibly. Behavioural activation for comorbid depression must respect these limits rather than overriding them.

Monitor and coordinate.

Review sleep problems, pain, orthostatic intolerance, medicines and practical support. Start symptom-targeted medicines cautiously when sensitivity or adverse effects are concerns; an antidepressant treats a defined indication, not the entire illness. Track delayed symptom flares, function, nutrition, mood and patient-chosen goals. Severe illness may require home-based care and reduced light, sound or touch exposure. Coordinate school or workplace accommodations, benefits and caregiver support. New focal neurological signs, marked weight loss or other deterioration need reassessment rather than automatic attribution to the existing diagnosis.



Memory aid: "Listen to the delay; work within the limit."

Supplementary psychiatric teaching • September 2026

Chronic fatigue psychiatric differential and support. Read across the labelled contrasts to distinguish the related concepts. Section companion: Consultation-liaison and systemic medicine psychiatry.

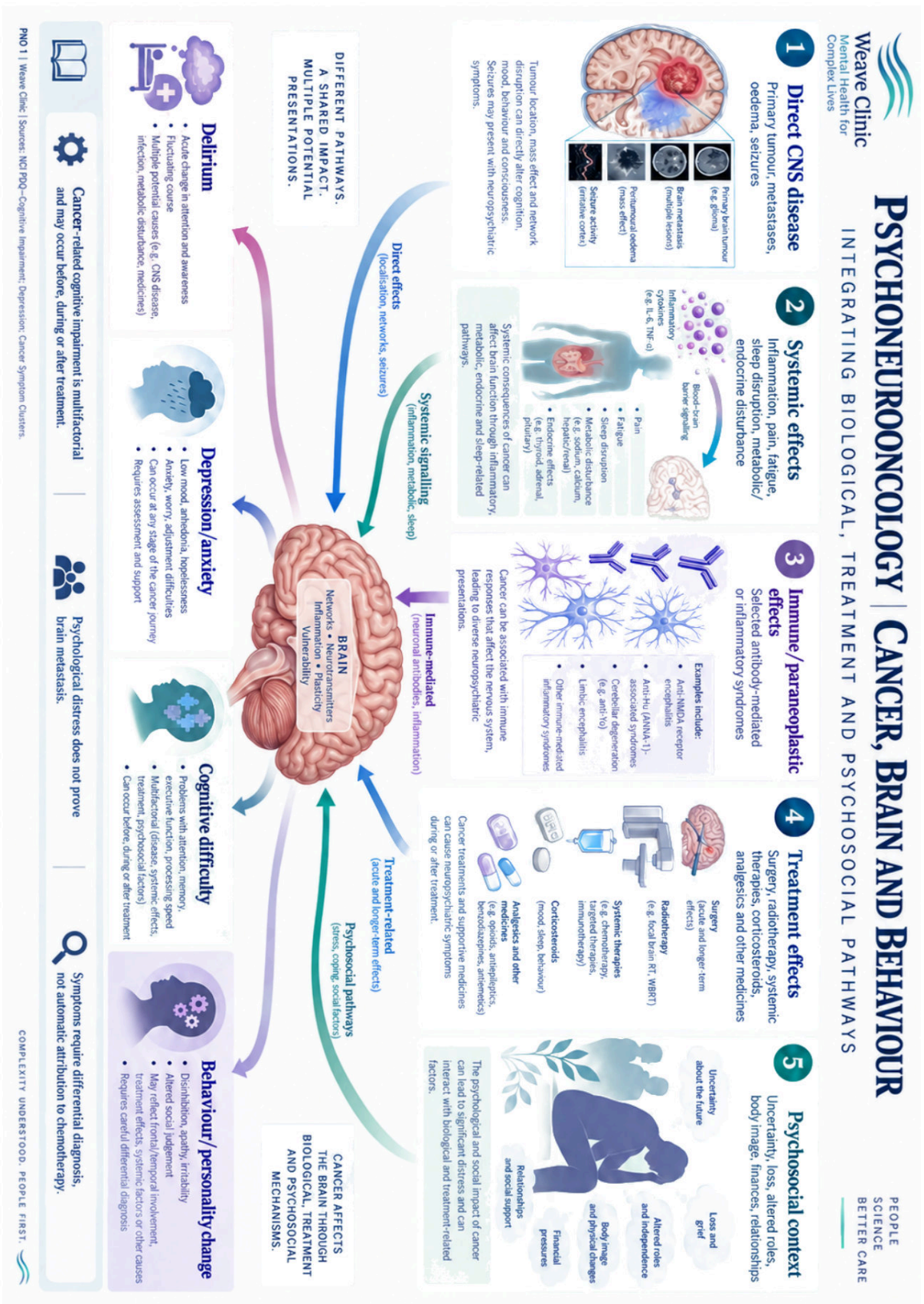
Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P24



Fibromyalgia versus chronic fatigue versus somatic symptom disorder versus depression. Read across the labelled contrasts to distinguish the related concepts. Section companion: Consultation-liaison and systemic medicine psychiatry.

Credits and references: retained within the illustration.



Psychoneurooncology - PNO-1. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Consultation-liaison and systemic medicine psychiatry.

Credits and references: retained within the illustration.

Weave Clinic
Mental health
for a more human cancer care

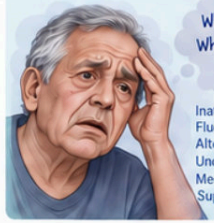
PSYCHONEUROONCOLOGY | ASSESSMENT AND INTEGRATED CARE

WHOLE PERSON CARE • SHARED EXPERTISE • BETTER OUTCOMES

PEOPLE
SCIENCE
COMPASSION
TOGETHER

DELIRIUM

Assess attention and arousal; identify and treat causes; supportive care and medication review.



Inattention
Fluctuating course
Altered arousal
Underlying causes
Medication review
Supportive care

PERSISTENT COGNITIVE COMPLAINTS

Functional assessment; evaluate reversible contributors; rehabilitation/compensatory strategies when appropriate.



Distributed cognitive domains

Not all cognitive changes are due to cancer treatment.

MOOD AND DISTRESS

Evidence-based psychological and pharmacological care, with interaction and organ-function review.

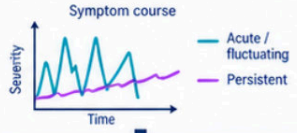


Psychological therapies
Medication when indicated
Review interactions
Consider organ function
Address existential distress
Support meaning and hope

1 ESTABLISH ONSET AND TRAJECTORY

Understand the time course and relationship to cancer and treatment.

Acute or fluctuating (hours to days)
- Suggests delirium, seizures, metabolic disturbance, infection or treatment effect



Persistent (weeks to months)
- Consider depressive disorder, anxiety, cognitive impairment or adjustment
- Explore relationship to cancer, treatment and life context

2 CHECK URGENT CAUSES

Identify and treat potentially reversible or life-threatening conditions.



3 DEFINE THE SYNDROME

Use a structured clinical assessment to clarify the main presentation.

Distress
- Common and understandable reaction to cancer
- Consider when it exceeds expected coping

Depressive disorder
- Persistent low mood and loss of interest
- Assess for suicidal ideation when indicated

Anxiety
- Worry, panic or heightened arousal
- Consider specific disorders (e.g. GAD, panic, adjustment)

Delirium
- Acute change, inattention, fluctuating course
- Identify and treat underlying causes

Cognitive impairment
- Subjective and/or objective cognitive change
- Consider multiple aetiologies

Assess suicide risk when indicated, and act promptly on any risk of harm.

4 REVIEW CONTRIBUTORS

Identify and address factors that can worsen or mimic neuropsychiatric symptoms.



Pain — Uncontrolled pain worsens mood, sleep and cognition

Sleep — Disturbed sleep is common and multifactorial

Fatigue — Distinguish cancer-related fatigue from depression or medical causes

Medicines — Review psychoactive drugs, steroids, antiemetics and interactions

Organ function — Consider hepatic or renal impairment

Endocrine changes — e.g. thyroid, adrenal, pituitary dysfunction

Support — Assess social, practical and spiritual support and carer needs

5 COORDINATE CARE

Integrated, patient-centred care with the right expertise at the right time.



Oncology — Cancer treatment plan and treatment-related effects

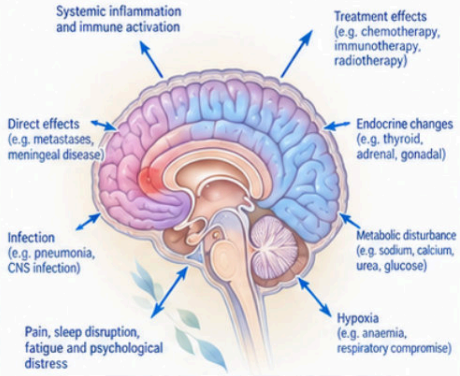
Psychiatry — Assessment and management of mental health conditions

Neurology — Seizures, focal deficits and complex cognitive syndromes

Palliative care — Symptom control, quality of life and end-of-life care

THE INTERCONNECTED BRAIN

Cancer, its treatments and the body can affect brain function through multiple mechanisms.



Schematic influences, not anatomical localisation.

INTEGRATED CARE THROUGH THE CANCER JOURNEY

Needs and priorities may change over time.



CRITICAL BOUNDARY

Do not assume every depressive symptom is an inevitable reaction to cancer, and do not label every cognitive complaint chemotherapy toxicity.

Look broadly, assess carefully, and keep an open mind.
People are more than their diagnosis or treatment.

RESPECTFUL CARE | Preserve autonomy • assess capacity for the specific decision • communicate with consent • support carers

Different minds A kinder tomorrow

PNO 2 | Weave Clinic | Sources: NCI PDQ—Delirium; Depression; Cognitive Impairment; Adjustment to Cancer.

Clinical knowledge. Human connection. A more hopeful tomorrow.

ILLUSTRATIONS

Psychoneurooncology - PNO-2. Use the visual organisation to recall the key distinctions and structure your answer.
Section companion: Consultation-liaison and systemic medicine psychiatry.

Credits and references: retained within the illustration.

AREA 04 OF 15 · P4-A02

Clinical neurology, headache and sleep neurology

8	8.0%	IV-31	IV-38
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 7

REVISION ORDER

The three headache slots first and as one block, because the classification slot opens both of the others. The two sleep slots next, and they share their pharmacology. The imaging and electrophysiology slot after that. Auditory event related potentials and neurological soft signs are independent of everything else here.

WHAT IS IN THIS AREA

Headache in three slots: migraine classified with its epidemiology, course and treatment, tension headache, and a classification slot that runs on to the acute and preventive treatment of the chronic tension type. Sleep in two, insomnia in old age and the orexins with the receptor antagonists. The registered name of this area stops there, and three of its eight slots are investigation questions: radiological and electrophysiological imaging, auditory event related potentials, and neurological soft signs. They are answered on their pages as they were set.

IV - 31

Describe the use of radiological and electrophysiological investigations in psychiatric illnesses. [5+5]

5 + 5 | CLINICAL NEUROLOGY, HEADACHE AND SLEEP NEUROLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **The principle**	These tests exclude and localise; they do not diagnose
3 **Radiological**	Structural first, functional only where it changes a decision
3 **Electrophysiological**	The record, the sleep study, the evoked potential
2 **Choosing, and limits**	Who to scan, and what a normal result means

THE PRINCIPLE

No psychiatric diagnosis is made by imaging or by a recording. They are ordered to exclude a treatable physical cause, to localise one that is suspected, and in research to test a mechanism. An answer that opens by listing machines rather than indications loses the first two marks.

INVESTIGATION	WHAT IT SHOWS	ORDERED IN PSYCHIATRY WHEN	THE CAVEAT
CT head	Bleed, calcification, gross lesion	Acute, uncooperative, or metal in situ	Poor for temporal lobe
MRI brain	Grey and white matter, hippocampus, small vessels	First episode with atypical features, late onset, focal signs, dementia	**Preferred structural test**
PET and SPECT	Regional metabolism or perfusion	Dementia subtype in doubt after MRI	Tertiary and research use
EEG	Slowing, epileptiform activity, periodic patterns	Delirium, suspected seizure, encephalitis, catatonia	**A normal record excludes little**
Polysomnography	Sleep architecture, apnoea, limb movements	Suspected apnoea, narcolepsy, parasomnia	Not for ordinary insomnia
Evoked potentials	Averaged, time-locked cortical responses	Research, and hearing or nerve conduction questions	**No diagnostic role yet**

Amber cells are the discriminators. Each row is an indication, not a machine, which is the form the question is asking for.

- **The patterns worth naming.** Temporoparietal hypometabolism in Alzheimer disease, frontal in frontotemporal dementia, occipital in Lewy body disease; triphasic waves in hepatic encephalopathy.
- **Indian practice.** The record and computed tomography are widely available; magnetic resonance is district level or above, and metabolic imaging is essentially tertiary, so the sequence has to be defensible with what is at hand.

WHAT EARNS THE MARKS

- **Indications written before modalities.** The examiner is checking clinical reasoning, not equipment knowledge.
- **Both halves answered separately.** Five marks of imaging and five of electrophysiology, not eight and two.
- **At least two named patterns.** A pattern shows the test was understood rather than listed.

SEE ALSO Slot IV - 36 auditory event related potentials Slot IV - 38 neurological soft signs

IV - 32

Classify migraine and discuss its epidemiology, course and treatment. [4+2+2+2]

4 + 2 + 2 + 2 | CLINICAL NEUROLOGY, HEADACHE AND SLEEP NEUROLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	Classification	The families, with the criteria that define each
2	Epidemiology	Who gets it, and the psychiatric company it keeps
2	Course	Onset, decline, and what turns it chronic
3	Treatment	Two arms, each chosen by the comorbidity present

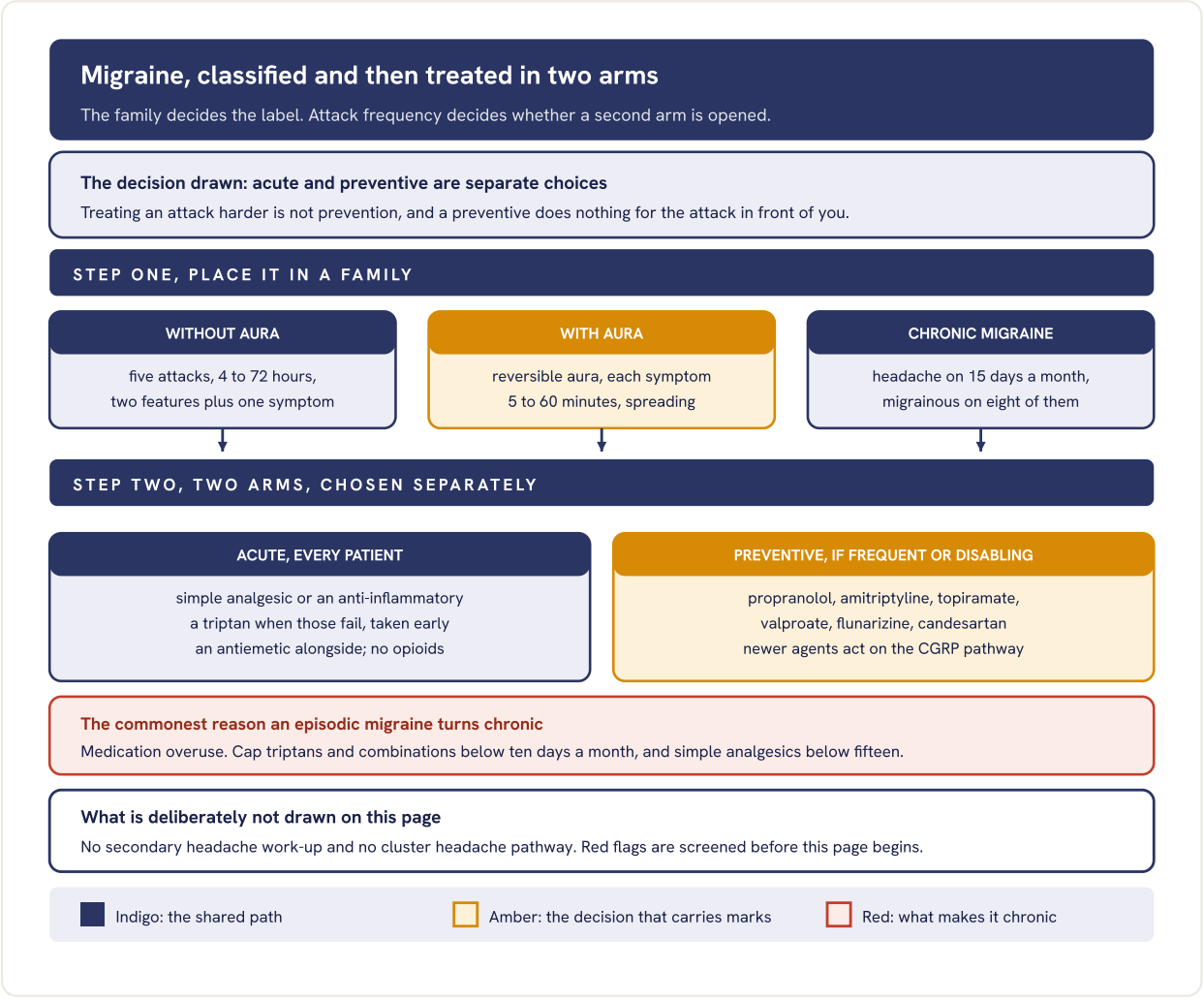


Fig 032.1 The three families most often asked about, then the two treatment arms kept apart, with medication overuse carried as a red strip because it is the modifiable driver of chronic migraine.

- **Epidemiology and course.** Two to three times commoner in women, onset in adolescence or early adult life, peak burden through the working years, and attacks usually easing after menopause.
- **Choosing the preventive by the comorbidity.** Amitriptyline where sleep is poor or tension-type headache overlaps; propranolol avoided in depression and asthma; valproate avoided in women of childbearing potential; flunarizine can cause depression and parkinsonism.

PITFALL

Migraine keeps psychiatric company: depression, anxiety and bipolar disorder are all commoner, and the relationship with depression runs both ways. Treating only the headache in a psychiatry answer misses marks the examiner has already allotted.

WHAT EARNS THE MARKS

- **Named families with their defining numbers,** not a list of headache types.
- **Acute and preventive answered as separate arms,** with the threshold for opening the second one.
- **One preventive chosen by comorbidity,** which is what a psychiatry examiner is looking for.
- **Medication overuse named as the driver of chronicity.**

SEE ALSO Slot IV - 35 classification of headache Slot IV - 33 tension type headache **SPRINT** Day 31, Neurology foundations for psychiatrists

WEAVE 100 · PAPER IV · C04

MIGRAINE AND PSYCHIATRY: A SHARED CLINICAL PLAN

Companion C04

MIGRAINE IS A NEUROLOGICAL DISORDER

Migraine involves susceptible brain networks and trigeminovascular signalling, including calcitonin gene-related peptide. Attacks are not simply dilated vessels, suppressed anger or a psychiatric symptom. Depression and anxiety commonly coexist and can increase disability, while recurrent unpredictable attacks can worsen mood and anticipatory fear. Shared susceptibility, sleep disruption and behavioural pathways contribute; association does not prove one disorder caused the other. Bipolar disorder and substance-related problems may also affect assessment and treatment choices.

RECOGNISE THE HEADACHE BEFORE FORMULATING THE DISTRESS

Typical untreated migraine lasts four to seventy-two hours in adults, with moderate/severe pain that may be unilateral, pulsating and aggravated by ordinary activity, accompanied by nausea or vomiting, or both light and sound sensitivity. Patients often prefer stillness and darkness.

Aura consists of reversible neurological symptoms, commonly visual, sensory or language symptoms that evolve over minutes; it is distinct from a vague prodrome or psychosis. First, atypical or persistent neurological symptoms require appropriate evaluation.

Chronic migraine means at least fifteen headache days monthly for over three months, with migraine features on at least eight; count headache and migraine days separately.

ASSESS FOUR LINKED TARGETS

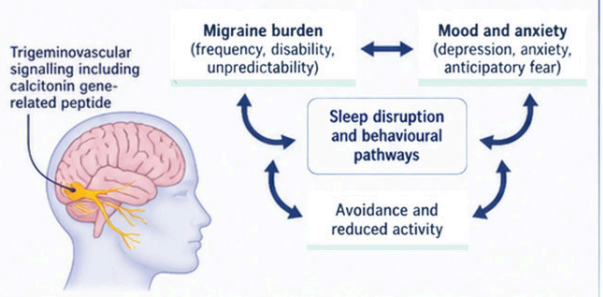
Headache: onset, phenotype, frequency, disability, menstrual relationship and previous acute/preventive trials.

Medication: count days of use, not merely tablet numbers; include combination products, caffeine, opioids and non-prescribed remedies.

Psychiatric state: screen for persistent mood/anxiety symptoms, suicidality, mania/hypomania and sleep disorder. Attack-related fatigue or transient irritability alone does not establish depression or bipolar disorder.

Behaviour and context: anticipatory avoidance, irregular sleep/meals, excessive trigger searching, work demands, support and access to treatment. A diary should inform decisions without becoming constant hypervigilance.

MIGRAINE, MOOD AND MANAGEMENT: A CONNECTED CYCLE



TWO TREATMENT LANES

Acute treatment
(triptan and/or non-steroidal anti-inflammatory drug or paracetamol, with antiemetic when needed)

Preventive treatment
(match to comorbidity and risk: for example amitriptyline, topiramate, propranolol, candesartan or specialist calcitonin gene-related peptide treatments)

Monitor and review

Headache days • Disability • Acute-use days • Adverse effects • Mood

PSYCHOLOGICAL CARE: PRACTICAL COMPONENTS

Explain the neurological diagnosis and the pain–threat–avoidance cycle.
Agree functional goals alongside fewer headache days.
Cognitive behavioural therapy identifies catastrophic predictions, reduces excessive avoidance and supports problem-solving and relapse plans.
Relaxation training practises a repeatable physiological skill; biofeedback teaches regulation using measured signals such as muscle tension.
Schedule regular meals, hydration, sleep and individually tolerable activity.
Distinguish reproducible personal triggers from broad fear-driven restriction.
These measures complement medical therapy and can help without a psychiatric diagnosis.
Treat a coexisting psychiatric syndrome on its own merits, rather than promise that curing anxiety will eliminate migraine.

ACUTE AND PREVENTIVE ARE DIFFERENT DECISIONS

Agree an early-attack plan using a suitable triptan and/or non-steroidal anti-inflammatory drug or paracetamol, with an antiemetic when needed.
Choose route according to vomiting and attack speed, and review vascular or other contraindications.
Avoid routine opioids.
Frequent or disabling attacks warrant preventive discussion even if acute treatment works.
Match selection to comorbidity and risk: amitriptyline may help sleep but causes anticholinergic effects, sedation and overdose concerns; topiramate can impair word finding/concentration and carries major reproductive risks; propranolol can cause fatigue and is unsuitable in asthma or important bradycardia.
Depression is not an automatic absolute contraindication to propranolol.
Consider candesartan or specialist calcitonin gene-related peptide treatments where appropriate.
Do not choose valproate casually because bipolar disorder coexists; reproductive safeguards remain essential.

PSYCHOTROPIC SAFETY AND OVERUSE

Antidepressant selection requires bipolar-risk review and attention to interactions.
Combining a triptan with a serotonergic antidepressant is not an automatic prohibition, but educate about serotonin-toxicity symptoms and review other serotonergic medicines.
Medication-overuse headache requires frequent headache plus regular overuse for over three months: thresholds depend on drug class, commonly ten days monthly for triptans/combination analgesics and fifteen for simple analgesics.
These are diagnostic thresholds, not safe-use targets.
Plan withdrawal/support and prevention rather than blame.

REVIEW AND RED FLAGS

Review headache days, disability, acute-use days, adverse effects and mood separately.
Thunderclap onset, fever, new focal signs, papilloedema or a major pattern change requires medical evaluation before routine psychiatric attribution.

MEMORY AID

Name the headache, assess the mood, match prevention, review use.

Supplementary medical teaching • September 2026

Migraine psychiatric overlaps. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-32.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · F23

HEADACHE MECHANISMS — MIGRAINE AND TENSION-TYPE HEADACHE

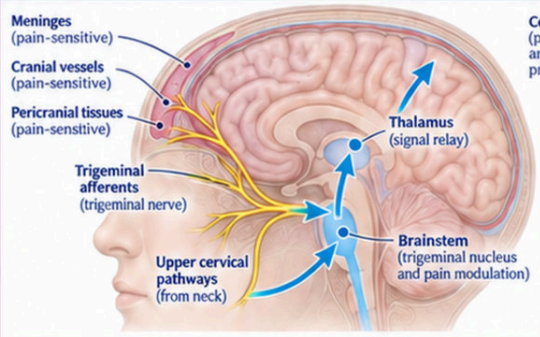
Foundation 23 • Normal function → mechanism → clinical recognition → management

NORMAL FUNCTION AND BACKGROUND

Normal: pain-sensitive structures include the meninges, cranial vessels and pericranial tissues; brain tissue itself is relatively pain-insensitive. Trigeminal afferents and upper cervical pathways carry head pain to brainstem, thalamus and cortex. Descending pathways modify this signal.

A **primary headache** is the disorder itself; a **secondary headache** results from another condition such as haemorrhage, infection or raised intracranial pressure.

HEAD PAIN PATHWAY AND MIGRAINE MECHANISM



Amplification in migraine

Altered sensory processing and increased network responsiveness lead to pain and sensory hypersensitivity.

Meninges, cranial vessels and pericranial tissues → Trigeminal afferents and upper cervical pathways → Brainstem (trigeminal nucleus) → Thalamus → Cortex (pain perception and sensory processing)

MIGRAINE

Migraine mechanism: altered sensory processing and trigeminovascular activation produce pain and sensory hypersensitivity; calcitonin gene-related peptide participates in this pathway. Migraine is not simply dilated blood vessels. Cortical spreading depolarisation, a slowly moving wave of altered cortical activity, is associated with aura. **Aura** consists of reversible neurological symptoms, commonly visual or sensory, often spreading gradually. New, atypical or persistent deficits require assessment for other causes.

Migraine without aura, adult criteria:

- at least five attacks lasting 4–72 hours untreated or unsuccessfully treated;
- at least two of unilateral pain, pulsating quality, moderate/severe intensity, or worsening with/avoidance of routing activity;
- during pain, nausea/vomiting or both light and sound sensitivity;
- no better explanation.

Pulsating activity-sensitive pain reflects sensitised pathways; nausea and sensory aversion accompany broader network activation. Children may have shorter attacks and bilateral pain.

Chronic migraine: headache on at least 15 days monthly for over three months, including at least eight days with migraine features or migraine-specific treatment response in an appropriate history.

TENSION-TYPE HEADACHE

Tension-type mechanism: peripheral muscle nociception and altered central pain processing contribute; muscle tension alone is not a complete explanation.

Typical episodic headache lasts 30 minutes to seven days, with at least two of:

- bilateral location;
- pressing/tightening quality;
- mild/moderate intensity;
- no worsening with routine activity.

No nausea or vomiting; at most one of light or sound sensitivity. Pericranial tenderness may occur.

Frequent episodic disease requires at least ten episodes on 1–14 days monthly for over three months.

Chronic disease occurs on at least 15 days monthly for over three months; mild nausea can occur, unlike episodic criteria.

CLINICAL RECOGNITION AND INVESTIGATION

Evaluate first: onset, maximum-intensity timing, duration, triggers, neurological symptoms, systemic illness, pregnancy, trauma and medication use.

Examine vital signs, neurological function and optic discs.

Keep a diary of headache days, disability, associated features and acute medication days.

No blood reference range confirms primary headache; routine imaging is unnecessary for a typical stable pattern with normal examination.

Investigate red flags:

- sudden thunderclap;
- fever/meningism;
- focal deficit;
- impaired consciousness;
- papilloedema;
- new headache with cancer/immunosuppression;
- or major pattern change.

New headache after age 50 requires consideration of secondary causes.

MANAGEMENT FLOW

Red flags present?

Sudden thunderclap, fever/meningism, focal deficit, impaired consciousness, papilloedema, new headache with cancer/immunosuppression, major pattern change, or new headache after age 50

Yes → Investigate for secondary cause and treat underlying condition

No → Manage as primary headache → Identify phenotype and overuse → Choose acute and preventive plans separately → Review diary and function

MANAGEMENT DETAILS

Migraine attack

- triptan plus a nonsteroidal anti-inflammatory drug or paracetamol where suitable; consider antiemetic treatment.
- avoid opioids.
- triptans require vascular contraindication review.

Migraine prevention

- for troublesome recurrent migraine may include propranolol, amitriptyline or topiramate, selected for comorbidity and risk.
- topiramate requires strict pregnancy precautions; propranolol may be unsuitable in asthma or bradycardia; amitriptyline can cause sedation and anticholinergic effects.
- specialist calcitonin gene-related peptide treatments or botulinum toxin are options for selected patients.

Tension-type attacks

- paracetamol or an appropriate nonsteroidal anti-inflammatory drug; regular sleep, activity, relaxation and physical approaches support care.
- persistent chronic headache may need preventive treatment and specialist review.

Medication-overuse headache

- requires headache on at least 15 days monthly with overuse for over three months:
  - triptans/opioids/combination analgesics on at least ten days monthly,
  - simple analgesics on at least fifteen.
- these are diagnostic thresholds, not safe targets.
- plan supported withdrawal and prevention; monitor after treatment changes.

MEMORY AID

Pattern first, warning signs always, medicine use counted.

Foundation 23 • Medical foundations • Source-checked supplementary teaching • September 2026.
Laboratory ranges and treatment protocols require local context.

Headache mechanisms migraine and tension-type headache. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Read with IV-32.

Credits and references: retained within the illustration.

IV - 33

Clinical features and management of Tension head ache. [10]

10 CLINICAL NEUROLOGY, HEADACHE AND SLEEP NEUROLOGY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 **Clinical features**	Bilateral, pressing, and not worsened by activity
2 **Grade it by frequency**	Infrequent, frequent episodic, chronic
3 **Management**	A capped acute plan, then a preventive if frequent
2 **Non-drug and mood**	Biofeedback, therapy, sleep, and the overuse trap

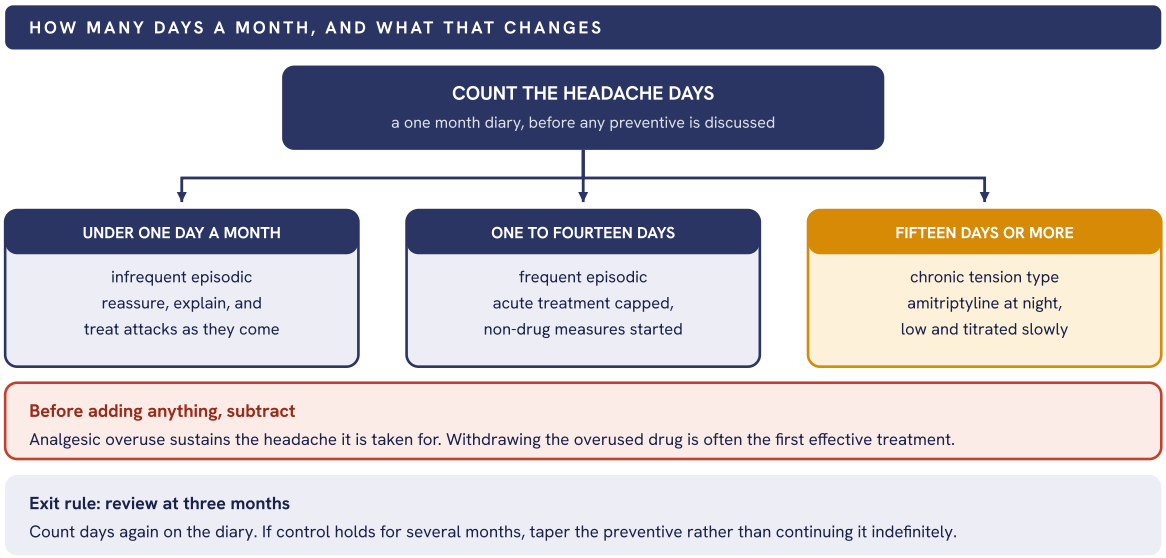


Fig 033.1 Headache days a month set the grade, and the grade decides whether a preventive is opened at all, with analgesic withdrawal placed before any addition.

- **The features that define it.** Bilateral, pressing rather than pulsating, mild to moderate, not worsened by activity, no vomiting, and at most one of light or sound sensitivity.
- **The one positive sign.** Pericranial tenderness on palpation, which also names the subtype and points to the physiotherapy and biofeedback half of treatment.

PITFALL

Amitriptyline is the preventive with the evidence here; the serotonin reuptake inhibitors have not shown benefit for this headache, even when mood is low.

WHAT EARNS THE MARKS

- **Features written as the criteria that exclude migraine,** not as a general description of head pain.
- **The frequency grade stated,** because it is what decides whether a preventive is justified.
- **Withdrawal of overused analgesics placed before any new drug.**

SEE ALSO Slot IV - 35 classification of headache Slot IV - 32 migraine SPRINT
Day 31, Neurology foundations for psychiatrists

WEAVE 100 · PAPER IV · C05

TENSION-TYPE VERSUS CLUSTER HEADACHE: RECOGNISE, THEN TREAT.

Companion C05

TWO DISTINCT PRIMARY HEADACHE DISORDERS

A primary headache disorder is diagnosed by its characteristic clinical pattern after assessing for a secondary cause. "Tension-type" does not prove muscle tension, stress or psychiatric illness is the cause. Peripheral pain mechanisms may contribute to episodic tension-type headache; altered central pain processing becomes more important in chronic disease. Cluster headache belongs to the trigeminal autonomic cephalalgias: severe trigeminal-distribution pain accompaniss cranial autonomic activation. Hypothalamic and brainstem networks contribute to its striking time pattern, it is not an extreme form of tension-type headache.

Tension-type headache

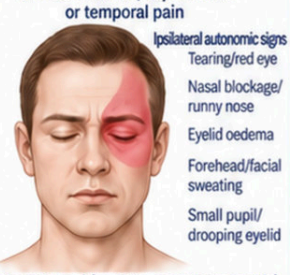
Bilateral pressing or tightening pain



Typically bilateral

Cluster headache

Unilateral orbital, supraorbital or temporal pain



Strictly unilateral (symptoms on the pain side)

SIDE-BY-SIDE RECOGNITION

Tension-type

- Usually bilateral pressing or tightening pain, mild to moderate, not worsened by ordinary activity.
- Episodic attacks last thirty minutes to seven days.
- Nausea and vomiting are absent, and at most one of light or sound sensitivity occurs.
- Pericranial tenderness may be present but is not required.
- The patient can often continue ordinary activity.

Cluster

- Severe or very severe, strictly unilateral orbital, supraorbital or temporal pain, typically fifteen to one hundred eighty minutes untreated.
- Attacks occur from once every other day to eight daily during active periods.
- Ipsilateral tearing/red eye, nasal blockage/runny nose, eyelid oedema, forehead/facial sweating or a small pupil/drooping eyelid may occur; restlessness or agitation is another defining accompaniment.
- Patients often pace rather than lie still.
- Symptoms are on the pain side, not randomly bilateral.

FREQUENCY AND DIAGNOSTIC LIMITS

Tension-type

- Frequent episodic tension-type headache occurs on one to fourteen days monthly on average for over three months; chronic tension-type headache occurs on at least fifteen days monthly for over three months.
- Chronic pain may last hours to days or be unremitting; mild nausea can occur, but not moderate/severe nausea or vomiting.
- Do not apply episodic exclusions mechanically to chronic disease.

Cluster

- Cluster diagnosis requires at least five characteristic attacks and exclusion of a better explanation.
- Episodic cluster occurs in bouts separated by remissions of at least three months; chronic cluster persists for at least one year without remission or with remissions shorter than three months.
- A diary records duration, frequency, side, autonomic features and treatment response.
- Migrainous features, medication overuse and other headache disorders can complicate classification.

TENSION-TYPE MANAGEMENT

- For occasional attacks, select paracetamol or a non-steroidal anti-inflammatory drug according to gastrointestinal, kidney and cardiovascular risk; avoid opioids.
- Frequent analgesic use can perpetuate headache.
- Persistent frequent/chronic disease needs preventive and behavioural planning: regular sleep and meals, activity, relaxation, and treatment of contributing musculoskeletal problems.
- Cognitive behavioural therapy and biofeedback can improve coping and reduce disability.
- **Amitriptyline** is a commonly supported preventive option: begin low, titrate slowly and monitor sedation, anticholinergic effects, orthostasis, cardiac risk and overdose safety.
- Antidepressant benefit here does not require depression.
- Treat comorbid depression independently; selective serotonin reuptake inhibitors should not be presented as reliably effective tension-headache prevention solely because they treat mood.

CLUSTER MANAGEMENT

- Acute attacks need a rapid treatment: prescribed high-flow oxygen through an appropriate non-rebreathing mask and/or subcutaneous or nasal triptan according to contraindications and sepeialist advice.
- Ordinary oral analgesics are too slow and usually ineffective.
- Prevention during a bout commonly uses verapamil under specialist supervision, with baseline and follow-up electrocardiography because conduction block can occur.
- Transitional treatment may bridge the delay before prevention works.
- Refractory or chronic disease needs headache-specialist care, lithium is a selected specialist option requiring renal, thyroid, interaction and concentration monitoring, not a casual substitute.
- Discuss severe distress and suicide risk compassionately.

WHEN TO REOPEN THE DIAGNOSIS

- Thunderclap onset, fever, new neurological deficit, altered consciousness, papilloedema, a new progressive pattern or new headache in a high-risk context requires investigation.
- A first cluster-like presentation merits specialist assessment and consideration of imaging.
- Do not diagnose either disorder merely from a normal scan or an anxious appearance.

MEMORY AID

Pressure with function preserved suggests tension-type; one-sided eye pain with autonomic signs suggests cluster.

Confirm the full pattern.

Supplementary medical teaching • September 2026

Tension-type and cluster headache. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-33.

Credits and references: retained within the illustration.

IV - 34

There is sudden surge of patients with insomnia in psychiatry OPDs. What is physiological insomnia? What are the causes of insomnia in old age? Discuss its management in detail of such a case. [2+3+5]

2 + 3 + 5 | CLINICAL NEUROLOGY, HEADACHE AND SLEEP NEUROLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Physiological sleep change**	What ageing alone does, and where disorder begins
3	**Causes in old age**	Five groups, screened in a single pass
4	**Management**	Behavioural first, drug last, shortest and lowest
1	**What not to prescribe**	The sedatives that buy sleep with falls

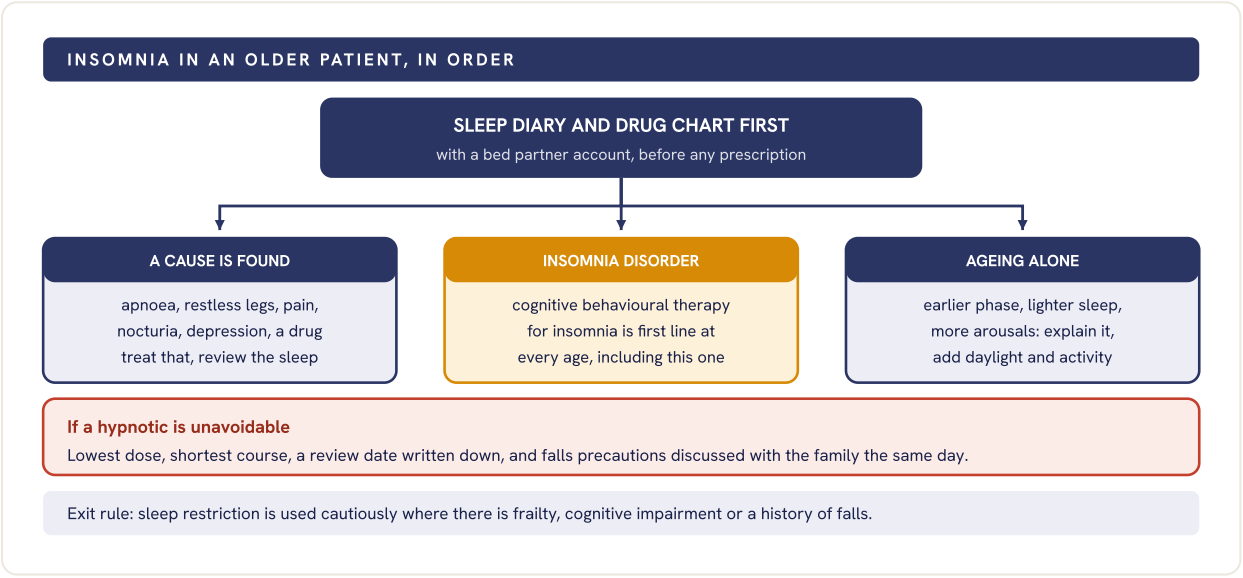


Fig 034.1 The diary and the drug chart decide which of three routes the case takes, with the hypnotic placed after all three rather than inside any of them.

- **Reading the stem.** Physiological insomnia is not a formal category. The defensible reading is normal age-related change: lighter sleep, an advanced phase and more night waking, without daytime impairment.
- **The five groups of cause.** Primary sleep disorders, medical illness, psychiatric illness including bereavement and alcohol, drugs given at the wrong hour, and circadian or behavioural factors.

PITFALL

Benzodiazepines and the Z drugs are potentially inappropriate in older adults: falls, fractures, confusion, dependence. Sedating antihistamines add delirium risk on top.

WHAT EARNS THE MARKS

- **Normal ageing separated from insomnia disorder** in the first two lines, which is what the first part asks.
- **Causes grouped, not listed.** Five headings beat twenty items for the same three marks.
- **Cognitive behavioural therapy named as first line,** with the drug chart reviewed before any drug is added.

SEE ALSO Slot IV - 37 orexin receptor antagonists **SPRINT** Day 17, Eating & sleep-wake disorders

IV - 35

Classify headache and describe acute and preventive treatment of chronic tension type headache. [4+3+3]

4 + 3 + 3 | CLINICAL NEUROLOGY, HEADACHE AND SLEEP NEUROLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

4 Classification	Three parts, then the primary families inside part one
3 The acute arm	Capped deliberately; the cap is itself the treatment
3 The preventive arm	The drug, the titration, the review, and what is added alongside

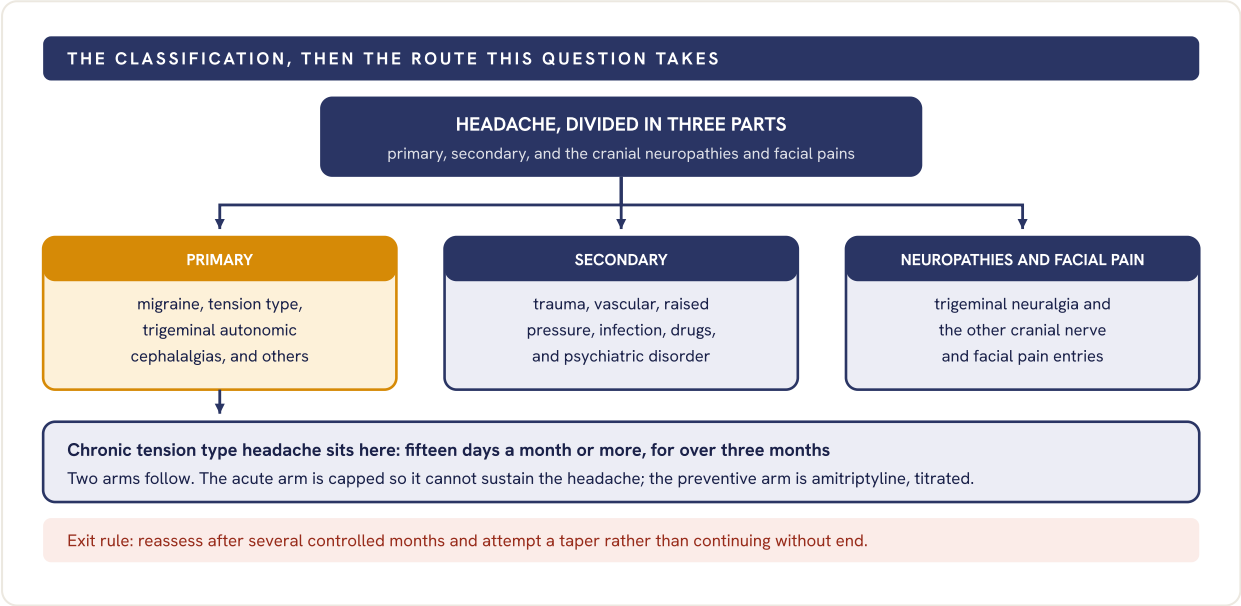


Fig 035.1 The three parts of the classification, with the route this question takes highlighted, so the four classification marks and the six treatment marks sit on one picture.

- **The acute arm, and why it is capped.** Simple analgesics only, kept below fifteen days a month; beyond that they sustain the headache they were taken for.
- **The preventive arm.** Amitriptyline at a low night dose, titrated slowly over weeks to the lowest effective dose, with mirtazapine or venlafaxine as alternatives where it cannot be tolerated.

PITFALL

Biofeedback, relaxation and cognitive therapy are additive to amitriptyline rather than a softer substitute for it, and they are what makes the taper possible later.

WHAT EARNS THE MARKS

- **Three parts named before any headache type.** Listing primary headaches only answers a third of the classification.
- **The chronic threshold stated in days and months.**
- **The cap on acute treatment written as treatment,** which is the line most answers omit.

SEE ALSO Slot IV - 33 tension type headache Slot IV - 32 migraine **SPRINT** Day 31, Neurology foundations for psychiatrists

IV - 36

Discuss about auditory event related potential (ERPs) and its use in psychiatry research. [3+7]

3 + 7 | CLINICAL NEUROLOGY, HEADACHE AND SLEEP NEUROLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	What it is	Averaged, time-locked, named by polarity and latency
2	The components	What each paradigm actually probes
3	Use in research	Endophenotype, mechanism, risk and staging
2	Limits	Group level, unspecific, and not diagnostic

THE DEFINITION

An event related potential is a voltage change in the ongoing record that is time locked to a stimulus, and pulled out of background activity by averaging many trials. Each component is named by its polarity and its approximate latency in milliseconds, and reported as amplitude and latency.

COMPONENT	PARADIGM	WHAT IT PROBES	MAIN PSYCHIATRIC FINDING
P50	Paired clicks, ratio of the second to the first	Sensory gating, early and automatic	**Poor suppression in schizophrenia and in relatives**
Mismatch negativity	Oddball with deviant tones, attention not required	Pre-attentive change detection	**Reduced in schizophrenia; among the most replicated**
P300	Oddball, the rare target counted	Attention and working memory updating	**Smaller amplitude, longer latency in schizophrenia**
N400	Sentences ending unexpectedly	Semantic processing	Abnormal where thought disorder is present

Amber cells are the discriminators. Naming the paradigm alongside the component is what separates understanding from a list of labels.

- **Why research uses them.** They are endophenotypes, present in unaffected relatives, so they index liability rather than illness. They probe mechanism, mismatch negativity being tied to glutamate function and P50 to nicotinic receptors, and they are studied for predicting transition in people at clinical high risk.
- **The honest limit.** These are group differences with overlapping distributions, altered by medication, attention, smoking and age. No component diagnoses an individual, and none is used clinically for that purpose.

WHAT EARNS THE MARKS

- **Averaging and time locking in the definition.** Without them this is just an ordinary recording.
- **Each component tied to its paradigm,** which is where the seven discussion marks sit.
- **Endophenotype used precisely,** meaning present in relatives, not merely abnormal in patients.
- **The limit stated.** A research tool described as a diagnostic test reads as unfamiliarity.

SEE ALSO Slot IV - 31 investigations in psychiatric illness Slot IV - 38 neurological soft signs

IV - 37

Discuss the role of orexins in sleep and wakefulness. How orexin receptor antagonists are helpful in sleep issues? [5+5]

5 + 5 | CLINICAL NEUROLOGY, HEADACHE AND SLEEP NEUROLOGY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The system	Two peptides, one small nucleus, two receptors
3 What they do	Hold the wake side of the switch steady
3 Blocking them	Removing wakefulness rather than adding sedation
2 Cautions	Narcolepsy, dreaming, next day, interactions

THE SYSTEM, IN ONE PARAGRAPH

Orexin A and B, also called hypocretins, come from a small group of neurons in the lateral hypothalamus and act at two receptors. They project to the noradrenergic, serotonergic, histaminergic and cholinergic arousal nuclei, and hold the wake side of the sleep-wake switch stable, which is why their loss produces narcolepsy with cataplexy rather than simple sleepiness.

DOMAIN	BENZODIAZEPINE RECEPTOR AGONIST	OREXIN RECEPTOR ANTAGONIST	WHAT SETTLES IT
Mechanism	Enhances inhibition across the brain	Removes one wake-promoting signal	**Subtraction, not sedation**
Sleep architecture	Deep sleep reduced, REM suppressed	Architecture better preserved, REM increased	**The main pharmacological claim**
Which insomnia	Mainly onset	Onset and maintenance	Maintenance is the gap it fills
Next day and falls	Psychomotor slowing, falls in the elderly	Less impairment at licensed doses	**Why it is proposed in old age**
Dependence	Tolerance, rebound, withdrawal	Low abuse potential, less rebound	Still prescribed with a review date

Amber cells are the discriminators. Every row follows from the first: blocking a wake signal is a different act from deepening inhibition.

- **The agents.** Suvorexant, lemborexant and daridorexant block both receptors. Beyond sleep, the same system links arousal to feeding and reward, which is why orexin is also studied in addiction.
- **Cautions.** Contraindicated in narcolepsy. Vivid dreams, sleep paralysis and next-day somnolence occur, metabolism is through cytochrome P450 3A4, and cost and availability limit use in Indian practice.

WHAT EARNS THE MARKS

- **The switch model named,** with orexin stabilising it rather than simply causing wakefulness.
- **Narcolepsy used as the proof of function,** which shows the physiology was understood.
- **Subtraction contrasted with sedation,** the single line that answers the second half.

SEE ALSO Slot IV - 34 insomnia in old age SPRINT Day 17, Eating & sleep-wake disorders

IV - 38

Neurological soft signs. [10]

10 CLINICAL NEUROLOGY, HEADACHE AND SLEEP NEUROLOGY

RGUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	Definition	Minor, non-localising, and no focal lesion behind them
3	Domains and tests	What is examined, and with what at the bedside
3	Why psychiatry cares	Neurodevelopmental evidence and the relatives
2	Limits	Unspecific, confounded, and not diagnostic

THE DEFINITION

Neurological soft signs are minor, non-localising abnormalities of motor coordination, sensory integration, the sequencing of complex acts and the primitive reflexes, found without a focal lesion or a defined neurological disorder.

DOMAIN	BEDSIDE TEST	A POSITIVE LOOKS LIKE	NOTE
Motor coordination	Rapid alternating movements, finger to thumb, tandem walk	Clumsy, slow, breaks down on speed	Easiest to elicit
Sensory integration	Stereognosis, graphaesthesia, two-point discrimination, face-hand test	Cannot name an object or a traced figure by touch	Needs a cooperative patient
Sequencing	Fist-edge-palm, and other three-step motor sequences	Order lost after a few repetitions	The most discriminating domain
Primitive reflexes	Grasp, snout, glabellar tap; mirror movements	Present, or not extinguishing on repetition	Age norms matter
Rating	Neurological Evaluation Scale, Cambridge Neurological Inventory	Scored, so change can be followed	Name one in the answer

Amber cells are the discriminators. A named scale and a named test are what turn a definition into an examination the candidate could actually perform.

- **Why they matter in psychiatry.** Commoner in schizophrenia, including in drug-naive first-episode patients and unaffected relatives, so they are read as an endophenotype and as neurodevelopmental evidence. They track with negative symptoms and poorer outcome.
- **The honest limit.** Not specific: rates rise in bipolar disorder, obsessive compulsive disorder and with ageing, and drug-induced extrapyramidal signs confound the motor domain.

WHAT EARNS THE MARKS

- **Non-localising written into the definition,** with the absence of a focal lesion stated.
- **Domains with an actual test in each.** Naming the fist-edge-palm sequence beats naming three more syndromes.
- **Drug-naive patients and relatives cited,** because that is what makes the endophenotype claim.
- **One named scale,** and one honest statement of non-specificity.

SEE ALSO Slot IV - 31 investigations in psychiatric illness Slot IV - 36 auditory event related potentials SPRINT Day 31, Neurology foundations for psychiatrists

WEAVE 100 · PAPER IV · C07

c07 — Neurological soft signs: examine the process, not just the score.

Companion C07

Overview

Skilled movement requires sensory input, motor planning, sequencing, coordination and inhibition to work together.
Neurological soft signs are subtle abnormalities that do not clearly localize to one focal lesion.
They are described in neurodevelopmental and psychiatric conditions, including schizophrenia, but also occur in healthy people.
They are neither specific diagnostic tests nor proof that symptoms are psychological.
New asymmetry, weakness, a sensory level or an unequivocal pathological reflex requires assessment for neurological disease.

Before testing

Explain and demonstrate each task, ensure comprehension, and establish handedness.
Check vision, hearing, strength, primary sensation, pain and joint movement first.
Record age, developmental history, fatigue, cooperation and medication exposure.
Sedation, tremor, extrapyramidal effects, cerebellar disease and peripheral neuropathy can mimic or magnify findings.
Compare both sides under the same conditions.
Use a standardized instrument rather than adding unrelated bedside observations into an improvised total.

Motor coordination: smoothness and timing

For rapid alternating movements, ask the patient to alternate palm and back of hand on the opposite palm, then increase speed.
Observe rhythm, amplitude and breakdown rather than speed alone.
For finger–thumb opposition, sequentially touch the thumb to each fingertip and reverse; look for missed fingers or overflow movement.
For finger–nose testing, alternate between the patient's nose and your moving finger, noting dysmetria (overshooting or undershooting the target) or intention tremor.
For tandem walking, ask for heel-to-toe steps while guarding against falls.
Poor performance may reflect coordination difficulties, but definite ataxia should not be dismissed as a soft sign.

Motor sequencing: keeping the rule

Demonstrate fist–edge–palm on a table, then ask the patient to repeat the sequence independently several times.
Observe order errors, perseveration, loss of rhythm and reliance on copying.
Test each hand separately if appropriate.
Alternating fist–palm patterns can increase demand.
A failure may reflect difficulty learning instructions, motor planning, working memory or movement execution; one abnormal sequence is not a diagnosis of frontal disease.

Sensory integration: recognizing a meaningful pattern

With eyes closed and primary sensation intact, place a familiar object in the hand for stereognosis and ask the patient to identify it.
For graphesthesia, trace a recognizable number on the palm, first demonstrating with eyes open.
Test two-point discrimination with randomized one- versus two-point contacts; thresholds depend on body site and technique, so avoid one universal cutoff.
For extinction, demonstrate that each side is detected alone, then stimulate both simultaneously.
Consistently missing one simultaneous stimulus suggests an attentional integration problem.
Naming failure must be distinguished from inability to recognize the object.

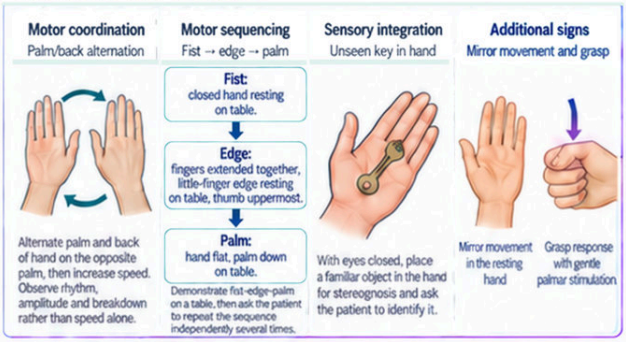
Additional signs: inhibition and developmental patterns

Observe associated mirror movements in the resting hand while the other performs a task.
A grasp response is elicited by gentle palmar stimulation; persistent involuntary gripping differs from following an instruction.
Snout or persistent glabellar responses can be recorded, but isolated primitive reflexes have limited specificity and vary with age.
Explain the procedure and avoid painful or intrusive manoeuvres.
Prominent new frontal release signs merit clinical evaluation.

Interpretation and relevance

The Neurological Evaluation Scale groups sensory integration, motor coordination, complex motor sequencing and additional signs; the Cambridge Neurological Inventory uses its own structure.
Follow the instrument's exact administration and scoring.
Findings may accompany developmental vulnerability, cognitive difficulty or illness burden at group level.
There is no laboratory reference interval or universally diagnostic total.
Management addresses the underlying disorder, medication adverse effects and functional needs; occupational or neurological assessment may help when daily skills are affected.
Repeat under comparable conditions if tracking change.

Key tasks and examples



Exclude weakness, sensory loss, comprehension and medication effects before interpretation.

MEMORY AID | **Demonstrate, compare sides, exclude confounders, interpret the pattern.**

Supplementary medical teaching • September 2026

Neurological soft signs. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-38.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · C06

c06 — Measuring mental processes: what the signal actually means

Companion C06

Key principles

- **Mental processes are inferred from converging observations:** what someone reports, what they do, and what their nervous system records.
- A laboratory measure samples a process under particular conditions.
- It does not read thoughts, replace the clinical interview, or automatically diagnose an individual.
- Reliability means repeatability; validity means measuring the intended construct.
- A reproducible group difference can still classify individuals poorly because distributions overlap.

Error-related negativity: detecting an action error

- During a flanker task, the participant presses a left or right key for the central arrow while ignoring distracting arrows.
- Speed pressure produces occasional errors.
- Scalp electroencephalography records voltage changes; many trials are aligned to the keypress and averaged separately for correct and incorrect responses.
- The error-related negativity is a brief frontocentral negative deflection, usually peaking within roughly 50–100 milliseconds after an erroneous response; timing depends on task and analysis.
- Compare the error waveform with the correct-response waveform.
- Voltage is measured in microvolts, not a diagnostic blood-test range.
- Medial frontal networks, including anterior cingulate and presupplementary motor regions, contribute to performance monitoring.
- The signal is associated with detecting a mismatch or need for control; subsequent slowing may represent adjustment but is not invariably beneficial.
- Scalp location is not proof that a single brain area generated the entire response.
- Record enough clean error trials, check accuracy and response speed, remove eye and muscle artefacts, and specify the reference and scoring window.
- Age, motivation, anxiety, medication and task difficulty affect interpretation.
- Research associations with compulsivity or anxiety are not stand-alone diagnostic thresholds.

Other electrical windows

- Event-related potentials average responses time-locked to a stimulus or action.
- P50 (a positive response around 50 milliseconds) suppression compares responses to paired clicks and probes sensory gating.
- Mismatch negativity compares frequent sounds with occasional deviants and probes auditory change detection.
- P300 (a positive response around 300 milliseconds) is often studied when a person detects rare targets; it reflects attention and context updating, not an intelligence score.
- N400 (a negative response around 400 milliseconds) varies with semantic expectancy.
- Names refer approximately to waveform polarity and latency, paradigms and populations alter them.
- Electroencephalography offers millisecond timing but imperfect source localization.
- Magnetoencephalography records magnetic fields from neuronal currents, with similar timing and demanding equipment.

Anatomy versus activity

- Structural magnetic resonance imaging shows tissue anatomy.
- Diffusion imaging measures water displacement and supports models of white-matter pathways, reconstructed tracts are not photographs of individual axons.
- Functional magnetic resonance imaging measures blood-oxygen-level-dependent contrast, an indirect vascular response to neural activity occurring over seconds.
- Task contrasts and resting-state correlations do not establish causation.
- Positron emission tomography uses radioactive tracers to investigate metabolism or molecular targets; tracer properties, radiation, cost and modelling constrain its use.

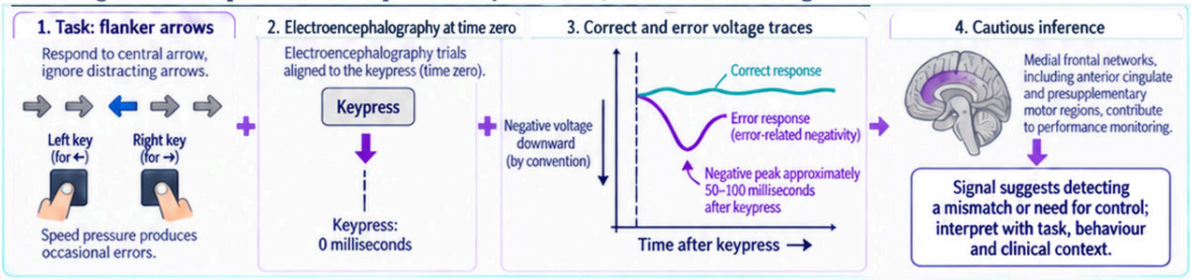
Behaviour outside the scanner

- Reaction time, accuracy, learning curves and computational models can separate candidate mechanisms such as decision speed or sensitivity to reward.
- Different mechanisms can nevertheless produce the same score.
- Eye tracking measures gaze; pupillometry, skin conductance and heart-rate variability sample arousal-related physiology without uniquely identifying an emotion.
- Ecological momentary assessment asks brief questions repeatedly in everyday life.
- Wearables and phone-derived activity patterns provide context but introduce missing data, device effects and privacy concerns.

Clinical interpretation

- Select a measure to answer a defined question; compare appropriate age, language and education norms; document sleep, substances and sensory limitations.
- Combine history, examination, functional change and validated assessment.
- Neurological investigation can identify epilepsy or structural disease when indicated.
- Most proposed psychiatric biomarkers remain research tools; an unusual trace alone does not prescribe treatment.

From signal to interpretation: complementary evidence, not automatic diagnosis



Multiple sources inform clinical interpretation



MEMORY AID Measure the signal, check the task, respect the limits.

Supplementary medical teaching • September 2026.

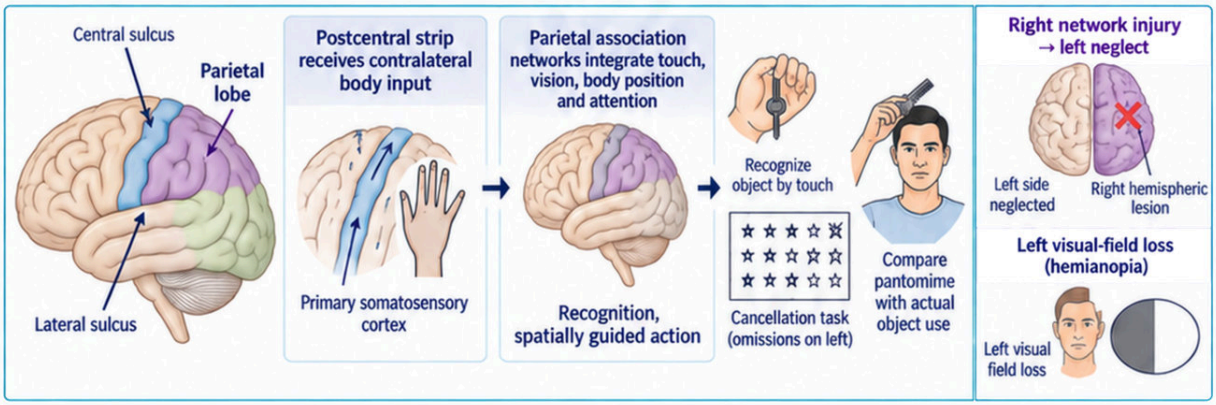
Error-related negativity and modern measurement. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Clinical neurology, headache and sleep neurology.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · C12

c12 — Parietal lobe: body, space and skilled action

Companion C12



Parietal lobe function and organization

The parietal lobe lies behind the central sulcus and above the lateral sulcus. Primary somatosensory cortex receives predominantly contralateral body information. Association networks integrate touch, vision, body position and attention so that we can recognize an object, locate ourselves in space and guide action. Dominant inferior parietal regions participate in learned actions and symbolic operations. Right-sided networks are particularly important for spatial attention, but these functions are distributed and lateralization varies.

Sensory loss versus integration failure

Primary cortical injury can impair discriminative sensation on the opposite side. Higher-order difficulty recognizing an object by touch is called astereognosis, but the term is meaningful only when basic sensation and sufficient hand movement are preserved. Failure to recognize a traced number is impaired graphesthesia. Weakness, peripheral sensory loss, unfamiliarity with the object or language difficulty can produce misleading results. Compare modalities and sides rather than declaring cortical disease from one failed item.

Neglect: a failure to attend

A right hemispheric attention-network lesion often produces left-sided neglect. The patient may omit food on the left of a plate, shave only the right face or collide with obstacles on the left. Neglect is not identical to visual-field loss: it may affect body awareness, touch, hearing, imagery and exploration. A patient can have both neglect and hemianopia. Reduced awareness of impairment, called anosognosia, can complicate safety and rehabilitation; it is not necessarily psychological denial.

Dominant parietal patterns

Difficulty writing, calculating, distinguishing fingers and identifying right from left forms the classic Gerstmann constellation. The complete pattern is uncommon and does not make localization infallible. Apraxia is difficulty performing a learned purposeful action despite adequate basic movement, comprehension and cooperation. It can reflect disruption of a broader parietofrontal network. Dressing and construction difficulties also depend on spatial processing and are not interchangeable with every form of apraxia.

Bedside HOW: sensation

1. First test light touch and joint-position sense with the patient looking away.
2. Once basic sensation is established, place a familiar key or coin in one hand with eyes closed and ask for identification; repeat on the other side.
3. Trace a number with a blunt stimulus after demonstrating the task.
4. For extinction, touch each side separately to establish detection, then both together; ask where the touch occurred.
5. Consistent omission during simultaneous stimulation suggests impaired attentional competition rather than complete primary sensory loss.

Bedside HOW: space and action

1. Ask the patient to cross out scattered targets, bisect horizontal lines and copy a simple figure; inspect omissions and spatial organization, not just the final score.
2. Use several tasks because no single test detects all neglect.
3. Observe dressing and reaching in real activity.
4. Ask the patient to demonstrate using a comb or saluting, then compare imitation and actual object use.
5. Establish comprehension and strength first.
6. Test writing, simple calculations, finger identification and right–left commands at an appropriate educational level.
7. Test visual fields independently; drawing failure may also reflect poor vision or weakness.

Conditions, investigations and care

Stroke is a major acute cause; tumour, trauma and neurodegenerative conditions can also affect parietal networks. Corticobasal syndrome may include asymmetric apraxia and cortical sensory dysfunction; posterior cortical atrophy may cause progressive spatial difficulty. These patterns require full assessment and are not diagnoses from one sign. Sudden focal dysfunction needs urgent stroke evaluation. Imaging and targeted tests investigate the cause; there is no laboratory reference interval for neglect or apraxia. Occupational therapy, structured scanning practice, environmental adaptation and caregiver teaching support function. Judge capacity and practical safety individually.

MEMORY AID

Feel, recognize, attend, act: test the missing step.

Supplementary medical teaching • September 2026

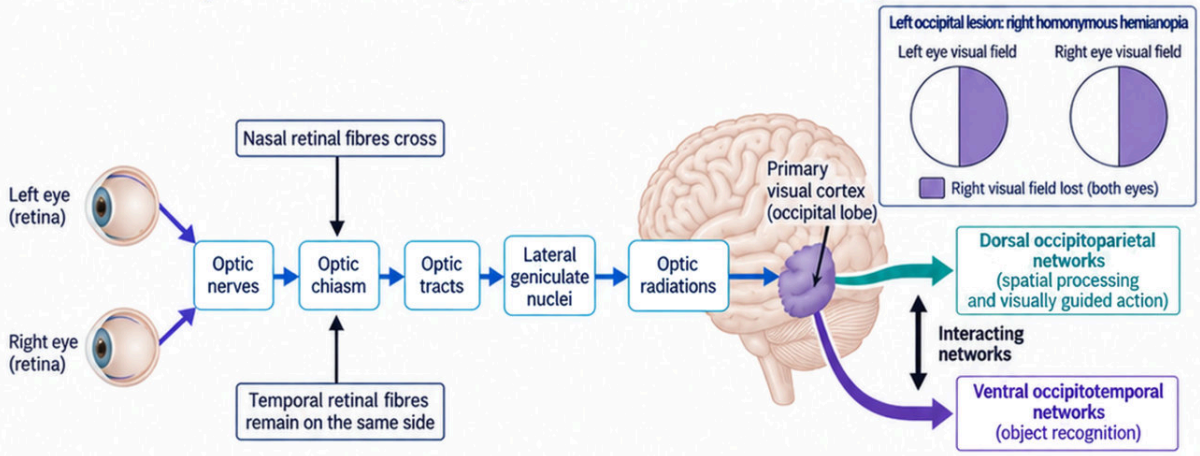
Parietal lobe functions lesions and testing. Use the labelled structures to connect anatomical location with function. Section companion: Clinical neurology, headache and sleep neurology.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · C13

c13 — Occipital lobe: seeing a field, then understanding it.

Companion C13



Anatomy and visual pathways

The occipital lobes form the posterior cerebral cortex. Retinal signals travel through the optic nerves, optic chiasm and optic tracts to the lateral geniculate nuclei, then through optic radiations to primary visual cortex. Nasal retinal fibres cross at the chiasm; temporal retinal fibres remain on the same side. Consequently, each occipital lobe represents the opposite visual hemifield from both eyes. This is a map of visual space, not simply one eye per hemisphere.

From basic vision to recognition.

Primary visual cortex processes organized visual input. Connected extrastriate regions analyse features such as form, colour and motion. Ventral occipitotemporal networks support object recognition; dorsal occipitoparietal networks support spatial processing and visually guided action. These pathways interact, and complex visual functions extend beyond the occipital lobe. Normal visual acuity does not guarantee normal visual recognition or spatial attention.

Field loss explains the behaviour.

A unilateral occipital lesion can produce contralateral homonymous **hemianopia:** loss of the same half of visual space in both eyes. A left occipital lesion therefore affects the right visual field of each eye. Macular sparing can occur but is not obligatory. Patients may miss words, collide with objects or appear inattentive. Neglect is an attentional disorder and must be tested separately; it may coexist with a field defect, Quadrantic defects can also arise in optic radiations, so they do not automatically localize to cortex.

Bilateral and association syndromes.

Bilateral primary visual cortical injury can cause cortical blindness despite preserved pupillary light reflexes because the reflex pathway branches before cortex. Some patients lack awareness of blindness and provide inaccurate visual accounts, termed Anton syndrome. This should not be mistaken automatically for deliberate fabrication. Visual agnosia means impaired recognition despite adequate elementary vision; compare recognition by touch or sound. Impaired familiar-face recognition is prosopagnosia, often involving right or bilateral occipitotemporal networks. Acquired cerebral colour-processing difficulty differs from congenital retinal colour-vision deficiency.

Positive visual symptoms.

Occipital seizures may produce brief, stereotyped visual phenomena such as coloured shapes or flashes, sometimes confined to one field. Migraine aura more often evolves gradually over minutes, but duration and pattern alone do not safely exclude other causes. Visual release hallucinations can occur with impaired vision and may have preserved insight. Hallucinations require assessment of eye disease, neurological disease, medicines and mental state; they are not automatically evidence of primary psychosis.

Bedside HOW.

Establish visual acuity with usual correction, examine pupils and eye movements, and ask about onset and persistence. For confrontation fields, sit opposite the patient, ask fixation on your nose, cover opposite eyes, and compare detection or counting of a target in each quadrant without allowing gaze shifts. Repeat the other eye. Document the pattern; formal perimetry is more sensitive and quantifies deficits. Test visual extinction using simultaneous bilateral targets only after confirming each is seen alone. Assess object matching and recognition, not naming alone; aphasia can cause naming failure. When testing faces or colours, establish adequate vision, familiarity and comprehension.

Investigate and support.

Sudden visual-field loss is a possible stroke even without weakness and needs urgent assessment. Progressive visual difficulty, recurrent spells or unexplained cortical visual signs require neurological oor ophthalmological evaluation. Magnetic resonance imaging, vascular assessment or electroencephalography are chosen according to the suspected cause. There is no laboratory threshold for cortical visual dysfunction. Treat the cause, provide visual rehabilitation and environmental adaptations, and assess reading, mobility and driving safety. Explain the visual mechanism before assigning a psychiatric label.

MEMORY AID

Check the eyes, map the field, test recognition.

Supplementary medical teaching • September 2026.

Occipital lobe functions lesions and testing. Use the labelled structures to connect anatomical location with function. Section companion: Clinical neurology, headache and sleep neurology.

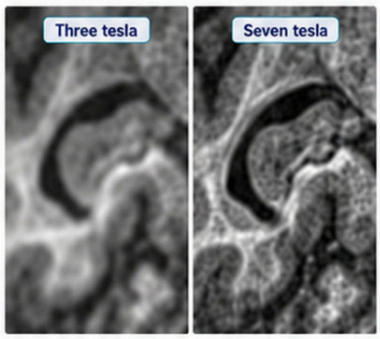
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WEAVE 100 · PAPER IV · R07

SEVEN-TESLA IMAGING AND PROTON SPECTROSCOPY

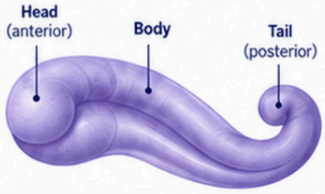
Finer structure and tissue chemistry: different questions, different limits

Seven-tesla structural imaging
Finer anatomical detail



Coronal view of the medial temporal lobe showing the hippocampus at three and seven tesla (illustrative, not from a single subject).

Hippocampus: long-axis view
Key regions and internal components

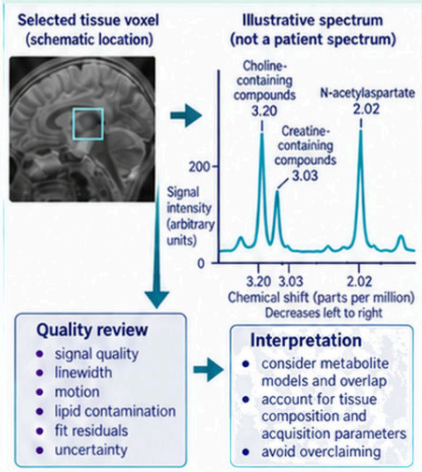


Internal components include:

- dentate gyrus
- cornu ammonis fields
- subiculum
- connected with adjacent entorhinal and parahippocampal cortices

The amygdala lies anteriorly and is a distinct structure (not shown).

From selected voxel to spectrum
Acquisition, quality review and interpretation



Quality review

- signal quality
- linewidth
- motion
- lipid contamination
- fit residuals
- uncertainty

Interpretation

- consider metabolite models and overlap
- account for tissue composition and acquisition parameters
- avoid overclaiming

WHAT THE MAGNET CHANGES

Magnetic resonance imaging uses a static magnetic field, radiofrequency excitation and spatial gradients to generate signals from tissue nuclei. Seven tesla describes field strength, not sevenfold image accuracy. Compared with lower fields, increased signal can support smaller voxels and finer detail, while altered susceptibility and relaxation affect contrast. The benefit depends on the sequence, coil, anatomy and acquisition time. No ionising radiation is used.

Higher field also brings challenges: radiofrequency-field inhomogeneity, susceptibility distortion near air–tissue interfaces, motion sensitivity and tissue-heating constraints. Implant compatibility must be checked specifically for the intended field and scanning conditions; safety at three tesla does not establish safety at seven. Expert protocols and quality review remain essential.

HIPPOCAMPUS: LOCATE BEFORE MEASURING

The hippocampus lies in the medial temporal lobe and curves from an anterior head through body to posterior tail. Its internal organisation includes the dentate gyrus, cornu ammonis fields and subiculum, connected with adjacent entorhinal and parahippocampal cortices. The amygdala lies anteriorly and is a distinct structure. These regions contribute to distributed memory networks; no single subfield is a diagnostic location for depression.

High-resolution imaging may improve the delineation of internal boundaries and subtle lesions. Segmentation divides an image into labelled regions for measurement; automated labels are estimates based on image information and an anatomical model. Different protocols may define boundaries differently. Head size, age, motion, partial-volume effects and scanner differences can influence apparent volume. A group-average difference does not classify an individual reliably.

MEMORY AID

Structure, spectrum, signal quality, then clinical significance.

HOW PROTON SPECTROSCOPY WORKS

Hydrogen nuclei in different chemical environments resonate at slightly different frequencies. Spectroscopy analyses these chemical shifts within a selected tissue volume, rather than primarily displaying anatomical contrast. After localisation, water suppression and acquisition, the time-domain signal is transformed into a frequency spectrum and fitted using metabolite models. The horizontal axis is chemical shift in parts per million; the vertical axis shows signal intensity. Approximate familiar resonances include N-acetylaspartate around 2.02, creatine-containing compounds around 3.03 and choline-containing compounds around 3.20 parts per million. Peaks may overlap: a labelled resonance is not necessarily a pure single molecule. Peak area, acquisition parameters, reference signal and tissue composition inform quantification; visual peak height alone is insufficient.

READ THE METABOLITES WITHOUT OVERCLAIMING

N-acetylaspartate is associated with neuronal metabolism and integrity. but a reduction is not simply a count of dead neurons. Choline-containing compounds relate to membrane metabolism; creatine-containing compounds relate to energy metabolism and are not an invariant denominator. Myo-inositol has osmotic and glial associations without being a cell-specific inflammation test.

Glutamate, glutamine, gamma-aminobutyric acid and glutathione can be studied with suitable sequences and fitting. Higher field can improve separation of overlapping resonances, but does not remove all technical uncertainty. Spectroscopy measures tissue metabolite pools; it does not directly measure moment-to-moment synaptic release or prove excessive excitation in an individual patient.

QUALITY COMES BEFORE BIOLOGICAL INTERPRETATION

Report voxel location, grey matter, white matter and cerebrospinal-fluid fractions, field strength, sequence, echo time and reference method. Inspect signal quality, linewidth, motion, lipid contamination, fit residuals and uncertainty. A clean-looking fitted curve can conceal poor data. Compare like protocols and account for medication, illness stage, smoking and other relevant differences before interpreting psychiatric associations.

CLINICAL USE VERSUS RESEARCH

Seven-tesla systems have authorised clinical applications in some jurisdictions; this does not authorise psychiatric diagnosis from a scan. Structural imaging answers a clinical question such as suspected neurological disease. Spectroscopy can support selected specialist neurological or tumour assessments, while psychiatric diagnosis, treatment selection and individual response prediction remain largely research applications.

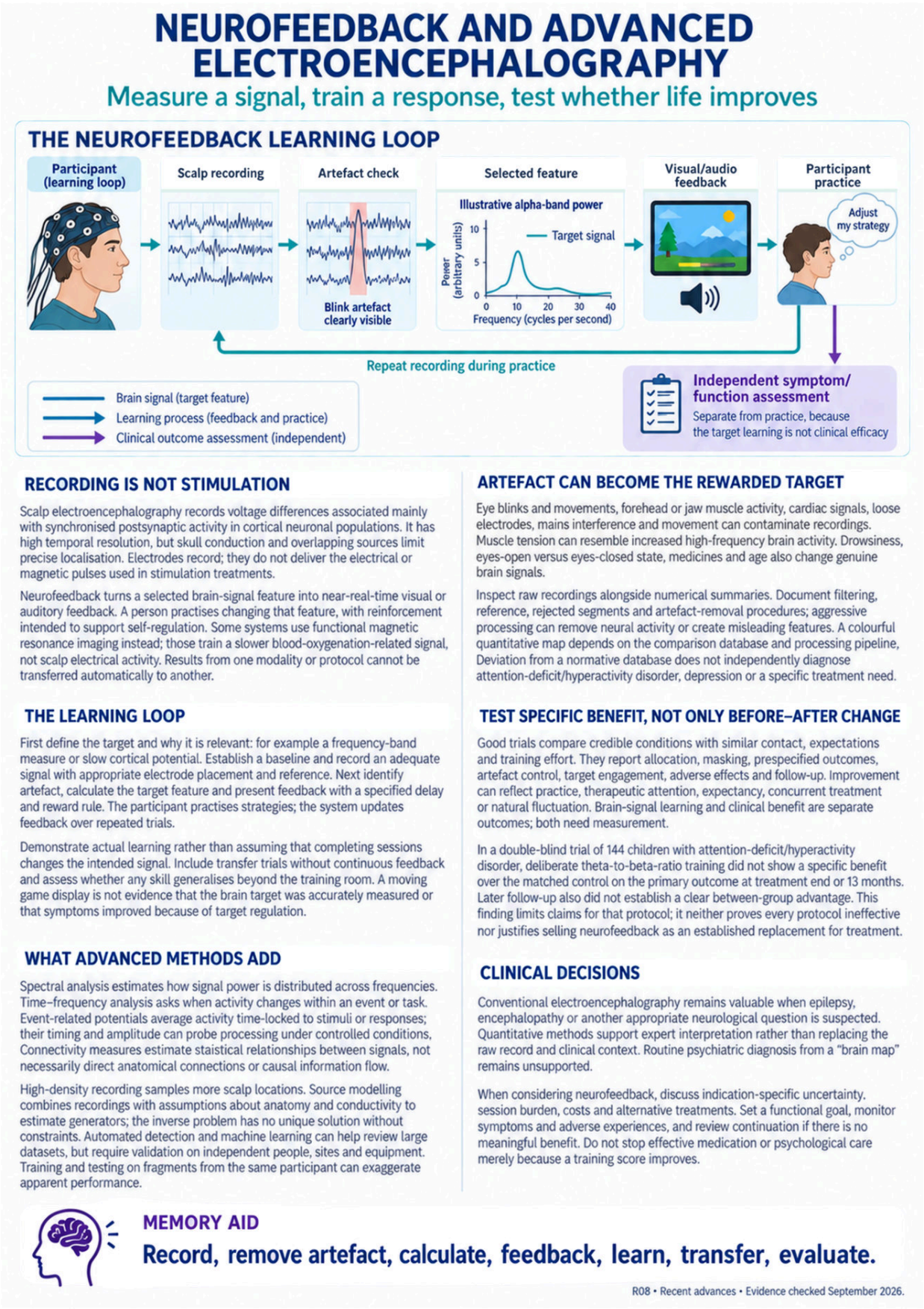
A 2025 paired three- and seven-tesla dataset with manual hippocampal subfield labels illustrates progress in measurement and validation. It does not itself establish a diagnostic biomarker. The practical question is whether the result adds reliable information that changes care beyond history, examination and established investigations.

R07 • Recent advances • Evidence checked September 2026.

Advanced neuroimaging and proton spectroscopy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Clinical neurology, headache and sleep neurology.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · R08



Neurofeedback and advanced electroencephalography. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Clinical neurology, headache and sleep neurology.

Credits and references: retained within the illustration.

AREA 05 OF 15 · P4-A07

Stroke, TBI and focal brain syndromes

8	7.9%	IV-39	IV-46
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 2 · HIGH 1 · SINGLE 5

REVISION ORDER

The five head injury slots as one block and in that order, because each adds an axis to the answer before it: manifestations, severity, prognosis, the post-concussional picture, then the lesion site. Cerebral small vessel disease next, it is where the stroke content sits. Subdural haematoma and Kluver-Bucy last, and both are self-contained.

WHAT IS IN THIS AREA

Head injury in five of the eight slots: the neuropsychiatric manifestations with their management, the severity indices with the sequelae, the definition with the prognostic factors, the post-concussional syndrome, and the personality change read against the site of the lesion. Cerebral small vessel disease and subdural haematoma carry the vascular and the surgical limb, and the Kluver-Bucy syndrome stands on its own.

IV - 39

Discuss the neuropsychiatric manifestations of head injuries and their management. [10]

10 STROKE, TBI AND FOCAL BRAIN SYNDROMES

KUHS, NTRUHS, RGUHS, TN-MGRMU - 5 APPEARANCES, 5 OF 78 SESSIONS

SKELETON

2	**Injury and severity**	Primary and secondary damage, and how severity is graded
4	**The syndromes**	Grouped by time from injury, not listed at random
3	**Management**	Non-drug first, then drugs at a lowered threshold
1	**Prognosis**	What predicts outcome, and the medicolegal line

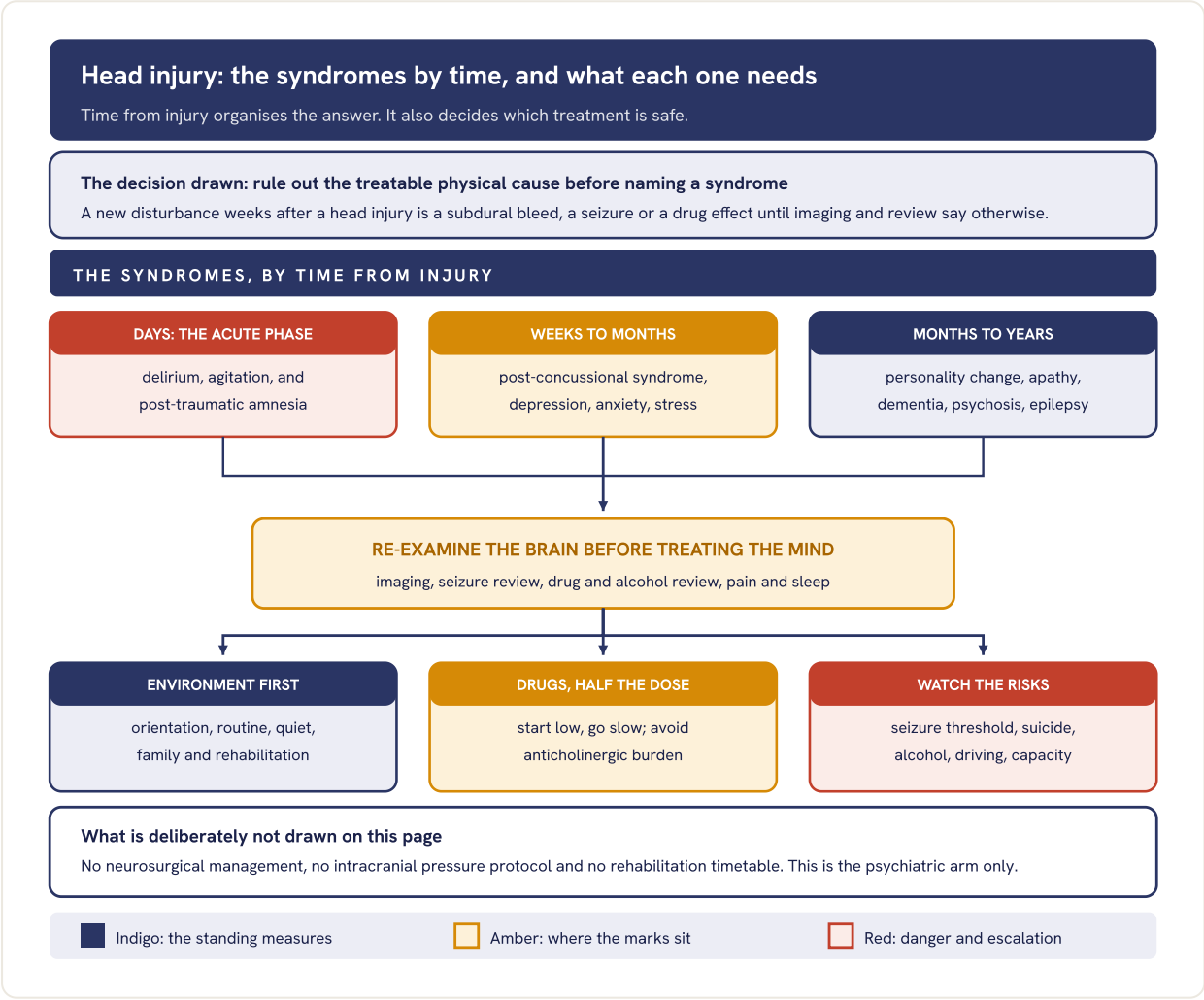


Fig 039.1 The syndromes grouped by time from injury, the physical review that must precede treatment, and the three arms of management that follow.

- **Cognitive and behavioural.** Slowed processing, poor attention and impaired executive function are the commonest and most disabling. Behavioural change follows lesion site: disinhibition and poor judgement from orbitofrontal damage, apathy from medial frontal damage, irritability and aggression from temporal damage.

- **Affective and psychotic.** Depression is the commonest psychiatric diagnosis after head injury and carries a raised suicide risk. Anxiety and post-traumatic stress disorder are common, and can coexist with amnesia for the event itself. Psychosis is uncommon, late, and more likely with temporal injury and post-traumatic epilepsy.
- **The prescribing rule.** The injured brain is more sensitive to every psychotropic. Start at roughly half the usual dose, titrate slowly, and prefer agents with low anticholinergic and low seizure liability. Benzodiazepines worsen cognition and disinhibition, so they are short term at most.

WHAT EARNS THE MARKS

- **Syndromes grouped by time from injury.** An unordered list of diagnoses reads as recall.
- **The physical review written before the psychiatric treatment.**
- **The lowered dosing threshold, stated as a rule** rather than left implied.
- **Depression named as the commonest,** with its suicide risk.

SEE ALSO Slot IV - 42 traumatic brain injury and prognosis Slot IV - 46 personality change by lesion site **SPRINT** Day 34, Stroke, TBI, delirium & focal brain syndromes

IV - 40

How will you quantify the severity of head injury? Discuss neuropsychiatric sequelae of head injury. [4+6]

4 + 6 | STROKE, TBI AND FOCAL BRAIN SYNDROMES

DNB, KNRUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

4	Quantifying severity	Three indices, each measuring a different thing
3	Early sequelae	Delirium, amnesia, the post-concussional cluster
2	Late sequelae	Cognitive, affective, behavioural, epileptic
1	The link	Which sequelae severity predicts, and which it does not

INDEX	MILD	MODERATE	SEVERE
Glasgow Coma Scale, at presentation	13 to 15	9 to 12	8 or below
Loss of consciousness	Under 30 minutes	30 minutes to 24 hours	Over 24 hours
Post-traumatic amnesia	Under 24 hours	1 to 7 days	Over 7 days
Structural imaging	Usually normal	Often abnormal	Abnormal

Amber marks post-traumatic amnesia, the single best predictor of outcome. Quoting all three indices, and saying which one predicts best, is what the four marks are for.

- **The three measure different things.** The coma scale is a snapshot at presentation and is degraded by alcohol, sedation and intubation. Post-traumatic amnesia is measured forwards, as the interval until continuous day to day memory returns, and it predicts outcome better than the admission score.
- **The sequelae, by domain.** Cognitive: attention, processing speed and executive function. Affective: depression is the commonest diagnosis, with anxiety and post-traumatic stress disorder. Behavioural: irritability, aggression, disinhibition, apathy. Late and uncommon: psychosis, and post-traumatic epilepsy.
- **The asymmetry.** Severity predicts cognitive impairment and epilepsy well, but post-concussional symptoms and depression follow mild injury just as often as severe. A normal scan and a good coma score never promise a good psychiatric outcome.

WHAT EARNS THE MARKS

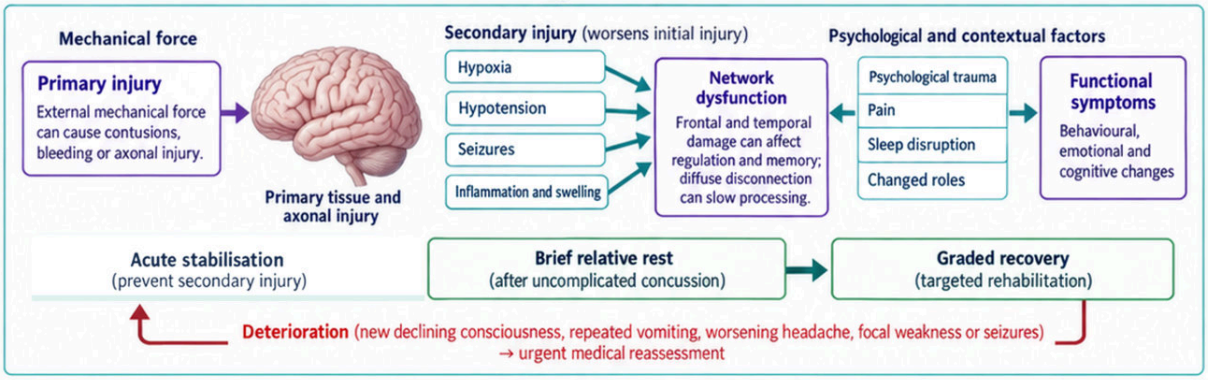
- **All three indices, with their bands.** Quoting the coma scale alone answers a quarter of the question.
- **Post-traumatic amnesia defined properly,** as the return of continuous memory rather than as confusion.
- **Sequelae grouped by domain,** and the honest line that mild injury still produces them.

SEE ALSO Slot IV - 42 traumatic brain injury and prognosis Slot IV - 44 post-concussional syndrome SPRINT Day 34, Stroke, TBI, delirium & focal brain syndromes

WEAVE 100 · PAPER IV · C09

c09 – Traumatic brain injury: psychiatric and behavioural assessment

Companion C09



Background and mechanisms

Brain function depends on connected networks, intact axons and stable oxygen and energy delivery. External mechanical force can cause contusions, bleeding or axonal injury. Secondary hypoxia, hypotension, seizures, inflammation and swelling may worsen the initial injury. Frontal and temporal damage can affect regulation and memory; diffuse disconnection can slow processing. Psychological trauma, pain, sleep disruption and changed roles also shape symptoms. A normal routine scan does not exclude concussion, and persistent symptoms do not prove continuing structural damage.

Common later presentations

Depression, anxiety and posttraumatic stress symptoms can coexist with the neurological injury. Irritability, impulsivity and poor frustration tolerance may reflect impaired regulation, overload or unmet needs. Apathy is reduced initiation and differs from depressed mood. Headache, dizziness, fatigue, poor sleep and concentration problems can form persistent postconcussion symptoms. These symptoms are real but nonspecific: assess vestibular dysfunction, migraine, sleep disorder, medicines and mood. Mania or psychosis is less frequent and requires careful differential diagnosis. No single personality pattern proves traumatic brain injury.

Describe severity accurately.

Acute classification uses the Glasgow Coma Scale after resuscitation, duration of loss of consciousness and duration of posttraumatic amnesia. Typical mild ranges are a score of 13–15, loss of consciousness up to 30 minutes and amnesia no longer than 24 hours. Sedation, intoxication and other injuries can confound scoring. Document the most concerning reliable feature rather than averaging incompatible categories. Acute severity informs risk but does not fully predict an individual's later psychiatric difficulties. Record prior injuries and the chronology of recovery.

Bedside HOW.

Reconstruct the event, acute records, loss of consciousness and first continuous memories, using an informant where available. Establish premorbid functioning, mood, substance use and employment demands. Assess orientation and attention before memory; then examine processing speed, executive control, language and neurological signs. Ask for practical examples: missed medicines, unsafe cooking, angry outbursts or inability to resume work. Screen mood, trauma symptoms, alcohol use and suicide risk. Formal neuropsychological testing can characterize persistent functional difficulty; interpret performance with language, education, effort, pain and fatigue. One low score does not establish cause, and compensation or litigation does not establish malingering.

Early confusion is not automatically a primary psychiatric illness.

During posttraumatic amnesia, the person may be disoriented, unable to form continuous new memories, restless or agitated. Pain, infection, withdrawal, sleep disruption or seizures can worsen this state. New declining consciousness, repeated vomiting, worsening headache, focal weakness or seizures needs urgent medical reassessment. Do not label unexplained deterioration as "behavioural." Hypoactive confusion can be missed because the patient is quiet.

Management matches the phase.

Acute care prevents secondary injury. After uncomplicated concussion, brief relative rest is followed by gradual, symptom-guided return to ordinary activity; prolonged strict inactivity is generally unhelpful. Avoid another head injury, and obtain appropriate clearance before contact sport or hazardous work. Provide a clear explanation, written pacing strategies, sleep regularity and targeted headache or vestibular treatment. Rehabilitation can teach external memory aids, task simplification and planned breaks. Treat depression, anxiety and posttraumatic stress with adapted evidence-based psychological and medication approaches.

For agitation, first reduce overstimulation, provide orientation, identify triggers and treat medical causes. Medication, when necessary, requires individualized specialist review because sedation and cognitive adverse effects can obstruct recovery. Start cautiously and review benefit against harm. Support caregivers, relationships and return-to-work planning. Reassess driving, capacity and safeguarding individually. There is no laboratory reference range or routine biomarker that confirms the cause of every persistent symptom.

MEMORY AID

Check deterioration, establish the timeline, treat the contributors.

Supplementary medical teaching • September 2026

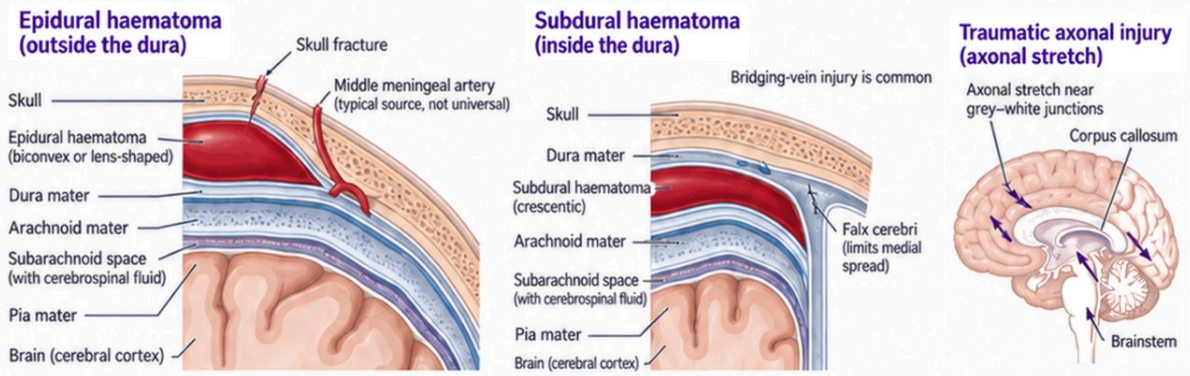
Traumatic brain injury psychiatric aspects. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-40.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · C18

C18 — Epidural versus subdural haematoma

Clinical comparison 18 • Epidural versus subdural, with traumatic axonal injury



NORMAL LAYERS AND PRESSURE

From outside inward: skull → dura mater → arachnoid mater → cerebrospinal-fluid-containing subarachnoid space → pia mater on the brain. Intracranial epidural and subdural collections separate tissues that normally lie closely together. The rigid skull has limited reserve: expanding blood or swelling can reduce cerebral perfusion and displace brain tissue. A small initial collection can enlarge; appearance at one time is not a guarantee of stability.

EPIDURAL: OUTSIDE THE DURA

Blood collects between skull and dura, often after a skull fracture injures a meningeal vessel. The middle meningeal artery is the classic source, especially with temporal injury, but venous sources also occur. Expansion can be rapid.

Computed tomography typically shows a biconvex or lens-shaped extra-axial collection. Dural attachment at cranial sutures usually limits spread across them. These are typical features, not absolute rules for every location or injury.

A lucid interval—initial impaired consciousness, apparent recovery, then deterioration—is memorable but not required. Headache, vomiting, declining responsiveness, new focal weakness or an enlarging pupil can signal dangerous expansion. Absence of a remembered loss of consciousness does not exclude it.

SMALL BOX: TRAUMATIC AXONAL INJURY

Rapid rotational/acceleration forces can stretch and disrupt axons, particularly near grey–white junctions, corpus callosum and brainstem. Diffuse axonal injury is the traditional term.

Impaired consciousness can exceed the visible computed-tomography abnormality; magnetic resonance imaging is more sensitive to many lesions. It can coexist with haematoma.

Management supports physiology and prevents secondary injury; there is no single clot to drain. Imaging distribution contributes to prognosis but cannot determine one person's outcome alone.

MEMORY AID

Locate the blood, watch the patient, escalate deterioration.

SUBDURAL: INSIDE THE DURA

Blood collects between dura and arachnoid, commonly after bridging-vein injury; cortical vessel injury can also contribute. Acute subdural bleeding may accompany substantial underlying brain injury and can deteriorate rapidly despite a venous source.

Computed tomography typically shows a crescentic collection that can cross sutures but is constrained by dural reflections such as the falx and tentorium. It may extend along these reflections. Acute blood is usually relatively dense; chronic fluid is often less dense, and rebleeding can produce mixed density. Density alone does not precisely date an injury.

Brain atrophy stretches bridging veins and increases vulnerability: older adults, people with recurrent falls and those taking anticoagulants are important risk groups. Chronic collections can become symptomatic weeks after a minor or unremembered injury through membrane formation, inflammation and recurrent bleeding.

Headache, gait decline, cognitive change, fluctuating confusion or weakness may be the presentation. Do not label every such change as dementia progression.

SHARED EMERGENCY PATHWAY

Stabilise airway, breathing and circulation, protect the spine where indicated, prevent hypoxia/hypotension and check glucose.

Record serial Glasgow Coma Scale components, pupil size/reactivity, limb findings and vital signs.

Obtain urgent noncontrast head computed tomography when indicated; review anticoagulant/antiplatelet exposure and clotting tests.

Coordinate reversal and neurosurgical care under the relevant protocol.

A worsening examination outranks reassurance from an earlier scan.

HOW SPECIALISTS DECIDE

Brain Trauma Foundation surgical guidance gives examples for acute lesions: epidural volume greater than 30 cubic centimetres generally requires evacuation regardless of coma score; acute subdural thickness greater than 10 millimetres or midline shift greater than 5 millimetres generally requires evacuation. These are decision thresholds, not safe limits below which deterioration can be ignored. Smaller lesions with neurological decline, abnormal pupils or raised intracranial pressure may also need surgery.

Selected stable small collections may undergo close specialist observation and repeat imaging. Acute evacuation and chronic drainage use different approaches; treatment depends on the whole picture. Chronic-subdural management may include selected endovascular strategies, but these do not replace urgent decompression when mass effect threatens life.

Supplementary medical teaching • September 2026

Epidural subdural and diffuse axonal injury. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-40.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · F19

HEAD INJURY AND SUBDURAL BLEEDING

Foundation 19 • Normal function → mechanism → clinical recognition → management

NORMAL PROTECTION AND PRESSURE

The skull encloses brain tissue, blood and cerebrospinal fluid. The dura mater is the tough outer meningeal layer; the arachnoid lies beneath it, and the pia closely covers the brain. Cerebrospinal fluid occupies the subarachnoid space. Bridging veins carry blood from the brain surface to dural venous sinuses. Because the adult skull is rigid, expanding blood or swelling eventually raises intracranial pressure and reduces cerebral perfusion. Compensation is limited. Typical supine adult intracranial pressure is about 5–15 millimetres of mercury; interpretation depends on measurement and clinical circumstances, and routine minor head injury does not require invasive monitoring.

PRIMARY AND SECONDARY INJURY

Primary injury occurs at impact: contusion, axonal stretching or tearing, vascular disruption and tissue damage. Acceleration–deceleration and rotation can cause injury without a visible skull fracture. Concussion is a mild traumatic brain injury with transient disturbance of brain function; loss of consciousness is not required, and computed tomography may be normal.

Secondary injury develops afterwards through hypoxia, hypotension, swelling, seizures, inflammation or expanding bleeding. Preventing these processes is a major treatment goal. A normal initial scan does not eliminate every cause of later deterioration.

WHERE IS THE BLOOD?

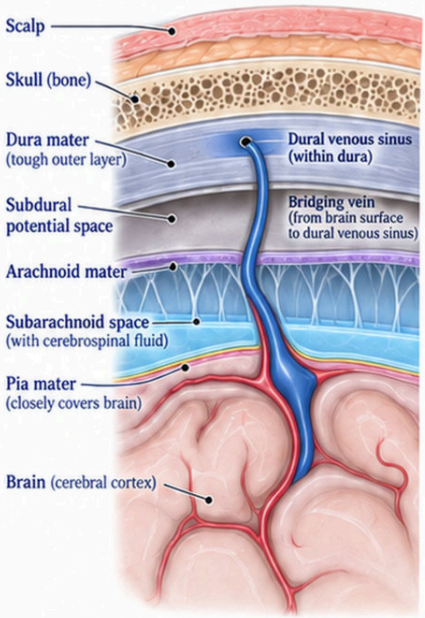
Epidural: between skull and dura, often lens-shaped on computed tomography.

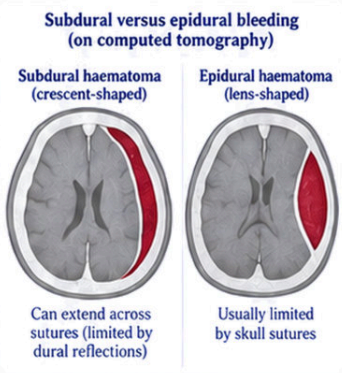
Subdural: between dura and arachnoid, usually crescent-shaped; bridging-vein injury is common, although other vessels may contribute.

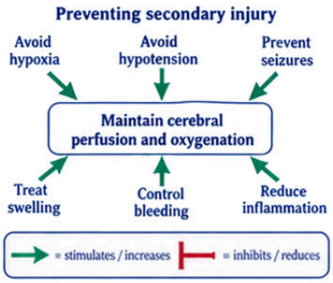
Subarachnoid: within the cerebrospinal-fluid space.

Intracerebral: within brain tissue.

Subdural blood can extend across sutures but is limited by dural reflections. Acute subdural bleeding may rapidly compress the brain. Chronic collections develop over weeks through inflammatory membranes and recurrent bleeding; older age, cerebral atrophy, anticoagulation and alcohol-related risk increase susceptibility. The initiating injury can be minor or forgotten.







RECOGNISE DETERIORATION

Headache, vomiting, confusion, reduced consciousness, new weakness, speech disturbance, seizures or unequal pupils demand reassessment. Chronic subdural haematoma may present with gait decline, slowed thinking, fluctuating confusion or a focal deficit rather than a dramatic trauma history. Herniation means brain displacement across rigid intracranial boundaries; it can be fatal. Do not attribute new behavioural or cognitive change to a psychiatric illness before evaluating neurological causes.

MEASURE AND INVESTIGATE

The **Glasgow Coma Scale** records eye opening, verbal response and motor response, summing to 3–15. After initial resuscitation, scores 13–15 are commonly termed mild, 9–12 moderate and 3–8 severe; sedation, intoxication, language and intubation can confound scoring. Report component scores and trends, not just the total.

Assess airway, breathing, circulation, pupils, focal signs and possible cervical injury. Urgent computed tomography is indicated by risk features such as low or declining consciousness, focal deficit, seizure, signs of skull fracture or repeated vomiting. Anticoagulation, age and mechanism influence imaging decisions. Follow an age-appropriate validated guideline; children have different thresholds. Check coagulation and blood count when relevant.

MANAGEMENT FLOW

Stabilise airway and cervical spine precautions when indicated → prevent hypoxia and hypotension → urgent imaging and neurosurgical discussion for significant bleeding or deterioration.

Reverse anticoagulation promptly when clinically indicated under the relevant protocol.

Large or symptomatic subdural collections may need surgical evacuation; selected small stable collections can be monitored with explicit clinical and imaging follow-up. Decisions use symptoms, scan findings and trajectory, not age alone.

Chronic subdural management may include specialised endovascular options in selected settings.

After concussion, give clear warning signs and a gradual, symptom-guided return to activity; avoid premature return to contact sport.

Persistent symptoms require reassessment and targeted rehabilitation.

Do not discharge solely because one consciousness score or scan appears reassuring.

MEMORY AID

Find the compartment, prevent secondary injury, watch for deterioration.

Foundation 19 • Medical foundations • Source-checked supplementary teaching • September 2026.
Laboratory ranges and treatment protocols require local context.

Head injury and subdural bleeding. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-40.

Credits and references: retained within the illustration.

IV - 41

Describe presentation of cerebral small vessel disease in psychiatry. How to manage such a case? [4+6]

4 + 6 | STROKE, TBI AND FOCAL BRAIN SYNDROMES | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	The vessel, the lesions, and what imaging shows
3 Presentation	Vascular depression, slowing, gait, and the cognitive profile
4 Management	The vessels first, then mood, then safety
1 Course	Stepwise decline, and what the family is told

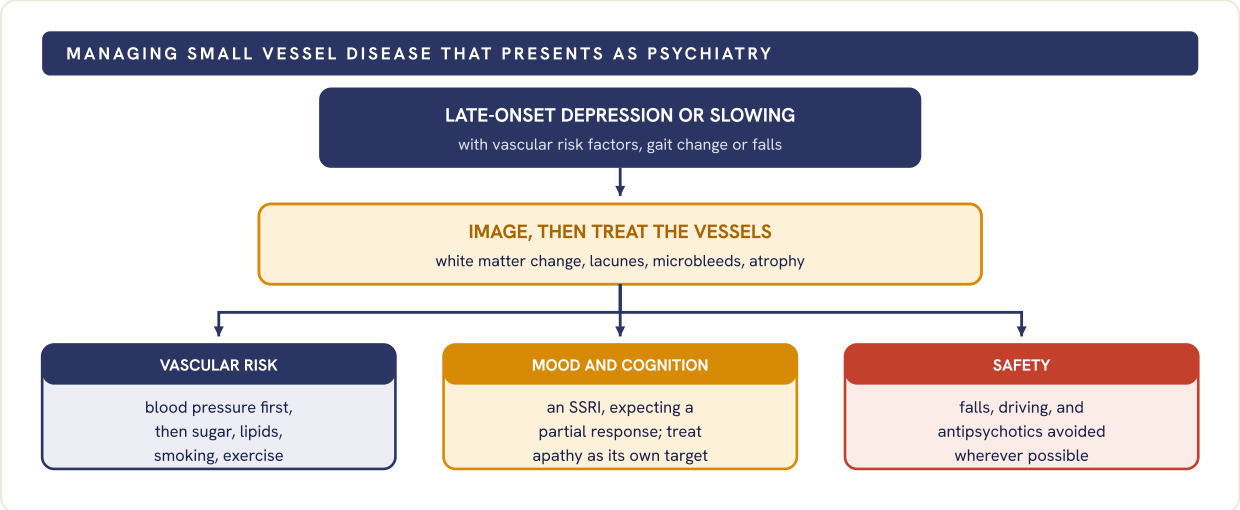


Fig 041.1 One presentation, one confirmatory step, and three arms of management with the vessels treated before the mood.

- **The presentation.** Late-onset depression with prominent apathy and psychomotor slowing, executive and processing-speed impairment with memory relatively spared, unsteady small-stepped gait, urinary urgency and emotional lability. Psychosis is uncommon and late.
- **Vascular depression.** The concept links late-onset depression, executive dysfunction and white matter disease, and it predicts a slower and less complete antidepressant response. Naming it, and saying what it predicts, is the mark.

PITFALL

Antipsychotics raise the risk of stroke and death in older people with vascular brain disease, so agitation is managed by environment and by treating pain, constipation and infection before any drug. Tricyclics are avoided for anticholinergic burden and postural drop, which turns into falls.

WHAT EARNS THE MARKS

- **Blood pressure named as the first treatment,** ahead of any psychotropic.
- **The cognitive profile stated as executive and speed,** not as amnesia.
- **The partial antidepressant response predicted in advance,** so it is not read as treatment failure.

SEE ALSO Slot IV - 52 depression after stroke Slot IV - 19 subcortical dementias SPRINT
Day 34, Stroke, TBI, delirium & focal brain syndromes

WEAVE 100 · PAPER IV · F18

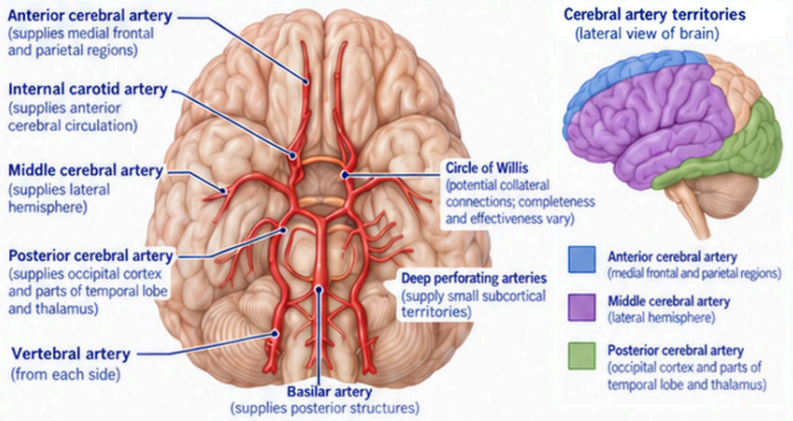
CEREBRAL CIRCULATION, STROKE AND SMALL-VESSEL DISEASE

Foundation 18 • Normal function → mechanism → clinical recognition → management

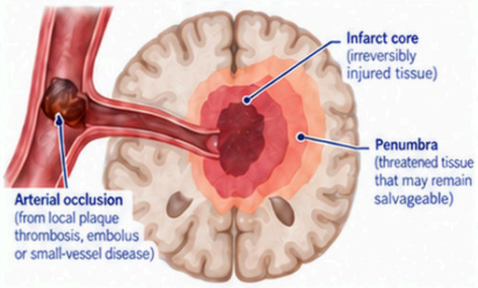
NORMAL BRAIN PERFUSION

Internal carotid arteries supply most anterior cerebral circulation; vertebral arteries join to form the basilar artery supplying posterior structures. The circle of Willis offers potential collateral connections, but completeness and effectiveness vary. The anterior cerebral artery mainly supplies medial frontal and parietal regions; the middle cerebral artery much of the lateral hemisphere; the posterior cerebral artery the occipital cortex and parts of the temporal lobe and thalamus. Deep perforating arteries supply small subcortical territories. Brain function requires continuous oxygen and glucose delivery. Autoregulation adjusts vascular resistance as perfusion pressure changes, within limits that shift with disease.

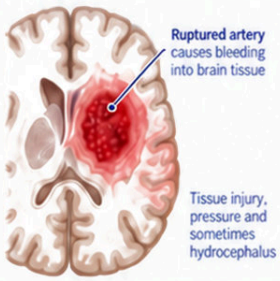
Arterial supply and circle of Willis



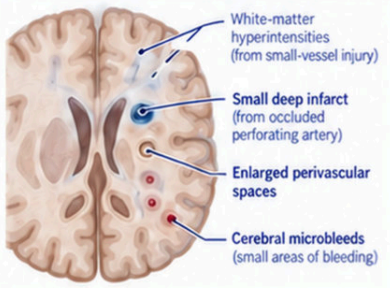
Arterial occlusion → core and penumbra



Vessel rupture → haemorrhage



Small-vessel disease → white-matter injury



WHAT GOES WRONG

Ischaemic stroke results from arterial blockage and inadequate perfusion. Local plaque thrombosis, emboli from the heart or proximal arteries, and small-vessel occlusion are major mechanisms. The infarct core is irreversibly injured tissue; the surrounding penumbra is threatened tissue that may remain salvageable. **Haemorrhagic stroke** results from vessel rupture, causing tissue injury, pressure and sometimes hydrocephalus. **Transient ischaemic attack** produces transient focal neurological dysfunction from ischaemia without acute infarction; resolved symptoms still require urgent assessment. Intracerebral and subarachnoid haemorrhage are distinct bleeding patterns.

SMALL VESSELS, CUMULATIVE INJURY

Ageing, hypertension, diabetes and other mechanisms injure perforating arteries, arterioles and capillaries. Imaging may show small deep infarcts, white-matter hyperintensities, microbleeds or enlarged perivascular spaces. These findings differ in mechanism and significance. Accumulated injury can disrupt networks, slowing processing and affecting gait, balance or executive function. Incidental white-matter changes do not alone establish vascular dementia; relate them to symptoms, functional change and alternative explanations. Cerebral amyloid angiopathy is another important small-vessel process, particularly associated with lobar bleeding.

TREAT AND PREVENT

Activate stroke services immediately. Eligible ischaemic stroke may receive intravenous thrombolysis within 4.5 hours; selected large-vessel occlusions may undergo thrombectomy, including selected patients up to 24 hours based on imaging and clinical criteria. Selected patients with unknown onset or beyond 4.5 hours may also qualify for imaging-guided thrombolysis under specialist protocols; never self-exclude from emergency assessment. Bleeding must be excluded before ischaemic-stroke antithrombotic decisions. **Haemorrhage** requires haemorrhage-specific blood-pressure, anticoagulant-reversal and neurosurgical assessment. Screen swallowing before oral intake. Provide early organised rehabilitation and prevent complications. Secondary prevention follows mechanism: antiplatelet treatment for suitable non-cardioembolic disease; anticoagulation for appropriate atrial-fibrillation-related disease after individualised timing; lipid lowering and risk-factor control. Smoking cessation, activity and adherence remain central. New or recurrent focal symptoms are an emergency, even when they resolve.

ANATOMY → CLINICAL FINDING

Sudden face or arm weakness, speech disturbance, visual loss, imbalance or sensory loss suggests stroke. Dominant middle cerebral territory injury may cause aphasia, an impairment of language; non-dominant injury may cause neglect, failure to attend to one side. Anterior cerebral injury often affects the opposite leg more than arm. Posterior cerebral injury may cause opposite visual-field loss. Brainstem or cerebellar stroke may cause diplopia, dysarthria, severe imbalance or crossed signs. Not every stroke follows a perfect territory pattern. Thunderclap headache, vomiting or reduced consciousness raises concern for haemorrhage but clinical signs alone cannot reliably distinguish bleeding from ischaemia.

URGENT INVESTIGATION

Record the exact last-known-well time → check glucose to exclude a mimic → neurological assessment and urgent brain imaging. Non-contrast computed tomography rapidly detects haemorrhage; early ischaemic stroke may initially be invisible. Angiography identifies large-vessel occlusion; perfusion imaging or magnetic resonance imaging can guide selected reperfusion decisions. Check blood count, coagulation, kidney function and cardiac rhythm. Atrial fibrillation can produce emboli. No universal normal scan or blood value excludes acute stroke. Blood-pressure targets vary by stroke type and reperfusion plan; do not reflexively normalise pressure without the stroke pathway.

MEMORY AID

Sudden focal change needs urgent imaging, even if it resolves.

Foundation 18 • Medical foundations • Source-checked supplementary teaching • September 2026.
Laboratory ranges and treatment protocols require local context.

Cerebral circulation stroke and small-vessel disease. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-41.

Credits and references: retained within the illustration.

IV - 42

What is traumatic brain injury? What are the prognostic factors that determine outcome of traumatic brain injury? Discuss the psychiatric sequelae of head injury. [2+3+5]

2 + 3 + 5 | STROKE, TBI AND FOCAL BRAIN SYNDROMES

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Definition**	What counts as traumatic brain injury, and the two damage phases
3 **Prognostic factors**	Before, during and after the injury
4 **Psychiatric sequelae**	By domain, with the commonest named as commonest
1 **What follows**	Rehabilitation, review, and the capacity question

THE DEFINITION

An alteration in brain function, or other evidence of brain pathology, caused by an external mechanical force. Primary damage is done at impact by contusion, laceration and diffuse axonal injury; secondary damage follows over hours from hypoxia, hypotension, oedema, haematoma and raised intracranial pressure, and it is the part that treatment can still prevent.

GROUP	FACTORS THAT WORSEN OUTCOME	WHY IT MATTERS
Before the injury	Older age, previous head injury, prior psychiatric illness, alcohol and substance use, low education and cognitive reserve	Fixed, but identifies who to follow
The injury itself	Low coma score, long post-traumatic amnesia, hypoxia and hypotension, haematoma, focal frontal or temporal lesion, seizures	**The strongest single group**
After the injury	Unemployment, poor family support, chronic pain, litigation, delayed or absent rehabilitation	**The group that is modifiable**

Amber marks the two rows worth arguing about: injury factors predict most strongly, but post-injury factors are the only ones treatment can change.

- **The sequelae.** Cognitive impairment of attention, speed and executive function; depression as the commonest diagnosis, with raised suicide risk; anxiety and post-traumatic stress disorder; personality change by lesion site; aggression and irritability; and late, uncommon, psychosis and post-traumatic epilepsy.
- **The honest qualifier.** Outcome is determined as much by what surrounds the patient as by what happened to the brain. Saying so, and naming rehabilitation and family support as treatment, is what lifts this answer.

WHAT EARNS THE MARKS

- **Primary and secondary damage separated in the definition.** Two marks are cheaply lost here.
- **Prognostic factors grouped into before, during and after.** An unsorted list scores lower than the same facts sorted.
- **Depression named as the commonest sequela,** with its suicide risk stated.

SEE ALSO Slot IV - 40 quantifying severity of head injury Slot IV - 39 management of head injury **SPRINT** Day 34, Stroke, TBI, delirium & focal brain syndromes

IV - 43

Subdural hematoma. [10]

10 STROKE, TBI AND FOCAL BRAIN SYNDROMES

KUHS, NTRUHS - 3 APPEARANCES, 3 OF 78 SESSIONS

SKELETON

2	What it is	Bridging veins, the subdural space, acute and chronic
2	Who gets it	The psychiatric populations that are already at risk
3	How it presents	The masquerade: dementia, depression, delirium
3	Diagnosis and management	Image early, refer early, and what happens after

THE DEFINITION

Bleeding into the space between dura and arachnoid, classically from torn bridging veins. Because the source is venous and low pressure, the collection can enlarge slowly, and the interval between the injury and the presentation may be long enough that neither patient nor family connects the two.

FEATURE	ACUTE	CHRONIC
Time from injury	Within three days	Three weeks or more
Typical patient	Any age, significant trauma	Older, alcohol dependent, on anticoagulants, epileptic
The injury	Always remembered	**Often trivial, and frequently not recalled at all**
Appearance on scan	**Crescentic, bright, crossing suture lines**	**Crescentic, dark or mixed, crossing suture lines**
Presentation	Falling consciousness, focal signs	Fluctuating confusion, headache, gait change, personality change
Management	Urgent neurosurgical referral	Referral; small collections may be observed

Amber cells hold the two discriminators. A crescentic collection that crosses suture lines is subdural; a lens-shaped one that does not is extradural. The forgotten injury is what makes the chronic form a psychiatric problem.

- **Why psychiatrists must know it.** The chronic form imitates whatever the patient is already thought to have. It presents as new or worsening dementia, as late-onset depression with apathy, or as unexplained delirium, and the populations at highest risk are exactly ours: older patients, alcohol dependence, falls, epilepsy and anticoagulation.
- **The rule that prevents the miss.** Any patient with fluctuating consciousness, a new focal sign, a headache that is new in character, or a cognitive decline that is faster than the diagnosis should allow, gets imaged. Attributing sudden change to a known chronic illness is how this is missed.

WHAT EARNS THE MARKS

- **Acute and chronic separated,** since they differ in patient, presentation and urgency.
- **The forgotten or trivial injury named.** It is the whole reason the diagnosis is delayed.
- **The imaging rule written as a threshold,** not as a general recommendation to investigate.

SEE ALSO Slot IV - 86 neurological complications of alcohol Slot IV - 92 delirium **SPRINT**

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

IV - 44

Post concussional Psychiatric manifestation. [10]

10 STROKE, TBI AND FOCAL BRAIN SYNDROMES

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition	What the syndrome is, and what it follows
3	The three clusters	Somatic, cognitive, affective, each named
2	Why it persists	What predicts persistence, and what does not
3	Management	Explain early, grade the return, treat what is treatable

THE DEFINITION

A cluster of symptoms following head injury, usually mild, with brief or no loss of consciousness. The classification asks for a history of head trauma together with three or more of headache, dizziness, fatigue, irritability, insomnia, difficulty with concentration or memory, and reduced tolerance of alcohol, stress or emotion.

- **The three clusters.** Somatic: headache, dizziness, fatigue, sensitivity to noise and light. Cognitive: poor attention, slowed processing, forgetfulness that is worse under pressure. Affective: irritability, anxiety, low mood, emotional lability, and preoccupation with the symptoms themselves.
- **Course and what predicts persistence.** Most patients recover within weeks to about three months. Persistence beyond that is predicted less by the severity of the injury than by prior psychiatric history, the expectation of harm, misattribution of ordinary symptoms to brain damage, ongoing litigation, and unemployment.
- **Management.** Explain early, and give a positive and specific prognosis. Advise graded return to activity rather than prolonged rest, treat headache and sleep directly, treat depression and anxiety on their own merits, and offer cognitive behavioural therapy where symptoms persist. Stop repeating normal investigations.

PITFALL

These symptoms are non-specific. They occur in healthy controls and after orthopaedic injury without any head trauma at all. Attributing every symptom to brain damage and dismissing them all as compensation seeking are the same error made in opposite directions, and both lose the patient.

WHAT EARNS THE MARKS

- **The criterion set, with the number of symptoms required.**
- **Persistence explained by maintaining factors,** not by injury severity.
- **Graded return named instead of rest.** Prolonged rest worsens this syndrome.
- **The even-handed line on litigation.** It is a maintaining factor, not a verdict on the patient.

SEE ALSO Slot IV - 40 quantifying severity of head injury Slot IV - 42 traumatic brain injury and prognosis **SPRINT** Day 34, Stroke, TBI, delirium & focal brain syndromes

IV - 45

Kluver-Bucy syndrome. [10]

10 STROKE, TBI AND FOCAL BRAIN SYNDROMES

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Definition and lesion	Where it was described, and what has to be damaged
4 The features	Named, each with what it looks like at the bedside
2 Causes	What actually produces it in human beings
2 Management	Treat the cause, contain the behaviour, support the family

THE DEFINITION

A behavioural syndrome that follows bilateral damage to the anterior temporal lobes, and to the amygdala in particular. Described by Kluver and Bucy in rhesus monkeys after bilateral temporal lobectomy. In human beings the complete syndrome is rare and the partial form is the rule, which is worth saying.

FEATURE	WHAT IT MEANS	HOW IT APPEARS
Visual agnosia	Failure to recognise objects by sight, with vision intact	Handles a familiar object as if it were new
Hyperorality	Examining everything by mouth	Mouthing objects, eating inedible things
Hypermetamorphosis	Compulsion to attend to every visual stimulus	Touches whatever enters the field of view
Placidity	Loss of fear and of anger	Flat, unreactive to threat or to rebuke
Altered sexual behaviour	Increased and indiscriminate	Public masturbation, inappropriate approaches
Dietary change	Hyperphagia and altered preference	Eats continuously, loses food preferences

Amber marks the two features candidates most often omit. Listing hyperorality and hypersexuality alone is the half answer that this question is set to catch.

- **Causes in practice.** Herpes simplex encephalitis is the commonest, because it targets the temporal lobes bilaterally. Also head injury, bilateral temporal lobectomy for epilepsy, frontotemporal dementia, anoxic brain injury and late Alzheimer disease.
- **Management.** Treat the underlying cause where it is treatable, and treat encephalitis urgently. Beyond that the work is behavioural: supervision, environmental control, protecting dignity, and family support. Selective serotonin reuptake inhibitors or a mood stabiliser may reduce disinhibition and sexual behaviour.

WHAT EARNS THE MARKS

- **The lesion named as bilateral and anterior temporal,** with the amygdala specified.
- **All the features, including visual agnosia and hypermetamorphosis.**
- **Herpes encephalitis named as the commonest human cause.**
- **The line that the human form is usually partial.** It shows the syndrome is understood, not recited.

SEE ALSO Slot IV - 46 personality change by lesion site Slot IV - 13 frontotemporal dementia SPRINT Day 34, Stroke, TBI, delirium & focal brain syndromes

WEAVE 100 · PAPER IV · C19

C19 — Klüver-Bucy syndrome

KLÜVER–BUCY SYNDROME: TEMPORAL NETWORKS AND BEHAVIOUR

Clinical syndrome 19 • Recognise the cluster, then find the cause

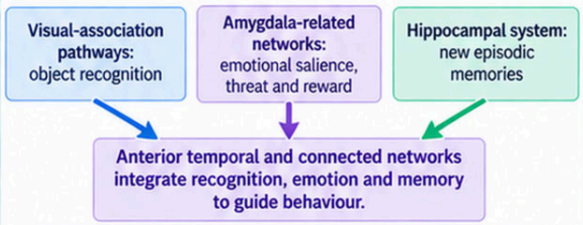
NORMAL NETWORKS

Anterior temporal and connected limbic networks link sensory recognition with emotional significance, memory and appropriate behaviour. The **amygdala** helps assign salience and learn threat associations; the hippocampal system supports new episodic memories; temporal visual-association pathways help recognise objects. These structures work with frontal control and wider networks. The amygdala is not a single isolated "fear switch".

WHAT THE SYNDROME MEANS

Klüver and Bucy described behavioural changes after bilateral temporal-lobe removal in monkeys. The human syndrome is classically associated with bilateral anterior temporal dysfunction, particularly amygdala-related networks. Human presentations are often incomplete and can include additional language, memory and cognitive deficits. Rare partial or transient presentations occur without obvious bilateral structural damage, including seizure-related dysfunction. A normal-looking region on one scan does not prove normal network function.

ANTERIOR TEMPORAL NETWORK MODEL



COMPLETE VERSUS PARTIAL

A **complete** presentation contains the full characteristic cluster. Partial describes an incomplete constellation; definitions vary across reports, and a feature count is not a validated stand-alone diagnostic **test**. Two striking behaviours alone do not establish a lesion or cause. Document which features are present, their timing and functional consequences.

SIX CLASSIC FEATURES: WHAT TO LOOK FOR

1. Visual agnosia



Inability to recognise familiar objects visually despite sufficient basic vision. Ask the person to identify an object by sight, then compare recognition by touch or sound. Exclude poor acuity, field loss, inattention and naming difficulty before calling it agnosia.

2. Hyperorality



Excessive oral exploration, mouthing objects or attempting to eat non-food material. Observe safely and obtain collateral history; do not test with dangerous objects.

3. Hypermetamorphosis



Excessive attention to and exploration of each new visual stimulus. Repeatedly shifting toward, grasping or handling objects is more specific than simply being distractible.

4. Placidity



Unusually reduced normal fear or anger responses, sometimes with poor judgement about danger. This is not proof of emotional well-being; irritability or agitation can coexist in an incomplete human presentation.

5. Altered sexual behaviour



Increased or socially inappropriate sexual activity or approaches. Assess change from baseline, context, consent and vulnerability without moral judgement.

6. Dietary change



Increased eating, indiscriminate intake or altered food preferences. This differs from oral exploration but can overlap with it.

Memory impairment commonly accompanies the syndrome but is not one of these six classical features.

CAUSE → ASSESSMENT → SUPPORT

Identify the cause

- Herpes simplex encephalitis
- Traumatic or hypoxic brain injury
- Neurodegeneration
- Temporal surgery
- Vascular injury
- Seizure-associated dysfunction
- Consider alternative diagnoses

Assess thoroughly

- History and examination
- Magnetic resonance imaging
- Electroencephalography
- Blood tests and cerebrospinal-fluid studies
- Urgent assessment if encephalitis suspected

Provide ongoing support

- Treat underlying cause
- Control seizures
- Safety, supervision and nutrition
- Protect dignity and consent
- Rehabilitation
- Reassess over time

CAUSES AND DIFFERENTIALS

Herpes simplex encephalitis is an important cause because temporal regions are vulnerable. Other causes include traumatic or hypoxic brain injury, neurodegeneration, temporal surgery, vascular injury and seizure-associated dysfunction. Do not claim a universal commonest cause across all populations.

Consider delirium, mania, frontal disinhibition, behavioural-variant frontotemporal dementia, isolated visual-recognition disorders, medication/substance effects and developmental conditions. Episodic hypersomnolence with behavioural change suggests a different syndrome, such as Kleine–Levin syndrome. Tempo, alertness, examination and collateral history help distinguish these possibilities.

ASSESSMENT → CAUSE → SUPPORT

Establish onset, prior function, seizures, fever, trauma and medicine exposure. Examine cognition, vision, language, memory and the full neurological system. Brain magnetic resonance imaging evaluates temporal and other lesions; electroencephalography investigates suspected seizures. Blood tests and cerebrospinal-fluid studies follow the clinical differential. Suspected encephalitis needs urgent hospital assessment and timely treatment; do not wait for a complete behavioural cluster. There is no diagnostic blood concentration or universal severity score for this syndrome.

MANAGEMENT AND FOLLOW-UP

Treat the underlying disease and control seizures where present. Provide structured routines, supervision, safe food/object access, swallowing/nutritional assessment when needed, privacy and protection of everyone's consent and dignity. Use calm redirection and caregiver education; record target behaviours and triggers. Rehabilitation addresses memory, communication and daily function.

Medication is symptom-targeted, with evidence largely from case reports and small series. Selected serotonergic medicines, carbamazepine or other agents may be considered by specialists, with adverse-effect and interaction monitoring; no drug reliably reverses the syndrome. Course depends on cause and extent of injury, so reassess rather than promise recovery or permanence.

MEMORY AID

Recognize the cluster, find the cause, protect dignity.

Supplementary medical teaching • September 2026

Kluver-Bucy syndrome. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-45.

Credits and references: retained within the illustration.

IV - 46

Discuss the personality changes in head injury in relation to the location in the brain. [5+5]

5 + 5 | STROKE, TBI AND FOCAL BRAIN SYNDROMES | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Why location matters	The frontal-subcortical circuits, named before the list
5	The syndromes by site	Four sites, four different people
2	Assessment	How the change is established, and against what
1	Management	What actually helps, and what does not

SITE	SYNDROME	THE PERSONALITY CHANGE	HOW THE FAMILY DESCRIBES IT
Orbitofrontal	Disinhibition	**Impulsive, tactless, socially inappropriate, facile jocularity, poor judgement**	Not the same person, but sharp as ever
Dorsolateral prefrontal	Dysexecutive	Poor planning and set shifting, perseveration, concrete thinking, reduced fluency	Cannot organise anything any more
Medial frontal and anterior cingulate	Apathy	**Loss of initiation and drive, flat affect, in the extreme akinetic mutism**	Sits all day, has to be started
Anterior temporal	Irritable and aggressive	Short fuse, emotional lability, aggression; bilateral damage gives the Kluver-Bucy picture	Explodes over nothing
Diffuse axonal	Slowed	Fatigue, slowness, irritability without a focal signature	Tired and short tempered

Amber marks the two poles that make the point of the question: the same injury produces a disinhibited patient from one site and an inert one from another.

- **Assessment.** Personality change is established by informant history set against premorbid personality, not by a cross-sectional interview. Neuropsychological testing and bedside frontal tasks such as verbal fluency support it, and serial review separates change from a bad week.

PITFALL

Formal cognitive testing is often entirely normal in orbitofrontal damage. A normal mini mental state examination therefore never excludes personality change after head injury, and the patient who tests well while his life falls apart is the classic presentation, not a contradiction.

WHAT EARNS THE MARKS

- **The circuits named before the syndromes are listed.** It converts a list into an explanation.
- **Disinhibition and apathy contrasted as different sites,** which is the whole point of the question.
- **Informant history named as the method of assessment.**

SEE ALSO Slot IV - 45 Kluver-Bucy syndrome Slot IV - 39 management of head injury SPRINT Day 34, Stroke, TBI, delirium & focal brain syndromes

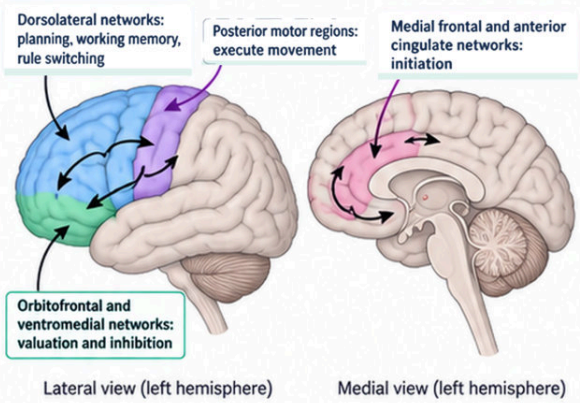
WEAVE 100 · PAPER IV · C10

c10 — Frontal lobe: choosing, initiating and regulating action

Companion C10

Overview and networks

The frontal lobe lies anterior to the central sulcus. Its posterior motor regions help execute movement; premotor and supplementary motor regions organize actions. Prefrontal regions work with parietal cortex, basal ganglia, thalamus and limbic structures to maintain goals, select responses and adjust behaviour. A "frontal" syndrome may arise from damaged connections outside the lobe. Anatomical regions are useful guides, not sealed compartments for personality.



Dorsolateral networks: hold the plan.

Working memory keeps information available while a task is completed. Planning, switching between rules and monitoring errors support organized behaviour. Dysfunction can cause poor problem solving, distractibility, perseveration and difficulty completing multistep activities. Someone may describe a plan correctly yet fail to carry it out in daily life. Slow performance alone is nonspecific and may reflect depression, fatigue, medication or motor impairment.

Orbitofrontal and ventromedial networks: value and restraint.

These systems link choices with reward, consequences and social context. Injury may produce impulsivity, tactlessness, risky decisions or diminished sensitivity to others. Do not equate all disinhibition with a focal lesion, or infer a person's morality from a scan. Obtain concrete examples of change from their established baseline. A normal conversational interview or memory score can miss severe everyday executive difficulty.

Medial frontal and anterior cingulate networks: start the action.

Dysfunction may produce apathy or abulia: reduced spontaneous speech and goal-directed activity despite potentially preserved ability when prompted. Extensive bilateral injury can cause akinetic mutism. Apathy differs from the sadness, guilt and hopelessness of depression, although both may coexist. Severe reduced responsiveness still requires assessment for delirium, catatonia, seizures and other medical causes.

Motor and language clues.

Primary motor injury causes predominantly contralateral weakness according to the affected body representation. Dominant inferior frontal language-network injury can contribute to effortful, nonfluent speech; comprehension, repetition and naming require separate assessment. Speech difficulty is not simply unwillingness to engage. A destructive frontal eye-field lesion can cause gaze preference toward the lesion; seizure-related eye deviation can differ, so do not use one rule without context.

Interpretation and conditions.

These bedside tasks use multiple networks and cannot diagnose a frontal lesion alone. Stroke, tumour, traumatic injury and frontotemporal degeneration are important causes; seizures, toxic-metabolic illness and psychiatric disorders may also affect executive performance. Abrupt onset favours an acute process; progressive behavioural change suggests a different investigation pathway. There is no diagnostic serum range for frontal dysfunction.

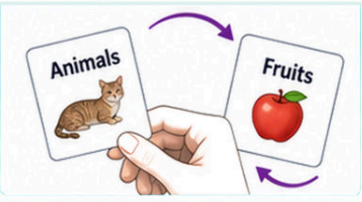
Bedside HOW.

Ask an informant about initiative, finances, organization, empathy and inhibition. Test attention first. Request months backward or backward digit span, adapting for language and education. Ask for words beginning with a specified letter for one minute, avoiding proper names and repetitions; interpret against appropriate norms, not a universal cutoff. Alternate between two categories to examine set shifting. Demonstrate a fist–edge–palm sequence, then ask independent repetition; note perseveration and comprehension. For a go/no-go task, agree a simple rule such as tapping once after one tap but withholding after two; observe errors after practice. Examine strength, gait and speech alongside cognition.

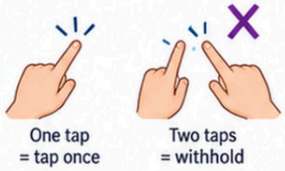
Investigation and care.

New focal signs or sudden behavioural change need urgent medical assessment. Brain magnetic resonance imaging, targeted laboratory testing and electroencephalography are selected by the presentation. Neuropsychological assessment can define strengths and support needs. Treat the underlying cause; simplify tasks, provide external prompts and predictable routines, reduce unsafe access when justified, and support caregivers. Capacity is decision-specific, not determined by a lobe label or one executive score.

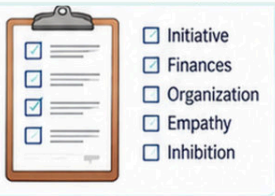
Set shifting: alternate categories



Go/no-go: withhold on two taps



Informant checklist (examples)



MEMORY AID

Plan, initiate, regulate: test each in daily life.

Supplementary medical teaching • September 2026

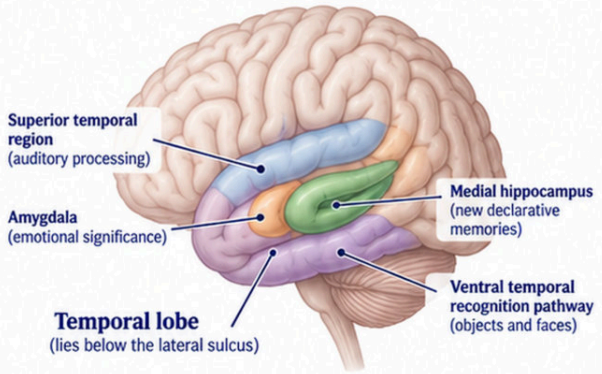
Frontal lobe functions lesions and testing. Use the labelled structures to connect anatomical location with function. Section companion: Stroke, TBI and focal brain syndromes.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · C11

c11 — Temporal lobe: memory, meaning and emotional significance

Companion C11



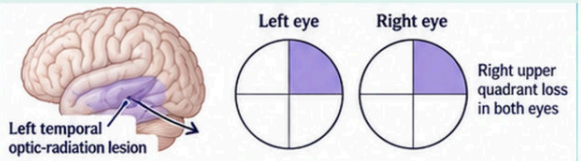
From attention to memory

Attention → Encoding (registration) → Consolidation (new episodic memories) → Later retrieval (uses frontal and distributed cortical networks)

These functions depend on connected systems rather than one isolated memory or emotion centre.

Possible accompanying visual-field sign

(not every temporal lesion)



Anatomy and functions

The **temporal lobe** lies below the lateral sulcus. Superior temporal regions participate in auditory processing; dominant temporal language networks help connect speech with meaning. Medial temporal structures, including the hippocampus and adjacent cortex, support formation of new declarative memories. The amygdala participates in learning emotional significance through wider networks. Ventral temporal pathways help recognize objects and faces. These functions depend on connected systems rather than one isolated memory or emotion centre.

Seizures and episodic symptoms

Temporal seizures may include a rising epigastric sensation, sudden fear, unusual familiarity, an unpleasant smell, behavioural arrest or automatisms. These features are suggestive, not diagnostic individually. Ask whether events are brief, stereotyped and associated with impaired responsiveness or post-event confusion. A normal routine electroencephalogram does not exclude epilepsy. Persistent anxiety or psychosis should not automatically be attributed to temporal epilepsy without the chronology and appropriate assessment.

Memory: registering is not storing.

Attention permits information to be registered; medial temporal systems help establish new episodic memories; later retrieval also uses frontal and distributed cortical networks. Bilateral medial temporal injury can cause striking anterograde amnesia with relatively preserved immediate span. Remote memories and procedural skills may be affected differently. Poor recall can also result from inattention, depression, language difficulty or impaired retrieval, so do not infer hippocampal damage from three-word recall alone.

Klüver–Bucy pattern.

Bilateral anterior temporal injury can produce a combination of hyperorality, altered dietary behaviour, reduced fear or placidity, abnormal sexual behaviour, visual recognition difficulty and excessive attention to visual stimuli. Human presentations are often incomplete. Encephalitis, trauma and degenerative disease can cause overlapping features. The syndrome is not synonymous with isolated hypersexuality, and it is not explained solely by removing an "aggression centre."

Language and recognition.

Dominant posterior temporal-network injury may produce fluent but poorly informative speech with impaired comprehension. Examine naming, repetition, reading and writing rather than treating all fluent speech as normal. Anterior temporal degeneration can erode knowledge of words, objects and people. Right or bilateral ventral temporal dysfunction can impair recognition of familiar faces; recognition by voice or contextual cues may remain possible. Hearing loss and visual impairment must be excluded before attributing a recognition failure to cortex.

Bedside HOW.

Establish hearing, attention and language before memory testing. Give several words, confirm adequate learning, then request delayed free recall after an intervening task; add cues or recognition choices to clarify the pattern. Use an age-, language- and education-appropriate standardized test when scores matter. Ask the patient to name everyday objects and explain their use; assess single-word meaning and progressively complex commands. Compare picture recognition with recognition by touch or description if a modality-specific deficit is suspected. Obtain an eyewitness account or safe event recording for recurrent spells. Check visual fields: temporal optic-radiation injury can cause a contralateral superior quadrantic defect.

Clinical relevance and management.

Medial temporal systems are vulnerable in several amnestic disorders, while herpes simplex encephalitis can prominently involve temporal regions. Acute fever, altered mental state, seizures or rapidly developing memory change needs emergency assessment; do not wait for a psychiatric explanation. Magnetic resonance imaging, electroencephalography, cerebrospinal fluid testing and targeted laboratory studies follow the suspected cause. Progressive semantic or behavioural change merits neurological and neuropsychological evaluation. Treat the underlying disorder; use written reminders, routines and communication support. There is no laboratory concentration or single bedside score that diagnoses "temporal lobe dysfunction."

MEMORY AID

Remember, understand, recognize: check attention and the timeline.

Supplementary medical teaching • September 2026

Temporal lobe functions lesions and testing. Use the labelled structures to connect anatomical location with function. Section companion: Stroke, TBI and focal brain syndromes.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · C14

C14 — Glasgow Coma Scale: assess, score and interpret

Companion C14

GLASGOW COMA SCALE: DESCRIBE THE RESPONSE

Clinical tool 14 • A structured observation, not a diagnosis

Conscious responsiveness depends on arousal networks and functioning cerebral systems, but a response also requires intact language, sensation and movement. The scale records eye opening, verbal response and best motor response. It measures what is observed at a particular time; it does not identify the cause, establish brain death or directly measure decision-making capacity.

CHECK → OBSERVE → STIMULATE → RATE

First stabilise airway, breathing and circulation; check glucose and protect the cervical spine when indicated. Check hearing, language, baseline disability, sedation, paralysis and local injuries.

Observe spontaneous eye opening, speech and movement on both sides. Speak clearly, then use a louder request if needed.

If necessary, a trained examiner applies an appropriate brief pressure stimulus; avoid injured sites and repeated damaging stimulation.

Use a consistent institutional technique, document it and stop when an adequate response is obtained.

EYE OPENING: FOUR LEVELS

4	**spontaneous:** eyes already open without stimulation.
3	**to sound:** opens after spoken or shouted request.
2	**to pressure:** opens only after pressure stimulation.
1	**none:** no opening despite adequate assessment, without a preventing factor.

Swollen or mechanically closed eyelids may make eye opening not testable; this is different from no neurological response.

VERBAL RESPONSE: FIVE LEVELS

5	**orientated:** correctly identifies name, place and date in context.
4	**confused:** converses coherently but is disorientated.
3	**words:** recognisable words without coherent conversation.
2	**sounds:** moaning or other sounds without recognisable words.
1	**none:** no audible response, without a preventing factor.

An endotracheal tube prevents normal speech testing. Record verbal response as not testable and explain why; do not convert an inability to speak into proven cerebral unresponsiveness.

GLASGOW COMA SCALE: SCORING AT A GLANCE

Eye opening (4 levels)		Verbal response (5 levels)		Best motor response (6 levels)	If any component not testable
4	spontaneous	5	orientated	6	obeys commands	Report the assessable components and reason; do not manufacture a comparable total.
3	to sound	4	confused	5	localises	
2	to pressure	3	words	4	normal flexion	
1	none	2	sounds	3	abnormal flexion	
		1	none	2	extension	
				1	none	

Total score (when all components testable)
Range 3–15
Example: eye 3 + verbal 4 + motor 6 = 13

No total score
Record reason

BEST MOTOR RESPONSE: SIX LEVELS

6	**obeys commands:** completes an appropriate two-part request.
5	**localises:** purposefully brings a hand toward a head/neck stimulus, above the clavicle, to remove it.
4	**normal flexion:** rapid elbow flexion/withdrawal without predominantly abnormal posturing.
3	**abnormal flexion:** slower stereotyped flexion, often with arm adduction and forearm rotation.
2	**extension:** elbow extension in response to stimulation.
1	**none:** no movement despite adequate assessment and no preventing factor.

Withdrawal moves away; localisation targets the stimulus. A grasp reflex alone does not establish command following. Observe both sides: record the best response for the score, but document asymmetry separately because a good side can conceal a focal deficit.

Localises to stimulus



Purposefully brings a hand toward a head/neck stimulus, above the clavicle, to remove it.

Withdrawal from stimulus



Rapid elbow flexion/withdrawal moving away from the stimulus.

REPORT COMPONENTS BEFORE THE TOTAL

When all components are testable, total range is 3–15. Example: eye 3 + verbal 4 + motor 6 = 13.

Different component patterns can produce the same total.

If a component is not testable, report the assessable components and reason; do not manufacture a comparable total.

Record time, stimulus, confounders and changes after resuscitation or medication.

INTERPRETATION AND ACTION

Conventional traumatic-brain-injury bands are 13–15 mild, 9–12 moderate and 3–8 severe, interpreted after initial resuscitation and with confounders considered.

A score of 15 does not exclude intracranial bleeding or important cognitive injury. Falling responsiveness, new asymmetry, abnormal pupils or respiratory compromise needs urgent reassessment.

A score of 8 or less prompts early skilled airway assessment; airway protection depends on the whole clinical state, not arithmetic alone.

OTHER TOOLS ANSWER OTHER QUESTIONS

Alert–voice–pain–unresponsive is a rapid screen, not a substitute for detailed serial scoring.

The Full Outline of UnResponsiveness score adds brainstem reflexes and respiratory pattern to eye and motor responses; four domains score 0–4, total 0–16.

Post-traumatic amnesia meaurees time until continuous new memory returns; use serial validated assessment.

Pupils, limb examination and vital signs remain essential alongside any scale.

MEMORY AID

Check confounders, describe components, repeat the examination.

Serial observation and trend
Record time, stimulus, confounders and changes after resuscitation or medication.

Falling responsiveness, new asymmetry, abnormal pupils or respiratory compromise needs urgent reassessment.

Supplementary medical teaching • September 2026.

Glasgow Coma Scale and assessment tools. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Stroke, TBI and focal brain syndromes.

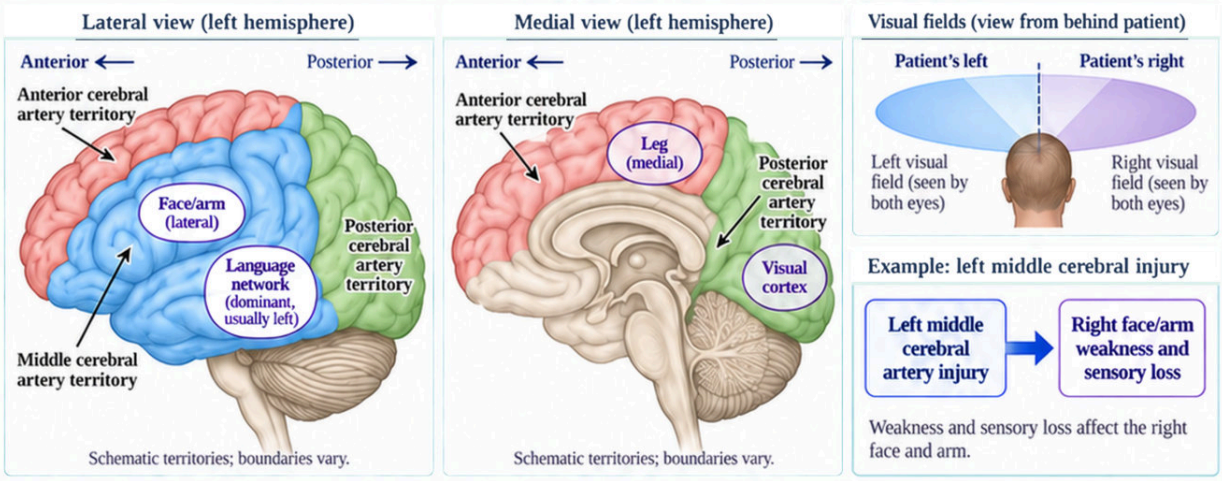
Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · C15

C15 — Stroke arterial territories and cortical localisation

STROKE LOCALISATION: CORTEX AND ARTERIAL TERRITORIES

Clinical map 15 • Function → examination → likely territory



NORMAL ORGANISATION

The internal carotid circulation supplies most anterior and lateral cerebral cortex through the anterior and middle cerebral arteries. Vertebral arteries join the basilar artery; posterior cerebral arteries usually arise from its terminal division. Communicating arteries connect these systems, but collateral capacity and arterial anatomy vary.

The motor and sensory maps place leg representation mainly on the medial hemisphere and face/arm representation mainly laterally. Visual cortex surrounds the calcarine sulcus. Language and spatial attention are distributed networks: a landmark is a useful starting point, not a complete functional boundary.

ANTERIOR CEREBRAL ARTERY: MEDIAL HEMISPHERE

Typical injury affects medial frontal/parietal cortex and adjacent connections. Contralateral leg weakness and sensory loss often exceed arm involvement. Medial frontal disruption may cause reduced initiation, abulia, gait difficulty or urinary incontinence. Extensive bilateral injury can produce profound loss of spontaneous activity. These features are neither obligatory nor unique to this artery.

Examine each leg against gravity, compare sensation, observe initiation and gait only when safe. Apparent unwillingness to move may reflect impaired initiation rather than simple muscle weakness.

MIDDLE CEREBRAL ARTERY: LATERAL HEMISPHERE

A large proximal occlusion may affect both cortex and deep motor pathways: contralateral face/arm weakness, sensory loss, visual-field loss and an acute gaze preference toward the injured hemisphere. The full pattern depends on branches and collaterals.

Dominant superior-division injury often impairs fluent speech production, with face/arm weakness. Dominant inferior-division injury often impairs comprehension with fluent but poorly informative speech; weakness may be limited. Extensive dominant injury can impair both expression and comprehension. The dominant hemisphere is usually left, but handedness alone cannot establish dominance.

Nondominant, commonly right-sided, network injury can cause contralateral spatial neglect and impaired awareness of deficit. Neglect is reduced attention to one side of space; it is not the same as a visual-field defect. Both may coexist.

POSTERIOR CEREBRAL ARTERY: VISION AND MEMORY

Occipital injury commonly causes contralateral homonymous visual-field loss: the same side of visual space is lost in both eyes. Central vision may be spared, but this is not guaranteed. Medial temporal involvement can impair memory. Deep branches may affect thalamus or midbrain, adding sensory or ocular findings.

Dominant occipital plus splenial injury can disconnect visual input from language networks, causing alexia without agraphia; difficulty reading despite preserved ability to write. Bilateral occipital injury can produce cortical visual impairment. Do not assume every visual complaint is ocular disease.

TEST THE NETWORKS

Language: assess spontaneous speech, naming, comprehension, repetition, reading and writing; distinguish aphasia from dysarthria, a motor-speech problem. Account for hearing, literacy and preferred language.

Visual fields: test each eye by confrontation while fixation is maintained.

Attention: use bilateral simultaneous stimulation and cancellation or line-bisection tasks; interpret with visual, sensory and motor limitations.

Cortical sensation: after confirming basic sensation, test recognition of a familiar object by touch or a traced figure. Apraxia is impaired learned action not explained by weakness, sensory loss or failure to understand the task.

LOCALISATION IS A HYPOTHESIS

Border-zone infarcts occur between arterial territories and may follow reduced perfusion or embolism. Multiple lesions, old deficits, swelling and anatomical variants blur textbook patterns. A clinical territory does not prove the clot location or distinguish infarction from haemorrhage.

Sudden focal symptoms → establish last-known-well time, check glucose and activate the stroke pathway → urgent brain and vascular imaging → specialist reperfusion assessment where eligible. Do not wait for every classical sign. A normal early noncontrast computed tomography scan does not exclude ischaemia. Record a structured stroke score, but a low score can still accompany disabling language, vision or posterior-circulation deficits.

MEMORY AID

Name the deficit, test the function, localize the network.

Supplementary medical teaching • September 2026.

Arterial territories and cortical localisation. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Stroke, TBI and focal brain syndromes.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · C16

C16 — Brainstem and cerebellar localisation

Companion C16

BRAINSTEM AND CEREBELLUM: READ THE CROSSED SIGNS

Clinical map 16 • Level + pathway + side

NORMAL ROUTES

The midbrain, pons and medulla contain cranial nerve nuclei, long motor/sensory pathways and networks for arousal, breathing and autonomic control. Corticospinal fibres largely cross in the lower medulla before reaching the opposite limbs. Body pain/temperature pathways have already crossed in the spinal cord; discriminative touch and position pathways cross in the medulla. Cranial nerve findings help identify the level.

The cerebellum compares intended with actual movement and adjusts timing, coordination and balance. Cerebellar hemispheres mainly coordinate ipsilateral limbs; midline regions influence stance and gait. Connections can produce exceptions, so use a cluster of signs rather than one finding.

WHY FINDINGS CAN BE CROSSED

A brainstem lesion can damage a cranial nerve nucleus or fascicle on its own side and a long pathway serving the opposite body. Example: left hypoglossal fascicle injury plus left corticospinal injury above its crossing → left tongue weakness with right limb weakness.

Crossed findings strongly suggest brainstem localisation, but their absence does not exclude it. Large or bilateral lesions may produce complex patterns.

MEDULLA: TWO USEFUL PATTERNS

Medial medullary injury can affect the pyramid, medial lemniscus and hypoglossal fibres: opposite limb weakness, opposite loss of vibration/position sense and same-side tongue weakness. On protrusion, a weak lower-motor-neuron tongue usually deviates toward the lesion. Anterior spinal and vertebral branches supply this region.

Lateral medullary injury can affect spinal trigeminal pathways, spinothalamic tract, vestibular nuclei, inferior cerebellar connections, sympathetic fibres and nucleus ambiguus. Look for same-side facial plus opposite-body pain/temperature loss, vertigonystagmus, same-side ataxia, Horner syndrome, dysphagia and hoarseness. Horner syndrome combines a small pupil and mild eyelid droop, sometimes reduced sweating. Vertebral or posterior inferior cerebellar artery disease can cause this pattern; not every case is a posterior inferior cerebellar occlusion. Major limb weakness is not the defining feature.

PONS AND MIDBRAIN

Pontine injury may combine same-side facial weakness or impaired eye abduction with opposite limb weakness or sensory change. A sixth-nerve fascicle lesion limits one eye's abduction; a sixth-nerve nucleus or adjacent gaze-centre lesion can impair conjugate gaze toward that side.

Lateral pontine disease may add hearing loss, vestibular symptoms and cerebellar signs; anterior inferior cerebellar artery supply is relevant.

Ventral midbrain injury can combine same-side oculomotor palsy—ptosis, impaired eye movements, sometimes a dilated pupil—with opposite weakness through cerebral-peduncle involvement. A pupil-sparing presentation does not rule out serious disease.

Bilateral ventral pontine injury can cause locked-in syndrome: profound paralysis and loss of speech despite preserved awareness, often with vertical eye movement/blinking available for communication.

EXAMINE THE CEREBELLUM

Finger–nose and heel–shin testing reveal dysmetria, overshooting or irregular trajectories. Rapid alternating movements assess rhythm and coordination.

Observe gaze-evoked nystagmus, broken pursuit, scanning/slurred speech, sitting balance and a broad-based gait; guard against falls.

Limb signs often point to the ipsilateral hemisphere; marked truncal instability suggests midline/vestibular connections. Weakness, sensory loss, visual impairment and poor comprehension can confound these tasks.

A **positive Romberg** test—disproportionate worsening on closing the eyes—suggests dependence on vision from impaired proprioceptive or vestibular input. It is not synonymous with cerebellar ataxia, which may already be evident with eyes open.

ACTION BEFORE A PERFECT LABEL

Sudden imbalance with new double vision, dysarthria, swallowing difficulty, severe truncal instability or crossed deficits warrants urgent stroke assessment.

Stabilise airway and swallowing safety: obtain brain and vascular imaging. Early imaging may miss small posterior lesions; persistent convincing findings require specialist reassessment.

Cerebellar swelling can compress the brainstem or obstruct the fourth ventricle, causing hydrocephalus.

Deteriorating consciousness requires emergency escalation.

MEMORY AID

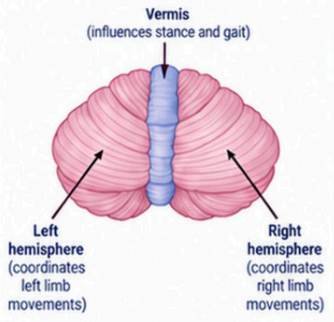
Check cranial nerves, long pathways and coordination together.

Brainstem localisation: an example above the motor crossing

A left medial medullary lesion can affect both pathways.

Left hypoglossal fascicle injury	Ipsilateral lower motor neuron tongue weakness (left side)	Left tongue weakness (tongue deviates left on protrusion)
Left corticospinal pathway injury above the lower-medullary crossing	Contralateral upper motor neuron limb weakness (right side)	Right limb weakness

Cerebellum (dorsal view)



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Brainstem cerebellar and crossed signs. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Stroke, TBI and focal brain syndromes.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · C17

C17 — Deep structures and lacunar localisation

Companion C17

DEEP BRAIN LOCALISATION: SMALL LESION, LARGE DEFICIT

Clinical map 17 • Capsule, thalamus, basal ganglia and penetrating arteries

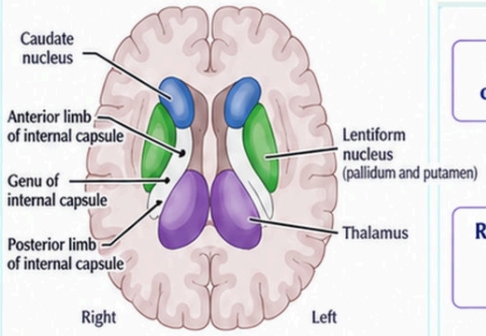
NORMAL ANATOMY

The internal capsule is a compact white-matter corridor carrying fibres between cortex, thalamus, brainstem and spinal cord. Its anterior limb lies between caudate and lentiform nucleus; its posterior limb lies between thalamus and lentiform nucleus. The genu is the bend. Corticobulbar fibres are associated with the genu and adjacent capsule; corticospinal and sensory fibres run through the posterior limb. These are useful approximations, not perfectly separated cables.

The thalamus relays and integrates sensory, motor, arousal and cognitive signals. Ventral posterolateral pathways carry body sensation; ventral posteromedial pathways carry facial sensation.

Basal ganglia loops help select, scale and suppress actions rather than directly supplying limb strength.

Axial view of deep brain structures



Small capsular lesion and opposite deficits

Left internal capsule injury

↓

Right face, arm and/or leg weakness

WHY PENETRATING-ARTERY LESIONS MATTER

Small penetrating branches supply deep structures: lenticulostriate branches from the middle cerebral artery, other anterior-circulation perforators, and posterior-circulation branches to thalamus and pons. Territory boundaries overlap.

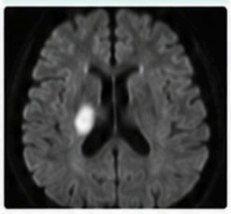
Chronic hypertension and diabetes promote small-vessel injury, but an apparently small deep infarct does not prove one mechanism; parent-artery disease and embolism can also contribute.

A recent small subcortical infarct is an acute lesion. **A lacune** is a small fluid-filled cavity from an old small infarct or haemorrhage. Do not use every small white-matter spot as a synonym for a lacune. Imaging, timing and clinical findings must be reconciled.

Acute lesion versus chronic lacune

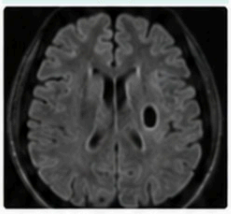
Illustrative schematic images — not patient scans.

Acute small infarct (recent)



Recently ischaemic lesion on magnetic resonance diffusion imaging (acute lesion).

Chronic lacune (old)



Small fluid-filled cavity from an old small infarct or haemorrhage (lacune).

FIVE CLASSIC LACUNAR SYNDROMES

1 Pure motor

Weakness of the opposite face, arm and/or leg without prominent cortical or sensory findings. Internal capsule, corona radiata or ventral pons can produce it. A tiny strategic lesion may disable several body regions because descending fibres are densely packed.

Possible sites: Internal capsule; Corona radiata; Ventral pons

2 Pure sensory

Opposite-sided numbness or impaired sensation, often from thalamic injury; other sensory-pathway sites can mimic it. Test several modalities rather than assuming all are equally affected.

Possible sites: Thalamus; Internal capsule; Other sensory-pathway sites

3 Sensorimotor

Combined opposite weakness and sensory loss, often involving adjacent thalamic and capsular pathways.

Possible sites: Thalamus; Internal capsule

4 Ataxic hemiparesis

Weakness with incoordination on the same affected side of the body, opposite the brain lesion; pontine or capsular lesions are examples. Incoordination is disproportionate to weakness.

Possible sites: Ventral pons; Internal capsule

5 Dysarthria–clumsy hand

Impaired articulation with reduced fine hand control; pontine or capsular pathways may be involved. Language formulation can remain intact despite poor speech clarity.

Possible sites: Ventral pons; Internal capsule

These are clinical patterns, not definitive lesion coordinates. More than one location can produce each syndrome.

CLUES BEYOND THE CLASSIC FIVE

Aphasia, neglect, a gaze preference or a homonymous field defect suggests cortical or broader network involvement and should prompt reconsideration of a simple lacunar label. Their absence supports but does not prove a deep lesion. Strategic thalamic or striatal injury can itself affect language, attention or behaviour.

Thalamic injury may later cause central neuropathic pain, including burning pain or pain from normally non-painful stimulation; not every sensory stroke develops this.

Bilateral paramedian thalamic injury may impair alertness and memory.

Sudden flinging movements can reflect disruption of contralateral subthalamic/basal-ganglia networks; hemiballismus is not exclusive to the subthalamic nucleus.

HOW TO TEST AND ACT

Compare face, arm and leg power, assess pronator drift, tone, reflexes and plantar responses. Map pinprick, light touch, vibration and joint position with eyes closed. Compare finger–nose and heel–shin performance after allowing for weakness. Test speech articulation separately from naming and comprehension; screen visual fields and neglect.

New focal deficits require the same urgent stroke pathway as a large cortical syndrome: glucose, last-known-well time, brain imaging and appropriate vascular/cardiac assessment. Magnetic resonance diffusion imaging can identify small acute lesions missed on computed tomography.

Treatment depends on timing, disability, haemorrhage exclusion and cause; "lacunar-looking" is not a reason to withhold assessment.

Rehabilitation, blood-pressure control, diabetes/lipid care, smoking cessation and mechanism-appropriate antithrombotic therapy reduce further harm.

MEMORY AID

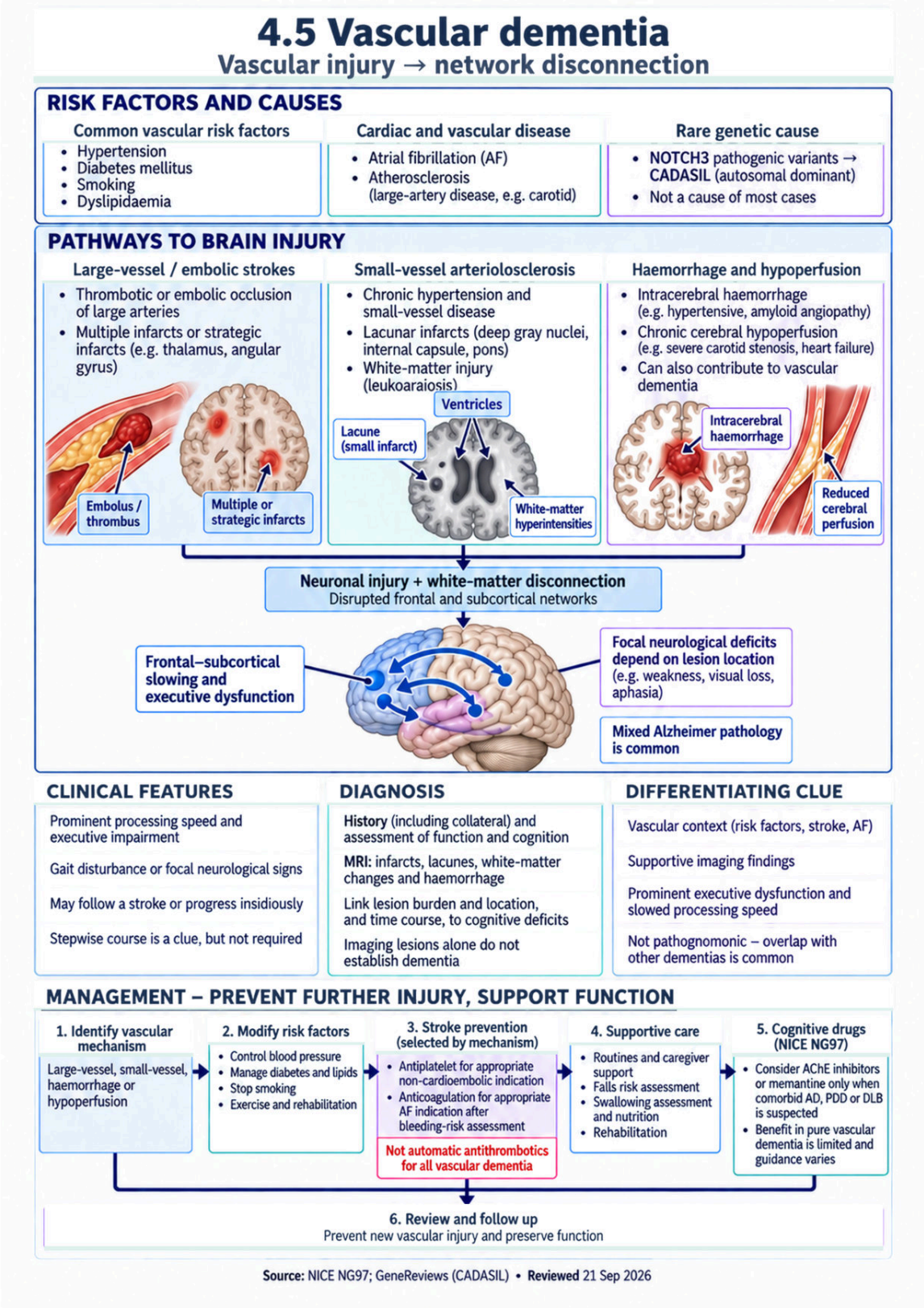
Small deep lesions can cause large deficits: assess urgently.

Supplementary medical teaching • September 2026.

Deep structures and lacunar localisation. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Stroke, TBI and focal brain syndromes.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · D4.5



Vascular dementia - Vessels networks and prevention. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Stroke, TBI and focal brain syndromes.

Credits and references: retained within the illustration.

AREA 06 OF 15 · P4-A10

Movement and degenerative disorders in psychiatry

7	6.7%	IV-47	IV-53
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 3 HIGH 2 SINGLE 2

REVISION ORDER

The four Parkinson slots together and hardest: they cross-link each other and the safe-agent rule is identical on all of them. Take the whole-disease slot first, it half writes the other three. The two multiple sclerosis slots next, they are one answer at two widths and they cross-link each other. Huntington last, and separately.

WHAT IS IN THIS AREA

Parkinson disease in four slots: the psychological and psychiatric features, the drug induced problems with their management, the relationship with psychosis, and one that asks for the whole disease from aetiology to prognosis. Multiple sclerosis in two, the neuropsychiatric aspects and the aetiology, features and management. Huntington disease takes the seventh. One of the Parkinson slots carries a second limb on the assessment and management of depression after stroke, which the board set there and which is answered on that page as it was set.

IV - 47

Describe the psychological and psychiatric features of Parkinson's disease. [10]

10 MOVEMENT AND DEGENERATIVE DISORDERS IN PSYCHIATRY

KUHS, NTRUHS, TN-MGRMU - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2	Why they occur	Part of the disease, not merely a reaction to it
4	The syndromes	Six, each with what makes it look different here
2	Sleep and the prodrome	The disorder that arrives years before the tremor
2	Assessment	Why the usual diagnostic cues mislead here

SYNDROME	HOW IT APPEARS IN PARKINSON DISEASE	WHY IT IS MISSED
Depression	Common; less guilt and self-reproach than primary depression, more dysphoria and worry	Slowness and flat face are read as mood
Anxiety	Frequent, often locked to the wearing-off periods between doses	Attributed to the situation, not to the dose cycle
Apathy	**Loss of motivation and initiation without sadness or self-criticism**	**Recorded as depression, then treated as depression**
Psychosis	Visual hallucinations first, insight often retained early; delusions later	Patients conceal them unless asked directly
Impulse control disorders	**Gambling, hypersexuality, compulsive shopping and eating, punding**	**Never volunteered, and rarely asked about**
Dementia	Late, subcortical and executive, with prominent visuospatial difficulty	Confused with dementia with Lewy bodies

Amber marks the two that are missed most often and cost most. Apathy is treated as depression that will not respond, and impulse control disorders are found only by a direct question to the patient and the family.

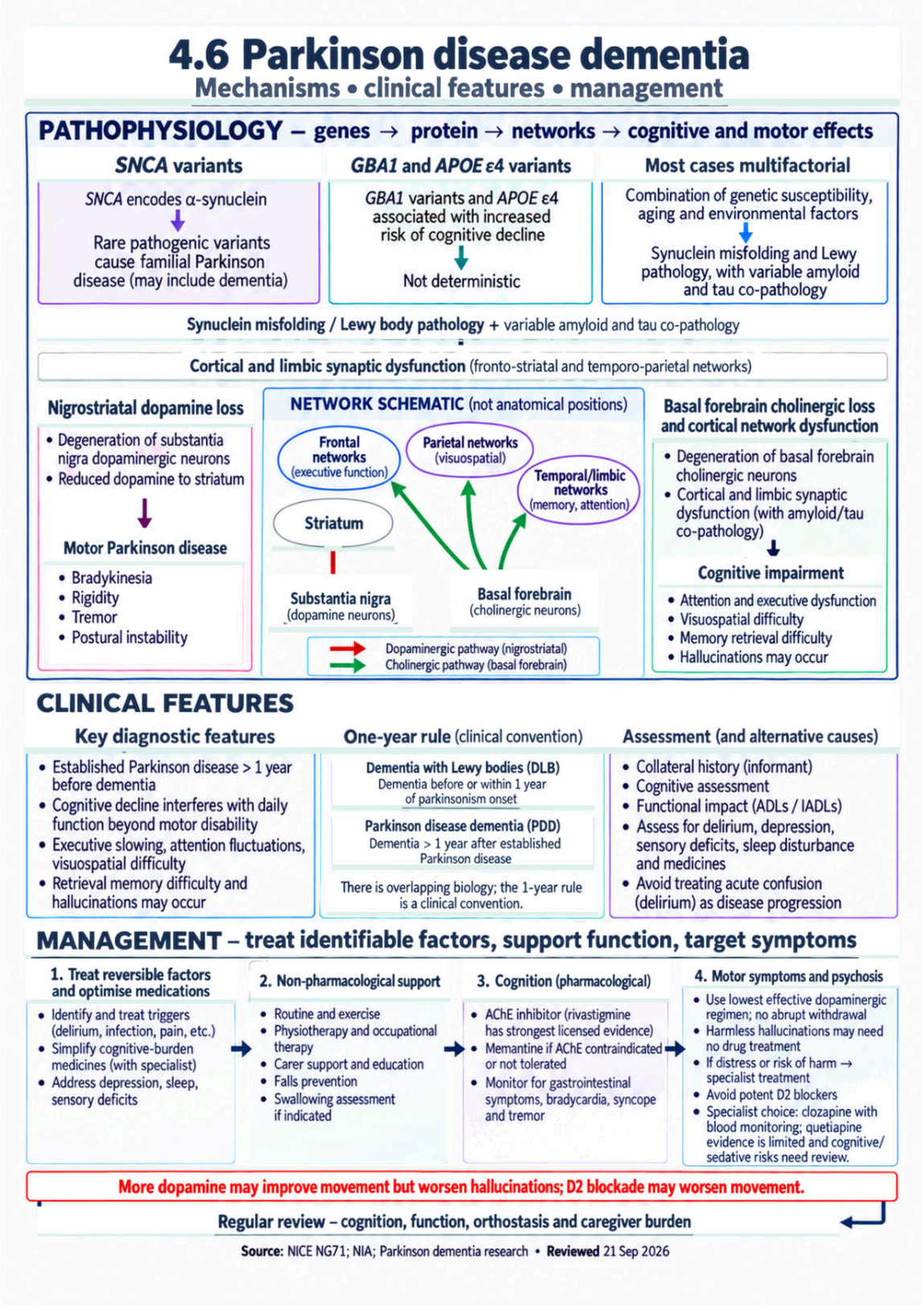
- **The prodrome.** Rapid eye movement sleep behaviour disorder, in which the normal atonia of dreaming sleep is lost and the patient acts out dreams, can precede motor Parkinson disease by years. Depression, anxiety, constipation and reduced smell belong to the same premotor phase.
- **Why assessment is hard.** Masked facies, bradykinesia, stooped posture, soft monotonous voice, fatigue and disturbed sleep all imitate depression, so the diagnosis has to rest on reported mood, anhedonia, hopelessness and guilt rather than on what is observed across the desk.

WHAT EARNS THE MARKS

- **Apathy separated from depression.** It is the discrimination this question is set to test.
- **Impulse control disorders named,** with the fact that they must be asked about.
- **Sleep behaviour disorder given as premotor,** not as a late complication.
- **The overlap problem in assessment, stated explicitly.**

SEE ALSO Slot IV - 49 drug induced problems and their management Slot IV - 53 aetiology, course and management

SPRINT Day 35, Neuropsychiatry of systemic & neurological disease



Parkinson disease dementia - Networks and treatment balance. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-47.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · F20

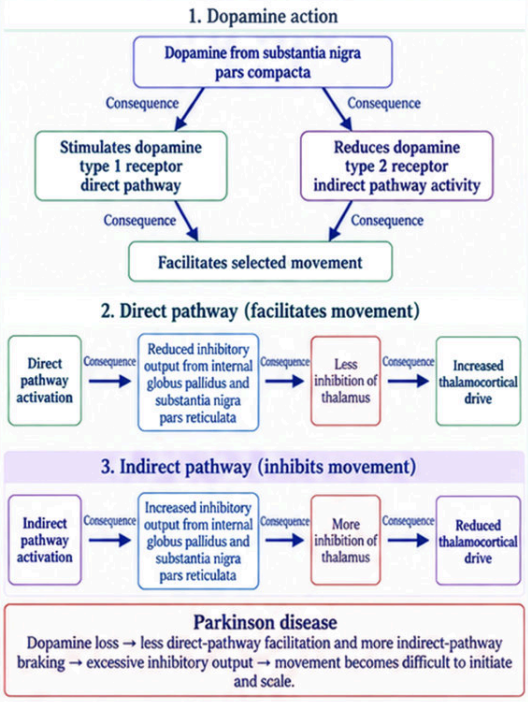
MOVEMENT CIRCUITS AND PARKINSON DISEASE

Foundation 20 • Normal function → mechanism → clinical recognition → management

NORMAL MOVEMENT SELECTION

The motor cortex plans and executes movement with feedback from the basal ganglia, cerebellum and sensory systems. Basal ganglia circuits help **select desired actions and suppress competing ones.** The striatum receives cortical input. Output from the internal globus pallidus and substantia nigra pars reticulata normally inhibits the thalamus. The simplified direct pathway reduces this brake, **facilitating selected movement;** the indirect pathway **increases it.** Dopamine from the substantia nigra pars compacta supports movement by stimulating dopamine type 1 receptor direct pathway and reducing dopamine type 2 receptor indirect pathway activity. This is a useful simplified circuit model, not a complete explanation of every motor symptom.

SIMPLIFIED MOVEMENT-CONTROL MODEL



Parkinson disease

Dopamine loss → less direct-pathway facilitation and more indirect-pathway braking → excessive inhibitory output → movement becomes difficult to initiate and scale.

WHAT DEGENERATES

Parkinson disease is a progressive neurodegenerative disorder involving loss of nigrostriatal dopamine neurons and broader neural systems. Alpha-synuclein aggregation and Lewy pathology are characteristic in many cases, but pathogenesis is complex and heterogeneous. Reduced dopamine shifts basal-ganglia output towards excessive inhibition of thalamocortical motor activity. Movement becomes difficult to initiate and scale. Non-dopaminergic degeneration contributes to autonomic, sleep, sensory and cognitive symptoms. Age, genetic susceptibility and environmental factors interact; one simple cause does not explain most cases.

MECHANISM → CLINICAL FINDING

- **Bradykinesia** means slowness with progressive reduction in speed or amplitude during repeated movement, not merely feeling slow.
- **Rigidity** is increased resistance to passive movement.
- **Rest tremor** occurs in a relaxed limb and may lessen during action, but is neither required nor specific.
- **Parkinsonism** is defined clinically by bradykinesia with rigidity or rest tremor.
- **Reduced automatic movement** causes decreased arm swing, facial expression and blinking.
- **Short steps, freezing** and impaired postural responses increase falls.
- **Small handwriting,** quiet speech, swallowing difficulty and drooling can occur.
- **Constipation,** reduced smell, dream enactment, orthostatic symptoms and other non-motor features may precede or accompany motor disease.
- **Tremor severity** does not directly measure total disability.

DIAGNOSE CLINICALLY

- Look for asymmetry, examination signs, progression and response to dopaminergic treatment.
- Review dopamine-blocking medicines and other causes of parkinsonism.
- Early recurrent falls, prominent early autonomic failure, vertical gaze palsy or a poor sustained levodopa response may suggest an atypical syndrome.
- Structural magnetic resonance imaging mainly evaluates alternatives when indicated; it does not routinely confirm typical Parkinson disease.
- Dopamine-transporter imaging can assist selected diagnostic uncertainties but does not reliably distinguish all degenerative parkinsonian disorders.
- There is no routine blood dopamine level or universal laboratory threshold that diagnoses the disease.
- Assess gait, falls, swallowing, nutrition, cognition, sleep and standing blood pressure.
- A fall of at least 20 millimetres of mercury systolic or 10 diastolic within three minutes of standing supports orthostatic hypotension; context and symptoms matter.

RESTORE FUNCTION, REVIEW REGULARLY

- Offer explanation, exercise and physiotherapy, occupational therapy, speech/swallow support and nutrition review.
- When motor symptoms affect quality of life, **levodopa** is generally the most effective symptomatic medicine. It crosses into the brain and is converted to dopamine; carbidopa or benserazide reduces conversion outside the brain. Dose timing matters.
- Dopamine agonists and monoamine oxidase typeB inhibitors may be appropriate for selected patients, with different benefit–harm profiles.
- Monitor nausea, orthostatic hypotension, somnolence, hallucinations and impulse-control problems, particularly with dopamine agonists.
- As disease progresses, **wearing-off** means benefit fades before the next dose; **dyskinesia** means involuntary movement, often related to dopaminergic treatment.
- Specialist adjustment may include dose scheduling, adjuncts or continuous delivery.
- Selected patients with troublesome fluctuations may benefit from deep-brain stimulation after multidisciplinary assessment; it is not a cure and does not treat every non-motor problem.
- Avoid abruptly stopping dopaminergic medicines because severe rigidity, fever and systemic deterioration can follow.
- Reassess goals, function, adverse effects and caregiver needs over time.

MEMORY AID

Too little dopamine leaves too much braking of movement.

Foundation 20 • Medical foundations • Source-checked supplementary teaching • September 2026.
Laboratory ranges and treatment protocols require local context.

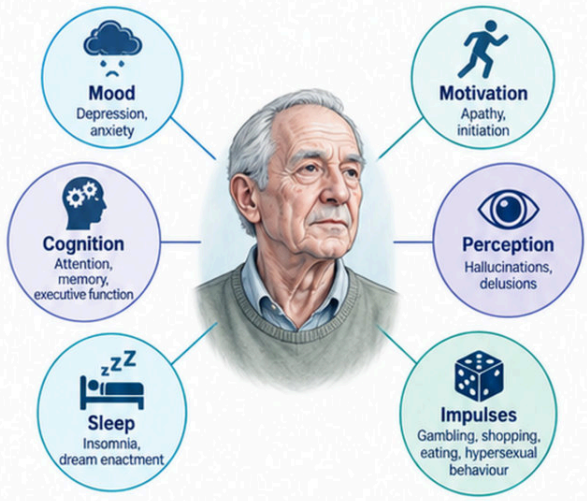
Movement circuits and Parkinson disease. Follow the labelled sequence and connections, then reconstruct the diagram from memory. Read with IV-47.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P13

PARKINSON DISEASE: MOOD, MOTIVATION AND PSYCHOSIS

Psychiatric companion • Examine the syndrome and the medicine cycle



THE MEDICINE CYCLE AND MENTAL SYMPTOMS

Take Parkinson medicine (at prescribed dose and time) → Improved movement and function → Possible wearing-off with increased anxiety

APPROACH TO PSYCHOSIS IN PARKINSON DISEASE

Hallucinations or delusions

↓

Check for delirium
Consider infection, dehydration, pain, constipation and medicines

If delirium suspected → Treat the underlying cause

↓ If no delirium

Specialist medicine review
Consider reducing potentially contributing Parkinson medicines

↓

Treat with antipsychotic only when clinically needed → Use specialist options. Avoid drugs that can worsen motor function (e.g. olanzapine).

WHY MENTAL SYMPTOMS OCCUR

Parkinson disease affects more than movement. Changes in brain networks, sleep, cognition and disability interact with dopaminergic treatment. Depression, anxiety, apathy, hallucinations, impulse-control problems and dementia require separate assessment because their treatments differ. Do not explain every symptom as a reaction to tremor or every hallucination as a drug adverse effect.

ASSESS BEYOND FACIAL EXPRESSION

A quiet voice, reduced facial movement, slowness and fatigue can resemble depression. Ask about felt sadness, pleasure, hopelessness, guilt and suicidal thoughts. Apathy is reduced motivation and initiation; it may occur without sadness, although it can coexist with depression. Ask what the person wants to do, whether they enjoy activities once started, and what prevents initiation.

Relate anxiety and mood to dose timing. Symptoms during wearing-off periods may improve with specialist adjustment of motor treatment; persistent anxiety still deserves psychological or psychiatric care. Record sleep, pain, falls, cognition and function. Obtain collateral information with consent because hallucinations, risky behaviours and cognitive changes may not be volunteered.

MOOD AND MOTIVATION

Use psychological treatment adapted to motor and cognitive needs: shorter sessions, written prompts, behavioural activation and support to practise between appointments. For depression requiring medication, choose an antidepressant according to prior response, sleep, autonomic symptoms, cognition and interactions. Review the complete Parkinson regimen, particularly monoamine oxidase inhibitors, before adding serotonergic treatment. Monitor mood, suicide risk, postural symptoms, sodium when indicated, falls and benefit.

For apathy, establish a predictable daily routine, provide external cues and divide tasks into achievable steps. Treat coexisting depression, sleep disturbance and medication-related sedation. Do not assume that escalating an antidepressant will correct isolated apathy.

MEMORY AID

Mood, motivation, medicines, perception, impulses, independence.

HALLUCINATIONS: CHECK, THEN ADAPT

Ask directly about visual experiences, delusions, insight, distress and behaviour. Abrupt change or fluctuating attention requires evaluation for delirium, including infection, dehydration, pain, constipation and medicines. Correct sensory problems and environmental triggers; improve lighting and reduce confusing visual stimuli.

Well-tolerated hallucinations with retained insight may need explanation, monitoring and safety planning rather than medication. For distressing or dangerous symptoms, obtain specialist advice before reducing potentially contributing Parkinson medicines. Balance psychiatric benefit against immobility and withdrawal effects. Do not abruptly stop dopaminergic treatment.

When antipsychotic treatment is necessary, specialist options include low-dose quetiapine in appropriate patients without cognitive impairment, or clozapine with the required blood and other safety monitoring. Quetiapine efficacy evidence is less secure than clozapine evidence. Both can cause sedation and postural hypotension. Pimavanserin is another jurisdiction-dependent option where authorised and available; check cardiac and interaction risks. Avoid assuming that all antipsychotics are suitable; strong dopamine blockade can markedly worsen motor function, and olanzapine is not recommended for Parkinson psychosis.

ASK ABOUT IMPULSES, NOT ONLY MOOD

Dopaminergic treatment, especially dopamine agonists, can produce gambling, compulsive shopping or eating, hypersexual behaviour and repetitive purposeless activity. Ask privately and sensitively, then involve trusted supporters with consent. Assess financial, sexual and safeguarding consequences. Develop immediate practical protections collaboratively.

Specialist gradual reduction of the contributing medicine is central; monitor motor deterioration and dopamine agonist withdrawal symptoms. Psychological treatment can help persistent behaviours after medication review. Do not moralise or ask the person simply to exercise willpower.

COGNITION, SLEEP AND FOLLOW-UP

Assess executive and visuospatial functioning when daily tasks decline; address delirium, depression and sedatives before concluding dementia. Cholinesterase inhibitor treatment may be appropriate for Parkinson dementia. Dream enactment needs injury prevention and sleep assessment; it is not automatically psychosis.

At follow-up, review mental symptoms alongside mobility, falls, daily independence and carer burden. A successful prescription must improve the person's overall function.

P13 • Psychiatric companion • Supplementary teaching.

Parkinson disease psychiatry. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-47.

Credits and references: retained within the illustration.

IV - 48

Huntington's disease. [10]

10 MOVEMENT AND DEGENERATIVE DISORDERS IN PSYCHIATRY

KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 **Genetics and pathology**	The repeat, the anticipation, the structure that atrophies
2 **The triad**	Movement, cognition, psychiatry, and which arrives first
4 **The psychiatric picture**	What it looks like, and where the risk sits
2 **Management**	Symptomatic only, plus the family and the testing question

GENETICS AND PATHOLOGY

Autosomal dominant, caused by an expanded CAG trinucleotide repeat in the huntingtin gene on chromosome 4. Forty repeats or more is fully penetrant, 36 to 39 carries reduced penetrance, and the intermediate range does not cause disease but is unstable when transmitted. Anticipation is greater through the father. The striatum atrophies, the caudate first, with loss of medium spiny neurons.

- **The triad.** Movement: chorea early, then dystonia, rigidity and bradykinesia as the disease advances. Cognition: a subcortical decline in speed, executive function and judgement, with insight often preserved painfully long. Psychiatry: frequently the first of the three to appear, sometimes by many years.
- **The psychiatric picture.** Depression is common and may precede the movement disorder. Suicide risk is markedly raised, with two peaks: around the time the diagnosis is made, and at the point independence is lost. Irritability and aggression cause most of the family burden. Apathy, obsessive and perseverative behaviour are common; psychosis is less so.
- **Management.** There is no treatment that changes the course. Chorea is treated with tetrabenazine or an antipsychotic when it is disabling. Depression, irritability and obsessive symptoms are treated conventionally and respond. Beyond drugs the work is genetic counselling, capacity, carer support and planning while the patient can still take part.

PREDICTIVE TESTING

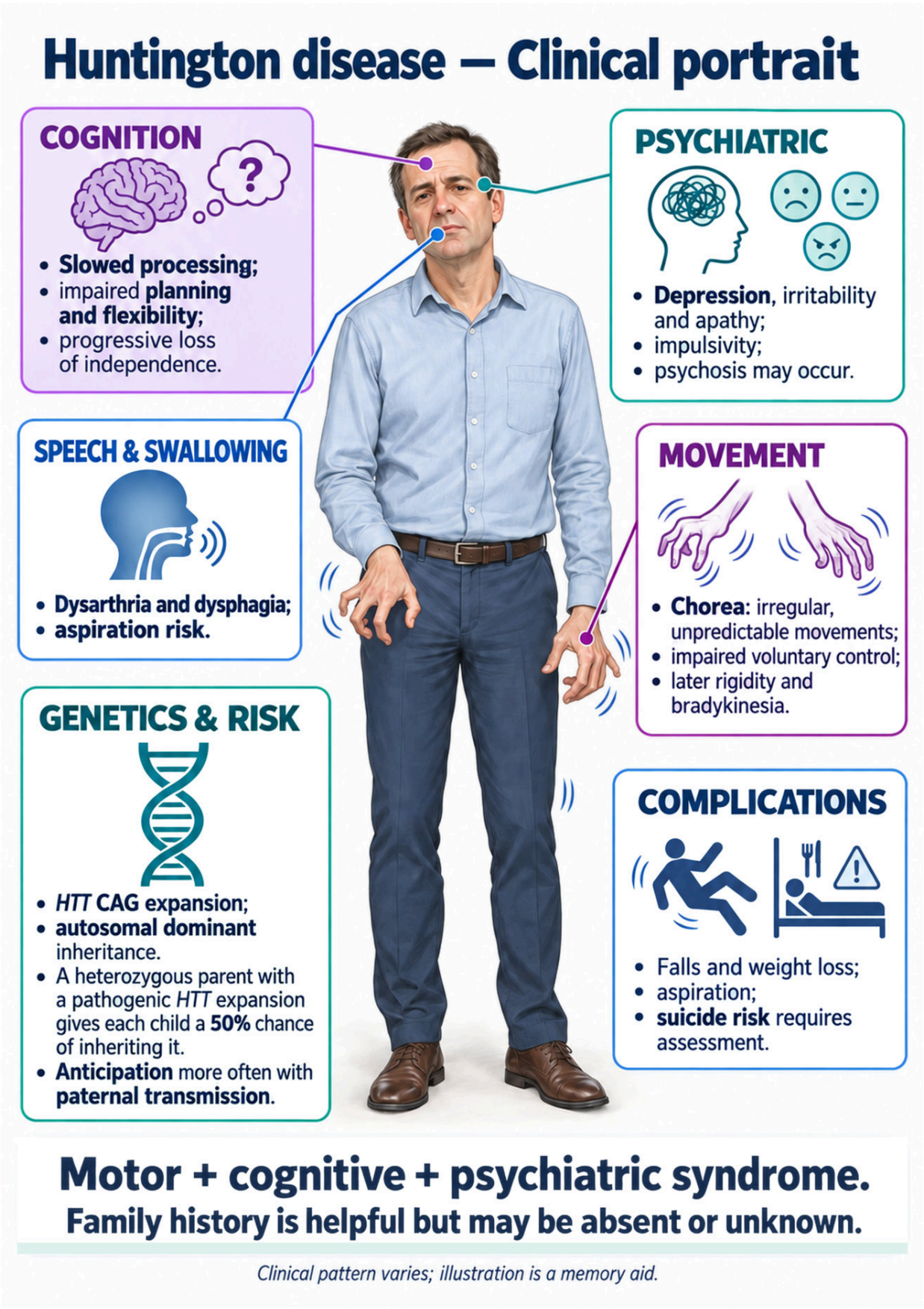
Never a routine blood test. It follows a protocol with counselling before and after, is not performed on children or on anyone who cannot consent for themselves, and is never done at the request of a relative, an employer or an insurer. The result changes a life whether it is positive or negative.

WHAT EARNS THE MARKS

- **The repeat, the gene and the chromosome,** with the penetrance threshold.
- **Psychiatric onset placed before the movement disorder.**
- **The two suicide peaks.** Naming when the risk rises is worth more than saying risk is raised.
- **Predictive testing handled as an ethical procedure,** not as an investigation.

SEE ALSO Slot IV - 82 Wilson disease Slot IV - 47 psychiatric features of Parkinson disease **SPRINT** Day 35, Neuropsychiatry of systemic & neurological disease

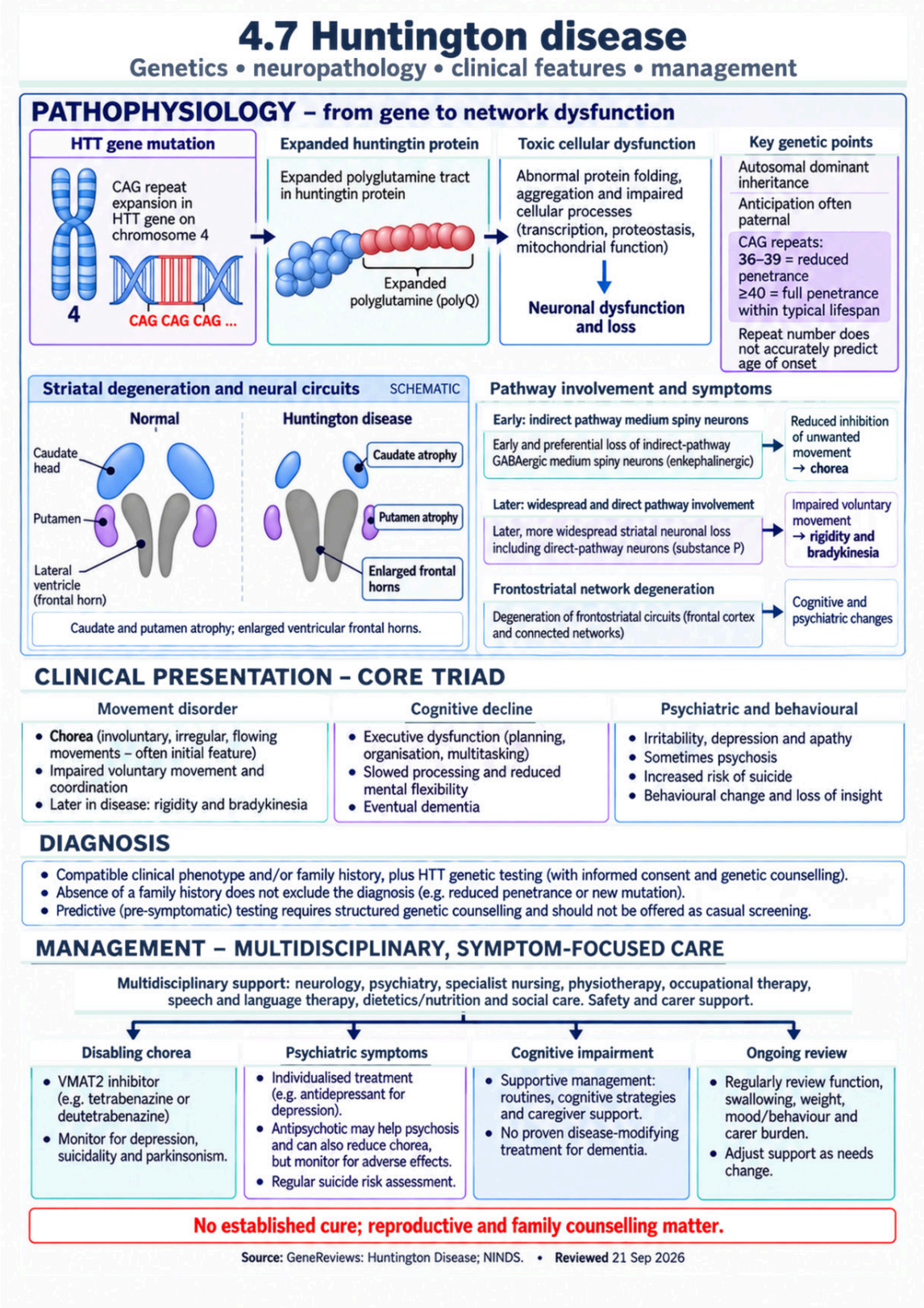
WEAVE 100 · PAPER IV · D4.13



Huntington disease - Clinical portrait risks and features. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-48.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · D4.7



Huntington disease - Gene to clinical triad. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-48.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · F21

HUNTINGTON DISEASE — FROM MOTOR SELECTION TO INHERITED NEURODEGENERATION

Foundation 21 • Normal function → mechanism → clinical recognition → management

1. NORMAL FUNCTION

Normal: the basal ganglia select useful actions and suppress competing movements. The striatum comprises caudate nucleus and putamen; its inhibitory medium spiny neurons communicate using gamma-aminobutyric acid. Cortical input, basal ganglia output and thalamic feedback form loops for movement, cognition and motivation. Huntingtin is a widely expressed protein involved in neuronal development, transport and cell maintenance.

3. WHAT GOES WRONG

What goes wrong: expansion of the cytosine–adenine–guanine repeat in the huntingtin gene on chromosome 4 produces an abnormally long glutamine sequence. Toxic mutant huntingtin disrupts cellular functions and progressively damages striatal and cortical networks. Early preferential injury to the indirect motor pathway weakens suppression of unwanted movements; wider later injury produces slowness and rigidity. This simplified circuit model does not explain every clinical feature.

4. INHERITANCE

Inheritance: autosomal dominant means one disease-associated allele can cause illness. A child of an affected heterozygous parent has a 50% chance of inheriting that allele at each pregnancy. Anticipation means earlier onset in successive generations, often associated with repeat expansion during paternal transmission. Repeat length influences onset but does not determine an individual's exact onset date or course.

5. REPEAT INTERPRETATION

Repeat interpretation: 26 or fewer repeats, usual non-disease range; 27–35, intermediate allele, usually unaffected but transmission may expand it; 36–39, reduced penetrance, disease may or may not develop; 40 or more, full penetrance over a usual lifespan. These are genetic categories, not blood concentrations or a severity score. The laboratory must interpret the assay and repeat structure.

2. FROM GENE REPEAT TO BRAIN INJURY AND SYMPTOMS

Cytosine–adenine–guanine in huntingtin gene (chromosome 4)



Expanded cytosine–adenine–guanine repeats

↓

Abnormally long glutamine sequence in huntingtin protein



↓

Toxic mutant huntingtin disrupts cellular functions

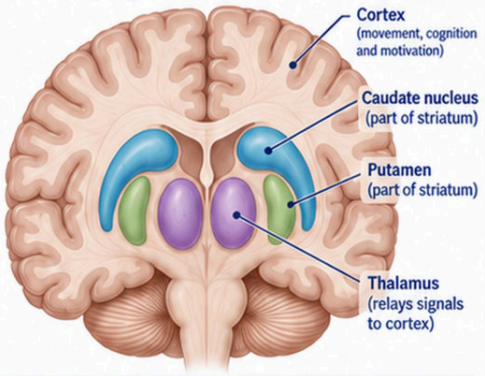
↓

Progressive striatal and cortical injury

↓

Movement, cognitive and behavioural symptoms

Basal ganglia in movement selection



Healthy movement selection

The striatum helps suppress competing movements so that useful actions are selected.

↓

Unwanted movements are suppressed

Damaged striatal suppression

Early injury to the indirect motor pathway weakens suppression of unwanted movements.

⇣

Unwanted movement escapes suppression

6. MECHANISM → RECOGNITION

Mechanism → recognition: reduced motor inhibition → chorea, irregular flowing involuntary movements. Loss of sustained motor control → inability to maintain tongue protrusion or a steady grip. Cortical–striatal injury → slowed processing, planning difficulty and impaired judgement. Network injury also causes apathy, irritability or depression, which can precede conspicuous movements. Progressive bulbar dysfunction → dysarthria, swallowing difficulty and aspiration. Increased expenditure plus swallowing problems → weight loss. Juvenile disease can feature rigidity and seizures rather than prominent chorea. Suicidal thinking requires direct assessment and support.

7. INVESTIGATIONS

Investigations: establish progressive motor, cognitive and functional changes, examine eye movements and gait, obtain a careful three-generation family history, and review drugs and acquired chorea causes. A negative known family history does not exclude disease. Diagnostic repeat testing follows informed consent and genetic counselling. Brain magnetic resonance imaging may show caudate and cortical atrophy but does not replace genetic confirmation. Consider treatable mimics, including Wilson disease, thyroid disease and drug-related movements, when the presentation warrants them. Predictive testing in an asymptomatic adult is a separate counselling-led process; do not conduct routine predictive testing in children. Symptomatic juvenile diagnostic evaluation is different.

8. MANAGEMENT FLOW

Management flow: define the person's disabling problem → rehabilitation and nutrition for everyone → add targeted treatment → reassess benefit and harms. Physiotherapy supports balance; occupational therapy supports daily function; speech and swallowing assessment guides food texture and aspiration precautions. Treat troublesome chorea with specialist-selected dopamine-depleting medication, such as tetrabenazine, or an appropriate antipsychotic when relevant coexisting symptoms favour it. Monitor depression, suicidality, sedation, rigidity, swallowing and interactions; reducing movement is not automatically improving function. Provide mood treatment, communication support, caregiver support, advance planning and reproductive counselling. Current routine treatment is symptomatic; do not promise a cure or reversal of the genetic disorder.

MEMORY AID

Movement, cognition and behaviour change; testing requires counselling.

→ Arrows show consequences, not individual synaptic connections.

Foundation 21 • Medical foundations • Source-checked supplementary teaching • September 2026.
Laboratory ranges and treatment protocols require local context.

Huntington disease. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-48.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P14

HUNTINGTON DISEASE: PSYCHIATRY ACROSS THE COURSE

Psychiatric companion • Protect safety, function and the person's choices

PARALLEL SUPPORTS

Trigger reduction → calmer care

Prompts → initiation

Mood treatment → reduced depressive burden



A NETWORK DISORDER WITH A FAMILY CONTEXT

Huntington disease progressively affects striatal and cortical networks involved in movement, cognition, motivation and behaviour. Depression, irritability, apathy and perseveration may precede obvious chorea. Psychiatric symptoms alone do not diagnose Huntington disease: assess the syndrome, neurological findings, family history and alternative causes, with specialist input.

The inherited condition affects the wider family through uncertainty, caregiving and testing decisions. A predictive genetic result does not specify the exact age of onset or future symptom pattern. Explain this uncertainty without minimising its consequences.

ASSESS CHANGE AND RISK

Compare current behaviour with the person's baseline. Ask about depression, pleasure, sleep, irritability, aggression, psychosis, substance use and suicidal thinking. Seek collateral information with consent, especially when insight, executive function or communication is impaired. Assess driving, money management, medication use, relationships, nutrition and swallowing as part of real-world function.

Suicide risk requires repeated review, particularly around diagnosis, functional loss and major transitions; these are heightened-risk contexts, not the only times risk occurs. Ask about intent, planning and access to means. Agree a practical safety plan and urgent support when needed. New behavioural deterioration also warrants a search for pain, infection, constipation, sleep disruption and adverse effects.

DISTINGUISH APATHY, DEPRESSION AND PERSEVERATION

Apathy is reduced initiation and motivation, often without sadness. Depression adds persistent low mood or loss of pleasure, hopelessness and other syndromal features. They can coexist. A person who starts little may still enjoy an activity once another person helps initiate it.

Perseveration is difficulty shifting from a thought or action; it is not always an intrusive, resisted obsession. Explain this distinction to carers so repeated questions are not interpreted simply as deliberate provocation. Reduce unnecessary choices, use a stable routine and redirect calmly instead of prolonged argument.

MEMORY AID

Check mood, reduce triggers, support initiation, protect choice.

TREAT MOOD AND BEHAVIOUR WITH A TARGET

Adapt psychological support to cognitive abilities, using simple plans, repetition and caregiver assistance with permission. Antidepressants may help a depressive syndrome and some anxiety or obsessive symptoms; choice depends on previous response, sleep, appetite, interactions and adverse effects. Review suicidality, activation, falls and whether function improves, not only whether the patient seems quieter.

For irritability, identify triggers such as rushed care, noise, frustration or fatigue. Slow communication, offer breaks and modify demands. Medication may be needed when severe symptoms persist, especially if aggression or psychosis creates danger. Antipsychotic choice should consider coexisting chorea but also sedation, swallowing difficulty, metabolic effects and worsening rigidity. Avoid using restraint or sedation as a substitute for understanding triggers and supporting carers.

No medicine reliably reverses apathy. Scheduled activities, external prompts and achievable goals are central; review sedating treatment and coexisting depression. Persistent psychosis warrants specialist treatment and ongoing assessment for medical contributors.

CHOREA TREATMENT CAN CHANGE MENTAL STATE

Medicines that reduce monoamine storage, such as tetrabenazine, can worsen depression and suicidality. Review current and previous depression before treatment, follow product-specific contraindications, and monitor mood during titration. Antipsychotics may reduce chorea when another indication is present, but motor benefit alone does not make their adverse effects acceptable. Coordinate neurological and psychiatric prescribing so the trade-offs are explicit.

COUNSELLING, CAPACITY AND CONTINUITY

Predictive testing is voluntary and follows specialist counselling before and after testing. Routine predictive testing of asymptomatic children is generally deferred; diagnostic testing of a symptomatic young person is a separate clinical question. Do not test an adult merely at a relative's request.

Assess capacity for the particular decision, support communication and plan future care while the person can participate. Include carers' needs, respite, occupational support and safeguarding. Review treatment goals as the disease changes; preserving comfort, dignity and relationships remains active care.

P14 • Psychiatric companion • Supplementary teaching.

Huntington disease psychiatry. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-48.

Credits and references: retained within the illustration.

IV - 49

Enumerate the neuropsychiatric manifestations of Parkinson's disease. Enumerate the drug induced psychological issues in Parkinson's disease. How will you manage them? [3+2+5]

3 + 2 + 5 | MOVEMENT AND DEGENERATIVE DISORDERS IN PSYCHIATRY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	The manifestations	Enumerated by domain, not as one long list
2	Drug induced problems	Which drug produces which problem
4	Management	Subtract before adding, then the two safe agents
1	The standing rule	What must never be stopped abruptly

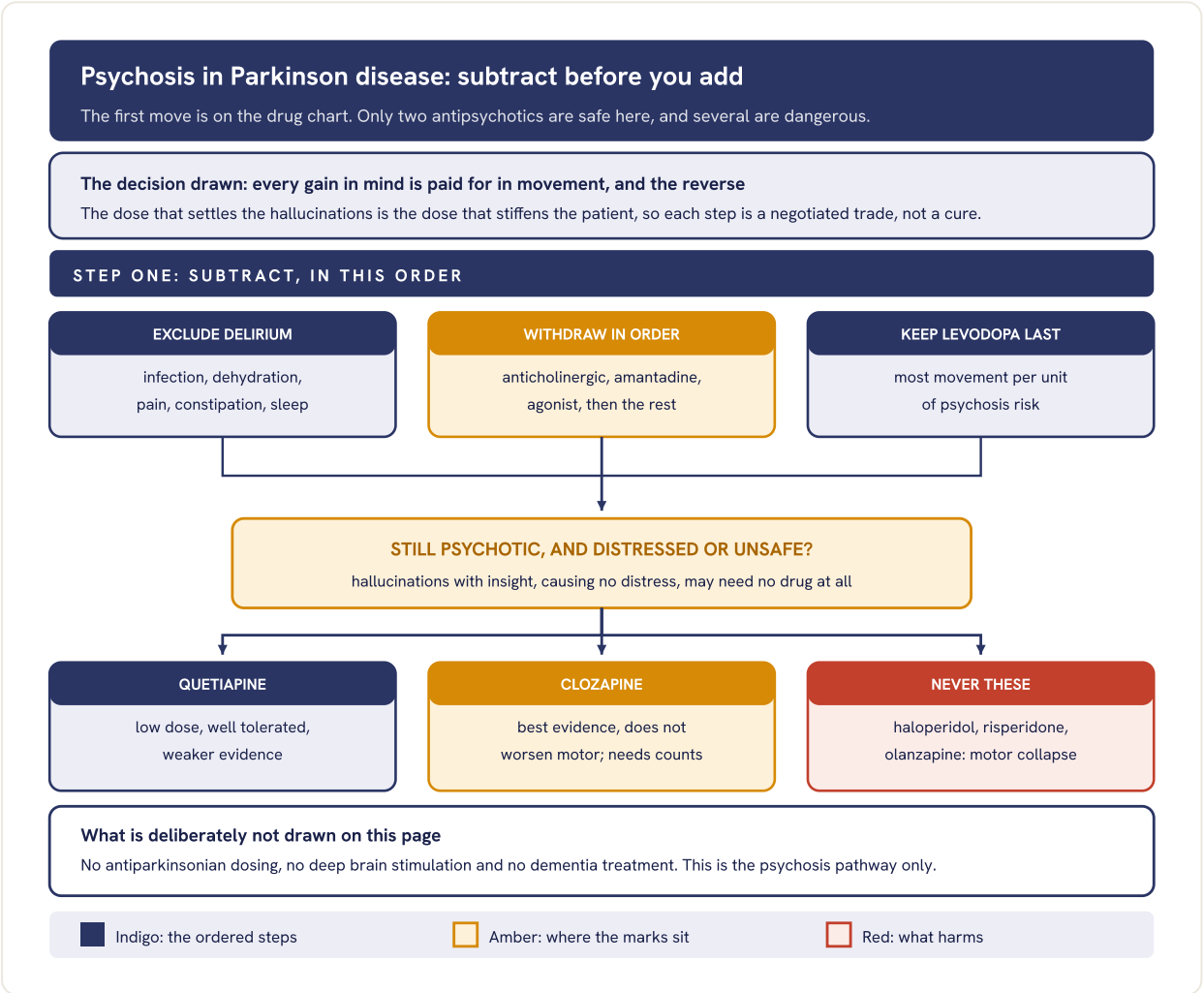


Fig 049.1 The withdrawal order that treats psychosis before any antipsychotic is added, the threshold for treating at all, and the two agents that are safe when it is crossed.

- **The manifestations, by domain.** Mood: depression, anxiety tied to wearing-off, apathy. Cognitive: executive and visuospatial decline, and dementia late. Perceptual: visual hallucinations and delusions. Sleep: rapid eye movement sleep behaviour disorder, insomnia, daytime somnolence. Behavioural: impulse control disorders and punding.

- **Which drug does what.** Dopamine agonists carry the highest risk of impulse control disorders, hallucinations and sudden sleep attacks. Levodopa causes psychosis, dyskinesia and, in excess use, dopamine dysregulation syndrome. Anticholinergics cause confusion and memory impairment, badly in the old. Amantadine causes hallucinations and confusion.
- **The standing rule.** Dopaminergic drugs are never stopped abruptly. Sudden withdrawal can precipitate a parkinsonism-hyperpyrexia syndrome that resembles neuroleptic malignant syndrome and can kill. Reduce stepwise, and watch the motor state at every step.

WHAT EARNS THE MARKS

- **The withdrawal order, written out.** It is the highest-value line on the page.
- **Delirium excluded first,** before any drug is blamed or added.
- **Clozapine and quetiapine named, and the dangerous agents named too.**
- **The rule against abrupt withdrawal,** with the syndrome it prevents.

SEE ALSO Slot IV - 47 psychiatric features of Parkinson disease Slot IV - 52 Parkinson disease and psychosis **SPRINT** Day 35, Neuropsychiatry of systemic & neurological disease

IV - 50

Write in detail about the Neuropsychiatric aspect of multiple sclerosis. [10]

10 | ASKED AT 15 MARKS | MOVEMENT AND DEGENERATIVE DISORDERS IN PSYCHIATRY

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

SKELETON

2	Why they occur	Lesion burden and disease context, both
4	The syndromes	Five, with the two that are specific to this disease
2	Iatrogenic	Steroids and the disease-modifying drugs
2	Management	What is treated, and how it is measured

SYNDROME	WHAT IT LOOKS LIKE	THE POINT TO MAKE
Depression	The commonest psychiatric diagnosis in multiple sclerosis	**Suicide risk is raised well above the general population**
Euphoria	Settled cheerful unconcern about real disability	Advanced disease, heavy frontal lesion load; not mania
Pseudobulbar affect	**Involuntary laughing or crying, detached from felt mood**	Distressing, often mistaken for lability of mood
Cognitive impairment	Processing speed first, then memory and executive function	**Occurs early, and tracks physical disability poorly**
Fatigue	The single commonest symptom of the disease	A symptom in its own right, not a depressive one

Amber marks the three that carry most of the marks: the suicide risk, the affect that is not a mood, and cognitive impairment that appears early and does not follow the walking stick.

- **Iatrogenic, and the rarer syndromes.** Psychosis and bipolar disorder are uncommon but more frequent than in the general population, and steroids and lesion site are considered first. High dose corticosteroids for relapse cause insomnia, elevated or irritable mood and occasionally psychosis, and they are the first thing to ask about in a patient with sudden mood change. Interferon beta has historically been linked with depression, though the association is debated; either way, depression here is treated rather than the treatment abandoned.
- **Management.** Depression responds to antidepressants and to psychological therapy. Pseudobulbar affect responds to antidepressants at doses lower than depression needs. Fatigue is treated as fatigue: exercise, sleep and heat avoidance.

PITFALL

Fatigue, slowed thinking, poor concentration and disturbed sleep all belong to multiple sclerosis itself. A depression scale weighted towards somatic items will therefore overdiagnose, so the diagnosis is made on mood, anhedonia, hopelessness and guilt instead.

WHAT EARNS THE MARKS

- **Euphoria and pseudobulbar affect distinguished from mania and from lability.**
- **Cognitive impairment placed early,** and uncoupled from physical disability.
- **Steroids named as a cause of acute mood change.**

SEE ALSO Slot IV - 51 aetiology, features and management of multiple sclerosis

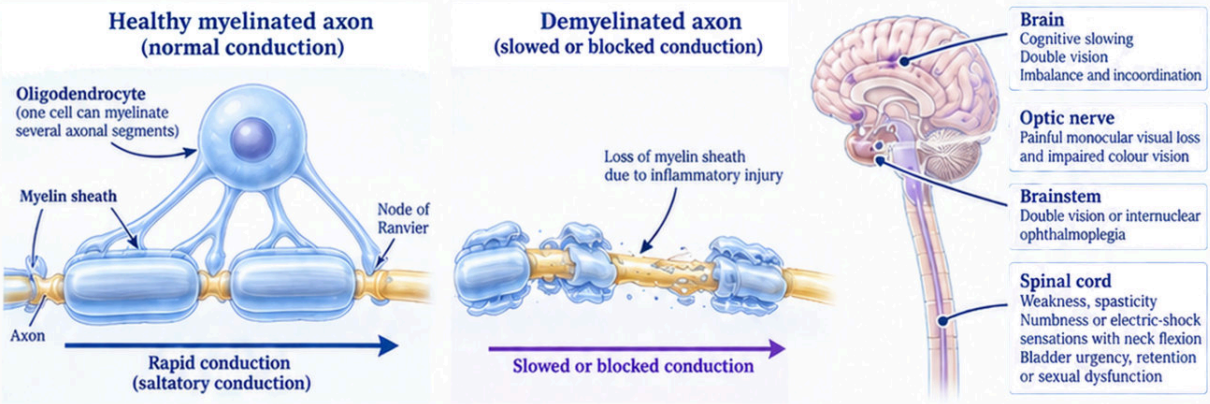
Slot IV - 47 psychiatric features of Parkinson disease SPRINT Day 35, Neuropsychiatry of systemic & neurological disease

WEAVE 100 · PAPER IV · F22

MYELIN AND MULTIPLE SCLEROSIS

INSULATION, INFLAMMATION AND CONDUCTION

Foundation 22 • Normal function → mechanism → clinical recognition → management



Normal function

Normal: an axon carries electrical impulses. Oligodendrocytes wrap central nervous system axons in myelin; one oligodendrocyte can myelinate several axonal segments. Schwann cells perform the corresponding task in peripheral nerves. Myelin increases membrane resistance and reduces capacitance, allowing current to travel efficiently between nodes of Ranvier. At each node, voltage-gated sodium channels regenerate the impulse: saltatory conduction. Myelin-producing cells also support axonal metabolism.

What goes wrong

What goes wrong: multiple sclerosis is an immune-mediated disease affecting brain, optic nerves and spinal cord. Inflammatory injury strips myelin and damages oligodendrocytes; axonal injury and neurodegeneration contribute to lasting disability. Demyelination slows or blocks conduction. Remyelination and resolution of inflammation permit partial recovery, while accumulated axonal loss limits recovery. Susceptibility reflects genetic and environmental factors, including Epstein–Barr virus exposure, smoking and other influences; it is not a simple Mendelian disorder, and no single exposure proves causation in an individual.

Mechanism → symptoms

Optic nerve inflammation → painful monocular visual loss and impaired colour vision.
Brainstem pathway injury → double vision or internuclear ophthalmoplegia.
Corticospinal injury → weakness, brisk reflexes and spasticity.
Sensory tract injury → numbness or electric-shock sensations with neck flexion.
Cerebellar injury → imbalance and incoordination.
Spinal autonomic pathway injury → bladder urgency, retention or sexual dysfunction.
Distributed network dysfunction → fatigue and cognitive slowing.

Warming can temporarily worsen old symptoms because demyelinated axons conduct less reliably; this does not automatically mean a new inflammatory attack.

Course

Course: relapsing-remitting disease has attacks with variable recovery. **Secondary progressive** disease follows a relapsing course with gradual disability accumulation. Primary progressive disease progresses from onset. Disease activity and progression must be described separately: new lesions or relapses can coexist with progression.

Assessment

Assessment: seek a typical neurological syndrome and objective findings; exclude infection, vascular lesions, vitamin B12 deficiency and other inflammatory demyelinating disorders. Magnetic resonance imaging assesses typical lesion location and activity. Cerebrospinal fluid oligoclonal immunoglobulin G bands indicate intrathecal antibody production but are not specific to multiple sclerosis. Optical coherence tomography and visual evoked potentials can support optic nerve involvement. Test aquaporin-4 or myelin oligodendrocyte glycoprotein antibodies when the clinical pattern suggests an alternative disorder.

Criteria update

Criteria update: the 2024 McDonald revisions, published in 2025, add the optic nerve as a fifth anatomical location and allow specified supportive biomarkers, including cerebrospinal fluid kappa free light chains and selected imaging signs. Diagnosis requires the full specialist algorithm and exclusion of better explanations; "two attacks are always necessary" is incorrect. There is no universal normal blood test or screening magnetic resonance lesion count that diagnoses this disease.

Management has three parallel jobs.

1. A significant relapse

Exclude infection and a temporary worsening first; high-dose methylprednisolone can accelerate recovery. National Institute for Health and Care Excellence guidance uses oral 500 milligrams daily for five days; specialist intravenous treatment is an alternative in selected situations. Monitor glucose, infection, sleep and mood. Severe steroid-resistant attacks may require plasma exchange.

2. Disease course

Early specialist disease-modifying therapy according to activity, prognostic risk, pregnancy plans, infection risk and monitoring capacity; corticosteroids are not long-term disease modification.

3. Symptoms and function

Individualised exercise and rehabilitation, fatigue/heat management, bladder review, spasticity and pain treatment, vaccination planning and smoking cessation. Review function, relapses, imaging and treatment toxicity regularly. Avoid attributing every new symptom to multiple sclerosis.

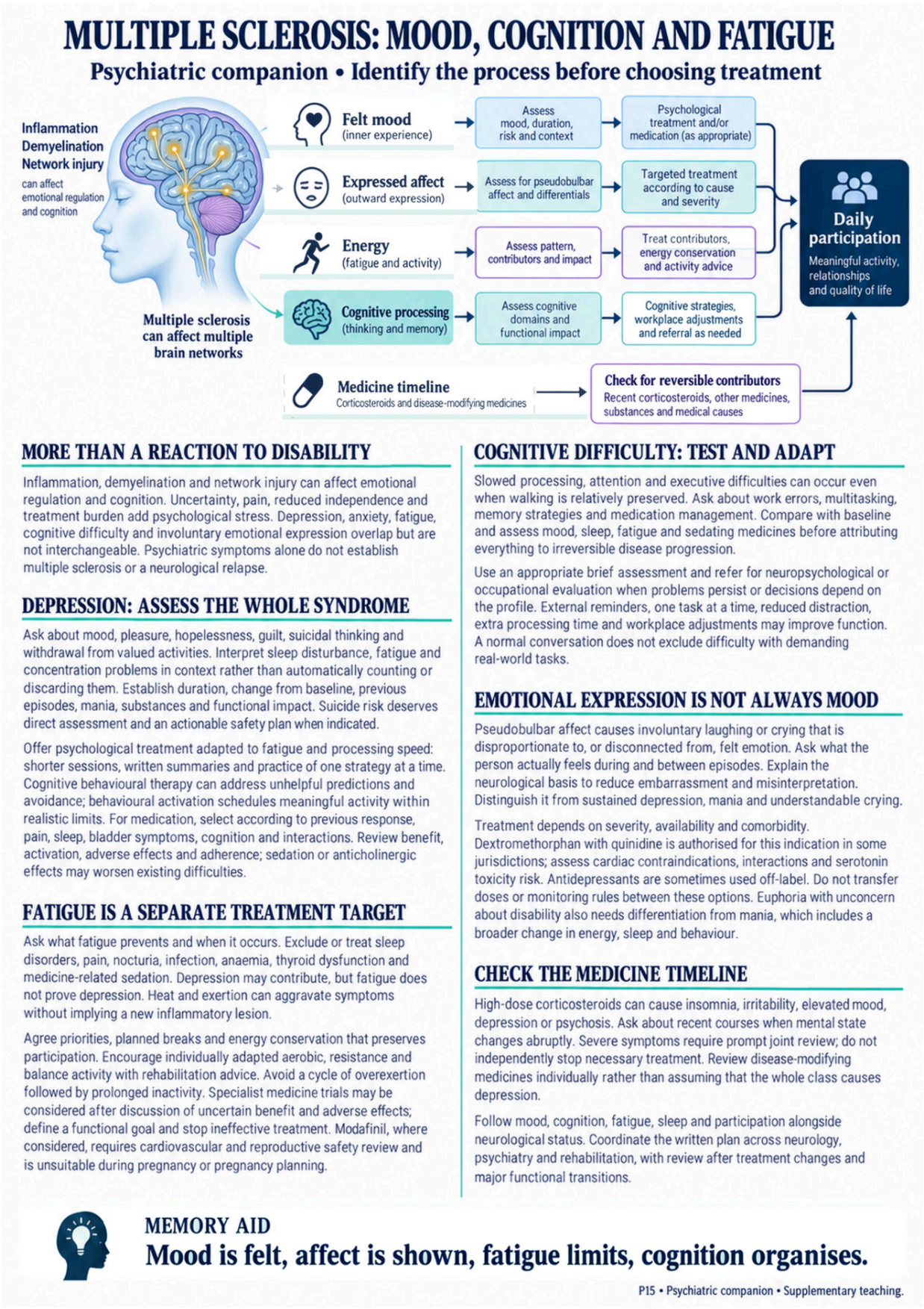
MEMORY AID — **Separate the relapse, the disease course and the symptom burden.**

Foundation 22 • Medical foundations • Source-checked supplementary teaching • September 2026.
Laboratory ranges and treatment protocols require local context.

Myelin and multiple sclerosis. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-50.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P15



Multiple sclerosis psychiatry. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-50.

Credits and references: retained within the illustration.

IV - 51

Etiology, clinical features and management of Multiple Sclerosis. [10]

10 MOVEMENT AND DEGENERATIVE DISORDERS IN PSYCHIATRY

KUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 Aetiology	Immune, genetic, environmental, in one line each
3 Clinical features	The patterns, the presentations, and the diagnostic rule
4 Management	Relapse, disease course, symptoms: three separate jobs
1 The psychiatric arm	Where a psychiatrist actually joins the team

AETIOLOGY AND THE DIAGNOSTIC RULE

An immune mediated demyelinating disease of the central nervous system. Susceptibility is genetic, with the HLA-DRB1 region carrying most of it, and environmental: low vitamin D, Epstein-Barr virus, smoking. Diagnosis rests on events disseminated in space and in time, with imaging and oligoclonal bands.

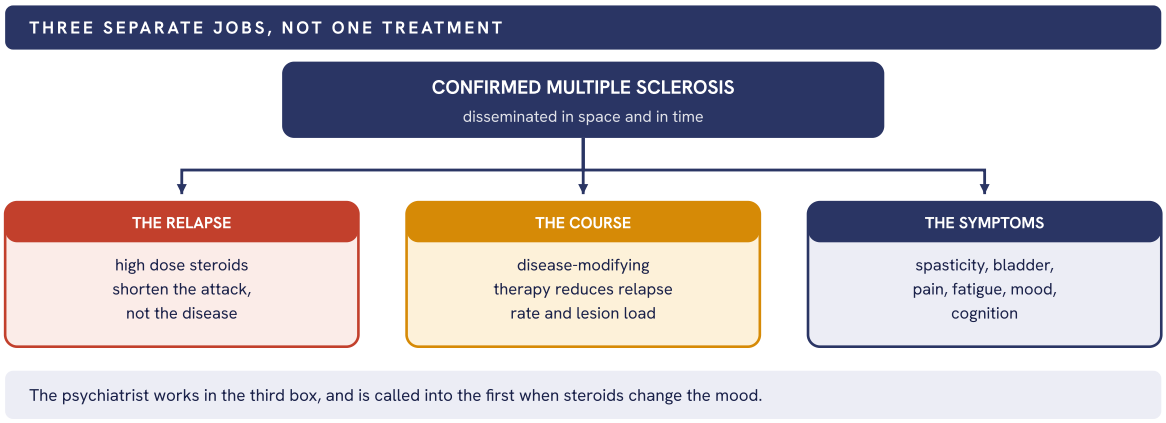


Fig 051.1 Management separated into the three jobs it actually is.

- **Patterns and presentations.** Relapsing-remitting is commonest at onset; many later become secondary progressive, and a minority are primary progressive. Presentations include optic neuritis, internuclear ophthalmoplegia, ataxia and bladder urgency.
- **Where the psychiatrist joins.** Depression, cognitive impairment, pseudobulbar affect, fatigue, and steroid induced mood change. Adjustment to a relapsing illness in a young adult, with its effect on work and family, is treatment rather than a side conversation.

WHAT EARNS THE MARKS

- **Dissemination in space and time,** stated as the diagnostic rule rather than implied.
- **The three jobs kept separate.** Steroids treat the relapse and not the disease, and saying so is the point.
- **The psychiatric contribution named,** which is what makes this a psychiatry answer.

SEE ALSO Slot IV - 50 neuropsychiatric aspects of multiple sclerosis Slot IV - 87 neuropsychiatric manifestations of HIV

SPRINT Day 35, Neuropsychiatry of systemic & neurological disease

IV - 52

a) Relationship between Parkinsons and psychosis. b) Write factors which decide assessment and management of depression in a patient with stroke. [5+5]

5 + 5 | MOVEMENT AND DEGENERATIVE DISORDERS IN PSYCHIATRY

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	**Parkinson and psychosis**	A relationship that runs in three directions
2	**What follows from it**	The two safe agents, and the ones that harm
3	**Depression after stroke**	The factors that decide the assessment
2	**Managing it**	What is treated, when, and alongside what

DIRECTION	WHAT HAPPENS	WHAT IT MEANS IN PRACTICE
Disease causes psychosis	Visual hallucinations and delusions arise from the illness itself, more with dementia and long duration	**Not simply a drug side effect**
Treatment causes psychosis	All dopaminergic drugs can, agonists most of all	Review the chart before adding anything
Antipsychotics cause parkinsonism	Dopamine blockade produces the motor syndrome, and unmasks or worsens the disease	**The relationship runs both ways**

Amber marks the two halves that make this a relationship, not a side effect. The safe agents are quetiapine and clozapine; haloperidol, risperidone and olanzapine harm.

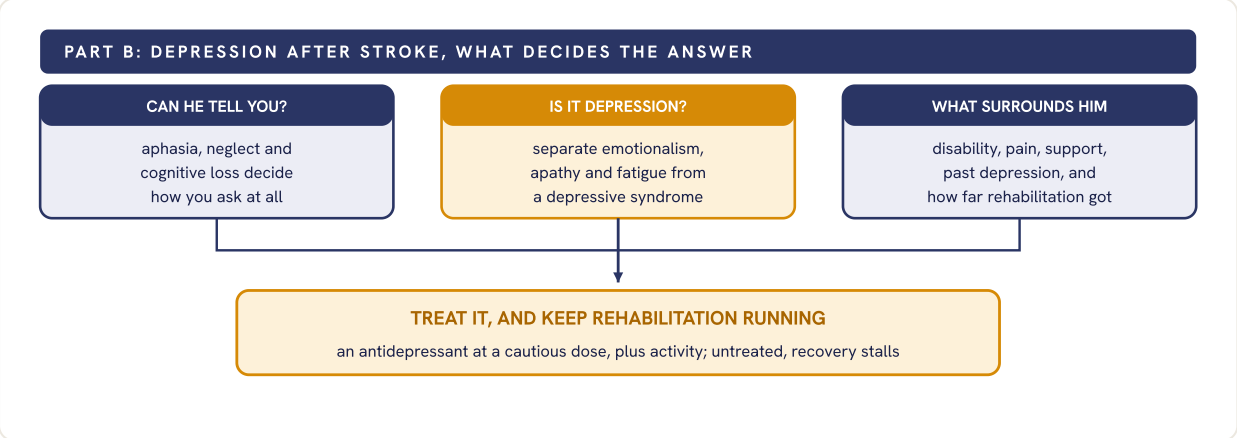


Fig 052.1 What decides the assessment, and treatment that runs alongside rehabilitation.

- **The two traps.** Emotionalism after stroke, sudden crying with little felt sadness, is not depression. And bleak realism about disability is not automatically illness: the diagnosis rests on anhedonia and hopelessness.

WHAT EARNS THE MARKS

- **Part (a) answered as a two-way relationship,** not as a list of drug side effects.
- **Communication named as the first assessment factor.** Aphasia changes the method.
- **Depression tied to rehabilitation outcome,** which is why it is treated promptly.

SEE ALSO Slot IV - 49 drug induced problems in Parkinson disease Slot IV - 41 cerebral small vessel disease **SPRINT** Day 35, Neuropsychiatry of systemic & neurological disease Day 34, Stroke, TBI, delirium & focal brain syndromes

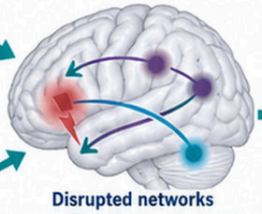
WEAVE 100 · PAPER IV · C08

c08 — Stroke: psychiatric and cognitive consequences

Companion C08

Premorbid vulnerability
- Prior small-vessel disease
- Previous mood disorder
- Disability, pain and social context

Stroke lesion
- Interrupted blood supply or haemorrhage
- Network disruption



Disrupted networks
A small strategic lesion can disconnect an important network

Mood Cognition Behaviour

Depression
Persistent low mood or loss of interest

Apathy
Reduced initiation, interest or goal-directed behaviour

Emotionalism
Disproportionate or difficult-to-control laughing or crying

Delirium
Acute, fluctuating disturbance of attention and awareness

Bedside communication support
Short sentences, supported choices, pictures or yes/no responses

Valid assessment
Consider premorbid state, use adapted tools, assess specific domains

Individualized rehabilitation and treatment
Support recovery, address psychiatric and cognitive consequences

1. Overview

- Normal mood, motivation and thinking depend on distributed cortical and subcortical networks, adequate perfusion and communication between regions.
- Stroke abruptly injures tissue through interrupted blood supply or haemorrhage.
- A small strategic lesion can disconnect an important network; a larger lesion may affect movement, language, attention and emotion together.
- Prior small-vessel disease, previous mood disorder, disability, pain and social context influence recovery.
- Lesion location helps interpretation but does not deterministically predict a psychiatric syndrome.

2. Depression: more than understandable sadness.

- Persistent low mood or loss of interest, hopelessness, guilt and suicidal thinking deserve assessment even when disability provides an understandable trigger.
- Sleep, appetite, energy and psychomotor changes overlap with stroke itself, so use the whole syndrome and change from baseline.
- Depression can reduce rehabilitation participation and quality of life.
- Screen repeatedly during recovery rather than assuming one early assessment is sufficient.
- A positive questionnaire requires a clinical interview, not an automatic diagnosis or prescription.

3. Look-alikes and coexistence.

- Apathy is reduced initiation, interest or goal-directed behaviour; the patient need not feel sad or worthless.
- Emotionalism involves disproportionate or difficult-to-control laughing or crying and may occur without sustained depressed mood.
- Fatigue is subjective exhaustion and can coexist with sleep disorders, medication effects or depression.
- Anxiety may involve recurrence fears, avoidance or panic.
- Delirium produces an acute, fluctuating disturbance of attention and awareness and prompts investigation for a medical cause.
- These conditions can coexist; do not force every symptom into one label.

MEMORY AID

Support communication, distinguish syndromes, restore function.

4. Cognition and behaviour.

- Slowed processing, impaired attention and executive dysfunction may undermine medication use, finances and planning.
- Memory problems can reflect poor encoding from inattention as well as damage to memory circuits.
- Aphasia impairs language, not necessarily intelligence or decision-making capacity.
- Neglect reduces attention to one side of space or body and can affect eating, dressing or safety.
- Mania, psychosis or major personality change may occur but are less common; establish timing, prior history, seizures, medicines and other causes before attributing them to stroke.

5. Bedside HOW.

- First establish a reliable communication method: short sentences, supported choices, pictures or yes/no responses checked for consistency.
- Ask the patient and an informant about premorbid mood, cognition, independence and current change.
- Test attention before interpreting memory; use cancellation or line bisection when neglect is suspected, and assess language separately.
- Adapt validated mood and cognitive tools for aphasia or sensory deficits.
- Ask directly about suicidal thoughts, hopelessness and safety.
- Observe actual rehabilitation tasks, not just a global cognitive score.
- Assess capacity for the particular decision with appropriate communication support.

6. Investigations follow the question.

- New focal deficits or sudden deterioration require urgent stroke reassessment.
- Review imaging, stroke type and location, neurological findings, oxygenation, glucose, medicines and infection.
- Consider electrolytes, thyroid function or other targeted tests when the presentation suggests them.
- Electroencephalography is useful when seizures are suspected, not routinely for every mood symptom.
- There is no blood-test cutoff or scan pattern that diagnoses poststroke depression.

7. Management supports recovery.

- Coordinate stroke prevention, rehabilitation, sleep and pain treatment, meaningful activity, communication therapy and caregiver support.
- Offer adapted psychological treatment and consider an antidepressant for a diagnosed depressive disorder according to severity, preference and interactions.
- Monitor falls, sodium disturbance and bleeding risk when relevant to the selected medicine and antithrombotic regimen.
- Routine fluoxetine solely to improve neurological recovery is not supported: large trials found no functional benefit and increased fractures.
- This does not prohibit treating established depression.
- Persistent emotionalism or severe apathy merits specialist assessment.
- Urgent suicidal risk, delirium or new neurological signs require immediate action.

Supplementary medical teaching • September 2026

Stroke psychiatric aspects. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-52.

Credits and references: retained within the illustration.

IV - 53

Describe the aetiology, neuropathology, clinical manifestations, course, prognosis and management of Parkinson's disease. [10]

10 | ASKED AT 20 MARKS | MOVEMENT AND DEGENERATIVE DISORDERS IN PSYCHIATRY

NTRUHS, RGUHS, TN-MGRMU - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2	**Aetiology and pathology**	What is lost, what accumulates, and what is inherited
3	**Clinical manifestations**	Motor and non-motor, both named
2	**Course and prognosis**	The staging, and what predicts a faster decline
3	**Management**	Motor, non-motor and surgical, kept apart

AETIOLOGY AND NEUROPATHOLOGY

Degeneration of dopaminergic neurons in the substantia nigra pars compacta, with Lewy bodies containing aggregated alpha-synuclein. Motor signs appear once much of the nigrostriatal projection is already lost. Mostly sporadic, with age the dominant risk factor; a minority are familial.

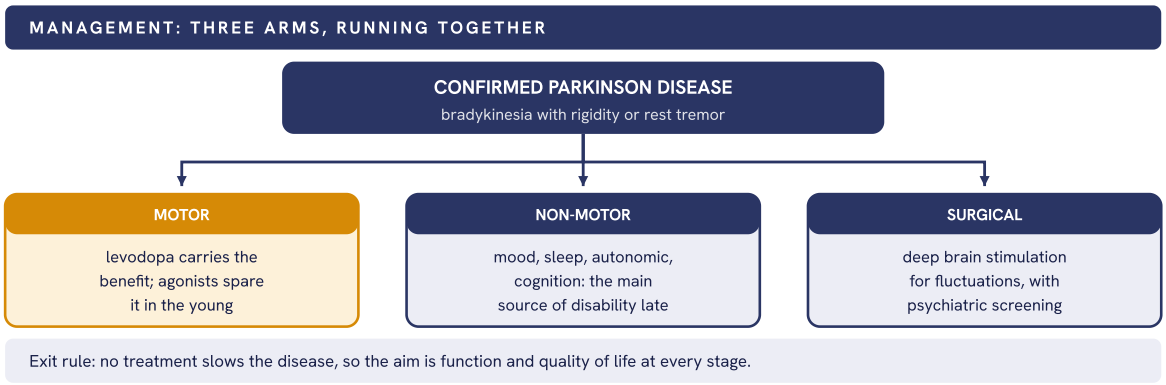


Fig 053.1 The three arms of management, with the non-motor arm marked as the main source of disability once the disease is established.

- **Manifestations.** Motor: bradykinesia is required, with rest tremor, rigidity and later postural instability; onset is asymmetrical. Non-motor: hyposmia, constipation, sleep behaviour disorder, depression and anxiety, often years earlier.
- **Course and prognosis.** Progressive, staged by the Hoehn and Yahr scale from unilateral disease to wheelchair dependence. Poorer with older onset, a rigid-akinetic picture, early falls, early cognitive impairment and poor levodopa response. The last two also warn the diagnosis may not be Parkinson disease.

WHAT EARNS THE MARKS

- **Alpha-synuclein and the Lewy body named,** with the site of degeneration.
- **Non-motor features given equal weight,** and placed before onset as well as after.
- **Prognostic markers listed,** with the two that should raise doubt about the diagnosis.

SEE ALSO Slot IV - 47 psychiatric features of Parkinson disease Slot IV - 49 drug induced problems **SPRINT** Day 35, Neuropsychiatry of systemic & neurological disease

AREA 07 OF 15 · P4-A05

Endocrine and metabolic psychiatry

6	6.2%	IV-54	IV-59
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 HIGH 2 SINGLE 3

REVISION ORDER

The three thyroid slots together, they are one answer at three widths and the lithium rule is stated identically on all three. Metabolic syndrome next, it is the one that carries the thresholds and they have to be exact. Diabetes and obesity last, and they share a behavioural spine.

WHAT IS IN THIS AREA

The thyroid in three slots: the psychiatric manifestations of thyroid disorders, hypothyroidism on its own, and a slot that adds the parathyroid axis. Then the metabolic limb, metabolic syndrome set against the second-generation antipsychotics with the first-generation comparison, obesity with the strategies for managing it, and the psychological and psychiatric aspects of diabetes mellitus.

IV - 54

Psychiatric manifestations of Thyroid disorders. [10]

10 ENDOCRINE AND METABOLIC PSYCHIATRY

KUHS, NTRUHS, RGUHS - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2 The principle	Thyroid state mimics, worsens and modifies psychiatric illness
3 Underactive	Depression, slowing, cognition, and the psychotic extreme
3 Overactive	Anxiety and mania, and the misleading elderly form
2 What to do	Who to test, what to correct, and the lithium rule

THYROID STATE	PSYCHIATRIC PICTURE	THE PRESENTATION THAT MISLEADS	TEST
Underactive	Depression, apathy, slowing, poor concentration, hypersomnia	A reversible dementia-like picture in an older patient	**TSH raised, free T4 low**
Severely underactive	Paranoid and hallucinatory psychosis, the picture called myxoedema madness	Read as late-onset schizophrenia	Same, with a long history
Overactive	Anxiety, irritability, insomnia, lability, distractibility	Diagnosed as panic disorder or as mania	**TSH suppressed, free T4 raised**
Overactive in old age	Apathy, low mood, weight loss, withdrawal	**Apathetic thyrotoxicosis, read as depression**	Check the pulse rhythm
Subclinical underactive	Low mood, and a poorer antidepressant response	Dismissed because free T4 is normal	TSH raised, free T4 normal

Amber cells are the discriminators. Each row is a presentation that reaches psychiatry before it reaches endocrinology.

- **Who to test.** First-episode psychosis, treatment-resistant depression, rapid cycling, new cognitive impairment, anxiety with prominent somatic features, and any postpartum presentation.
- **The lithium rule.** Lithium causes hypothyroidism and goitre, so check thyroid function at baseline and periodically. Finding it is a reason to add thyroxine, not a reason to stop the lithium.

PITFALL

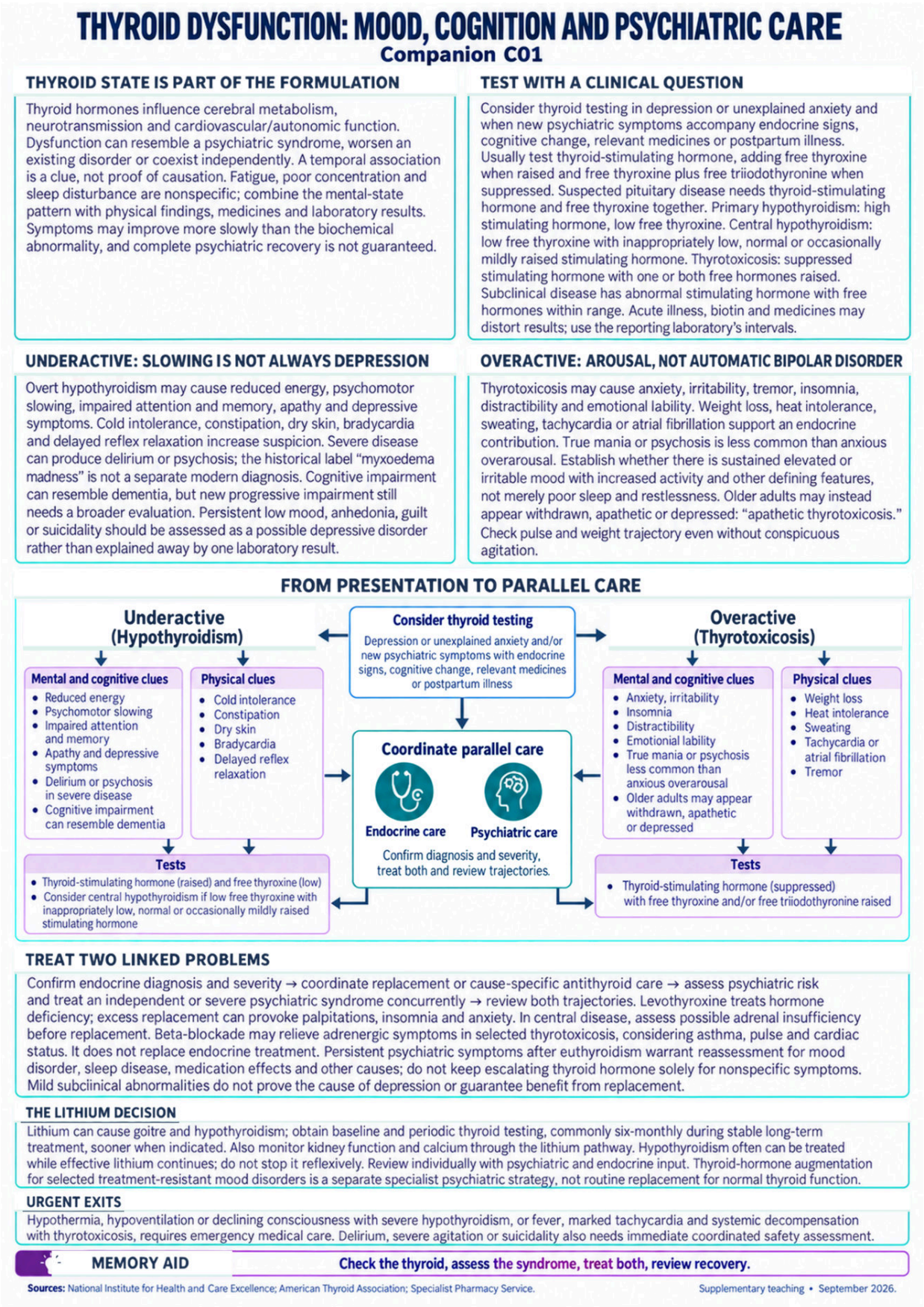
Correcting the thyroid state and stopping there. Symptoms improve with replacement but often lag behind the biochemistry, so the psychiatric illness still needs treating and reviewing on its own timetable.

WHAT EARNS THE MARKS

- **Both directions answered,** with equal weight; most answers give hypothyroid depression and stop.
- **Apathetic thyrotoxicosis named,** which is the discriminating fact on this page.
- **A testing rule, not a testing list.** Naming who to screen shows clinical judgment.

SEE ALSO Slot IV - 56 hypothyroidism Slot IV - 59 thyroid and parathyroid dysfunction SPRINT Day 33, Endocrine & metabolic psychiatry

WEAVE 100 · PAPER IV · C01



Thyroid psychiatric aspects. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-54.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · F01

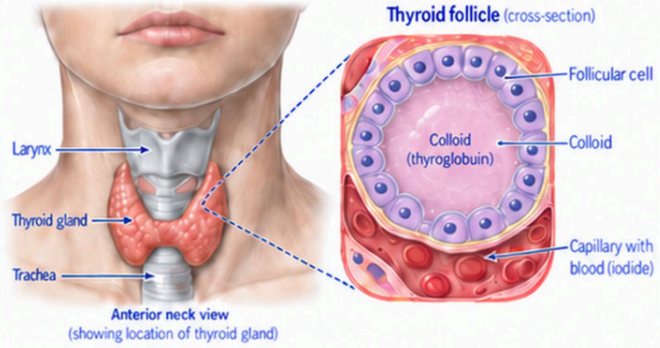
THYROID: THE BODY'S METABOLIC REGULATOR

Foundation 01 • Normal function → disease mechanism → clinical pattern

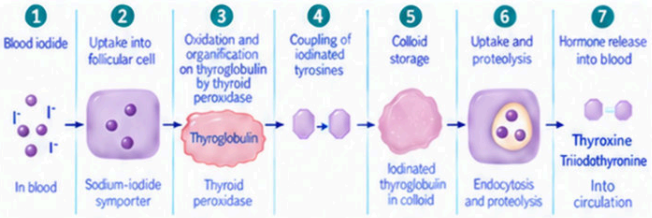
NORMAL PHYSIOLOGY

The thyroid is a follicular gland in the neck. **Labelled synthesis:** blood iodide → uptake into follicular cell → oxidation and organification on thyroglobulin by thyroid peroxidase → coupling of iodinated tyrosines → colloid storage → uptake and proteolysis → hormone release into blood.

The gland releases mainly thyroxine; tissues convert much of it into the more active triiodothyronine. Most circulating hormone is protein-bound; free hormone is available to tissues. Thyroid hormone changes gene expression, energy use and heat production, supports growth and brain development, and increases the heart's responsiveness to sympathetic stimulation.



Thyroid hormone synthesis (in follicular cell and colloid)

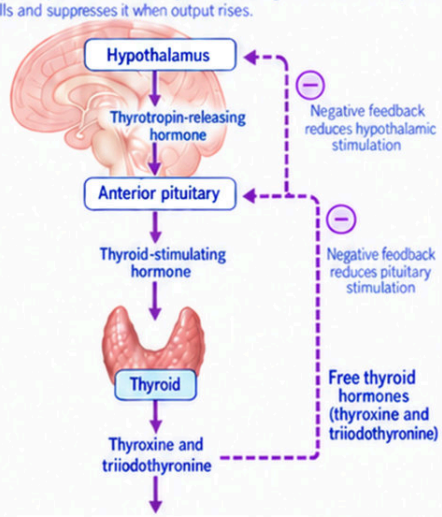


THE CONTROL LOOP

Hypothalamus → thyrotropin-releasing hormone → anterior pituitary → thyroid-stimulating hormone → thyroid → thyroxine and triiodothyronine → tissue effects.

Free thyroid hormones → negative feedback → reduce hypothalamic and pituitary stimulation.

An intact pituitary therefore raises thyroid-stimulating hormone when thyroid output falls and suppresses it when output rises.



Tissue effects

- Gene expression
- Energy use and heat production
- Growth and brain development
- Increased cardiac responsiveness to sympathetic stimulation

TOO LITTLE: HYPOTHYROIDISM

Primary gland failure: autoimmune Hashimoto disease, iodine deficiency, thyroid surgery or radioiodine; some medicines also interfere. Pituitary/hypothalamic disease causes central hypothyroidism.

- Reduced heat production → cold intolerance
- Slower gut motility → constipation
- Reduced cardiac drive → bradycardia
- Reduced energy turnover → fatigue and modest weight gain
- Dry skin, hair thinning and delayed relaxation of tendon reflexes support the pattern.
- Tissue glycosaminoglycan accumulation contributes to non-pitting puffiness.
- Menstrual disturbance and impaired fertility can occur.
- Symptoms are nonspecific: confirm the biochemical pattern.

TOO MUCH: THYROTOXICOSIS

Thyrotoxicosis means excess thyroid-hormone action from any cause. **Hyperthyroidism** specifically means increased synthesis: **Graves** disease stimulates the thyroid-stimulating-hormone receptor; toxic nodules produce hormone autonomously. **Thyroiditis** leaks stored hormone and is not increased synthesis; excess replacement is another cause.

- Higher heat production → heat intolerance and sweating
- Catabolism → weight loss despite appetite
- Increased cardiac responsiveness → palpitations, tachycardia or atrial fibrillation
- Faster intestinal transit → frequent stools
- Tremor, proximal muscle weakness and goitre may occur.
- Graves eye disease reflects autoimmunity, not simply a high hormone concentration.

READ THE LABORATORY PATTERN

Example adult intervals: thyroid-stimulating hormone 0.27–4.2 milli-international units/L; free thyroxine 12–22 picomoles/L; free triiodothyronine 3.1–6.8 picomoles/L. These are assay-specific examples; use the reporting laboratory's range. Pregnancy, age and acute illness change interpretation.

Condition	Thyroid-stimulating hormone	Free thyroid hormones
Primary hypothyroidism	High	Free thyroxine LOW
Subclinical hypothyroidism	High	Free thyroxine NORMAL
Primary hyperthyroidism	Low	Free thyroxine and/or free triiodothyronine HIGH; at least one is high.
Subclinical hyperthyroidism	Low	Both free hormones NORMAL
Central hypothyroidism	Inappropriately low or normal; occasionally mildly raised	Free thyroxine LOW

A normal thyroid-stimulating hormone result does not exclude central hypothyroidism.

INVESTIGATE WITH A QUESTION

- Usually begin with thyroid-stimulating hormone; add free thyroxine if raised, and both free hormones if suppressed.
- Suspected pituitary disease requires thyroid-stimulating hormone plus free thyroxine initially.
- Thyroid peroxidase antibodies support autoimmune hypothyroidism; receptor antibodies support Graves disease.
- Imaging addresses nodules or uncertain cause, not every abnormal blood test.
- Ask about biotin, medicines and acute illness; assay interference can mislead.

BASIC MANAGEMENT

- Replace deficient hormone with levothyroxine and monitor; central disease needs specialist assessment, including adrenal status.
- Treat hyperthyroidism with cause-appropriate antithyroid medication, radioiodine or surgery; beta-blockade may relieve adrenergic symptoms.
- Antithyroid drugs do not correct simple hormone leakage from thyroiditis.
- Monitor for over-treatment and educate about medication adverse effects.
- Pregnancy requires a separate pathway.

EMERGENCIES

Hypothermia with reduced consciousness in severe hypothyroidism, or fever with marked tachycardia and systemic decompensation in thyrotoxicosis, needs urgent hospital care.

Sources: American Thyroid Association; National Institute for Health and Care Excellence guideline145; National Institute of Diabetes and Digestive and Kidney Diseases; Gloucestershrie Hospitals laboratory.

Medical foundation01 • Supplementary to original PaperIV questions54/56/58 • 21September2026 • v1

Thyroid physiology and dysfunction. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-54.

Credits and references: retained within the illustration.

IV - 55

Mental health strategies for managing Obesity. [10]

10 | ENDOCRINE AND METABOLIC PSYCHIATRY | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Frame the problem**	The link runs both ways, and is often iatrogenic
3	**Assess three things**	The drug chart, the eating pattern, the measurements
3	**Psychological strategies**	Matched to what the assessment actually found
2	**Prescribing and beyond**	Switch, augment, refer, and monitor on a schedule

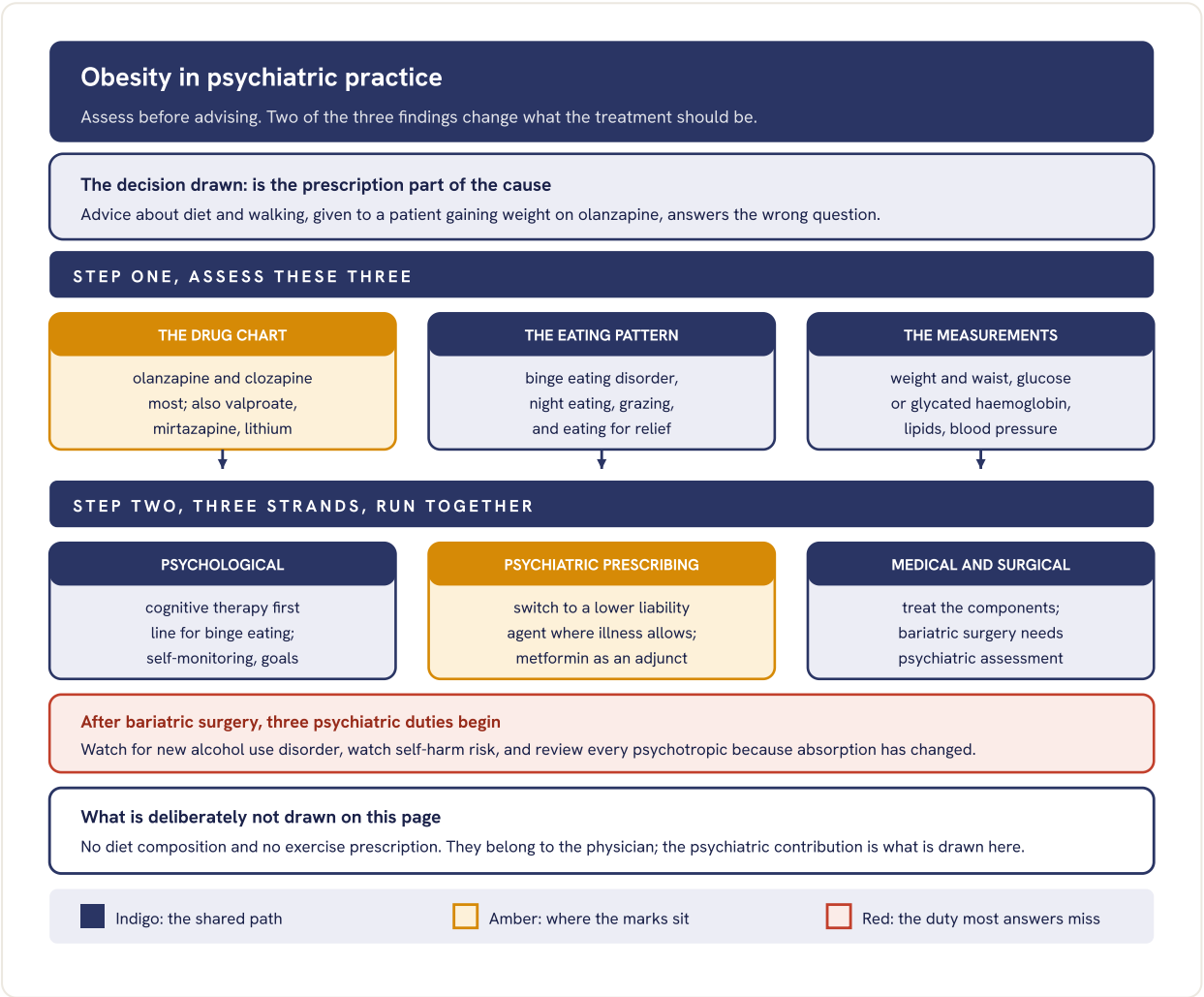


Fig 055.1 Three things assessed before anything is advised, then three strands of management run together, with the post-surgical duties carried as a red strip because they fall to psychiatry specifically.

- **Frame it first.** The link runs both ways: depression raises the risk of obesity and obesity raises the risk of depression, and in psychiatric patients the treatment is frequently part of the cause.
- **Weight stigma is a clinical variable.** Shame-based advice reduces attendance and worsens eating, so language and framing belong in the treatment plan rather than beside it.

WHAT EARNS THE MARKS

- **The drug chart reviewed first,** which is the single most psychiatric move on the page.
- **Binge eating disorder screened for and treated,** with cognitive therapy named as first line.
- **A monitoring schedule, not just a list of tests.**
- **The post-surgical duties,** which almost no answer reaches.

SEE ALSO Slot IV - 57 metabolic syndrome and antipsychotics Slot IV - 58 psychiatric aspects of diabetes **SPRINT** Day 33, Endocrine & metabolic psychiatry

WEAVE 100 · PAPER IV · F09

ENERGY, OBESITY AND METABOLIC RISK

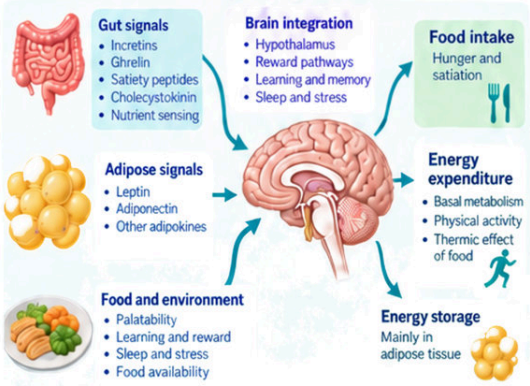
Foundation 09 • Regulation → adipose dysfunction → clustered risk

NORMAL ENERGY REGULATION

Food energy supports basal metabolism, movement and the thermic cost of processing food; surplus can be stored, mainly in adipose tissue. Body weight reflects energy balance over time, but both intake and expenditure are biologically regulated. The brain integrates nutrient, gut and adipose signals with learning, sleep, stress and food availability. Insulin and leptin signal energy state; gut signals influence hunger and satiation. Adipose tissue is an endocrine organ, not merely inert storage.

Weight loss can increase hunger and reduce expenditure. Maintenance therefore needs continuing support; energy balance does not imply that obesity is a simple failure of willpower.

REGULATION OF ENERGY BALANCE



WHY IT MAY BE SILENT

Metabolic abnormalities often cause no symptoms until complications develop. Mechanical loading can contribute to joint pain and reduced mobility. Upper-airway narrowing predisposes to obstructive sleep apnoea; ectopic hepatic fat can cause metabolic dysfunction-associated steatotic liver disease. Cardiovascular disease and type 2 diabetes risk rise. Breathlessness or fatigue still needs assessment rather than automatic attribution to weight.

MEASURE ADIPOSITY AND RISK

Body mass index = weight in kilograms ÷ height in metres squared.

Standard adult categories: healthy weight 18.5–24.9 kg/m²; overweight 25.0–29.9 kg/m²; obesity ≥30 kg/m². This is a screening measure, not a direct fat measurement; muscle mass, age and body composition affect interpretation.

National Institute for Health and Care Excellence uses lower practical thresholds for South Asian and several other higher-risk backgrounds: overweight 23.0–27.4 kg/m²; obesity ≥27.5 kg/m²: Other national systems differ: label the framework rather than mixing cutoffs.

Waist-to-height ratio adds information about central adiposity; ≥0.5 indicates increased risk in the National Institute for Health and Care Excellence adult assessment framework. Measure waist consistently.

Assess blood pressure, glucose/glycated haemoglobin, lipid profile, organ complications, medicines, sleep and relevant secondary causes.



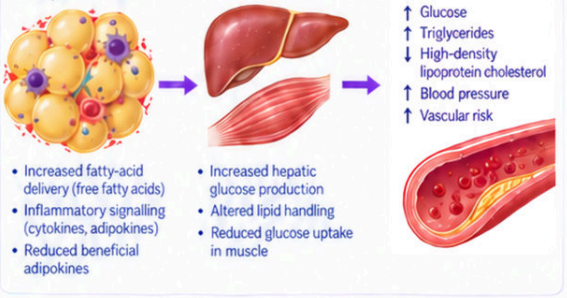
Measure waist circumference consistently (at midway between the lower rib and iliac crest, at the end of a normal breath, with a non-stretch tape).

WHAT CHANGES IN OBESITY

Obesity involves excess adiposity that can impair health. Genetic susceptibility, environment, sleep, activity, medicines and some diseases interact. Fat distribution matters: visceral and ectopic fat are particularly associated with metabolic dysfunction. Enlarged/dysfunctional adipose tissue can increase fatty-acid delivery and inflammatory signalling, promoting insulin resistance in liver and muscle.

The pancreas initially compensates with more insulin; glucose rises when secretion cannot meet demand. Altered liver lipid handling contributes to high triglycerides and low high-density lipoprotein cholesterol. Vascular dysfunction and multiple neurohormonal/renal pathways contribute to hypertension. These processes overlap but are not identical in every person.

FROM ADIPOSE DYSFUNCTION TO CLUSTERED RISK



- Increased fatty-acid delivery (free fatty acids)
- Inflammatory signalling (cytokines, adipokines)
- Reduced beneficial adipokines
- Increased hepatic glucose production
- Altered lipid handling
- Reduced glucose uptake in muscle

METABOLIC SYNDROME: FIVE COMPONENTS

	Component	Harmonised 2009 criteria (any THREE out of FIVE)	International Diabetes Federation 2005 criteria central obesity PLUS any TWO other components
1	Increased waist circumference	Using population-specific thresholds; commonly ≥90 cm for South Asian men and ≥80 cm for South Asian women.	Required (with population-specific thresholds).
2	Triglycerides	≥150 mg/dL (1.7 mmol/L), or treatment for this abnormality.	≥150 mg/dL (1.7 mmol/L), or treatment.
3	High-density lipoprotein cholesterol	<40 mg/dL (1.0 mmol/L) in men or <50 mg/dL (1.3 mmol/L) in women, or relevant treatment.	<40 mg/dL (1.0 mmol/L) in men or <50 mg/dL (1.3 mmol/L) in women, or treatment.
4	Blood pressure	Systolic pressure ≥130 mmHg **OR** diastolic pressure ≥85 mmHg, or antihypertensive treatment.	Systolic pressure ≥130 mmHg or diastolic pressure ≥85 mmHg, or treatment.
5	Fasting glucose	≥100 mg/dL (5.6 mmol/L), or treatment for elevated glucose.	≥100 mg/dL (5.6 mmol/L), or treatment.

- **Any THREE** of these **FIVE** identify a risk cluster, not one disease mechanism.
- Central obesity is not mandatory in this harmonised definition; the older International Diabetes Federation definition required it.
- A person can have important risk without reaching three criteria.

BASIC MANAGEMENT

Use respectful, individualised nutrition, activity, sleep and behavioural support with sustainable follow-up. Treat blood pressure, lipids, glycaemia, sleep apnoea and other complications directly. Consider weight-management medicines or metabolic/bariatric surgery when current eligibility, benefits, harms and preferences support them; nutritional and long-term monitoring remain essential.

Review potentially weight-promoting medicines without abrupt changes. Judge benefit through function, metabolic health and complication reduction as well as weight.

Avoid crash dieting and promises that one diet, supplement or hormone test explains every case.

Measure the waist, pressure, fats and glucose.

Sources: National Institute for Health and Care Excellence; National Heart, Lung and Blood Institute; 2009 harmonised metabolic syndrome consensus.

Energy balance obesity and metabolic syndrome. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-55.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P07

OBESITY, METABOLIC RISK AND PSYCHIATRIC TREATMENT

Psychiatric companion 07 • Preserve mental stability and physical health

EXPLAIN WITHOUT BLAME

- Weight is influenced by appetite regulation, genetics, sleep, activity, environment, illness and medicines.
- Some psychotropics increase appetite, reduce activity through sedation or alter metabolic regulation; glucose disturbance is not always proportional to weight change.
- Stigma can worsen avoidance and disengagement.
- Ask permission to discuss weight, use neutral language and agree meaningful health goals.

ASSESS THREE THINGS TOGETHER

The prescription

- Document weight before treatment, timing of changes, dose, response and previous alternatives.
- Olanzapine and clozapine have substantial metabolic liability; lower-risk drugs still require monitoring.
- Valproate, lithium and some antidepressants may contribute.
- Do not assume an entire drug generation has one risk profile.

The eating/sleep pattern

- Ask about binge eating, restriction, night eating, emotional triggers, food insecurity, alcohol and sleep-disordered breathing.
- Binge-eating disorder is not diagnosed from body size alone.
- Assess depression, anxiety, trauma and executive difficulties that affect routines.

The measurements

- Record weight, body mass index, waist circumference, blood pressure, glucose or glycated haemoglobin, and lipid profile.
- Consider ethnicity and central adiposity in risk interpretation.
- Metabolic syndrome clusters risks: treat each abnormal component even if the person does not meet the full syndrome definition.

CONVERGE TO PARALLEL INTERVENTIONS

Psychiatric interventions

- Optimise psychiatric treatment
- Consider lower-metabolic-risk options
- Monitor for relapse during changes
- Maintain mental stability

Behavioural interventions

- Nutrition and activity support
- Address eating patterns and sleep
- Use realistic goals and practical changes
- Treat eating disorders with evidence-based approach

Metabolic interventions

- Treat hypertension, dyslipidaemia and diabetes
- Consider adjunctive weight treatments
- Coordinate with primary and metabolic care
- Monitor and act early

MEMORY AID:
Review the medicine, understand the pattern, measure the trend, act early.

MONITOR EARLY ENOUGH TO ACT

National Institute for Health and Care Excellence antipsychotic guidance recommends weight weekly for the first six weeks, then at twelve weeks, one year and annually. Recheck pulse/blood pressure, glucose or glycated haemoglobin and lipids at twelve weeks, one year and annually; assess waist annually. Baseline measurements precede treatment.

Increase frequency for rapid change, symptoms or established disease rather than waiting for the next routine date.

Plot trends and discuss them. New thirst, polyuria, dehydration or acute illness requires prompt glucose assessment. Assign responsibility for results and intervention; recording an abnormal number is not the same as treating it.

MONITORING TIMELINE

Measure	Baseline (before treatment)	Weekly (first six weeks)	Twelve weeks	One year	Annually
Weight	✓	✓	✓	✓	✓
Pulse	✓	–	✓	✓	✓
Blood pressure	✓	–	✓	✓	✓
Glucose or glycated haemoglobin	✓	–	✓	✓	✓
Lipids	✓	–	✓	✓	✓
Waist circumference	✓	–	–	✓	✓

✓ = assess – = not routinely required at this time point

INTERVENE ON PARALLEL TRACKS

- Offer accessible nutrition, activity and behavioural support adapted to energy, finances, disability and psychiatric symptoms.
- Use realistic goals, self-monitoring and practical environmental changes; avoid punitive restriction.
- Treat an eating disorder with its own evidence-based approach.
- Manage blood pressure, dyslipidaemia and diabetes through coordinated medical care.
- Review whether a lower-metabolic-risk psychiatric medicine could preserve benefit.
- Explain the risks of relapse and adverse effects during switching.
- An effective clozapine regimen may be essential; metabolic harm deserves active treatment without automatically sacrificing psychiatric stability.
- Do not make abrupt changes or present lifestyle advice as the only answer to a medication effect.

ADJUNCTIVE WEIGHT TREATMENT

- Metformin has evidence for antipsychotic-associated weight gain, including prevention in selected higher-risk situations. Recent guidance supports earlier consideration rather than waiting for severe established obesity.
- Decide with the patient and medical team, considering renal function, gastrointestinal tolerability, vitamin B12 status, contraindications and local prescribing arrangements. It does not replace nutrition/activity support or monitoring.
- Other obesity treatments, including glucagon-like peptide-1 receptor agonists, may be appropriate under relevant indications and specialist assessment. Eligibility, adverse effects, access and long-term follow-up matter; do not assume all weight-loss treatments have equivalent psychiatric evidence or are suitable for every person.

BARIATRIC AND LONG-TERM CARE

- When surgery is considered, assess understanding, expectations, eating patterns, substance use, current psychiatric stability and support. A psychiatric diagnosis alone is not an automatic exclusion.
- Afterwards, review medication absorption and clinical response, nutrition, alcohol use and self-harm risk. Formulation changes require individual pharmacological review, not a blanket rule that every tablet stops working.
- Follow function, quality of life, psychiatric relapse, metabolic trends and treatment burden. Share the plan across psychiatry and primary/metabolic care.

Sources: National Institute for Health and Care Excellence; metformin prevention and management guidelines.

Obesity and metabolic psychiatry. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-55.

Credits and references: retained within the illustration.

IV - 56

Hypothyroidism. [10]

10 ENDOCRINE AND METABOLIC PSYCHIATRY KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition and levels	Primary, central, and the subclinical band
3	Causes	Autoimmune, iodine, iatrogenic, and the psychiatric drug
3	Features and diagnosis	Body and mind, then the order the tests are read in
2	Treatment	Replace, retest, and the rule about lithium

THE DEFINITION, IN THREE LEVELS

Deficient thyroid hormone action. Primary disease is failure of the gland, with a raised thyroid stimulating hormone and a low free T4. Central disease is pituitary or hypothalamic. Subclinical disease is a raised stimulating hormone with a normal free T4.

CAUSE GROUP	EXAMPLES	THE CLUE	NOTE
Autoimmune	Hashimoto thyroiditis	Goitre, other autoimmune disease	**Commonest where iodine is sufficient**
Iodine deficiency	Endemic goitre	Geography and diet	Still relevant in parts of India
Iatrogenic	Thyroidectomy, radioiodine, amiodarone, interferon	The history gives it away	Ask before testing
Psychotropic	Lithium	Rising stimulating hormone on treatment	**Treat, do not stop the lithium**
Transient	Postpartum thyroiditis	Months after delivery, often after a thyrotoxic phase	**Mimics postnatal depression**

Amber cells are the discriminators. Two of them are the reason this endocrine question sits in a psychiatry paper.

- **Features and testing.** Fatigue, cold intolerance, weight gain, constipation, bradycardia, slow-relaxing reflexes; mentally, depression and slowing. Test the stimulating hormone, then free T4.
- **Treatment.** Levothyroxine once daily on an empty stomach, apart from calcium, iron and antacids, retested after about six weeks and titrated to the stimulating hormone. Start low where the patient is old or has cardiac disease.

PITFALL

Myxoedema coma. Hypothermia, hyponatraemia, hypoventilation and a falling conscious level in a patient whose slowness was being treated as depression.

WHAT EARNS THE MARKS

- **Three levels defined,** subclinical disease included; it is where most psychiatric overlap sits.
- **Lithium named as a cause,** with the rule that replacement is added rather than lithium stopped.
- **Test order given,** stimulating hormone first, which shows the biochemistry is understood.

SEE ALSO Slot IV - 54 psychiatric manifestations of thyroid disorders Slot IV - 59 thyroid and parathyroid dysfunction

SPRINT Day 33, Endocrine & metabolic psychiatry

IV - 57

What is metabolic syndrome? Discuss the emergence of metabolic syndrome due to second-generation antipsychotics as compared with first-generation antipsychotics. [3+7]

3 + 7 | ENDOCRINE AND METABOLIC PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 Define it	Five components, three needed, and the Asian waist
3 The comparison	The answer is which drug, not which generation
2 Mechanism	The receptors that drive appetite and insulin
2 What follows	A monitoring schedule, then what to change

THE DEFINITION

Three of five: central obesity by waist, triglycerides 150 or more, high density lipoprotein under 40 in men or 50 in women, blood pressure 130 over 85 or more, fasting glucose 100 or more milligrams per decilitre, or treatment for any. South Asian waist is lower: 90 centimetres in men, 80 in women.

AGENT	METABOLIC LIABILITY	EVIDENCE AND COMMENT	GENERATION
Clozapine, olanzapine	Highest	Largest weight and lipid change in the major pragmatic trial	**Second**
Chlorpromazine, thioridazine	Intermediate to high	Low potency older drugs also cause substantial gain	**First**
Haloperidol, fluphenazine	Lower	The comparison that produced the class story	First
Aripiprazole, ziprasidone, lurasidone	Lowest	The usual switch targets when weight is the problem	**Second**

Amber cells are the discriminators. Rows three and five break the generation story, and saying so is the discussion the seven marks are for.

- **Mechanism.** Histamine H1 and serotonin 5-HT2C blockade drive appetite and weight, muscarinic M3 blockade impairs insulin secretion, and there are direct effects on insulin sensitivity.

STATUS: WHERE THIS SITS TODAY

Settled: liability is drug-specific, clozapine and olanzapine are the worst, and monitoring at baseline, three months and then yearly is standard. Still argued: how much of the excess is illness and lifestyle rather than drug, and where the newer weight-lowering agents belong.

WHAT EARNS THE MARKS

- **Three of five stated,** with the lower South Asian waist threshold named.
- **The generation framing challenged,** using a low potency older drug as the counterexample.
- **Receptor mechanism given,** histamine and serotonin for appetite, muscarinic for insulin.

SEE ALSO Slot IV - 55 mental health strategies for obesity Slot IV - 58 psychiatric aspects of diabetes **SPRINT** Day 33, Endocrine & metabolic psychiatry

WEAVE 100 · PAPER IV · X04

X04 — Metabolic syndrome: count the components, treat the risk.

METABOLIC SYNDROME: FIVE COMPONENTS, ONE INTEGRATED PLAN

What the cluster means. Metabolic syndrome groups abdominal adiposity, raised triglycerides, low high-density lipoprotein cholesterol, raised blood pressure and dysglycaemia. It signals increased cardiovascular and type 2 diabetes risk, often without symptoms. It is neither a single disease nor a substitute for assessing each component and overall cardiovascular risk. Important abnormalities deserve treatment even when fewer than three criteria are present.

From normal physiology to dysfunction. Insulin normally promotes glucose uptake in muscle and fat and restrains liver glucose production. Visceral adiposity, excess fatty-acid delivery and inflammatory signalling can impair these responses. Compensatory insulin secretion may initially maintain glucose; later pancreatic beta-cell insufficiency permits hyperglycaemia. Increased liver triglyceride production, altered lipoprotein handling and vascular, renal and sympathetic effects help link dyslipidaemia and hypertension. Genetics, sleep, activity, diet, medicines and social circumstances interact; body weight alone does not explain every case.

Name the definition: adult harmonised criteria, 2009. Any THREE of FIVE qualify; central obesity is not obligatory. Use population-appropriate waist thresholds. For South Asian adults:

1. Waist circumference: men at least 90 centimetres; women at least 80 centimetres.
2. Triglycerides: at least 150 milligrams per decilitre, equivalent to 1.7 millimoles per litre, or treatment specifically for raised triglycerides.
3. High-density lipoprotein cholesterol: below 40 milligrams per decilitre in men, equivalent to 1.0 millimole per litre; below 50 in women, equivalent to 1.3 millimoles per litre; or treatment specifically for this abnormality.
4. Blood pressure: systolic at least 130 **OR** diastolic at least 85 millimetres of mercury, or antihypertensive treatment in someone with hypertension.
5. Fasting glucose: at least 100 milligrams per decilitre, equivalent to 5.6 millimoles per litre, or treatment for raised glucose.

These are syndrome thresholds, not laboratory reference intervals or automatic thresholds for prescribing. A statin prescription for raised low-density lipoprotein cholesterol does not automatically establish the triglyceride or high-density-lipoprotein criterion. Count each component once.

Harmonised 2009 diagnostic thresholds for metabolic syndrome (South Asian adults)

Component	Threshold (or treatment)
Waist circumference	Men at least 90 centimetres; women at least 80 centimetres.
Triglycerides	At least 150 milligrams per decilitre, equivalent to 1.7 millimoles per litre, or treatment specifically for raised triglycerides.
High-density lipoprotein cholesterol	Below 40 milligrams per decilitre in men, equivalent to 1.0 millimole per litre; below 50 in women, equivalent to 1.3 millimoles per litre; or treatment specifically for this abnormality.
Blood pressure	Systolic at least 130 **OR** diastolic at least 85 millimetres of mercury, or antihypertensive treatment in someone with hypertension.
Fasting glucose	At least 100 milligrams per decilitre, equivalent to 5.6 millimoles per litre, or treatment for raised glucose.

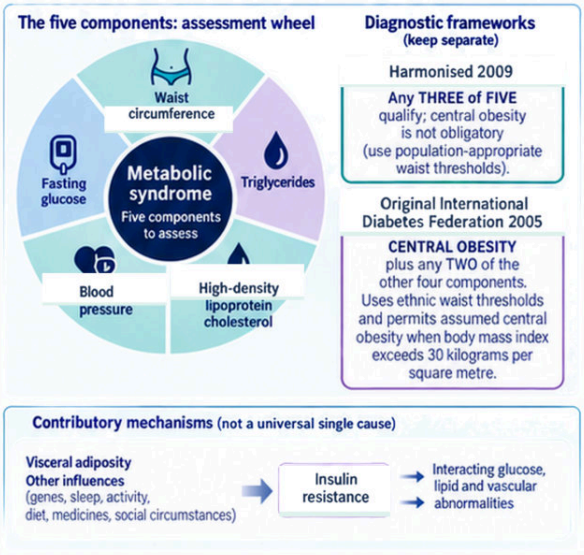
Do not mix frameworks. The original International Diabetes Federation definition, 2005, requires CENTRAL OBESITY plus any TWO of the other four components. It uses ethnic waist thresholds and permits assumed central obesity when body mass index exceeds 30 kilograms per square metre. The harmonised framework removed the compulsory-waist requirement. State which definition you use. Glycated haemoglobin helps diabetes assessment but does not replace fasting glucose in this five-component table. Adult thresholds do not apply unchanged to children.

Assess before labelling. Measure waist with a consistent anatomical protocol and blood pressure with an appropriate cuff; repeat abnormal readings. Record weight trajectory, fasting glucose and lipids, medicines, smoking, alcohol, diet, activity, sleep and family history. Assess kidney function and other tests according to comorbidity and planned treatment. Confirm diabetes or hypertension through their own diagnostic pathways. Severe symptomatic hyperglycaemia, chest pain or neurological deficits require urgent assessment, not routine lifestyle follow-up.

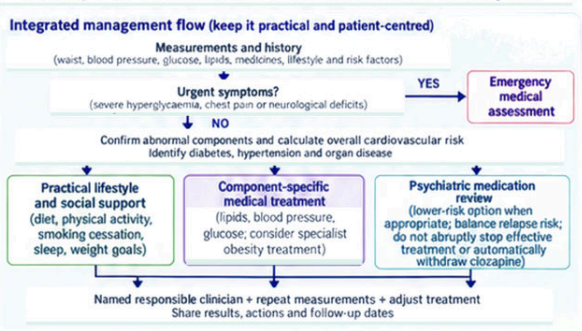
Psychiatric relevance. Risk reflects illness, disadvantage, behaviour and drug effects. Clozapine and olanzapine have high metabolic liability; risk differs between individual drugs within both generations. Appetite stimulation, sedation and direct metabolic effects can contribute. Changes may occur before major weight gain. Discuss a lower-risk option when clinically appropriate, balancing relapse risk; do not abruptly stop an effective antipsychotic or automatically withdraw clozapine.

Integrated management flow. Confirm components and assess urgency → estimate cardiovascular risk and identify diabetes, hypertension and organ disease → agree diet, feasible physical activity, smoking cessation, sleep and weight goals → treat abnormal components alongside psychiatric care. Choose lipid-lowering treatment by cardiovascular indication; do not prescribe merely to raise high-density lipoprotein cholesterol. Treat blood pressure and glucose under their respective guidance. Consider specialist obesity treatment when indicated. Metformin may be considered for antipsychotic-related weight gain or prevention under an appropriate prescribing protocol; it is not a universal metabolic-syndrome tablet. Review kidney function, gastrointestinal tolerability and vitamin B12 risk when using it.

Monitor with ownership. Before antipsychotic treatment record weight, waist, blood pressure, glucose or glycated haemoglobin, and lipids. National Institute for Health and Care Excellence monitoring: weight weekly for six weeks, at twelve weeks, one year and annually; waist annually; blood pressure, glucose and lipids at twelve weeks, one year and annually. Abnormal results or rapid change require earlier review. Name the clinician responsible; share results, actions and follow-up dates.



Memory aid: "Measure five, name the framework, treat every risk."



Supplementary clinical teaching • September 2026.

Metabolic syndrome - diagnostic criteria and integrated psychiatric care. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-57.

Credits and references: retained within the illustration.

IV - 58

Discuss the psychological and psychiatric aspects in the management of diabetes mellitus. [10]

10 ENDOCRINE AND METABOLIC PSYCHIATRY

KUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	**The two-way link**	Each raises the other, and control pays for it
3	**What to screen for**	Four things, and two of them are not depression
3	**Treatment**	Drug choices that respect weight and glucose
2	**The model of care**	Collaborative care, education, and the family

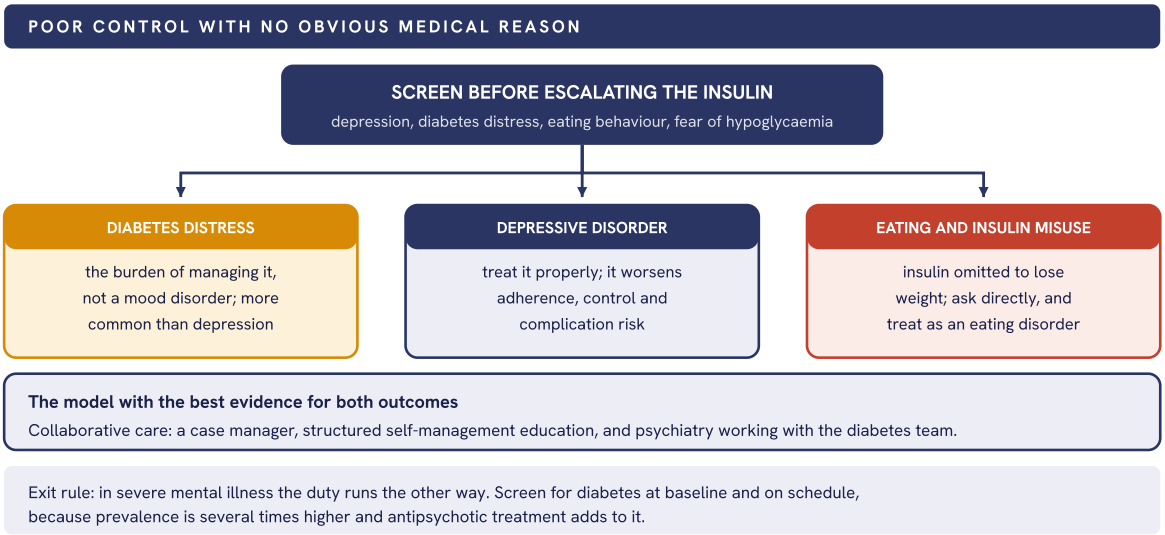


Fig 058.1 Screening placed before any escalation of the diabetic treatment, with distress separated from depression because they need different interventions and only one of them needs a drug.

- **Drug choices.** Selective serotonin reuptake inhibitors are preferred, sertraline commonly. Avoid the weight-promoting options where possible, and consider duloxetine where painful neuropathy coexists.
- **The interactions to know.** Serotonergic antidepressants can lower glucose further, olanzapine and clozapine worsen control, and steroids given for anything else will undo a month of adjustment.

PITFALL

Treating distress as depression. Distress responds to education, problem solving and a change in the regimen, and an antidepressant given instead treats nothing.

WHAT EARNS THE MARKS

- **Distress named and separated from depression.** It is the discriminating fact on this page.
- **Insulin omission asked about directly,** because it is dangerous and rarely volunteered.
- **Collaborative care named as the model,** not simply liaison in general terms.

SEE ALSO Slot IV - 57 metabolic syndrome and antipsychotics Slot IV - 55 mental health strategies for obesity

SPRINT Day 33, Endocrine & metabolic psychiatry

WEAVE 100 · PAPER IV · F08

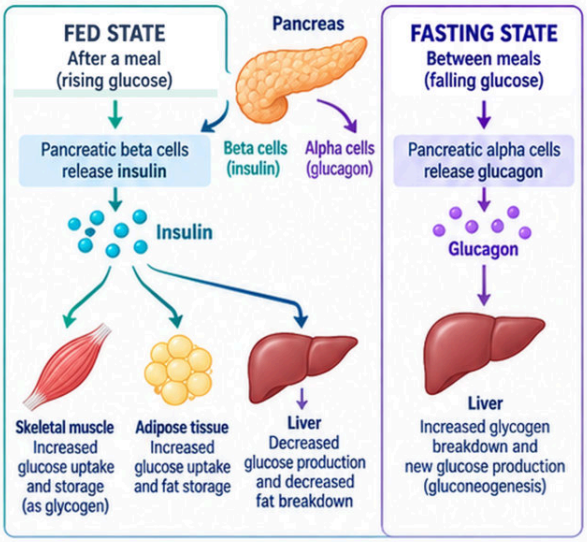
GLUCOSE AND DIABETES: FUEL SUPPLY WITHOUT CONTROL

Foundation 08 • Insulin, glucagon and the consequences of imbalance

NORMAL PHYSIOLOGY

Glucose supplies energy to tissues. After a meal, rising glucose stimulates pancreatic beta cells to release insulin. Insulin promotes glucose uptake in skeletal muscle and adipose tissue, supports energy storage and suppresses hepatic glucose production and fat breakdown. The brain and red blood cells use largely insulin-independent uptake; insulin is not a universal 'key' for every cell.

During fasting, falling insulin and increased glucagon from pancreatic alpha cells favour hepatic glycogen breakdown and new glucose production. Counter-regulatory hormones help defend glucose during stress. The liver, pancreas, kidneys and peripheral tissues together maintain balance.



Brain
Uses largely insulin-independent glucose uptake (essential energy supply)

Red blood cells
Use largely insulin-independent glucose uptake (insulin is not a universal 'key')

WHAT FAILS

Type 1 diabetes: immune-mediated beta-cell destruction usually produces severe insulin deficiency. Insulin replacement is essential.

Type 2 diabetes: insulin resistance in liver, muscle and adipose tissue combines with progressive beta-cell dysfunction. Increased hepatic glucose output and impaired disposal raise blood glucose. Genetics, adiposity, age and environment contribute; it is not simply eating sugar.

Other causes include pancreatic disease, medicines and specific genetic disorders. Gestational diabetes has pregnancy-specific testing and management. Diabetes type cannot always be inferred from age or body size alone.

MECHANISM → SYMPTOM

- Glucose exceeds renal reabsorptive capacity → glucose in urine draws water → polyuria, dehydration and thirst.
- Urinary calorie loss plus insulin deficiency/catabolism → weight loss and weakness.
- Osmotic effects in the lens → fluctuating blurred vision.
- Impaired host defence and tissue repair → infections and slow wound healing.
- Long-term hyperglycaemia and related vascular risk damage retina, kidneys and nerves; cardiovascular disease risk also increases.
- Early type 2 disease may be asymptomatic.

DIAGNOSTIC VALUES: NONPREGNANT ADULTS

Any qualifying criterion can establish diabetes with appropriate confirmation:

- Glycated haemoglobin ≥6.5% (48 mmol/mol), using a standardised laboratory method.
- Fasting plasma glucose ≥126 mg/dL (7.0 mmol/L), after at least 8 hours without caloric intake.
- Two-hour plasma glucose ≥200 mg/dL (11.1 mmol/L) after a 75-g oral glucose load.
- Random plasma glucose ≥200 mg/dL (11.1 mmol/L) with classic symptoms or hyperglycaemic crisis.

Without unequivocal hyperglycaemia, confirm with a second abnormal result. Home glucose meters are not the standard diagnostic method.

Other glycaemic ranges (American Diabetes Association)

Category	Glycated haemoglobin	Fasting plasma glucose	Two-hour plasma glucose
Intermediate risk	5.7–6.4%	100–125 mg/dL	140–199 mg/dL
Below these thresholds	<5.7%	<100 mg/dL (<5.6 mmol/L)	<140 mg/dL (<7.8 mmol/L)

Fasting definitions differ between organisations; label the system used. Low glucose is a separate abnormality, not a desirable normal result.

WHAT EACH TEST TELLS YOU

Plasma glucose describes that moment; glycated haemoglobin reflects preceding months, weighted toward recent weeks. Anaemia, altered red-cell turnover, haemoglobin variants, pregnancy and some renal settings can distort glycated haemoglobin. Use appropriate alternatives when the result and clinical picture disagree.

Assess blood pressure, lipids, kidney function/urine albumin, feet and eyes for complications. Ketones, electrolytes and acid–base tests are urgent when acute decompensation is suspected.

ACUTE DANGER: HIGH OR LOW

Marked insulin deficiency → lipolysis → ketones → metabolic acidosis: diabetic ketoacidosis. Hyperosmolar hyperglycaemic state causes profound dehydration and hyperosmolality, usually with less ketosis. Both need urgent fluids, insulin/electrolyte management and treatment of the precipitant under protocol. Ketoacidosis can occur without extreme glucose elevation.

Hypoglycaemia alert: glucose <70 mg/dL (3.9 mmol/L); <54 mg/dL (3.0 mmol/L) is clinically significant. Sweating, tremor and palpitations reflect counter-regulation; confusion, seizures or coma reflect insufficient brain fuel. Severe hypoglycaemia means assistance is required, regardless of the measured value.

BASIC MANAGEMENT

Use education, individualised nutrition/activity, glucose monitoring and cardiovascular-risk reduction. Type 1 needs insulin; type 2 treatment is selected for glucose, weight, cardiovascular/kidney disease and safety. Set individual targets rather than confusing diagnostic thresholds with goals. Teach hypoglycaemia treatment and sick-day plans; unconscious patients must not be given food or drink by mouth. Regular review prevents complications and identifies treatment burden.

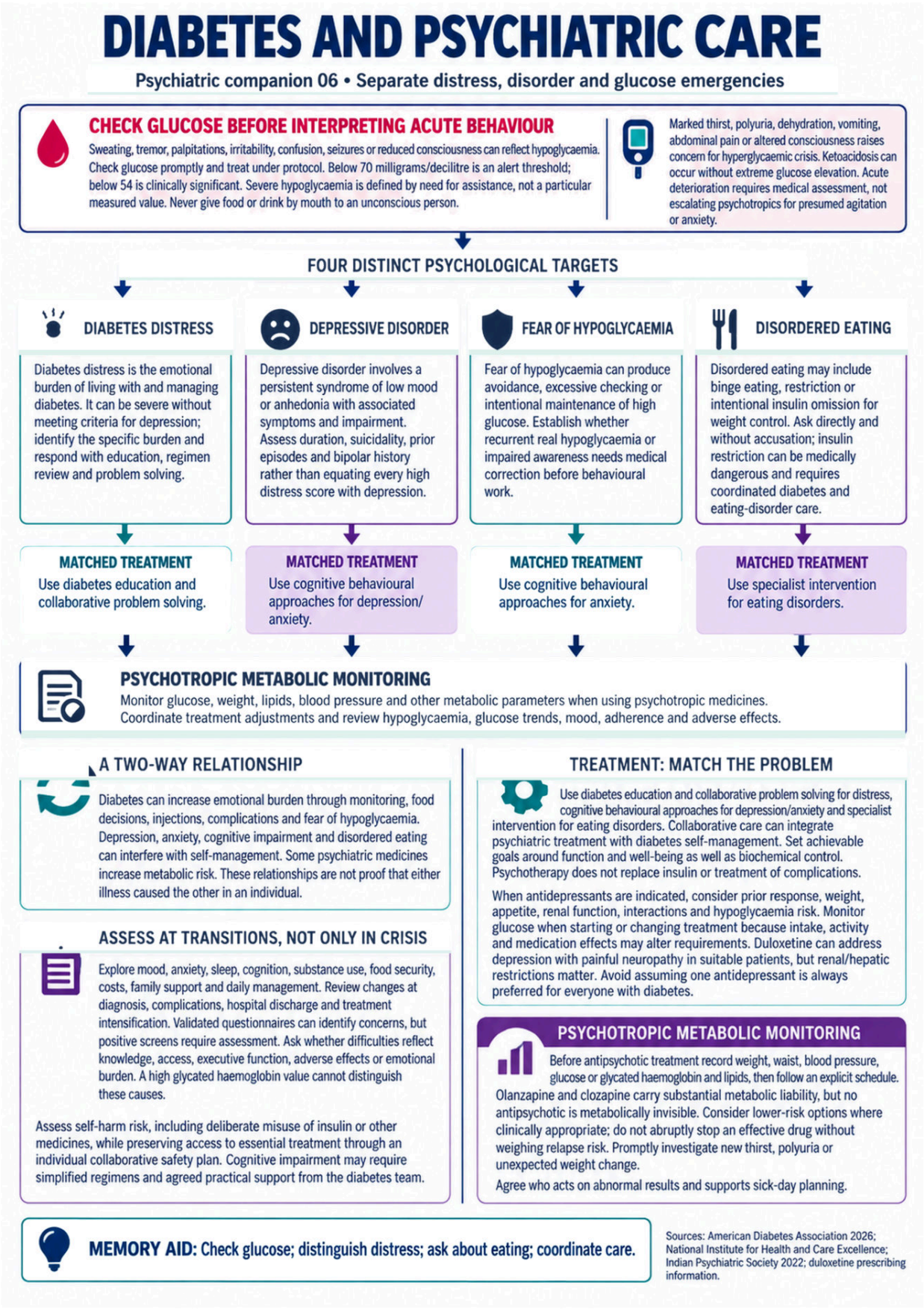
Supply, storage, spill: follow the glucose.

Sources: American Diabetes Association 2026 Standards of Care; National Institute of Diabetes and Digestive and Kidney Diseases.

Glucose regulation and diabetes mellitus. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-58.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P06



Diabetes psychiatry. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-58.

Credits and references: retained within the illustration.

IV - 59

Discuss the psychiatric disorders due to thyroid and parathyroid glands dysfunction. [10]

10 ENDOCRINE AND METABOLIC PSYCHIATRY

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

SKELETON

2	The organising rule	Each gland acts through one measurable number
3	Thyroid, compactly	Both directions, and the form that misleads in old age
3	Parathyroid and calcium	High and low, with the signs that give each away
2	What to do	Who to test, and how symptoms follow correction

THE ORGANISING RULE

Both glands reach the brain through a single number that a routine test reports: the thyroid stimulating hormone, and serum calcium. So the answer is structured as two axes, each with an over and an under state, and each with a psychiatric picture that tracks the level rather than the gland.

STATE	PSYCHIATRIC PICTURE	THE PHYSICAL GIVEAWAY	TEST
Thyroid, under	Depression, slowing, cognitive impairment; psychosis when severe	Cold intolerance, slow reflexes, bradycardia	Stimulating hormone high
Thyroid, over	Anxiety, lability, insomnia; apathy and low mood in the elderly	Tremor, weight loss, irregular pulse	**Stimulating hormone suppressed**
Calcium, high	Depression, apathy, poor concentration; delirium and psychosis as it rises	Thirst, polyuria, constipation, stones, bone pain	**Calcium and parathyroid hormone**
Calcium, low	Anxiety, irritability, lability; psychosis and seizures when severe	Tetany, and the Chvostek and Trousseau signs	**Calcium low, magnesium checked**
Calcium, low and long	Parkinsonism with psychiatric features	Basal ganglia calcification on imaging	Imaging, not biochemistry alone

Amber cells are the discriminators. The calcium rows are what this stem is really testing; the thyroid rows are the setting for them.

- **Two causes to name.** Primary hyperparathyroidism is most often a single adenoma, and lithium raises parathyroid hormone and calcium as well as causing hypothyroidism. Hypoparathyroidism is most often a consequence of thyroid surgery.
- **Who to test.** Unexplained depression with thirst and constipation, any delirium, new psychosis in an older patient, unexplained seizures, and every patient on long-term lithium.

WHAT EARNS THE MARKS

- **Both glands answered,** with the parathyroid given real space; most answers write only about the thyroid.
- **Symptoms tied to the level,** so severity explains why the same gland gives depression in one patient and delirium in another.
- **Lithium named on the calcium axis as well as the thyroid axis.**
- **The lag after correction,** which stops the answer promising instant recovery.

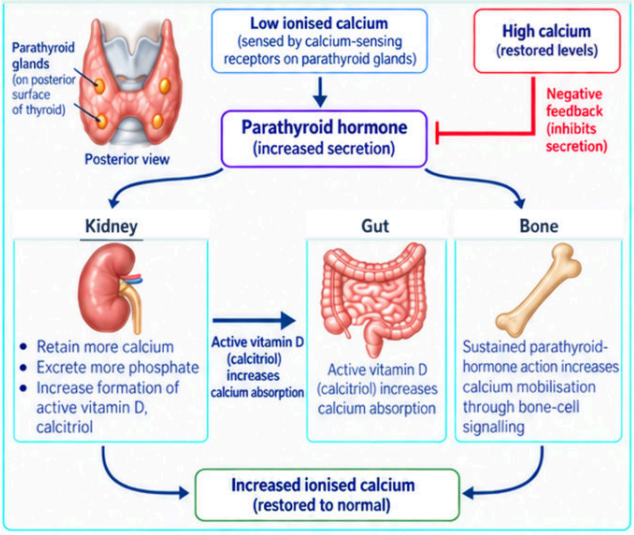
SEE ALSO Slot IV - 54 psychiatric manifestations of thyroid disorders Slot IV - 56 hypothyroidism **SPRINT** Day 33, Endocrine & metabolic psychiatry

WEAVE 100 · PAPER IV · F07

PARATHYROID AND CALCIUM: AN EXCITABILITY CONTROL SYSTEM

Foundation 07 • Sensor → bone, kidney and gut → feedback

THE CALCIUM CONTROL SYSTEM



NORMAL PHYSIOLOGY

- The small parathyroid glands usually lie behind the thyroid but have a different job: their calcium-sensing receptors respond to ionised calcium. Falling calcium stimulates parathyroid hormone; rising calcium suppresses it.
- Calcium supports nerve signalling, muscle contraction, cardiac function, clotting and the skeleton. Most body calcium is stored in bone.
- In blood, some is free and biologically active, some bound to albumin and some complexed with other ions. A change in albumin can change total calcium without the same change in active calcium.

THE THREE-ORGAN RESPONSE

- **Low ionised calcium** → increased parathyroid hormone.
- **Kidney:** retain more calcium, excrete more phosphate and increase formation of active vitamin D, calcitriol.
- **Gut:** calcitriol increases calcium absorption.
- **Bone:** sustained parathyroid-hormone action increases calcium mobilisation through bone-cell signalling.
- Restored calcium → negative feedback at the gland.
- Magnesium is needed for normal hormone secretion; a chronic, severe deficiency can cause hypocalcaemia that is resistant to calcium replacement alone.

PAIR CALCIUM, HORMONE AND SYMPTOMS

Calcium level	Parathyroid hormone level	Typical pattern and symptom mechanisms
High calcium	Raised or inappropriately unsuppressed	Parathyroid-dependent process (e.g. primary hyperparathyroidism). Symptoms from high calcium: reduced neuromuscular excitability (weakness, lethargy), impaired renal concentrating ability (polyuria, thirst, dehydration), reduced gut motility (constipation), kidney stones, skeletal fragility, confusion or arrhythmia (if marked).
High calcium	Suppressed	Investigate non-parathyroid causes (e.g. malignancy, vitamin D-mediated disorders, medicines). Suppressed parathyroid hormone directs investigation away from primary gland excess.
Low calcium	Low or inappropriately normal	Secretion failure or severe magnesium disorder. Symptoms from low calcium: increased nerve/muscle excitability (perioral or fingertip tingling, cramps, tetany), carpopedal spasm, seizures, laryngospasm or prolonged cardiac repolarisation.
Low calcium	High	Appropriate response; consider vitamin D deficiency, renal disease or hormone resistance.

TOO MUCH CALCIUM

- **Primary hyperparathyroidism** usually reflects autonomous gland secretion, often from an adenoma. Calcium is high but parathyroid hormone is raised or inappropriately unsuppressed.
- **Secondary hyperparathyroidism** is a response to another problem, such as vitamin D deficiency or chronic kidney disease: calcium is not necessarily high. Long-standing stimulation can become autonomous tertiary disease.
- **Other hypercalcaemia** causes include malignancy, vitamin D-mediated disorders and medicines. A suppressed parathyroid hormone directs investigation away from primary gland excess. Familial hypocalciuric hypercalcaemia can mimic primary disease and matters before considering surgery.
- High calcium reduces neuromuscular excitability → weakness and lethargy.
- Impaired renal concentrating ability → polyuria, thirst and dehydration.
- Reduced gut motility → constipation.
- Kidney stones and skeletal fragility can reflect prolonged mineral imbalance.
- Marked elevation may cause confusion or arrhythmia.

TOO LITTLE CALCIUM

- Causes include postsurgical hypoparathyroidism, vitamin D deficiency, kidney disease and severe magnesium deficiency.
- Low extracellular calcium increases nerve/muscle excitability → perioral or fingertip tingling, cramps and tetany.
- Carpopedal spasm, seizures, laryngospasm or prolonged cardiac repolarisation can signal severe disease.
- Chvostek and Trousseau signs support assessment but are not definitive tests.

READ THE PAIR, NOT ONE NUMBER

- Illustrative laboratory interval: total calcium 2.20–2.63 mmol/L; parathyroid hormone 1.6–6.9 pmol/L when calcium is normal. Use local methods and reference ranges; a normal-range hormone can be abnormal for a high calcium.
- High calcium → unsuppressed hormone → parathyroid-dependent process.
- High calcium → suppressed hormone → investigate non-parathyroid causes.
- Low calcium → low/inappropriately normal hormone → secretion failure or severe magnesium disorder.
- Low calcium → high hormone → appropriate response; consider vitamin D deficiency, renal disease or hormone resistance.
- Albumin adjustment is imperfect. Measure ionised calcium when the clinical setting makes total calcium unreliable: pH affects protein binding. Interpret this alongside the clinical context, not in isolation.

INVESTIGATE AND MANAGE

- Repeat/confirm abnormalities with paired calcium and hormone, albumin, magnesium, phosphate, renal function and vitamin D assessment.
- Review medicines; evaluate bone and renal complications.
- Urinary calcium can help distinguish selected causes.
- Neck imaging localises glands for surgery after biochemical diagnosis; it does not establish the diagnosis.
- Treat underlying cause and severity.
- Selected primary hyperparathyroidism requires surgery; others need specialist monitoring or medical therapy.
- Hypercalcaemia dehydration requires carefully monitored rehydration, respecting heart/kidney status.
- Hypocalcaemia needs calcium and cause-specific vitamin D or magnesium treatment.
- Severe symptoms, arrhythmia or major calcium derangement requires urgent monitored hospital treatment, not routine oral supplementation.

Pair calcium with its hormone: check the context.

Sources: National Institute of Diabetes and Digestive and Kidney Diseases; National Institute for Health and Care Excellence; Society for Endocrinology; Gloucestershire Hospitals laboratory.

Parathyroid function and calcium balance. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-59.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P05

CALCIUM, PARATHYROID FUNCTION AND PSYCHIATRY

Psychiatric companion 05 • A biochemical disorder can imitate a psychiatric syndrome

WHY CALCIUM MATTERS

Extracellular calcium influences neuronal and muscle excitability. Parathyroid hormone, kidney function, vitamin D and magnesium help regulate its concentration. Both high and low calcium can alter mental state, but psychiatric symptoms are nonspecific. Do not diagnose a parathyroid disorder from behaviour, or assume a measured abnormality explains every symptom.





Calcium influences neuronal excitability and neurotransmission

HIGH CALCIUM: SLOWING WITH SYSTEMIC CLUES

Hypercalcaemia can accompany fatigue, low mood, reduced concentration and apathy; severe or rapidly developing abnormalities may cause delirium and occasionally psychotic symptoms. Ask about thirst, frequent urination, constipation, nausea, weakness and kidney stones. Dehydration can worsen the disturbance. A new depressive or cognitive presentation with these physical clues warrants biochemical assessment rather than psychiatric treatment alone.

A high calcium result with parathyroid hormone that is elevated or inappropriately nonsuppressed suggests a parathyroid-dependent process. A suppressed hormone result directs attention to other causes, including malignancy and vitamin-related mechanisms. High calcium is not synonymous with primary hyperparathyroidism.

Common features of high calcium

- Fatigue, low mood, reduced concentration, apathy
- Delirium (severe) and occasionally psychotic symptoms
- Thirst, frequent urination, constipation, nausea, weakness
- Kidney stones
- Dehydration can worsen the disturbance



Kidney stone (hypercalcaemia)

LOW CALCIUM: INCREASED EXCITABILITY

Hypocalcaemia may cause anxiety-like distress, irritability, tingling, cramps, tetany, seizures or confusion. Neuromuscular symptoms can be mistaken for panic. Ask about recent neck surgery, renal disease, malabsorption and medicines. Severe symptoms require urgent medical care. Chvostek or Trousseau signs can support the examination but cannot replace calcium measurement or establish the cause.

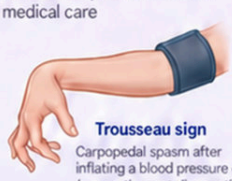
Long-standing disturbances can be associated with intracranial calcification and movement or cognitive findings; do not infer this complication from one low result.

Common features of low calcium

- Anxiety-like distress and irritability
- Tingling, cramps, tetany, seizures or confusion
- Neuromuscular symptoms can be mistaken for panic
- Recent neck surgery, renal disease, malabsorption, medicines
- Severe symptoms require urgent medical care



Chvostek sign
Facial muscle twitch after tapping facial nerve (supportive, not diagnostic)



Trousseau sign
Carpopedal spasm after inflating a blood pressure cuff (supportive, not diagnostic)

High calcium
Slowing
Systemic clues
Consider cause

CALCIUM–PARATHYROID HORMONE INTERPRETATION

Calcium result	Parathyroid hormone (PTH)	Likely interpretation
High	Elevated or inappropriately nonsuppressed	Parathyroid-dependent process (e.g. primary hyperparathyroidism, including lithium-associated)
High	Suppressed	Non-parathyroid causes (e.g. malignancy, vitamin-related mechanisms)
Low	Elevated	Appropriate response (e.g. vitamin D deficiency, renal disease, malabsorption)
Low	Low or inappropriately normal	Parathyroid failure or other causes (e.g. after neck surgery, autoimmune)

Low calcium
Increased excitability
Neuromuscular clues
Consider cause

MEASURE AND INTERPRET TOGETHER

Check calcium using the laboratory's reference interval, albumin, renal function, phosphate, magnesium and parathyroid hormone as indicated. Total calcium is influenced by albumin; albumin-adjustment formulas are imperfect, particularly in acute illness. Ionised calcium can clarify selected situations, with careful sample handling and attention to acid-base effects. Repeat unexpected stable results; do not delay urgent care for a symptomatic severe disturbance.

Typical adult total-calcium intervals are around 2.2–2.6 millimoles/litre, but local assay ranges govern interpretation. Degree, rate of change and clinical state all matter. Electrocardiography is useful when symptoms, marked abnormalities or interacting medicines raise cardiac concern: low calcium can prolong repolarisation, while high calcium can shorten it.



Low calcium: prolonged repolarisation
High calcium: shortened repolarisation

LITHIUM CONNECTS THE ENDOCRINE AND PSYCHIATRIC PLANS

Lithium can alter calcium-sensing and promote hyperparathyroidism/hypercalcaemia. Check calcium before treatment and periodically alongside renal and thyroid function: Specialist Pharmacy Service guidance includes six-monthly calcium in stable treatment. New thirst, polyuria, constipation or cognitive change deserves review rather than waiting for the routine visit.

Lithium

Possible contributor



May alter calcium-sensing and promote hyperparathyroidism / hypercalcaemia

A raised result calls for confirmation and endocrine/psychiatric assessment. Stopping lithium does not always reverse established parathyroid disease, while abrupt withdrawal can destabilise mood. Decide continuation, adjustment, alternative treatment or endocrine intervention jointly, considering severity and prior psychiatric benefit. Suspected lithium toxicity requires urgent evaluation separately from chronic calcium elevation.

TREAT THE CAUSE AND REASSESS THE MIND

Symptomatic or severe calcium disturbance requires medical treatment and monitoring. Hydration, calcium replacement or calcium-lowering treatment depends on the direction and cause, kidney/cardiac status and severity; there is no universal psychiatric-ward fluid prescription. Correct magnesium where relevant and treat the underlying disorder.

Persistent depression, anxiety or psychosis may still need psychological or pharmacological care after correction. Review baseline illness, residual symptoms and function; recovery need not mirror the laboratory timetable. Consider seizure threshold, cardiac repolarisation and sedation when selecting psychotropics. Record who follows calcium, hormone results and mental state.



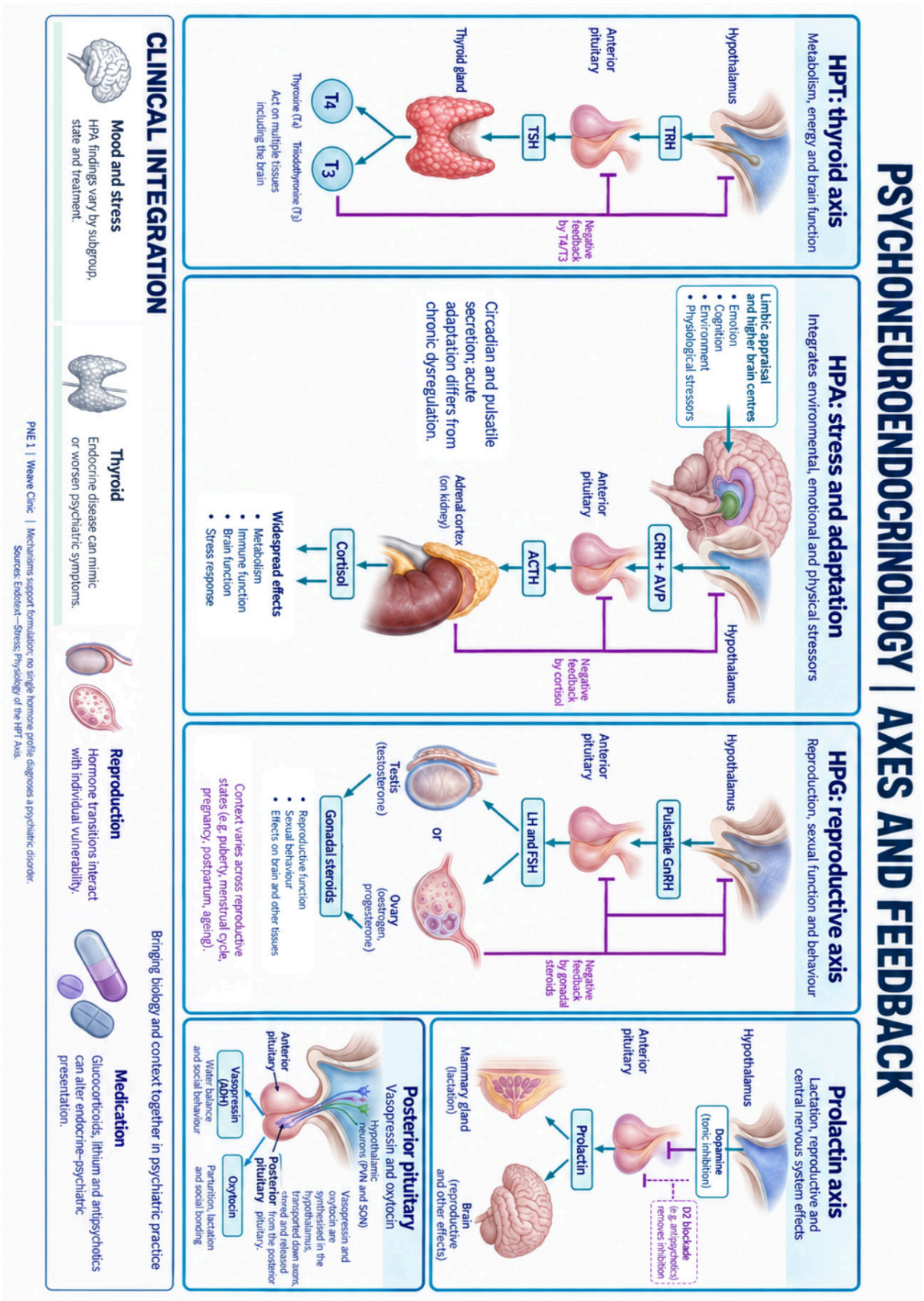
MEMORY AID:
Measure calcium;
interpret the hormone;
review lithium;
reassess symptoms.

Sources: Indian Psychiatric Society endocrine guidance 2022; National Institute for Health and Care Excellence parathyroid guidance; Society for Endocrinology emergency guidance. Original Paper IV Question 59, page 82 plus verified expansion.

September 2026.

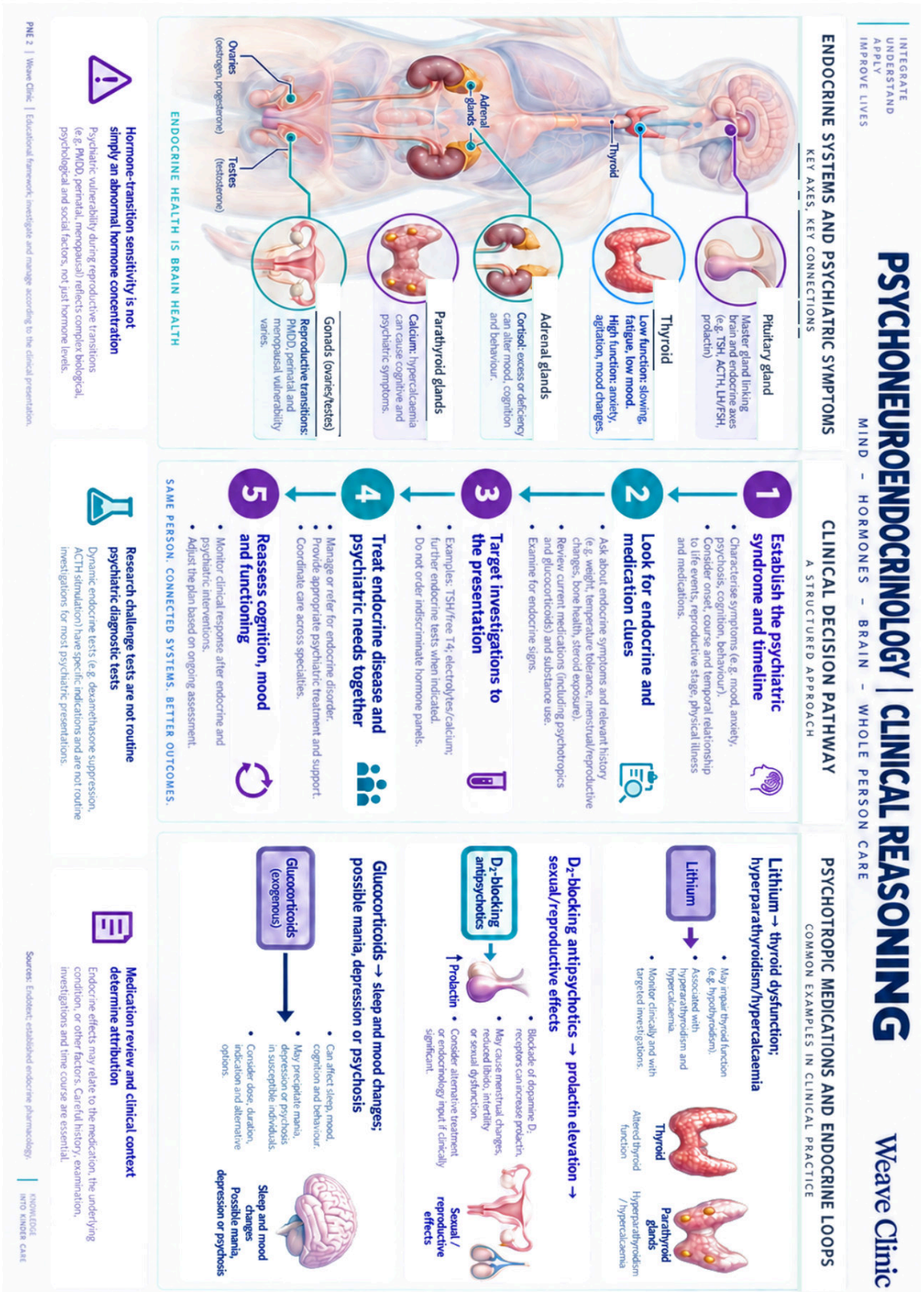
Parathyroid-calcium psychiatric manifestations. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-59.

Credits and references: retained within the illustration.



Psychoneuroendocrinology - PNE-1. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Endocrine and metabolic psychiatry.

Credits and references: retained within the illustration.



Psychoneuroendocrinology - PNE-2. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Endocrine and metabolic psychiatry.

Credits and references: retained within the illustration.

AREA 08 OF 15 · P4-A13

Recent advances and landmark studies

6	5.9%	IV-60	IV-65
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 1 SINGLE 5

REVISION ORDER

The critique slot first: it is the shape the others take and the trials it names carry into three of them. Genetics and the post-traumatic stress slot next, both are advances questions with named studies behind them. The bipolar slot after that, because half of it is classification. Rehabilitation and catatonia are independent, and both cross-link into other areas of this book.

WHAT IS IN THIS AREA

Recent advances asked five different ways: in psychological rehabilitation, in psychiatric genetics with the models of genetic study, in the diagnosis of post-traumatic stress disorder, in the treatment of bipolar affective disorder with the classification first, and as a critique of whether therapeutics have improved prognosis at all. The sixth slot asks for the current status of catatonia in DSM-5 and ICD-11 with its neurobiology and its drug treatment, which is a clinical question the boards set inside this area and is answered on its page as it was set.

IV - 60

Describe the recent advances in psychological rehabilitation in persons with major mental illness. [10]

10 RECENT ADVANCES AND LANDMARK STUDIES

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

SKELETON

2	What changed	From occupying time to restoring a role
5	The advances	Four, each with the evidence behind it
2	Where they stand	Available here, or still a trial finding
1	What is unsolved	One sentence, honest

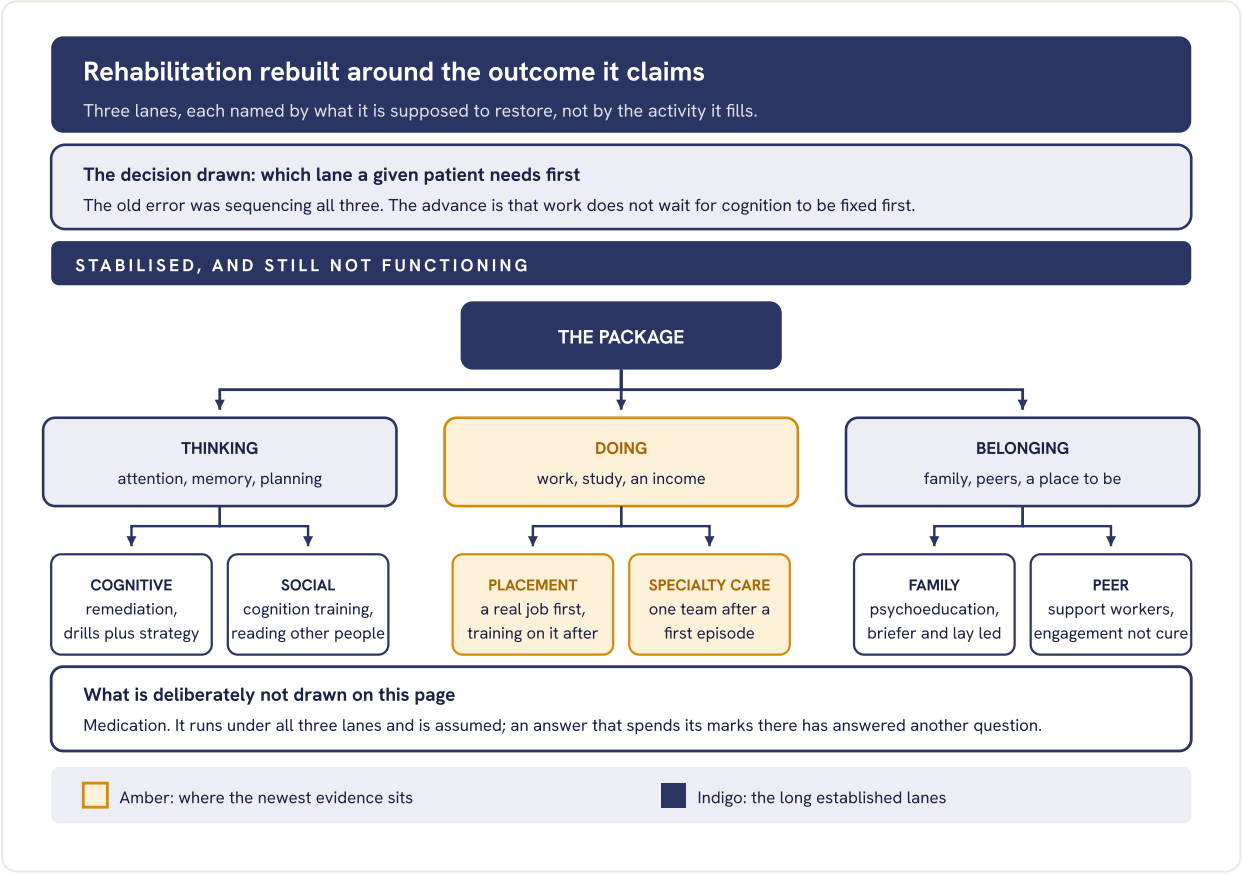


Fig 060.1 The package sorted by the outcome each lane claims, which is what makes place-then-train visible as a change of principle rather than a change of timetable.

ADVANCE	WHAT IT DOES	EVIDENCE SO FAR	STATUS
Cognitive remediation	Drills and strategies for attention, memory and planning	**Meta-analyses show durable cognitive gain, and functional gain when paired with rehabilitation**	In use where staffed
Individual placement and support	A competitive job first, training on the job after	**Randomised trials favour it over train-then-place services**	Established, thinly available
Coordinated specialty care	One team for drugs, family, work and study after a first episode	The RAISE trial improved quality of life and engagement against usual care	Few Indian sites
Task shifted community care	Lay health workers deliver the package at home	The COPSI trial found modest benefit, larger at rural sites	Trial stage here

STATUS: AVAILABLE, AND NOT AVAILABLE

Cognitive remediation and family work need only trained staff, and they travel. Supported employment and coordinated specialty care need an employer network and a funded team, so they stay in a few centres. What is unsolved: none of these has been shown to change long term recovery, only functioning while the programme runs.

WHAT EARNS THE MARKS

- **Place-then-train named as a reversal,** the clearest advance on the page.
- **Each advance tied to its evidence,** not a list of programme names.
- **An Indian trial named,** which almost no answer offers.
- **The unsolved line,** separating functioning from recovery.

SEE ALSO Slot IV - 64 have advances improved prognosis Slot IV - 02 principles of rehabilitation **SPRINT** Day 39, Community psychiatry and public health

IV - 61

Describe the different models of genetic study in psychiatry. Discuss the recent advances in psychiatric genetics. [5+5]

5 + 5 | RECENT ADVANCES AND LANDMARK STUDIES

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The classical models	Family, twin, adoption, and what each isolates
3	The molecular models	Linkage, candidate gene, association, sequencing
3	What has advanced	Loci, structural variants, cross-disorder overlap
2	What has not	Prediction for one patient, and why

- **The models, and what each isolates.** Family studies give recurrence risk in relatives but cannot separate genes from a shared home. Twin studies compare identical with fraternal concordance and yield heritability. Adoption studies separate rearing from inheritance. Linkage tracks co-segregation within families and finds large effect loci. Candidate gene association tested single guesses and mostly failed to replicate. Genome-wide association tests millions of common variants and needs enormous samples. Sequencing finds the rare coding variants the others miss.

ADVANCE	WHAT IT DOES	EVIDENCE SO FAR	STATUS
Genome-wide association	Common variants of tiny individual effect	Hundreds of loci in schizophrenia; the strongest signal sits in the histocompatibility region	Established research finding
Rare and structural variants	Large deletions and rare coding variants	**The 22q11.2 deletion carries a large risk; sequencing converges on synaptic and chromatin genes**	Useful in syndromic cases
Cross-disorder overlap	Shared variance across diagnoses	Substantial genetic correlation between schizophrenia, bipolar disorder and depression	Changing how boundaries are read
Pharmacogenomics	Metabolism and hypersensitivity typing	**CYP2D6 and CYP2C19 status guides dose; HLA-B 15:02 typing before carbamazepine**	The one advance in routine use

STATUS: THE GAP BETWEEN DISCOVERY AND THE CLINIC

A polygenic risk score sums thousands of variants and predicts well across a population while telling an individual patient almost nothing, so it stays a research instrument. The findings that have reached practice are not the loci but the pharmacogenomic ones, where a single typed variant changes a dose or prevents a severe drug reaction.

WHAT EARNS THE MARKS

- **Each classical model paired with what it isolates,** not simply named.
- **The candidate gene era described as a failure,** which is the honest history.
- **Population prediction separated from individual prediction,** the line that carries the last two marks.

SEE ALSO Slot IV - 63 bipolar disorder and recent advances SPRINT Day 38, Recent advances and landmark studies

IV - 62

Discuss the recent advances and the future of the diagnosis of post-traumatic stress disorder. [5+5]

5 + 5 | RECENT ADVANCES AND LANDMARK STUDIES

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Where the diagnosis moved	Out of anxiety, into trauma and stressor related
3	The two systems now	Four clusters against three, and complex PTSD
3	Measurement	The reference interview, and the new questionnaire
2	What the future rests on	Markers, and why none of them diagnoses yet

- **The two systems have diverged, not converged.** DSM-5 moved the disorder out of the anxiety disorders and widened it: four clusters, being intrusion, avoidance, negative alterations in cognition and mood, and altered arousal, plus a dissociative subtype and a separate preschool form. ICD-11 narrowed it instead to three: re-experiencing in the here and now, avoidance, and a persistent sense of current threat, and added complex PTSD alongside. Applied to the same sample the two produce different, overlapping populations.

ADVANCE	WHAT IT DOES	EVIDENCE SO FAR	STATUS
DSM-5 restructuring	Four clusters, with dissociative and preschool subtypes	Factor analytic work supports more than the original three clusters	In routine use
ICD-11 narrowing, with complex PTSD	Three core clusters, and a separate complex form carrying disturbances of self-organisation	**Latent class studies separate the two forms rather than grading one**	In routine use
The International Trauma Questionnaire	Measures the ICD-11 construct, both forms	Validated across many languages and settings	In routine use
Biological markers	Hippocampal volume, cortisol response, startle, methylation	**Group differences only, and no useful accuracy in an individual**	Research only

STATUS: WHAT THE FUTURE ACTUALLY RESTS ON

There is no validated diagnostic biomarker for this or for any other primary psychiatric disorder, and the candidates listed above have been studied for decades without becoming one. The realistic near future is better structured assessment: a clinician administered interview as the reference standard, dimensional measurement alongside the category, and repeated sampling after a trauma rather than a single cross-sectional judgement.

WHAT EARNS THE MARKS

- **The divergence stated as a divergence.** Most answers describe DSM-5 and assume ICD-11 followed it.
- **Complex PTSD placed in the right system,** with its distinguishing features named.
- **Markers reported as group findings,** which is the difference between a current answer and a hopeful one.

SEE ALSO Slot IV - 65 catatonia in the two systems **SPRINT** Day 38, Recent advances and landmark studies

IV - 63

Classify bipolar affective disorder and discuss the recent advances in its treatment. [5+5]

5 + 5 | RECENT ADVANCES AND LANDMARK STUDIES

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The classification	Types and specifiers, in one frame
3	Where the systems differ	The mixed episode, category against specifier
3	Advances in treatment	The agents that changed the depressed phase
2	What the trials removed	The antidepressant question, answered

- **The classification.** Both systems recognise bipolar type I, bipolar type II and cyclothymic disorder, with residual other specified and unspecified categories. Episodes are then described by pole, by the presence of psychotic symptoms, and by course modifiers such as rapid cycling and seasonal pattern.
- **Where they differ.** DSM-5 abolished the mixed episode and replaced it with a mixed features specifier that attaches to a manic, hypomanic or depressive episode. ICD-11 kept the mixed episode as a category. The DSM route makes mixed depression visible in a way the older criteria did not.

ADVANCE	WHAT IT DOES	EVIDENCE SO FAR	STATUS
Cariprazine	D3 preferring partial agonist, licensed across the phases	Randomised trials in acute mania and in bipolar depression	Marketed in India
Lumateperone	Newer agent aimed at the depressed phase	Randomised placebo controlled trials, alone and as an adjunct	Approved abroad, access limited here
Antidepressants reconsidered	An antidepressant added to a mood stabiliser	**The STEP-BD programme found no advantage over placebo added to the stabiliser**	Practice already changed
Psychoeducation and rhythm therapy	Structured relapse prevention, and stabilised daily routine	**Randomised trials show fewer relapses across follow up**	In use where staffed

STATUS: WHERE THE REAL CHANGE HAPPENED

The depressed phase is where nearly all recent movement sits, because it carries most of the disability and had the fewest licensed options. Lithium and the older agents remain the backbone of maintenance. What is unsolved is prevention of the depressive relapse, which every maintenance trial finds harder than mania.

WHAT EARNS THE MARKS

- **The mixed presentation handled as the point of difference,** not as a footnote to the list of types.
- **Each new agent tied to the phase it treats,** since none of them is a general advance.
- **A named trial for the negative finding.** Saying antidepressants are unhelpful without it is an opinion.
- **The unsolved line:** depressive relapse, which the status strip names.

SEE ALSO Slot IV - 74 lithium in youths with bipolar disorder Slot IV - 73 long acting injections in mood disorders

SPRINT Day 38, Recent advances and landmark studies

IV - 64

Have the advances in therapeutics over the last few decades improved the prognosis of mental disorders? Write a critique. [10]

10 | RECENT ADVANCES AND LANDMARK STUDIES | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The question restated	Prognosis of what, measured how
4	Domain by domain	Where it improved, and where it did not
2	Why the record looks better than it is	Publication bias and the outcomes chosen
2	The verdict	What actually changed outcome, and it was rarely a molecule

- **Restate before answering.** Prognosis is not one measurement. Symptom response, relapse, social function and death have moved in different directions, and an answer that treats them as one cannot be right about any of them.
- **Why the published record reads better than the clinic.** Analysis of the drug regulator's own trial database showed that positive antidepressant trials were published and negative ones largely were not, so the apparent effect in the literature exceeds the effect in the submitted data.

DOMAIN	WHAT CHANGED	THE EVIDENCE	VERDICT
Acute symptom control	Reliable control of mania, psychosis and catatonia	Decades of trials; ECT and clozapine remain the most effective treatments in psychiatry	Genuinely improved
Newer against older drugs	Second generation antipsychotics	**CATIE and CUtLASS found no advantage on effectiveness or on quality of life**	No gain
Long term function	Employment and social recovery in schizophrenia	Cohort comparisons across decades show little change	Largely unchanged
Physical mortality	Life expectancy in severe mental illness	**One to two decades of life lost, mostly cardiovascular, and the gap has not closed**	The strongest counter-argument

STATUS: WHAT DID MOVE THE PROGNOSIS

Four things, and only one of them is new. Clozapine in resistant schizophrenia, including its effect on suicidal behaviour. Lithium in bipolar disorder, including its anti-suicidal signal. Electroconvulsive therapy in severe and refractory illness. And the reorganisation of services: early intervention after a first episode, and the move out of long stay institutions. The largest gains came from how care was arranged rather than from what was prescribed.

WHAT EARNS THE MARKS

- **Prognosis broken into separate outcomes** in the first two lines.
- **Two named negative trials.** A critique without them is an assertion.
- **The mortality gap raised,** which is the fact that decides the question and is the one most answers omit.
- **A verdict that credits service organisation,** not a closing sentence about the future being bright.

SEE ALSO Slot IV - 60 advances in psychological rehabilitation Slot IV - 75 me too drugs and new agents SPRINT

Day 38, Recent advances and landmark studies

IV - 65

What is the current status of catatonia in DSM-5 and ICD-11? Discuss briefly its neurobiology and the pharmacological options for treatment. [3+3+4]

3 + 3 + 4 | RECENT ADVANCES AND LANDMARK STUDIES

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	Where it sits now	A DSM-5 specifier against an ICD-11 grouping
3	Neurobiology	GABA, glutamate, dopamine, and the motor circuit
4	Treatment	Lorazepam, then ECT, and what must be stopped

- **The status.** DSM-5 does not make catatonia an independent disorder: it is a specifier on another mental disorder, a catatonic disorder due to a medical condition, or unspecified catatonia, on three or more of twelve features. ICD-11 made it a grouping in its own right, with forms associated with another mental disorder, substance induced, and secondary.
- **Neurobiology.** Reduced GABA-A signalling in orbitofrontal and prefrontal cortex is the best supported account and explains the benzodiazepine response. Glutamatergic overactivity is supported by catatonia in anti-NMDA receptor encephalitis. Dopaminergic hypoactivity explains the resemblance to neuroleptic malignant syndrome.

OPTION	WHAT IT DOES	EVIDENCE SO FAR	STATUS
Lorazepam challenge	1 to 2 mg, response within minutes to hours; both a test and a treatment	**Open trials and large case series; high response rates in the non-malignant form**	First line everywhere
Continued lorazepam	Titration onward, often to 6 to 16 mg a day in divided doses	Observational; sedation is less than the dose suggests while catatonia persists	In routine use
Electroconvulsive therapy	Induced seizure, and the first line in malignant catatonia	**A long observational record and no alternative in the malignant form**	Established
NMDA antagonists	Amantadine or memantine when benzodiazepines fail	Case series only	Second line, weak evidence

WHAT MUST BE STOPPED

Antipsychotics. A dopamine blocker continued in a catatonic patient risks the malignant form or neuroleptic malignant syndrome, and the two are already hard to separate. Treat the cause too: encephalitis, metabolic derangement, withdrawal.

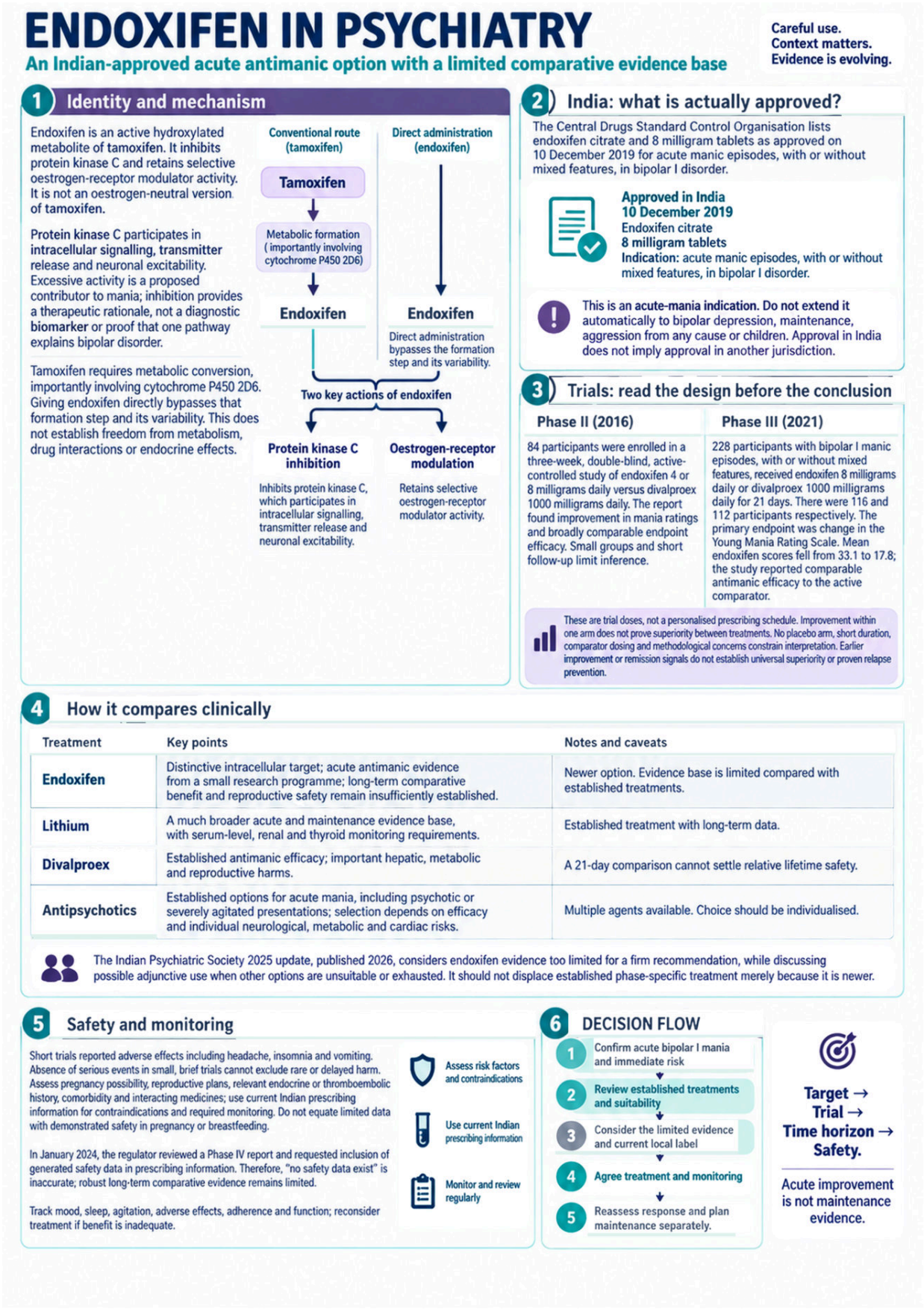
WHAT EARNS THE MARKS

- **The two systems contrasted,** specifier against independent grouping, which is the current status.
- **Each transmitter tied to a clinical observation,** not listed as three hypotheses.
- **The challenge dose given as both test and treatment,** which is the point of it.
- **Stopping the antipsychotic,** which most answers never write down.

SEE ALSO Slot IV - 73 neuroleptic malignant syndrome on a depot

Slot IV - 62 diagnosis of post-traumatic stress disorder **SPRINT** Day 38, Recent advances and landmark studies

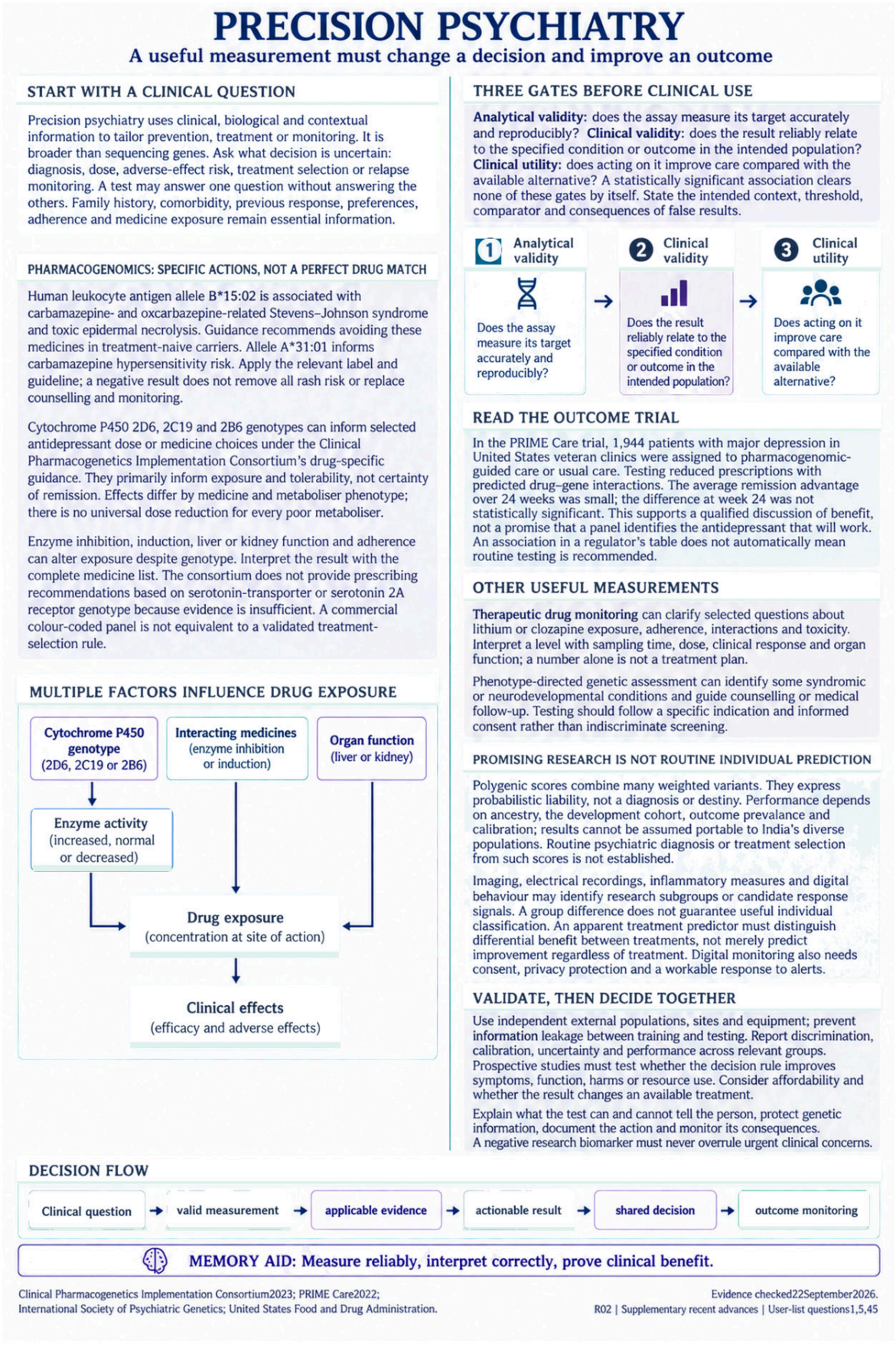
WEAVE 100 · PAPER IV · R01



Endoxifen in psychiatry. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Recent advances and landmark studies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · R02



Precision psychiatry and clinical translation. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Recent advances and landmark studies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · R03

R03 — Nanoparticle drug delivery: reaching the brain is only one step.

The delivery problem.

A medicine may be active in a laboratory yet fail clinically because it degrades, distributes to the wrong organs, cannot reach brain tissue or causes toxicity before an effective concentration is achieved. Nanocarriers aim to alter this journey. They are delivery systems, not automatically new therapeutic mechanisms. Nanometre-scale properties can change distribution and biological interactions, but small size alone does not guarantee brain entry or safety.

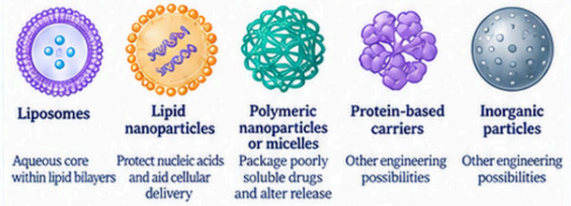
Understand the blood–brain barrier.

Brain microvascular endothelial cells have tight junctions and specialised transport systems. Pericytes, basement membrane and astrocytic end-feet support the neurovascular unit. Efflux transporters can return some molecules toward blood. A carrier must survive circulation, reach the barrier, cross or release a suitable payload, distribute through tissue and deliver active drug to the intended cells. Binding to an endothelial receptor is not proof of transport across the endothelium; uptake into a vesicle can end in degradation rather than release into brain.

Know the carrier choices.

Liposomes enclose an aqueous space within lipid bilayers and can carry different payloads in the core or membrane. Lipid nanoparticles can protect nucleic acids and aid cellular delivery. Polymeric nanoparticles or micelles can package poorly soluble drugs and alter release. Protein-based carriers and inorganic particles have other engineering possibilities. These categories overlap and individual formulations behave differently. Size distribution, shape, surface charge, coating, payload stability and manufacturing consistency all matter; there is no single optimal particle for every disease.

Examples of nanocarrier types (not exhaustive)



Targeting is a sequence, not a promise.

Surface ligands may bind receptors such as transferrin-related transport systems, aiming for receptor-mediated transcytosis. Surface adsorption and blood proteins can change how the carrier behaves; a protein corona may mask a designed ligand. Passive accumulation in a leaky tumour barrier is variable and is not equivalent to crossing an intact blood–brain barrier. A second targeting step may be required to reach a neuron, glial cell or tumour cell after tissue entry. For intracellular nucleic-acid delivery, escape from endosomal compartments can be another bottleneck.

Release must match the goal.

Encapsulation may reduce premature degradation or peak exposure. Release can follow diffusion, carrier breakdown or experimental triggers such as acidity, enzymes, heat or ultrasound. Too-early release loses targeting; too-late release leaves the active drug unavailable. Intranasal delivery and focused-ultrasound barrier opening are investigated approaches, but neither makes a nanoparticle universally brain-selective. Ultrasound with microbubbles modifies barrier permeability and requires its own safety assessment.

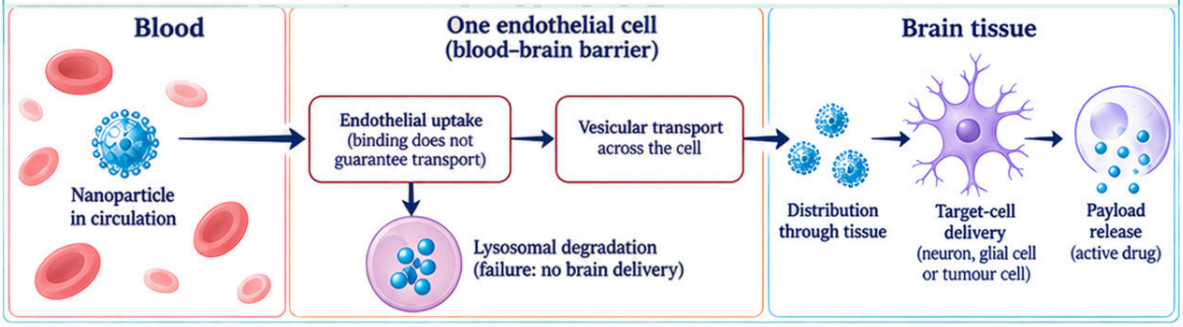
Established technology versus experimental psychiatry.

Nanomedicines have established uses outside psychiatry, including selected liposomal and protein-bound cancer treatments and lipid-based nucleic-acid delivery. Their approval validates a specific product and indication, not all nanoparticles. Human glioblastoma studies have investigated ultrasound-assisted delivery of albumin-bound paclitaxel; this is an early translational example, not evidence for routine psychiatric treatment. Brain-targeted carriers for psychiatric drugs, genes or biological agents remain predominantly investigational. A long-acting antipsychotic injection is not automatically a nanoparticle brain-targeting system, and prolonged release does not prove preferential brain delivery.

Judge the evidence and risks.

Ask whether a study measured total brain-associated material or active drug within target tissue; particles trapped in vessels can inflate apparent delivery. Compare free drug, empty carrier and relevant controls. Require clinical outcomes beyond imaging fluorescence or animal behaviour. Examine dose, repeated exposure, immune or infusion reactions, organ accumulation, degradation products and off-target toxicity. Manufacturting changes can alter the product, so quality control and reproducibility are central. Regulators evaluate the specific formulation's safety, effectiveness and quality; "nano" is not an approval category or a guarantee of benefit. No routine Indian psychiatric availability is asserted here.

From blood to brain: a possible route, and a common failure point



Established formulations for specific nonpsychiatric indications

- Selected liposomal and protein-bound cancer treatments.
- Lipid-based nucleic-acid delivery.
- Approval validates a specific product and indication, not all nanoparticles.

Investigational brain-targeted psychiatric delivery

- Brain-targeted carriers for psychiatric drugs, genes or biological agents remain predominantly investigational.
- Human glioblastoma studies have investigated ultrasound-assisted delivery of albumin-bound paclitaxel; this is an early translational example, not evidence for routine psychiatric treatment.
- A long-acting antipsychotic injection is not automatically a nanoparticle brain-targeting system, and prolonged release does not prove preferential brain delivery.

Memory aid: "Protect, reach, cross, release, prove benefit."

Supplementary clinical teaching • September 2026.

Nanoparticle drug delivery. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Recent advances and landmark studies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · R05

R05 — New directions in schizophrenia pharmacotherapy: target, evidence, clinical role

- **Name the target, name the outcome, check the evidence, monitor the person.**

Memory aid

Start with the unmet problem.

Established antipsychotics mainly influence dopamine type 2 receptor signalling and can improve positive symptoms, but cognitive impairment, persistent negative symptoms, functional disabiiity and adverse effects remain major concerns. A new mechanism is useful only if it improves a meaningful outcome with acceptable harm. Separate treatment of acute psychosis, relapse prevention, negative symptoms and cognition; success in one domain does not prove success in all.

Muscarinic signalling: an approved change of approach.

Xanomeline-trospium, marketed as **Cobenfy**, received United States approval in September2024 for schizophrenia in adults. Xanomeline activates muscarinic acetylcholine receptors, with activity relevant to type1 and type4 receptors; the precise therapeutic mechanism is not fully established. Trospium is a predominanity peripheral muscarinic antagonist intended to reduce peripheral cholinergic adverse effects. The combination does not depend on direct dopamine type2 receptor blockade, although downstream dopamine circuits may still be affected. "Beyond direct blockade" does not mean dopamine has become irrelevant.

What the evidence supports.

Two pivotal five-week placebo-controlled studies supported the original approval, showing improvement in total Positive and Negative Syndrome Scale scores. These acute trials do not by themselves establish superiority to clozapine, effectiveness in treatment-resistant schizophrenia, independent treatment of primary negative symptoms or restoration of cognition. The 2025 adjunctive ARISE trial did not meet its primary endpoint when the combination was added to an existing atypical antipsychotic. An approved monotherapy indication must not be converted into an evidence-free add-on recommendation.

A different adverse-effect profile still needs monitoring.

Nausea, vomiting, gastrointestinal symptoms, urinary retention and changes in heart rate or blood pressure are clinically relevant. Review anticholinergic medicines and the current product label, including contraindications involving urinary or gastric retention, untreated narrow-angle glaucoma and moderate or severe hepatic impairment. Assess renal and hepatic function as required by the label. Lower liability for some dopamine-blockade-related effects is not absence of adverse effects. Choice must account for the patient's comorbidity, prior response, preferences and practical ability to take treatment.

Other targets: plausible does not mean proven.

Selective muscarinic type4 positive allosteric modulation aims to amplify the receptor's response to acetylcholine. Emraclidine's two phase2 EMPOWER trials failed their primary endpoints in 2024, illustrating that one muscarinic drug's success does not validate every drug in the class. Trace amine-associated receptor1 agonism is another strategy affecting monoaminergic signalling without direct dopamine type2 blockade; early promise requires confirmation rather than assumption of approval. Glutamatergic strategies include enhancing glycine-related signalling at the N-methyl-D-aspartate receptor. The glycine transporter1 inhibitor iclepertin failed to improve the primary cognitive outcome in the phase3 CONNEX programme. Mechanistic elegance cannot replace a positive clinical trial.

Match the domain before selecting a treatment.

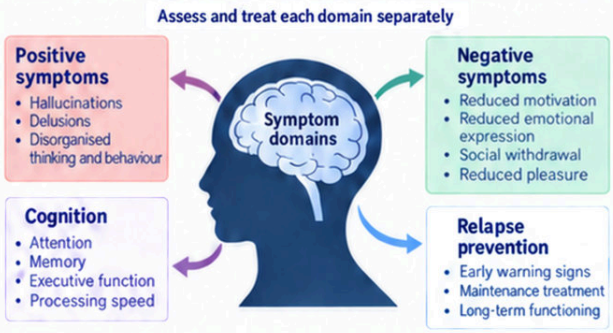
For apparent negative symptoms, assess depression, persistent psychosis, sedation, drug-induced movement problems and environmental deprivation before calling them primary negative symptoms. For cognition, address sleep, substance use, anticholinergic burdon and medical causes, and offer appropriate cognitive remediation and rehabilitation. Cariprazine, clozapine and long-acting formulation remain important background options for selected clinical problems; they should not be displaced merely because a newer target exists. Confirm adequate trials, adherence and diagnosis when evaluating treatment resistance.

Use an evidence ladder.

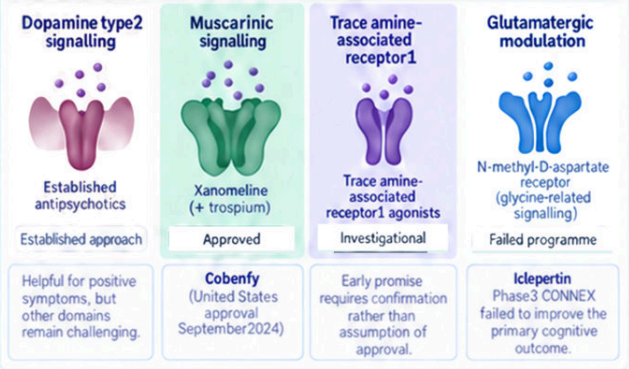
Target engagement → controlled symptom benefit → functional benefit and durability → comparative effectiveness and real-world safety. These are separate questions. Record whether results are peer-reviewed or sponsor topline reports, what comparator was used and which population was studied. A change in total symptom score is not automatically a change in employment or independent living. Monitor symptoms, functioning, adverse effects, cardiometabolic health and adherence through shared care. Approval and access differ by jurisdiation; this sheet makes no claim of Indian approval, marketing or availability for experimental agents or Cobenfy.

Symptom domains in schizophrenia

Assess and treat each domain separately



Mechanisms and example agents

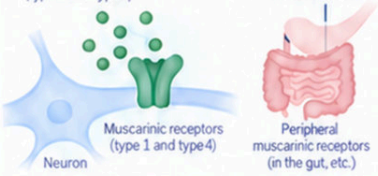


Muscarinic example: Cobenfy (xanomeline–trospium)

Xanomeline activates muscarinic acetylcholine receptors (type1 and type4)

+

Trospium reduces peripheral cholinergic adverse effects



Key points

- Approved in the United States in September2024 for schizophrenai in adults.
- Precise therapeutic mechanism is not fully established.
- Does not depend on direct dopamine type2 receptor blockade, although downstream dopamine circuits may still be affected.
- "Beyond direct blockade" does not mean dopamine has become irrelevant.

Selected other programmes

Emraclidine

- Selective muscarinic type4 positive allosteric modulation.
- Two phase2 EMPOWER trials failed their primary endpoints in 2024.

Failed programme

Trace amine-associated receptor1 agonists

- Affecting monoaminergic signalling without direct dopamine type2 blockade.
- Early promise requires confirmation rather than assumption of approval.

Investigational

Iclepertin

- Glycine transporter1 inhibitor.
- Failed to improve the primary cognitive outcome in the phase3 CONNEX programme.
- Mechanistic elegance cannot replace a positive clinical trial.

Failed programme

The evidence ladder



No automatic path from a novel target to a cure.

Supplementary clinical teaching · September 2026.

New directions in schizophrenia pharmacotherapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Recent advances and landmark studies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · R06

R06 — Psychedelic-assisted therapy: a treatment context, not just a molecule.

Memory aid:
"Prepare safely, support the session, integrate carefully, measure lasting benefit."

Define the intervention.

Psychedelic-assisted approaches combine administration of a psychoactive drug with structured preparation, supervised support during its effects and follow-up integration. Protocols differ in the amount and type of psychotherapy. Psilocybin is converted to psilocin, which acts principally through serotonin type2A receptor agonism. Lysergic acid diethylamide is another classic psychedelic. 3,4-methylenedioxymethamphetamine is an entactogen with monoamine-releasing effects, not simply a classic serotonergic psychedelic. Ketamine-based treatments have different mechanisms and approvals and should not be treated as interchangeable evidence.

Proposed mechanisms are not outcome measures.

Changes in perception, emotional processing and patterns of network activity may create opportunities for psychological learning. Neuroplasticity is an important research hypothesis, but a subjective breakthrough or altered scan does not establish durable recovery. Drug effects, expectations, therapeutic relationships and the treatment environment may all contribute. Trials evaluate the package delivered, not an abstract molecule detached from its context.

Preparation comes before administration.

Establish diagnosis, prior adequate treatments, capacity for informed consent, expectations and a safety plan. Review cardiovascular disease, blood pressure, pregnancy, neurological illness, substances and interacting medicines. Many trials exclude people with psychotic disorders, bipolar vulnerability or unstable medical disease; findings cannot be automatically generalised to these groups. Discuss the possibility of fear, distress, lack of benefit and symptom deterioration. Medication changes require individual medical supervision; patients should not stop antidepressants or mood stabilisers on their own to enter treatment.

The supported session.

Use a regulated clinical or research setting with trained staff, a clear protocol, physiological monitoring and an emergency response plan. Maintain privacy, documented boundaries and consent; altered consciousness increases vulnerability to suggestion and exploitation. Support should not impose interpretations, promise recovered memories or pressure disclosure. Monitor distress, confusion, blood pressure, pulse and other drug-specific risks. Acute anxiety, nausea and headache can occur; severe agitation, mania, psychosis, suicidality or medical instability require assessment and treatment rather than being dismissed as necessary healing.

Integration and follow-up.

After the acute effects subside, help the person reflect on the experience without treating every perception as factual. Connect any useful learning to achievable behavioural goals and ongoing care. Review mood, sleep, functioning, adverse experiences and suicidal thinking over time. Repeated administration and maintenance schedules remain protocol-dependent; a dramatic session is not proof of a permanent cure. People who do not improve still need evidence-based treatment and support.

Read trials critically.

Psilocybin depression studies and 3,4-methylenedioxymethamphetamine-assisted post-traumatic stress disorder trials have reported benefits, but the evidence is heterogeneous. Distinctive acute effects can reveal allocation, weakening blinding and increasing expectancy bias. Ask how the control condition, therapist contact, concomitant treatment, missing data and adverse events were handled. Compare prespecified primary outcomes with exploratory findings. Sponsor-reported phase3 results are not the same as a complete peer-reviewed dataset; uncontrolled extensions cannot establish comparative long-term benefit. Benefit in carefully selected participants does not demonstrate safety for routine unsupervised use.

Regulatory position: check the jurisdiction and date.

As checked in September2026, United States guidance for psychedelic clinical investigations is final; guidance, breakthrough designation or permission to conduct a trial is not marketing approval. Do not present psilocybin or 3,4-methylenedioxymethamphetamine as generally approved United States psychiatric medicines. Australia permits specifically authorised specialist psychiatrists to access psilocybin for treatment-resistant depression and 3,4-methylenedioxymethamphetamine for post-traumatic stress disorder under a restricted framework with ethics approval and prescribing requirements. This is not unrestricted access or approval of every product. No Indian clinical availability or regulatory approval is asserted here; local law and authorisation must be verified for any proposed use.

The treatment pathway (part of a package, not a single dose)



What contributes to the observed outcome?



Regulatory position (as checked in September 2026)

United States
Guidance for psychedelic clinical investigations is final; guidance, breakthrough designation or permission to conduct a trial is not marketing approval. Do not present psilocybin or 3,4-methylenedioxymethamphetamine as generally approved United States psychiatric medicines.

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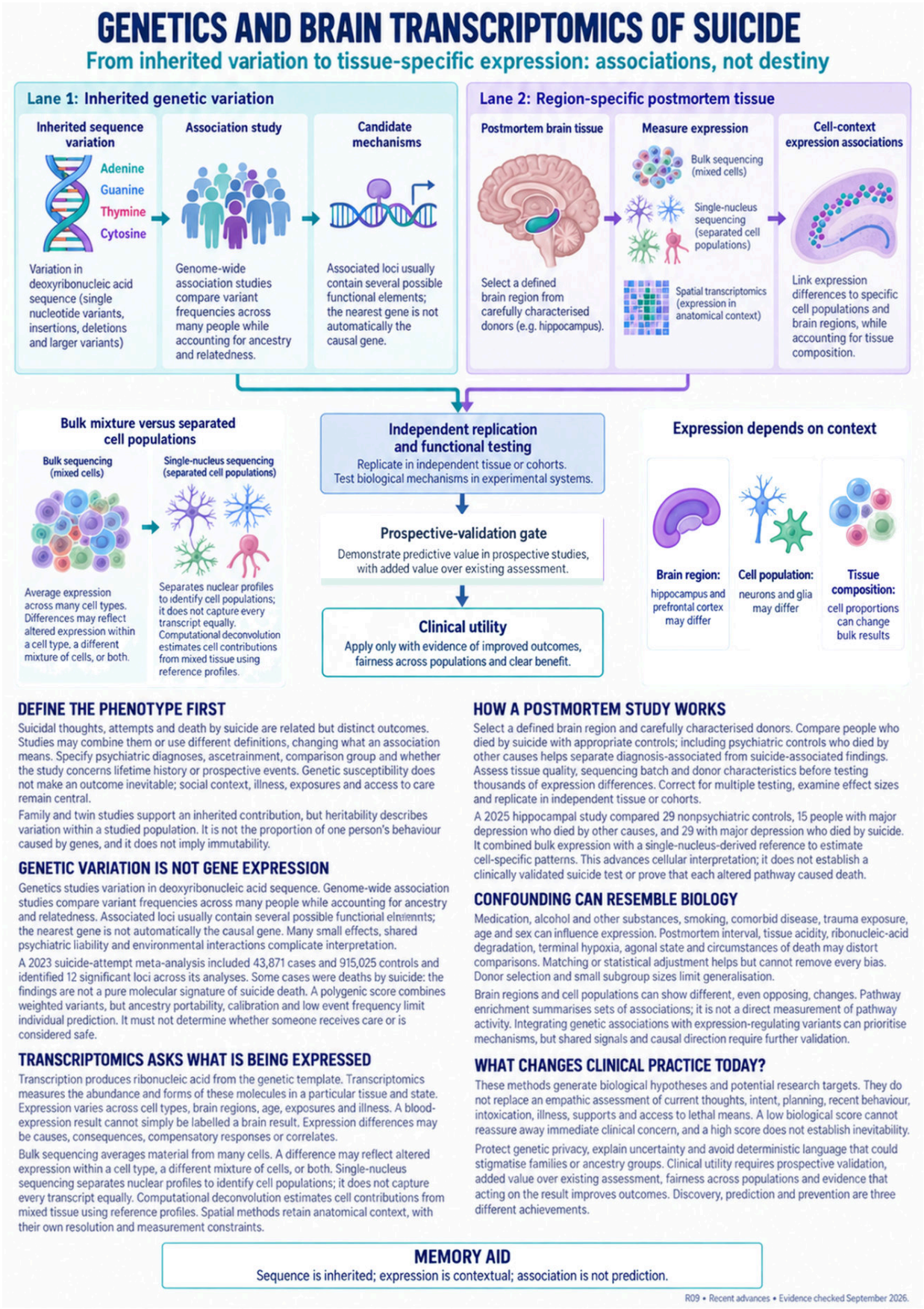
India
No Indian clinical availability or regulatory approval is asserted here; local law and authorisation must be verified for any proposed use.

Supplementary clinical teaching • September 2026

Psychedelic-assisted therapy. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Recent advances and landmark studies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · R09



Genetics and brain transcriptomics of suicide. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Recent advances and landmark studies.

Credits and references: retained within the illustration.

AREA 09 OF 15 · P4-A12

Drug emergencies, interactions, TRD and neurostimulation

6	5.6%	IV-66	IV-71
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

SINGLE 6

REVISION ORDER

The four stimulation slots as one block; they declare their own division in the fragment comments and reading them together is how that division becomes visible. Take the mechanism slot first. The two resistance slots next, they are one answer split, and one of them contains the other's whole question as a sub-part.

WHAT IS IN THIS AREA

Brain stimulation in four slots: the neurosurgical options with repetitive transcranial magnetic stimulation, a critique of the stimulation treatments as a group, the physical treatments with the rTMS procedure in detail, and the mechanisms with the other mechanical methods. The other two are the resistance pair, add-on treatment set against augmentation, and the strategies for the patient who has not responded to standard therapy.

IV - 66

In contrast to single drug treatment in psychiatry, there is some inclination towards add-on drug treatment when a patient does not show adequate response to a drug. What is add-on drug treatment, and how does it differ from augmentation treatment? Critically evaluate the add-on approach in the clinical setting. [2+3+5]

2 + 3 + 5 | DRUG EMERGENCIES, INTERACTIONS, TRD AND NEUROSTIMULATION

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Both terms defined**	One sentence each, and the test that separates them
3	**The grid**	Examples, evidence and risk, side by side
3	**The critique**	Where combining is justified and where it is drift
2	**The rule before either**	An adequate first trial, and a plan to stop

DOMAIN	ADD-ON COMBINATION	AUGMENTATION	WHAT SETTLES IT
What is added	A second drug of the same class, both treating the disorder	A drug that is not itself a treatment for the disorder	**Ask what the added drug would do if given alone**
Worked examples	Mirtazapine or bupropion with a reuptake inhibitor	Lithium, liothyronine, aripiprazole, quetiapine	**Lithium is augmentation, never combination**
Evidence	The CO-MED trial found no gain from starting combined, with more adverse effects	Lithium and liothyronine carry the oldest data; atypicals carry approval	**Augmentation has the stronger trial base**
Main risk	Serotonin toxicity and additive adverse effects	Metabolic, thyroid and renal monitoring	**Same class stacking is the riskier of the two**
When to prefer	Partial response with a residual symptom the second drug covers	Partial response where the first drug is worth keeping	Both need an adequate first trial

Amber cells are the discriminators. The first row is the whole distinction; the rest of the grid only shows what follows from it.

- **The critique, honestly.** Add-on is justified when a specific residual symptom is named and the second drug is chosen to cover it, with a date set to review. It is drift when a drug is added because response was disappointing and nothing was reconsidered.
- **What combining hides.** An undiagnosed bipolarity, an unmentioned substance, a dose never raised, a trial never run long enough. Every one of these looks like resistance and none of them is.

WHAT EARNS THE MARKS

- **The test in the first row of the grid,** not two memorised lists of drugs.
- **A named trial on each side,** which is what turns evaluate into critically evaluate.
- **A stopping plan.** Almost no answer says how a drug that was added comes off again.

SEE ALSO Slot IV - 70 the resistance ladder SPRINT

Day 37, Psychopharmacology II: emergencies, resistance and neurostimulation

IV - 67

What are the options for neurosurgery in psychiatry? Write in brief about the indications and mechanism of action of rTMS. Critically evaluate the efficacy of rTMS in psychiatry. [2+2+2+4]

2 + 2 + 2 + 4 | DRUG EMERGENCIES, INTERACTIONS, TRD AND NEUROSTIMULATION

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Neurosurgical options	Ablative procedures, then the stimulation alternatives
2	Indications for rTMS	Who is offered it, and after what has failed
2	Mechanism	Induced current, frequency, and the circuit claim
4	Critical evaluation	Effect size, the ECT comparison, blinding, durability

THE NEUROSURGICAL OPTIONS, IN TWO GROUPS

Ablative: anterior cingulotomy, anterior capsulotomy, subcaudate tractotomy and limbic leucotomy. Non-ablative and reversible: deep brain stimulation and vagus nerve stimulation. In India, Section 96 of the Mental Healthcare Act 2017 permits psychosurgery only with the person's informed consent and the approval of the concerned Board.

- **Indications for rTMS.** A depressive episode that has not responded to antidepressant treatment, where the illness is not severe enough or urgent enough to need ECT. Obsessive compulsive disorder carries a separate approval, and there is trial evidence in auditory hallucinations and negative symptoms.
- **Mechanism.** A rapidly changing magnetic field over the scalp induces a current in the cortex beneath it and depolarises neurons. High frequency over the left dorsolateral prefrontal cortex is excitatory, low frequency over the right is inhibitory. The lasting effect is attributed to plasticity resembling long term potentiation and to downstream change in prefrontal and limbic circuits.
- **The critique.** Sham controlled trials and meta-analyses show a real but moderate antidepressant effect, below that of ECT. Blinding is imperfect because scalp sensation and muscle twitch differ from sham. Benefit needs maintenance, and durability is poorly studied. Theta burst stimulation was non-inferior to standard high frequency treatment in the THREE-D trial, which cut session time rather than raising the effect.
- **And the practical limit.** Four to six weeks of daily attendance at a centre that owns the machine. In Indian practice that is the constraint, not the evidence.

WHAT EARNS THE MARKS

- **Neurosurgery split into ablative and reversible,** with the statutory safeguard named.
- **Frequency tied to direction of effect,** which is the mechanism mark.
- **The comparison with ECT made explicitly.** An answer that praises rTMS without placing it has not evaluated anything.
- **Blinding named as a methodological problem,** not as a criticism of the operators.

SEE ALSO Slot IV - 69 the rTMS procedure and its complications Slot IV - 68 a critique of brain stimulation SPRINT Day 37, Psychopharmacology II: emergencies, resistance and neurostimulation

IV - 68

Write a critique of the use of brain stimulation treatments in psychiatry. [10]

10 DRUG EMERGENCIES, INTERACTIONS, TRD AND NEUROSTIMULATION

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**What is being critiqued**	Name what the phrase lumps together
4	**The evidence, tier by tier**	The ladder, drawn, with each rung graded
2	**The method problems**	Sham, blinding, durability of effect
2	**Access and ethics**	What is reachable here, and why consent is statutory

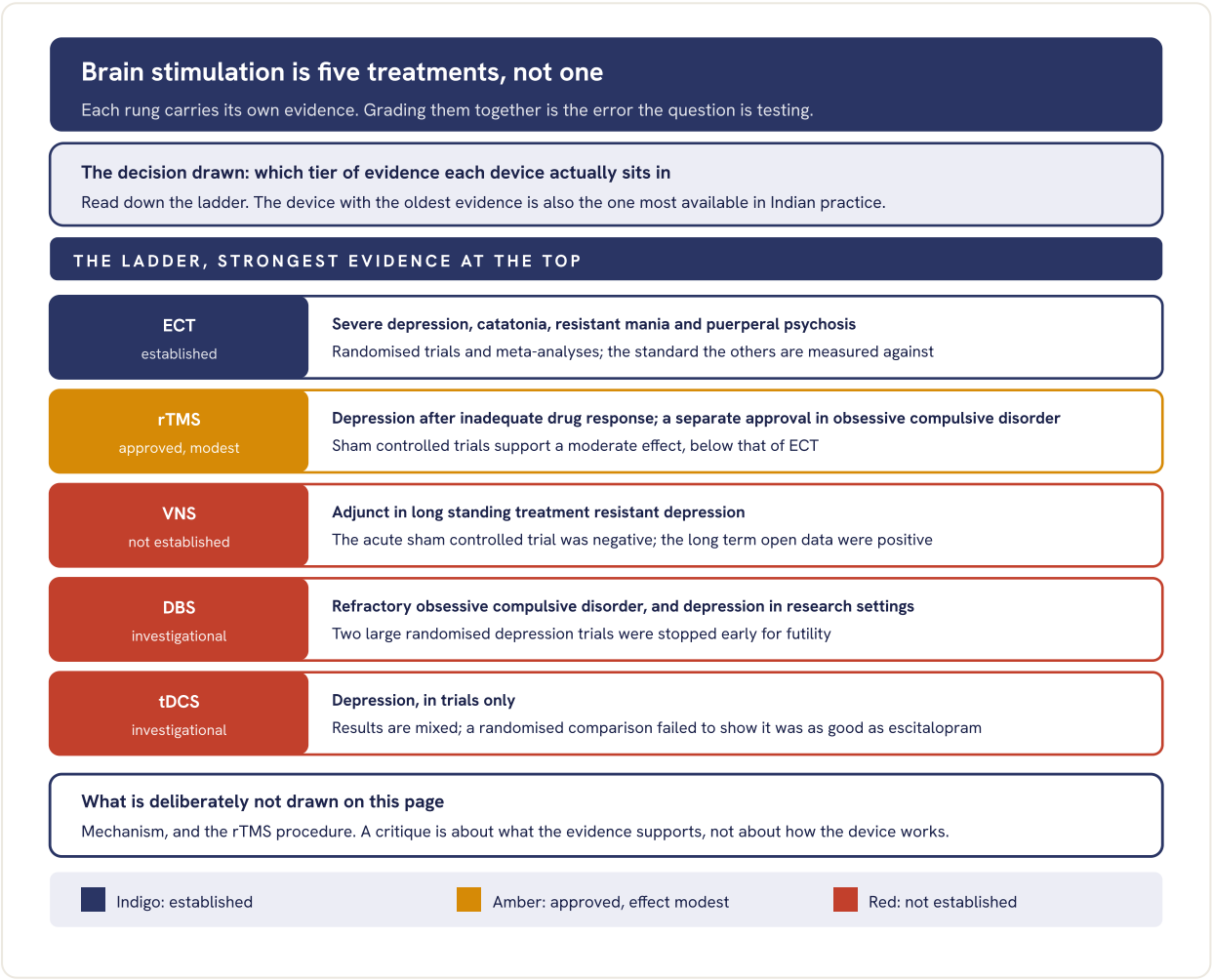


Fig 068.1 The family drawn as a graded ladder rather than a list, so that the critique is legible before a word of it is written.

- **The method problems.** Sham is the hard one: scalp sensation, muscle twitch and daily attendance cannot be blinded, so expectancy is built into every positive trial. Durability is barely studied, and maintenance schedules are empirical rather than trialled.
- **Access and ethics.** Cost and equipment concentrate these treatments in a few centres. The leucotomy era is why consent and Board approval for psychosurgery are written into statute rather than left to professional judgement.

WHAT EARNS THE MARKS

- **Refusing the category.** The first sentence should say that brain stimulation is not one evidence base.
- **Every rung graded,** including the two negative trial results most answers omit.
- **Sham named as the methodological weakness,** which is the difference between a critique and a complaint.

SEE ALSO Slot IV - 71 mechanism of brain stimulation Slot IV - 67 rTMS indications and efficacy **SPRINT**

Day 37, Psychopharmacology II: emergencies, resistance and neurostimulation

IV - 69

Describe the various physical treatment methods used in psychiatry. Discuss the indications, mechanism of action, procedure and complications of repetitive transcranial magnetic stimulation. [10]

10 | ASKED AT 15 MARKS | DRUG EMERGENCIES, INTERACTIONS, TRD AND NEUROSTIMULATION

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The catalogue	Grouped by whether a seizure is induced
2	Indication and mechanism	Who is offered it, and the induced current
4	The procedure	Threshold, target, dose, course
2	Complications and exclusions	What goes wrong, and who cannot have it

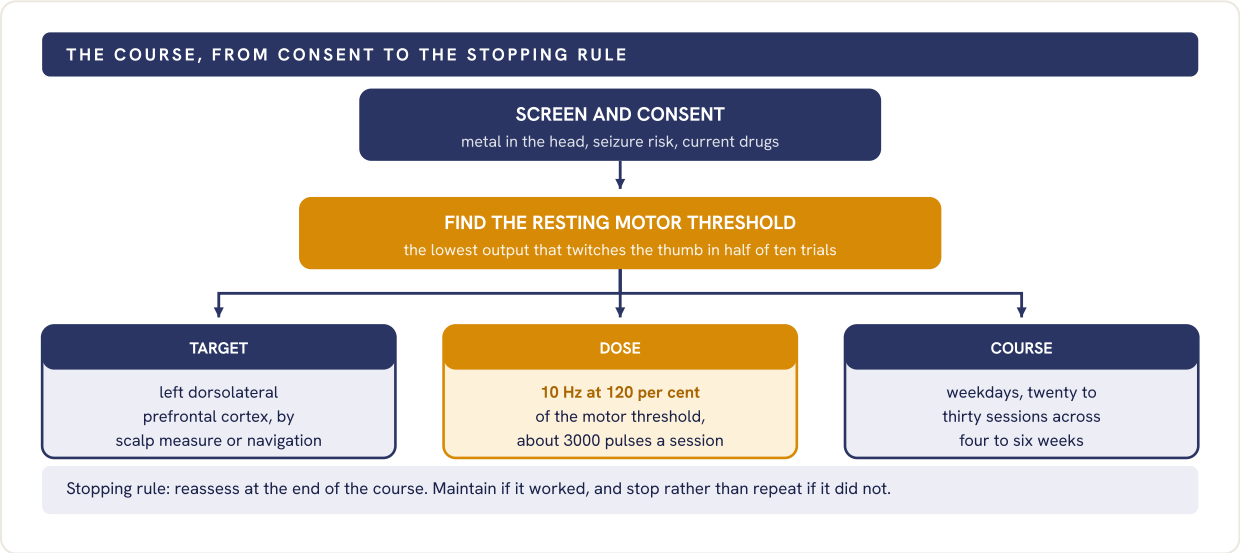


Fig 069.1 The threshold sits in amber because every later number on the page is a percentage of it, which is the step candidates omit.

- **The catalogue.** A seizure is induced: electroconvulsive therapy, magnetic seizure therapy. The cortex is stimulated without one: repetitive and deep transcranial magnetic stimulation, transcranial direct current stimulation. Implanted: vagus nerve and deep brain stimulation. Ablative: the psychosurgical procedures. Neither: bright light and sleep deprivation.
- **Complications and exclusions.** Scalp pain and headache early in the course, transient hearing change without earplugs, syncope, and a switch to mania in bipolar illness. Seizure is the serious risk: rare within published safety limits, and the reason for the exclusions. Do not treat a patient with ferromagnetic metal near the head, an implanted stimulator lead, or an untreated seizure risk.

WHAT EARNS THE MARKS

- **The catalogue grouped by a principle,** not listed alphabetically.
- **Motor threshold named as the reference for the dose,** which is the procedure mark.
- **Seizure separated from the nuisance effects,** and tied to the exclusion list rather than to a rate.

SEE ALSO Slot IV - 67 rTMS indications and efficacy Slot IV - 71 mechanism of brain stimulation **SPRINT**

Day 37, Psychopharmacology II: emergencies, resistance and neurostimulation

IV - 70

Discuss the various strategies to manage patients showing resistance to standard therapy. What is add-on drug treatment, and how is it different from augmentation treatment? [4+3+3]

4 + 3 + 3 | DRUG EMERGENCIES, INTERACTIONS, TRD AND NEUROSTIMULATION

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Before calling it resistance	Diagnosis, adherence, dose, duration
4	The ladder	Optimise, switch, augment, combine, then somatic
2	The two terms	Add-on against augmentation, and the test
2	Choosing on the ward	Severity and urgency, not the failure count

STRATEGY	WHAT IT MEANS	EVIDENCE	WHAT SETTLES IT
Optimise	Maximum tolerated dose, extended to six or eight weeks	Many so-called non-responders have had neither	**Do this before anything is added**
Switch	To another drug, within or across class	Remission rates were similar across switch options in the sequenced trial	Tolerability usually decides which
Augment	Add a drug that is not itself an antidepressant	Lithium and liothyronine carry the oldest data; atypicals carry approval	**The strategy with the best trial base**
Combine	Add a second antidepressant	Starting combined gave no advantage and more adverse effects	**The weakest evidence of the four**
Somatic	ECT, rTMS, or a ketamine based agent	ECT for severe, psychotic or urgent illness; rTMS for moderate resistance	**Severity and urgency, not the number of failures**

Psychological augmentation belongs on this ladder too: adding cognitive therapy performed comparably to adding a drug in the sequenced trial, and is the option least often offered.

- **The two terms.** Augmentation adds a drug that is not itself a treatment for the disorder, such as lithium or liothyronine. Add-on combination adds a second drug of the same class, so both are treating the illness. The test is simple: ask what the added drug would do if it were given alone.
- **Before any of it.** Resistance means two adequate trials in the current episode. Adequate means dose and duration, in a patient who took the drug, with the diagnosis rechecked for bipolarity, psychosis and substance use. Miss this and the ladder is climbed for the wrong illness.

WHAT EARNS THE MARKS

- **The pseudoresistance check written first,** before a single strategy is named.
- **The ladder given in order,** with the evidence attached to each rung rather than to the set.
- **The one line test for the two terms,** which is worth more than two lists of drug names.

SEE ALSO Slot IV - 66 add-on treatment critically evaluated Slot IV - 68 a critique of brain stimulation SPRINT Day 37, Psychopharmacology II: emergencies, resistance and neurostimulation

IV - 71

Discuss the mechanism of action of brain stimulation in psychiatry. What are the other mechanical methods used in psychiatric treatment, and what are their indications? [5+5]

5 + 5 | DRUG EMERGENCIES, INTERACTIONS, TRD AND NEUROSTIMULATION

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The organising question	Does the device fire neurons, or only bias them
3	Mechanism, device by device	Seizure, induced current, subthreshold shift
3	The other methods	Named, each with what it is used for
2	The honest limit	Where the mechanism is still not settled

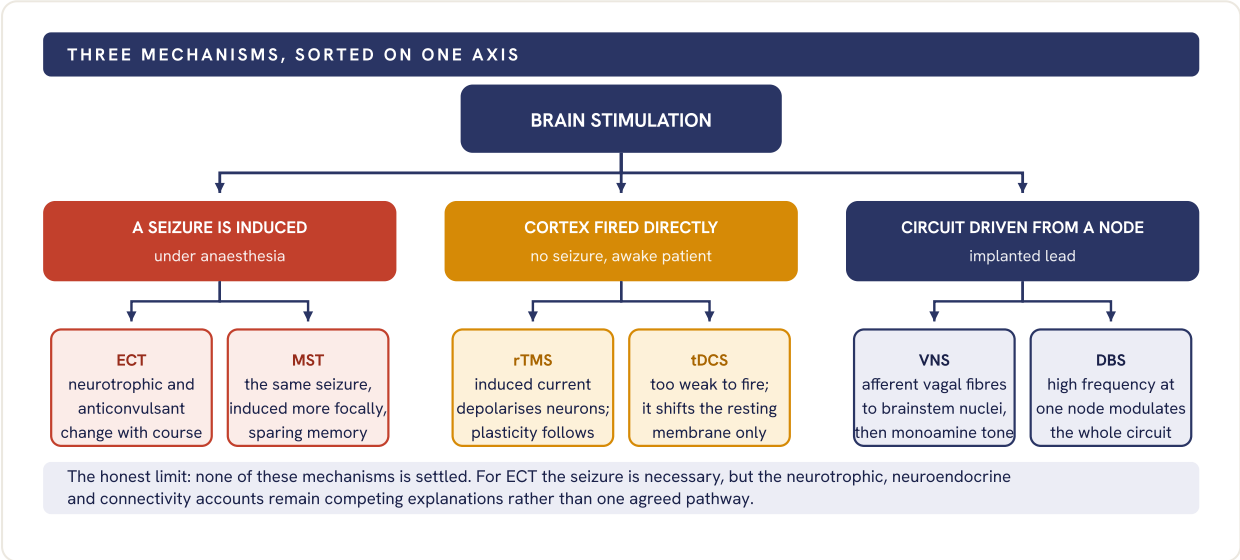


Fig 071.1 Every device sorted on one question, whether it fires neurons or only biases them, so the mechanism answer has a structure instead of six paragraphs.

- **The organising question.** Does the method induce a seizure, fire cortical neurons directly, or only bias the membrane so that whatever the brain is already doing becomes more or less likely? Sorting the devices on that axis is the mechanism answer.
- **The other mechanical methods, with indications.** Electroconvulsive therapy for severe depression with food refusal or high suicide risk, catatonia, resistant mania and puerperal psychosis. Bright light for seasonal depression. Sleep deprivation for a rapid but short lived lift, as an adjunct. Psychosurgery for refractory obsessive compulsive disorder and depression, under consent and Board approval.

WHAT EARNS THE MARKS

- **One axis stated before any device is described.** Six unsorted mechanisms is a mid-band answer.
- **The subthreshold point:** direct current stimulation does not fire neurons, and saying so separates it from the rest.
- **Each other method paired with its indication,** not listed on its own.
- **The limit strip along the foot,** which shows the field is understood rather than recited.

SEE ALSO Slot IV - 68 a critique of brain stimulation Slot IV - 69 the rTMS procedure **SPRINT**
Day 37, Psychopharmacology II: emergencies, resistance and neurostimulation

AREA 10 OF 15 · P4-A11

Psychopharmacology: principles and drug classes

5	5.0%	IV-72	IV-76
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

RECURRENT 1 SINGLE 4

REVISION ORDER

The two injectable slots together, and give the emergency limb its own time: it is the one map in this area that prints a drug dose under pressure. Lithium in youths next, where the comparison is the part that earns the marks. The me-too slot and vortioxetine last, and they share a critical-appraisal shape.

WHAT IS IN THIS AREA

The long acting injectables in two slots, one comparing the first and second generation depots and one that runs from the Indian preparations through their use in mood disorders to a suspected neuroleptic malignant syndrome. Then lithium in young people with bipolar disorder, the me-too drugs with two recent introductions to the Indian market, and vortioxetine for the acute treatment of major depressive disorder.

IV - 72

Discuss the advantages of depot antipsychotics. Compare the advantages and disadvantages of first generation with second generation depot antipsychotics. [4+6]

4 + 6 | PSYCHOPHARMACOLOGY: PRINCIPLES AND DRUG CLASSES

DNB - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	What a depot is for	Adherence made visible, and steadier levels
2	The rest of the case	Relapse and admission, and what it does not fix
4	The grid	Domain by domain, discriminators marked
2	Choosing between them	Who gets which, and what decides it here

DOMAIN	FIRST GENERATION DEPOT	SECOND GENERATION INJECTION	WHAT SETTLES IT
Agents	Fluphenazine, haloperidol, flupentixol, zuclopenthixol decanoate	Risperidone, paliperidone palmitate, olanzapine pamoate	Paliperidone runs to three monthly
Starting up	Test dose, then slow titration under oral cover	Risperidone needs three weeks of oral cover; paliperidone loads on day one and day eight	**Only paliperidone needs no oral overlap**
Movement effects	Higher acute and tardive burden	Lower, dose related, never absent	**The one pharmacological reason to prefer the newer class**
Metabolic burden	Modest	Weight and lipid rise, worst with olanzapine	**The price paid for the movement gain**
Specific hazard	Effects persist for weeks after the last dose	Post-injection delirium with olanzapine pamoate	**Three hours of observation, for that one drug**

Amber cells are the discriminators. Cost is the sixth domain and the one left off the grid: first generation esters are generic and stocked, the newer injections are several times dearer.

- **Why a depot at all.** Non-adherence stops being invisible. A missed injection is a recorded event on a known date; a missed tablet is a guess.
- **The rest of the case.** Steadier levels, first-pass metabolism bypassed, and fewer relapses and admissions than the same drug taken orally in mirror-image and pragmatic studies.
- **What a depot does not fix.** It cannot be taken back, so an adverse effect resolves over weeks. It does not treat refusal, and it is not an answer to non-response.

WHAT EARNS THE MARKS

- **Advantage given as a mechanism.** Adherence made visible is the sentence that earns the first four marks.
- **A grid, with the movement and metabolic trade named as a trade.**
- **The observation rule attached to one drug,** which separates a read answer from a recalled one.

SEE ALSO Slot IV - 73 long acting injections and neuroleptic malignant syndrome **SPRINT**

Day 36, Psychopharmacology I: principles and core drug classes

IV - 73

Enumerate the long acting injectable antipsychotic preparations available in India. Describe their role in mood disorders. How would you manage a suspected neuroleptic malignant syndrome in a patient already on long acting antipsychotic treatment? [3+2+5]

3 + 2 + 5 | PSYCHOPHARMACOLOGY: PRINCIPLES AND DRUG CLASSES

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3 What is available	Both classes, named, with the dosing interval
2 Role in mood disorders	Maintenance in bipolar I, and the limit of the evidence
4 Managing the emergency	The flowchart: withhold, support, treat by severity
1 Rechallenge	The rule that prevents the second episode

- **Available in India.** First generation esters: fluphenazine decanoate, haloperidol decanoate, flupentixol decanoate and zuclopenthixol decanoate, given two to four weekly. Second generation injections: risperidone microspheres two weekly, paliperidone palmitate monthly and three monthly, and olanzapine pamoate.
- **Role in mood disorders.** Maintenance, never acute treatment. Randomised maintenance trials support risperidone long acting injection in bipolar I where relapses are manic and adherence is poor, with little effect on depressive relapse. No injection treats bipolar depression.
- **The severe lane, with its ceiling.** Intravenous dantrolene 1 to 2.5 mg per kg, may be repeated, to a maximum of 10 mg per kg per day.

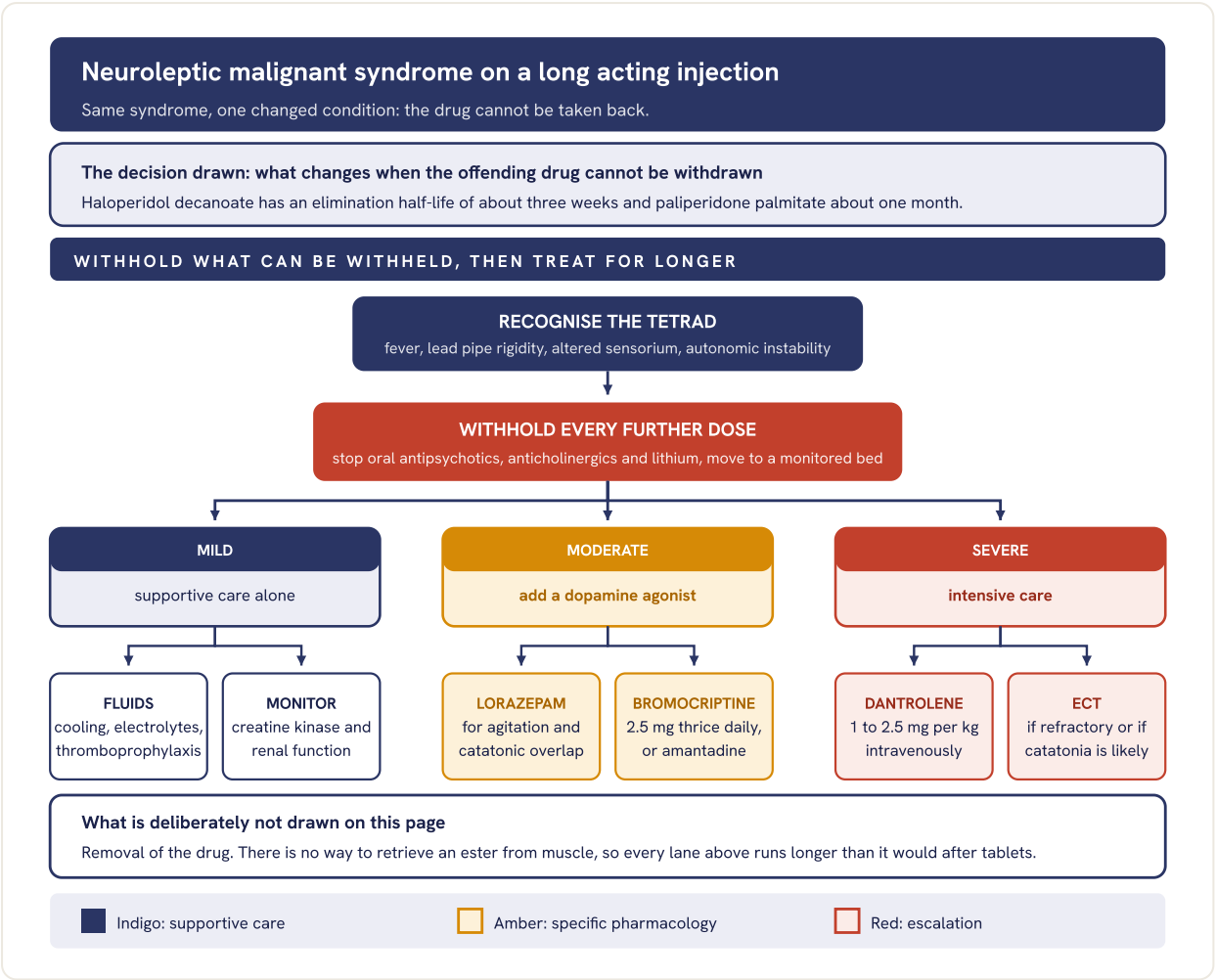


Fig 073.1 The ordinary algorithm redrawn for a drug that cannot be withdrawn, with the release half-life in the framing band so the longer course reads as a consequence.

RECHALLENGE

Never the same depot. Wait at least two weeks after full resolution, longer when the last injection was recent, then restart a lower potency oral agent slowly, watching temperature, pulse and creatine kinase.

WHAT EARNS THE MARKS

- **Both classes named with their intervals,** not the phrase "depot antipsychotics".
- **The framing band of the figure:** the drug cannot be withdrawn, so the course runs longer. Almost no answer says this.
- **The rechallenge box,** which most answers never reach.

SEE ALSO Slot IV - 72 depot antipsychotics compared Slot IV - 65 catatonia **SPRINT**

Day 37, Psychopharmacology II: emergencies, resistance and neurostimulation

IV - 74

"Lithium has advantages over other mood stabilisers in youths with bipolar disorder." Discuss this statement in the context of lithium being used as a first line mood stabiliser in adults with bipolar disorder. [10]

10 PSYCHOPHARMACOLOGY: PRINCIPLES AND DRUG CLASSES

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The claim, unpacked	What an advantage would have to mean in a child
3 What supports it	Licence, maintenance data, and the case against valproate
3 What weakens it	The head to head trial and the monitoring burden
2 The position to defend	Where the adult claim transfers and where it stops

ANSWER THE QUESTION THAT WAS ASKED

An advantage in a child must be one of three things: better efficacy in a head to head trial, a safety profile that suits a growing body, or an outcome that matters over decades. Lithium wins the third, argues the second, loses the first.

- **The licence and the record.** Lithium holds a paediatric licence for bipolar disorder in the United States and the longest paediatric record of any mood stabiliser. Maintenance levels of 0.6 to 1.0 mmol per litre are the usual target.
- **The comparison that matters.** Against valproate the case is strong, and strongest in girls: neural tube defects and neurodevelopmental harm after intrauterine exposure, polycystic ovary syndrome, weight gain, hepatotoxicity.
- **The head to head result.** The Treatment of Early Age Mania trial randomised children with mania to lithium, divalproex or risperidone. Risperidone gave the higher response rate, at the cost of weight and prolactin. Acute anti-manic evidence does not favour lithium.
- **The monitoring burden is the real limit.** A narrow index in a body that plays sport and gets gastroenteritis; levels pushed up by anti-inflammatories, thiazides and angiotensin converting enzyme inhibitors; toxicity above 1.5 mmol per litre; thyroid and renal tests an adolescent resents.
- **What does not transfer.** The anti-suicidal signal that anchors lithium in adults is adult data. A reasonable expectation in youths, not an established finding.

WHAT EARNS THE MARKS

- **The definition block:** naming what would count as an advantage before arguing about one.
- **The valproate comparison made sex specific,** which is where the paediatric case is won.
- **The head to head trial named against the claim,** not only the evidence that flatters it.
- **A stated position:** maintenance agent, not the acute first choice.

SEE ALSO Slot IV - 63 bipolar disorder and recent advances in treatment Slot IV - 76 vortioxetine in acute depression

SPRINT Day 36, Psychopharmacology I: principles and core drug classes

IV - 75

What are me too drugs? Discuss critically two new psychotropic drugs introduced in the Indian market. [2+4+4]

2 + 4 + 4 | PSYCHOPHARMACOLOGY: PRINCIPLES AND DRUG CLASSES

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Me too, defined	The definition, and the test that settles it
4 The first agent	What is genuinely new and what is only claimed
4 The second agent	The receptor claim and the trials behind it

AGENT	WHAT IT DOES	EVIDENCE SO FAR	STATUS
Vortioxetine	Reuptake inhibition plus direct action at five serotonin receptors	**Placebo controlled trials, plus a cognitive signal on the digit symbol test**	Marketed in India, second line
Cariprazine	Partial agonist preferring the dopamine D3 receptor	**Trials in schizophrenia, mania and bipolar depression; beat risperidone on negative symptoms**	Marketed in India, specialist use
Escitalopram	The active enantiomer of citalopram, nothing else	A chiral switch; its small edge on the parent is disputed	Routine use, the standard example
Desvenlafaxine	The active metabolite of venlafaxine	No demonstrated advantage over the parent	Marketed, a follow-on

Rows one and two answer the question. Rows three and four are the contrast that makes the critique land: two drugs that reached the same market with no claim to novelty at all.

- **Me too, defined.** A drug close in structure or pharmacology to one already marketed, offering little therapeutic gain over it, developed for market share and patent life rather than unmet need. The test is a head to head trial: does it beat the incumbent on efficacy, safety or convenience?
- **Where the two stand.** Vortioxetine is ordinary by effect size; its distinct case is a cognitive signal, real but modest and still argued. Cariprazine has the stronger claim: D3 preference is a genuine departure, and its bipolar depression data do not exist for the drugs it replaces.

STATUS: WHAT A MARKETING LICENCE CERTIFIES

Approval anywhere, India included, rests on efficacy and acceptable safety, not on superiority to what is already on the shelf. A me too drug can be licensed, promoted and priced as new without ever having beaten the drug it copies.

WHAT EARNS THE MARKS

- **The definition followed immediately by the test.** Defining me too without saying how it would be recognised is half a mark.
- **Two named agents with their evidence in the same row,** not a paragraph of mechanism.
- **A different verdict for each,** which is what critically means.

SEE ALSO Slot IV - 76 vortioxetine in acute depression Slot IV - 64 therapeutic advance and prognosis SPRINT Day 38, Recent advances and landmark studies

IV - 76

Write briefly about the safety and efficacy of vortioxetine for the acute treatment of major depressive disorder. [5+5]

5 + 5 | PSYCHOPHARMACOLOGY: PRINCIPLES AND DRUG CLASSES

DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	What it is	Multimodal pharmacology, dose, and where it is placed
3	Efficacy	The placebo controlled trials, then the cognition claim
3	Safety	Nausea, sexual function, stopping, interactions
2	The honest verdict	What is proven, and what is only marketed

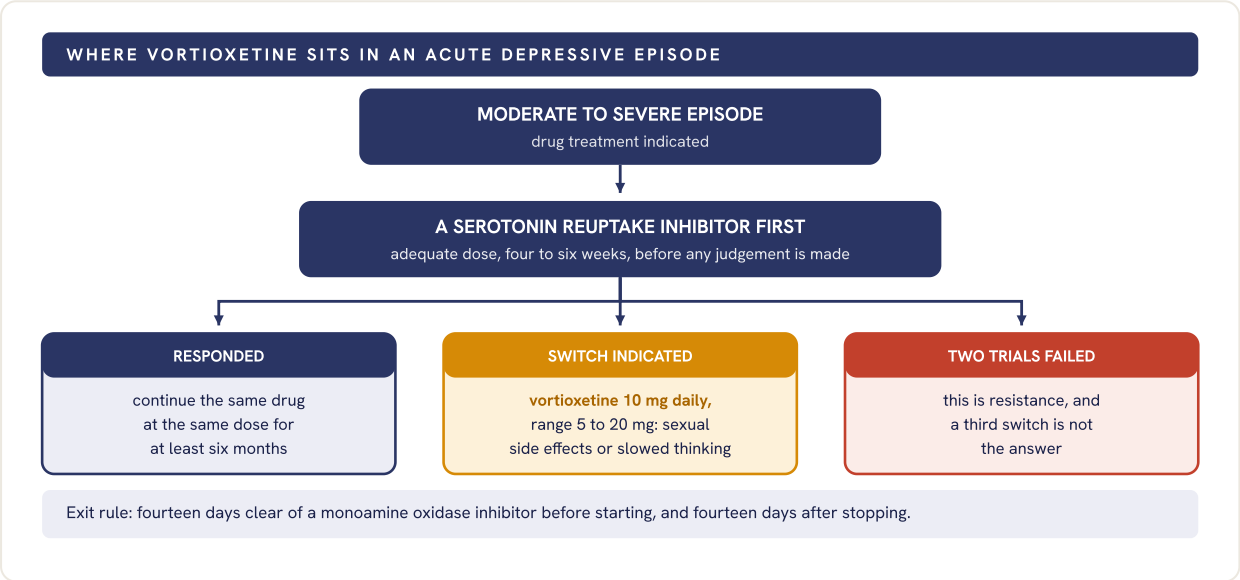


Fig 076.1 The drug placed rather than praised: vortioxetine enters at the switch, on two named grounds, and does not enter at the point where resistance has already been established.

- **Efficacy.** Short term placebo controlled trials separate on the Montgomery Asberg scale, and meta-analysis puts the effect with the other second generation antidepressants. The distinct claim is cognitive: gain on the digit symbol test, partly independent of mood change, where duloxetine did not separate. Real, modest, still argued.
- **Safety.** Nausea is commonest, dose related, worst at 20 mg and in the first fortnight. Little weight gain, less sexual dysfunction than the reuptake inhibitors, no meaningful QT effect, and few discontinuation symptoms given a half-life near sixty-six hours. Watch hyponatraemia in the elderly and reduce the dose with strong CYP2D6 inhibitors.

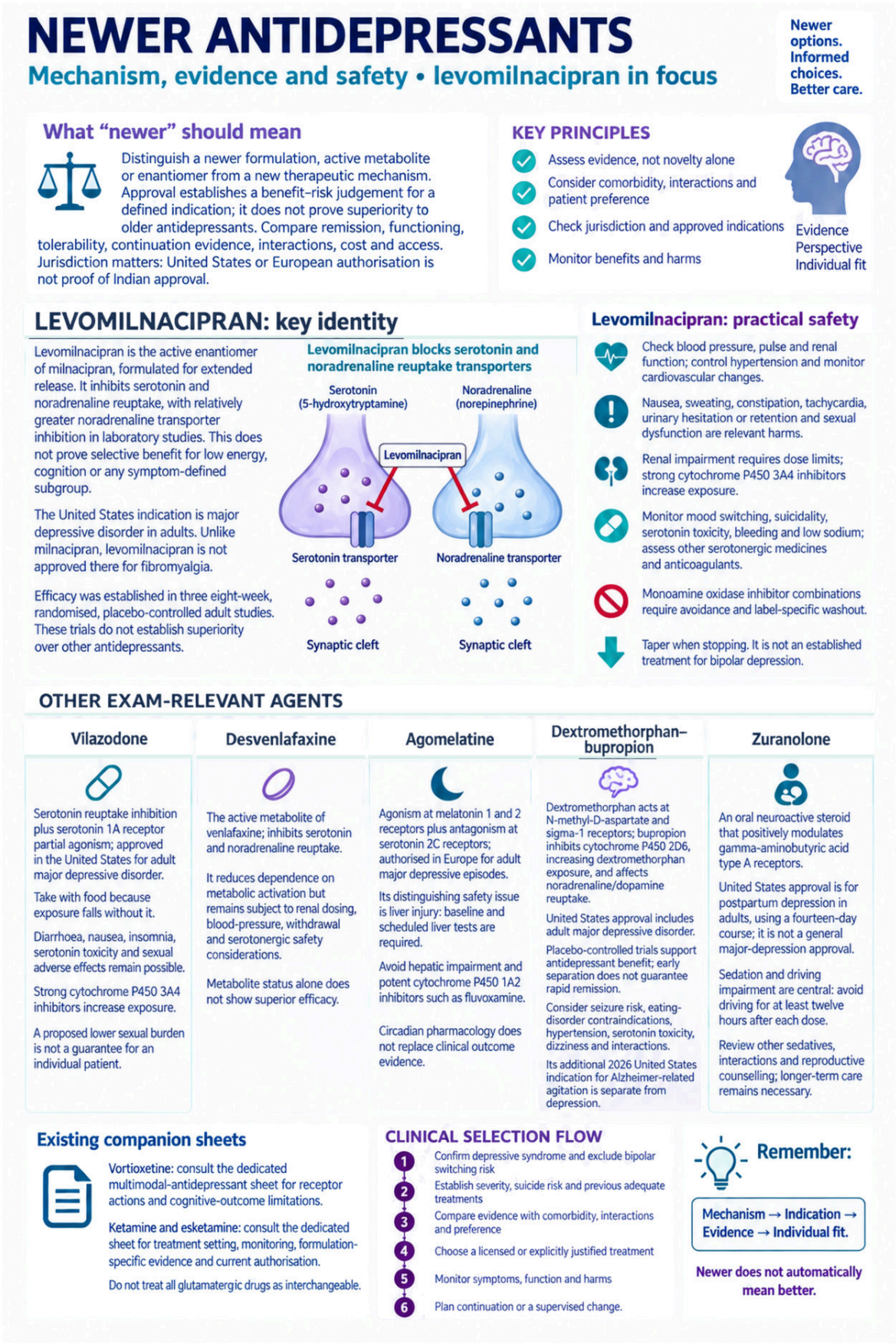
WHAT EARNS THE MARKS

- **Multimodal stated as pharmacology,** reuptake inhibition plus direct receptor action, not as a slogan.
- **The cognitive claim reported with its limit.** Naming the measure and the dispute is the top band answer.
- **The two grounds for the switch on the flowchart,** which is what the drug is actually for.

SEE ALSO Slot IV - 75 me too drugs and new agents in India Slot IV - 70 resistance and augmentation strategies

SPRINT Day 38, Recent advances and landmark studies

WEAVE 100 · PAPER IV · R04



Newer antidepressants with levomilnacipran. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Psychopharmacology: principles and drug classes.

Credits and references: retained within the illustration.

AREA 11 OF 15 · P4-A01

Epilepsy and psychiatry

5	4.9%	IV-77	IV-81
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

SINGLE 5

REVISION ORDER

The two classification slots together, they are the same answer at two widths and the operational classification writes both. The psychiatric manifestations slot next, it is the one most often set here. Absence seizures after that, and it is the one slot in this area that carries a drug dose. Magnetic seizure therapy last, and separately.

WHAT IS IN THIS AREA

The classification of epilepsy in two slots, one asking for it alone and one running on to the clinical features and management of complex partial seizures. Then the psychiatric manifestations of epilepsy with their management, and the management of absence seizures. The fifth slot asks for magnetic seizure therapy, which is a neurostimulation question the boards set inside the epilepsy area and is answered on its page as it was set.

IV - 77

Management of absence seizures. [10]

10 | EPILEPSY AND PSYCHIATRY | KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Confirm the syndrome	Absence, not inattention: the record settles it
2	What decides the drug	Absence alone, or absence with tonic-clonic seizures
4	First line treatment	The named agent, the weight band, what it does not cover
2	Review and withdrawal	Seizure freedom, a clean record, then a slow taper

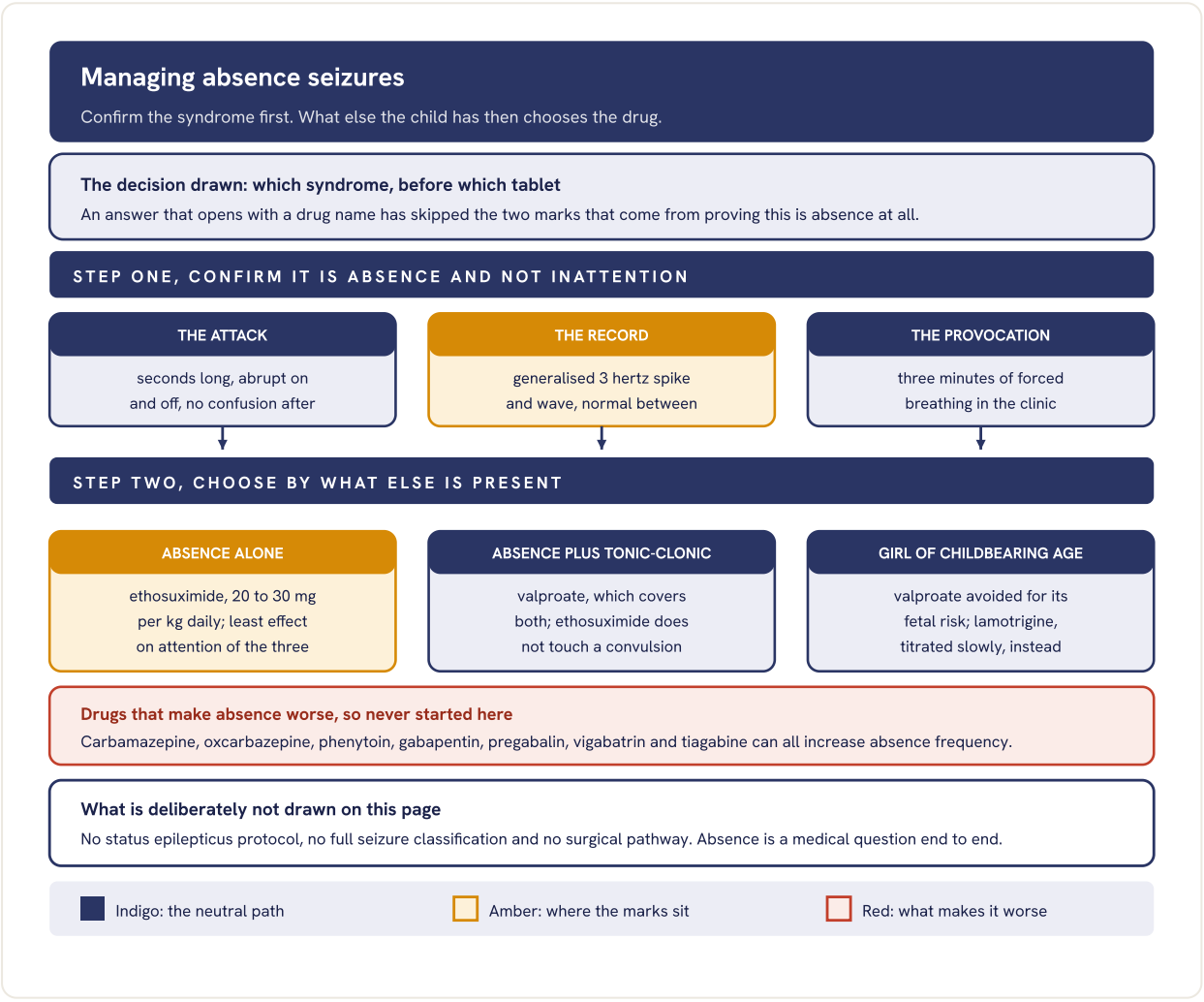


Fig 077.1 Confirmation first, then the drug chosen by what else the child has, with the aggravating drugs carried as a red strip because starting one is the commonest management error here.

- **Choosing between the three.** The three-arm comparative trial in childhood absence epilepsy found ethosuximide and valproate more effective than lamotrigine, and ethosuximide less impairing of attention. Where it is not stocked, valproate does this work.
- **Review and withdrawal.** Titrate to seizure freedom, confirmed by hyperventilation at each visit. After two seizure-free years with a clean record, taper slowly.
- **Attention does not always follow the record.** Inattention and academic underachievement often persist after the seizures stop, and need their own assessment.

PITFALL

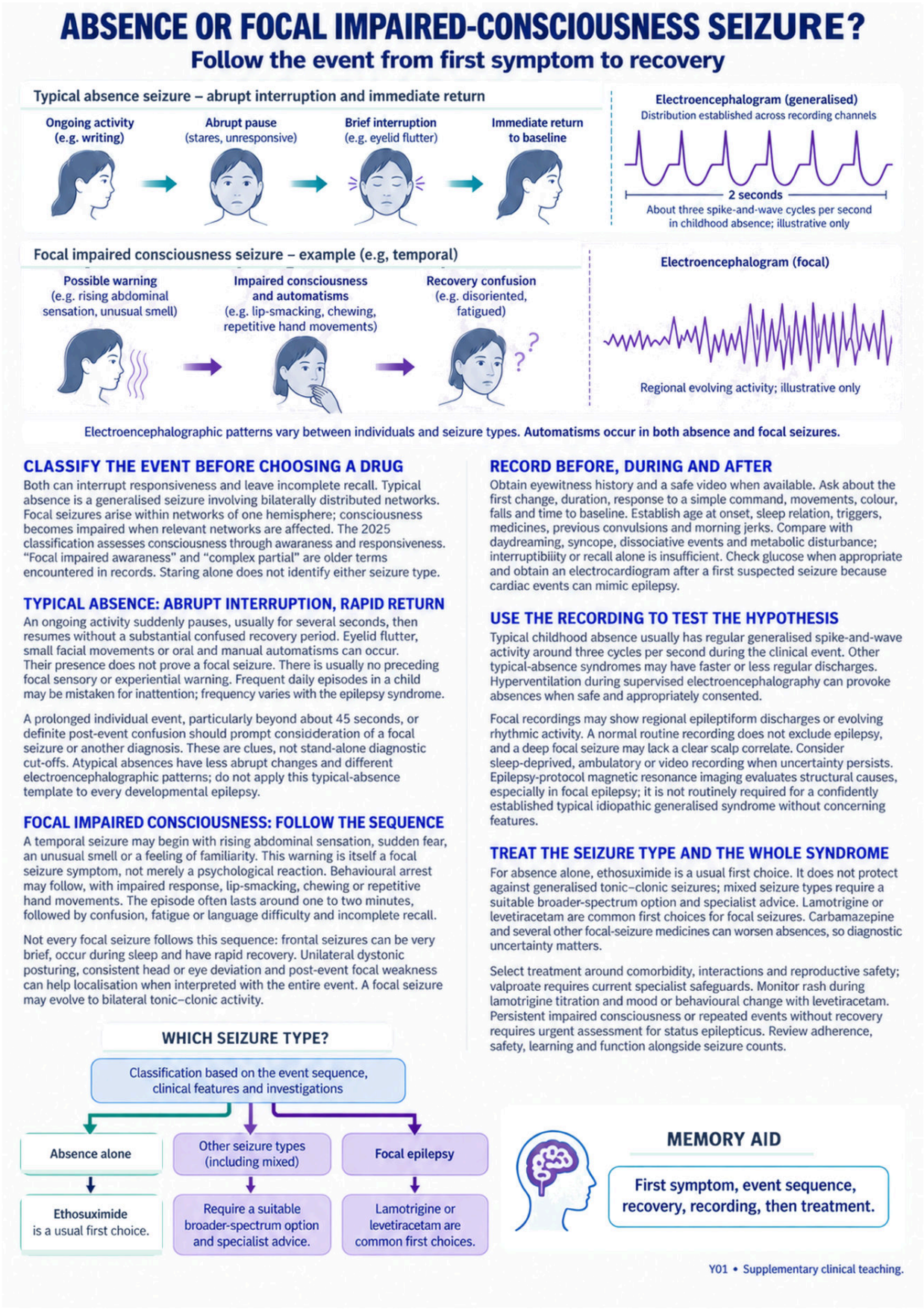
The child called a daydreamer. Absence is not attention deficit disorder, a stimulant does not treat it, and a sodium channel blocker started for a wrongly labelled focal seizure makes it worse.

WHAT EARNS THE MARKS

- **Confirmation written before any drug.** Attack description, the record, and provocation at the bedside.
- **The branch rule stated.** Ethosuximide when absence stands alone, valproate when a convulsion has also occurred.
- **The aggravating list named.** Most answers list treatments and never say what is contraindicated.
- **The withdrawal rule.** Two seizure-free years and a slow taper is the last mark.

SEE ALSO Slot IV - 80 classification of epilepsy Slot IV - 79 psychiatric manifestations of epilepsy **SPRINT** Day 30, Epilepsy & psychiatry

WEAVE 100 · PAPER IV · Y01



Absence versus focal impaired-consciousness seizures. Read across the labelled contrasts to distinguish the related concepts. Read with IV-77.

Credits and references: retained within the illustration.

IV - 78

What is magnetic seizure therapy? Discuss its role in psychiatry. [4+6]

4 + 6 | EPILEPSY AND PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition	A therapeutic seizure induced magnetically, under anaesthesia
2	Why it differs	The skull does not resist a magnetic field
4	Role and evidence	Where it has been tried, and what the trials show
2	Status and limits	Investigational, scarce, and honestly stated as such

THE DEFINITION

Magnetic seizure therapy is the induction of a generalised therapeutic seizure by a high frequency, high intensity magnetic field delivered through a coil, with the patient anaesthetised and relaxed exactly as for electroconvulsive therapy. It is a convulsive treatment, not a stronger form of repetitive transcranial magnetic stimulation.

DOMAIN	ELECTROCONVULSIVE THERAPY	MAGNETIC SEIZURE THERAPY	WHAT FOLLOWS FROM IT
Energy delivered	Electric current across the scalp	Magnetic field through the scalp	**Skull and scalp do not attenuate a magnetic field**
Spread of the field	Diffuse, reaching deep structures	Focal, mainly superficial cortex	**Medial temporal structures are relatively spared**
Recovery	Slower reorientation after the fit	Faster reorientation, replicated	The most consistent trial finding
Memory	Retrograde and anterograde loss	Less autobiographical memory loss	**The whole rationale for the technique**
Efficacy in depression	Established, largest effect available	Not shown to match it	Why it stays a research treatment

Amber cells are the discriminators. The comparison is about where the field goes, and every other row follows from that one fact.

- **Role in psychiatry.** Treatment-resistant depression is the main indication studied, with smaller work in bipolar depression, schizophrenia and suicidal ideation.
- **Status, stated honestly.** Randomised comparisons with electroconvulsive therapy are few and small. The cognitive advantage is consistent; equivalent efficacy is not established. It remains investigational and is not in routine Indian practice.

WHAT EARNS THE MARKS

- **Named as a convulsive treatment.** Calling it a stronger form of repetitive stimulation loses the definition marks.
- **The physical reason for the difference.** Focality follows from the field, and the cognitive claim follows from focality.
- **Evidence separated from promise.** Better cognition is shown; matched efficacy is not.

SEE ALSO Slot IV - 77 management of absence seizures SPRINT Day 27, ECT & neurostimulation

IV - 79

Discuss psychiatric manifestations of epilepsy and their management. [6+4]

6 + 4 | EPILEPSY AND PSYCHIATRY | DNB - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	The organising axis	Time relative to the seizure names the syndrome
4	The syndromes	Peri-ictal, postictal, interictal, and drug induced
3	Management	Fix the epilepsy, then treat, watching the interaction
1	The two traps	Forced normalisation and the enzyme inducer

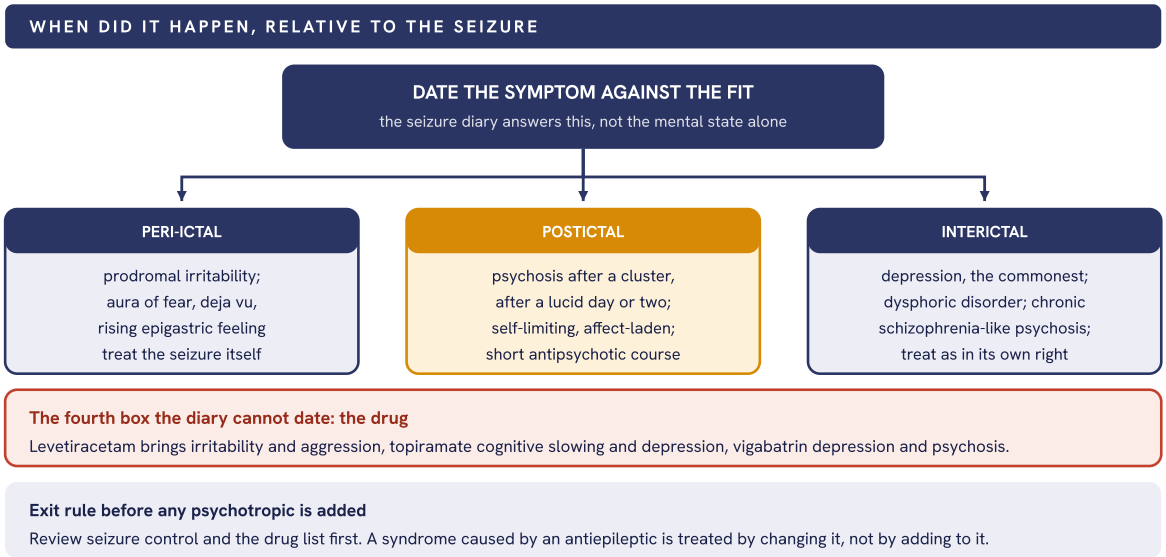


Fig 079.1 The syndromes sorted by their time relation to the seizure, with the drug-induced group set apart because it is the one the seizure diary cannot date.

- **Prescribing safely.** Selective serotonin reuptake inhibitors are the antidepressants of choice. Clozapine and chlorpromazine are the most epileptogenic antipsychotics.
- **The interaction that decides doses.** Carbamazepine, phenytoin and phenobarbitone induce enzymes and lower psychotropic levels. Valproate inhibits, so lamotrigine started alongside it needs half the usual dose to avoid a serious rash.

PITFALL

Forced normalisation. Psychosis can appear as the record normalises and the seizures stop, so raising the antiepileptic further can make the picture worse.

WHAT EARNS THE MARKS

- **The time axis named before any syndrome is listed.** A flat list of symptoms cannot earn the six description marks.
- **Postictal psychosis with its lucid interval,** separated from chronic interictal psychosis.
- **The drug reviewed before a drug is added,** and one named enzyme interaction.

SEE ALSO Slot IV - 81 complex partial seizures Slot IV - 77 management of absence seizures **SPRINT**

Day 30, Epilepsy & psychiatry

WEAVE 100 · PAPER IV · F17

NEURONAL SIGNALLING AND EPILEPSY

Foundation 17 • Normal function → mechanism → clinical recognition → management

NORMAL NEURONAL SIGNALLING

A neuron maintains ion gradients across its membrane. Opening voltage-gated sodium channels produces rapid depolarisation; potassium movement helps repolarisation. At a synapse, calcium entry triggers neurotransmitter release. Glutamate commonly mediates excitation; gamma-aminobutyric acid commonly mediates inhibition in the mature brain. Networks normally balance excitation, inhibition and synchronisation. A typical resting membrane potential is about minus 70 millivolts, varying by cell type and conditions; this is a physiological concept, not a routine patient test. Seizures arise from abnormal excessive or synchronised activity within susceptible neuronal networks, not simply from one universally deficient chemical.

SEIZURE IS AN EVENT; EPILEPSY IS A PREDISPOSITION

An acute symptomatic seizure occurs in close relation to a provoking brain or systemic insult, such as severe hypoglycaemia, major electrolyte disturbance, acute stroke or infection. It does not automatically establish epilepsy. The International League Against Epilepsy clinical definition allows epilepsy after two unprovoked or reflex seizures more than 24 hours apart; one such seizure with an estimated recurrence probability of at least 60% over ten years; or identification of an epilepsy syndrome. Aetiologies include structural, genetic, infectious, metabolic, immune and unknown categories; more than one may apply.

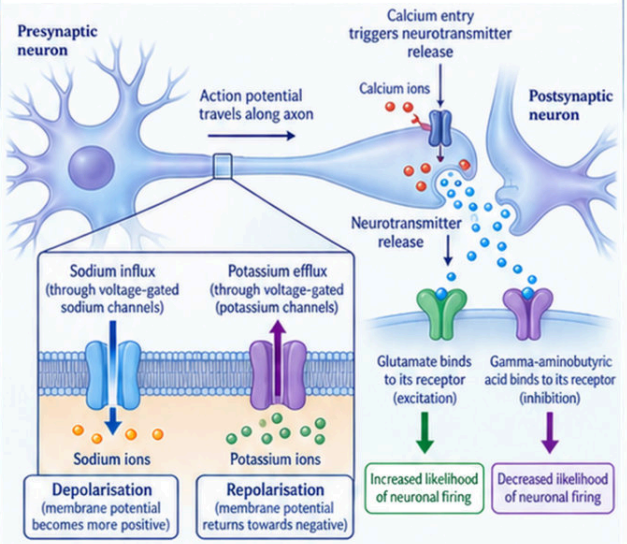
CLASSIFY WHAT HAPPENED

The 2025 seizure framework has four main classes; focal, generalised, unknown whether focal or generalised, and unclassified. Focal seizures arise within networks of one hemisphere and may affect movement, sensation, autonomic function or experience; consciousness can be preserved or impaired. A focal seizure can evolve to bilateral tonic–clonic activity. Generalised seizures engage bilaterally distributed networks and include absence, myoclonic and tonic–clonic seizures. Tonic means sustained stiffening; clonic means rhythmic jerking; myoclonus means a brief shock-like jerk. A convulsion describes motor activity and is not synonymous with every seizure. Postictal means the recovery period after a seizure; temporary confusion or focal weakness may occur.

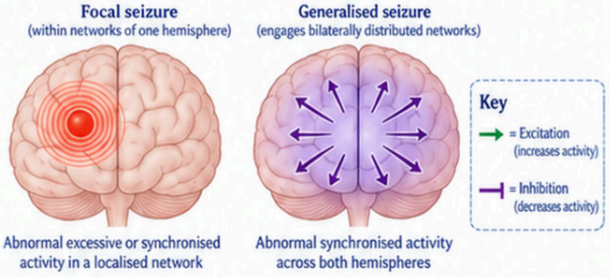
FIRST EVENT → EVALUATION

Obtain an eyewitness account or safe video, sequence of symptoms, duration and recovery; ask about sleep, substances, medicines and previous subtle events. Examine, check glucose and relevant electrolytes and obtain a twelve-lead electrocardiogram because cardiac syncope can mimic a seizure. Electroencephalography supports classification but a normal recording does not exclude epilepsy. Magnetic resonance imaging with an epilepsy protocol looks for structural causes when indicated. There is no universal blood test or single normal electroencephalogram value that rules epilepsy in or out. Consider syncope, metabolic events, sleep disorders and functional seizures; these can coexist with epilepsy.

NEURONAL SIGNAL TRANSMISSION



NEURONAL NETWORKS AND SEIZURES



MANAGEMENT AND SAFETY

Dbring a convulsive seizure: protect from injury, time the event, do not restrain or put anything in the mouth, and position safely once possible. A convulsive seizure lasting five minutes, or repeated seizures without recovery, requires the status-epilepticus emergency pathway, including prompt benzodiazepine treatment and escalation by protocol. Persistent unexplained altered consciousness can reflect non-convulsive status and requires urgent assessment.

Confirmed epilepsy → choose an antiseizure medicine matched to seizure type, syndrome, comorbidities and reproductive risks → review efficacy and adverse effects. Lamotrigine or levetiracetam are common first options for focal seizures; slow lamotrigine titration reduces serious rash risk, while levetiracetam nequires monitoring for behavioural adverse effects. Some medicines worsen particular generalised seizure types. Valproate requires specialist reproductive-risk safeguards; do not assume it is interchangeable for everyone. Explain adherence, sleep regularity, bathing and water safety, driving rules and sudden unexpected death in epilepsy. Failure of two appropriate, tolerated treatment regimens warrants specialist drug-resistant-epilepsy assessment, including surgery or other therapies where suitable.

MEMORY AID

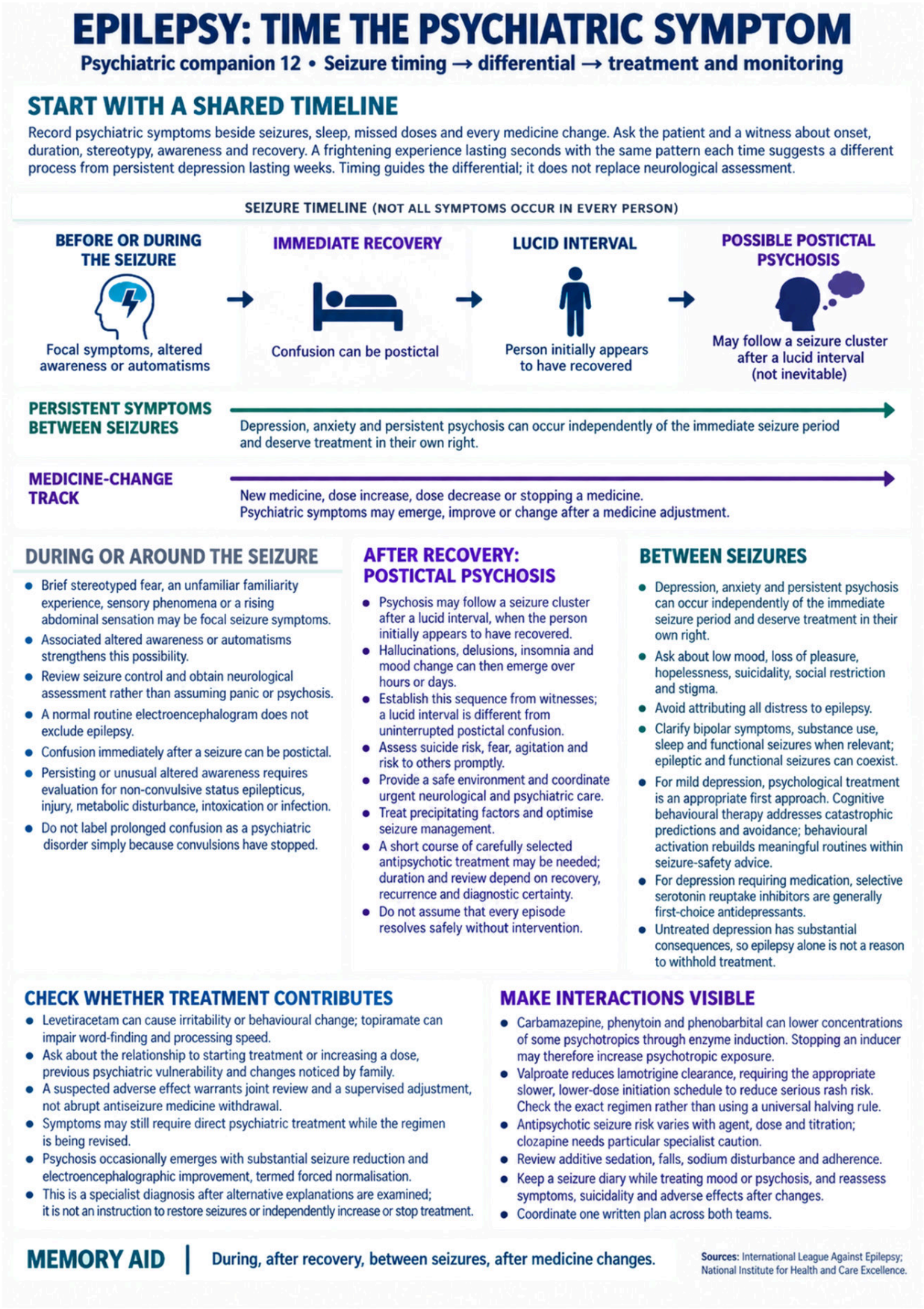
Describe the event, classify the network, match the treatment.

Foundation 17 • Medical foundations • Source-checked supplementary teaching • September 2026.
Laboratory ranges and treatment protocols require local context.

Neuronal signalling and epilepsy. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-79.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P12



Epilepsy psychiatry. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-79.

Credits and references: retained within the illustration.

IV - 80

How is epilepsy classified. [10]

10 | EPILEPSY AND PSYCHIATRY | KUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **The framework**	Three levels, with cause and comorbidity at each
3 **Level one, the seizure**	Onset: focal, generalised, or unknown
3 **Levels two and three**	Epilepsy type, then the named syndrome
2 **Cause and comorbidity**	Six aetiological groups, asked at every level

THE FRAMEWORK

The current classification is the operational scheme of the International League Against Epilepsy, published in 2017. It works in three levels: the seizure type, then the epilepsy type, then the epilepsy syndrome where one can be named. Aetiology and comorbidity are asked at every level, not appended at the end.

LEVEL	THE QUESTION IT ANSWERS	THE CATEGORIES	WHAT IT DECIDES
Seizure type	Where does the seizure begin	Focal, generalised, unknown onset	**Drug choice**
Focal, subdivided	Is awareness kept, and is onset motor	Aware or impaired awareness; motor or non-motor; focal to bi-lateral tonic-clonic	**Replaces simple and complex partial**
Generalised, subdivided	Motor or not	Tonic-clonic, tonic, clonic, my-oclonic, atonic, spasms; absence typical or atypical	Sodium channel blockers avoided
Epilepsy type	What pattern does this person have	Focal, generalised, combined, unknown	Prognosis and counselling
Syndrome	Does a named entity fit	Age at onset, seizure types, record, imaging taken together	**Prognosis and duration of treatment**
Aetiology	Why is it happening	Structural, genetic, infectious, metabolic, immune, unknown	Treatable cause found or excluded

Amber cells are the discriminators. The scheme is diagnostic in sequence, so each level narrows what the next one can be.

- **The terms retired in 2017.** Partial became focal; simple partial and complex partial became focal aware and focal impaired awareness. Use both names.
- **The Indian entry point.** Neurocysticercosis is the commonest identified cause of new focal seizures in adults here, and sits in the infectious group alongside tuberculoma, so imaging is part of classifying rather than an afterthought.

WHAT EARNS THE MARKS

- **Three levels named as levels,** in order, before any category is listed.
- **Both halves of the focal split.** Awareness and motor onset, with the retired terms mapped across.
- **The six aetiological groups,** stated as running through every level rather than as a final list.

SEE ALSO Slot IV - 81 complex partial seizures Slot IV - 77 management of absence seizures SPRINT
Day 30, Epilepsy & psychiatry

IV - 81

Classification of Seizure disorder. Discuss clinical features and management of Complex Partial Seizures. [10]

10 | EPILEPSY AND PSYCHIATRY | RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Place it in the scheme**	Focal impaired awareness in the current terms
3 **Clinical features**	Aura, automatisms, and the signs that lateralise
2 **Confirming it**	Record, epilepsy protocol imaging, video when unsure
3 **Management**	First drug, the resistance rule, then surgical referral

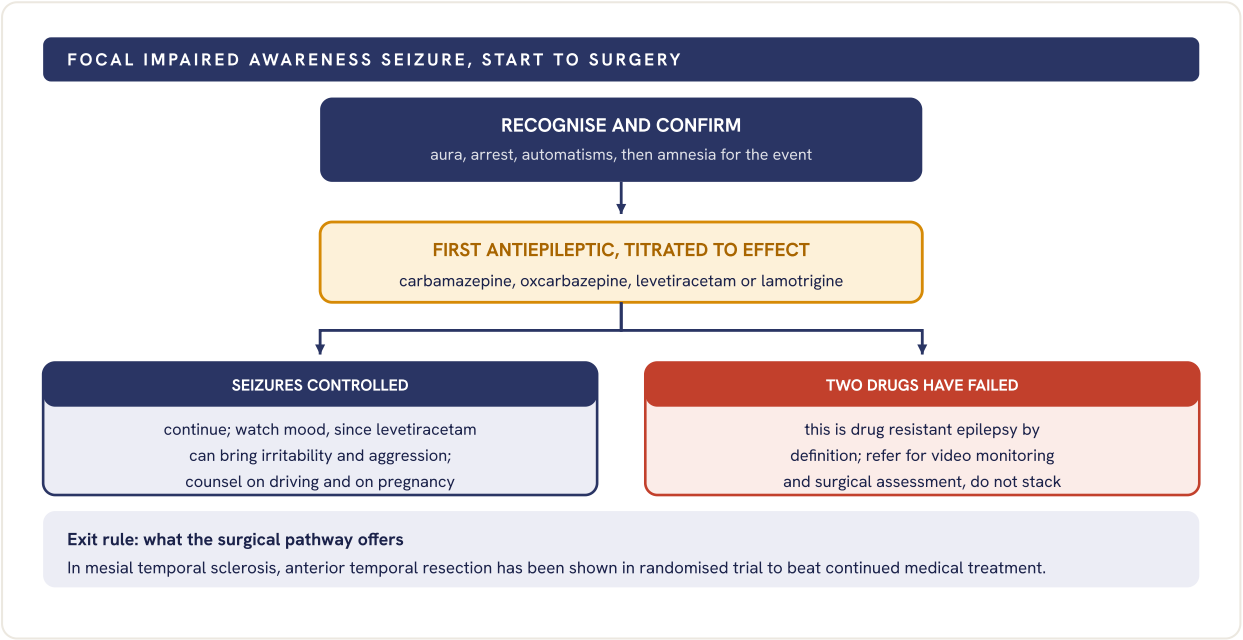


Fig 081.1 One drug, then a decision point that most answers never reach: two failed drugs is a definition, not a setback, and it routes the patient to assessment rather than to a third tablet.

- **Features, in order.** Aura of rising epigastric sensation, fear or deja vu; motionless stare; lip smacking, chewing and fumbling; one to two minutes; then amnesia.
- **What lateralises.** Dystonic posturing points away from the focus, automatisms and nose-wiping towards it, dysphasia after the fit to the dominant hemisphere.

PITFALL

Automatisms with amnesia get read as dissociation or panic: absence has no aura and no confusion after it, and a panic attack is remembered.

WHAT EARNS THE MARKS

- **The current name given alongside the old one.** Focal impaired awareness seizure, formerly complex partial.
- **At least one lateralising sign,** which is what separates a description from an assessment.
- **Drug resistance defined by two failed drugs,** and the referral that follows it.

SEE ALSO Slot IV - 80 classification of epilepsy Slot IV - 79 psychiatric manifestations of epilepsy **SPRINT** Day 30, Epilepsy & psychiatry

WEAVE 100 · PAPER IV · R10

NEWER ANTISEIZURE MEDICINES AND PSYCHIATRY

Match the seizure • monitor behaviour • separate neurological and psychiatric evidence

Start with the epilepsy, not the newest drug

Classify focal, generalised or unknown-onset seizures and identify the syndrome. Choice also depends on age, reproductive plans, organ function, interactions, comorbidity and previous response. "Newer" includes several generations; a common antiseizure effect does not imply a common psychiatric indication. Compare trials within their seizure population, rather than ranking drugs from unrelated studies.

MECHANISM FAMILIES AND CLINICAL MATCHING

Levetiracetam and brivaracetam bind synaptic vesicle protein 2A, altering transmitter release. Levetiracetam has broad seizure applications; brivaracetam is licensed for focal-onset seizures. Both can cause irritability, aggression, depression or psychotic symptoms. Switching may help some patients, but brivaracetam is not behaviourally risk-free. Adding it to levetiracetam did not provide additional benefit in registration trials.

Lacosamide enhances slow inactivation of voltage-gated sodium channels. It treats focal seizures and has an adjunctive indication for primary generalised tonic-clonic seizures in relevant jurisdictions. Watch dizziness, ataxia and cardiac conduction slowing; prolongation of the interval from atrial activation to ventricular activation. Obtain an electrocardiogram when cardiac disease or interacting conduction-slowing medicines make this relevant.

Eslicarbazepine stabilises inactivated sodium channels and treats focal seizures. Dizziness, rash and low sodium are important; confusion may reflect hyponatraemia rather than a primary psychiatric relapse. Check renal dosing and contraceptive interactions. It is not an established mood stabiliser.

Perampanel non-competitively blocks the alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid glutamate receptor. It treats focal seizures and is an adjunct for primary generalised tonic-clonic seizures. Serious aggression, hostility and other behavioural reactions warrant explicit counselling and early review, especially during titration. Alcohol can worsen impairment and behavioural effects.

Cenobamate combines effects on persistent sodium currents and gamma-aminobutyric acid type A receptor signalling; its exact therapeutic mechanism is not fully established. It treats adult focal seizures. Slow titration reduces the risk associated with drug reaction with eosinophilia and systemic symptoms. It shortens the electrocardiographic Q-to-T interval, spanning ventricular depolarisation and repolarisation, and is contraindicated in familial short QT syndrome. Review sedation and substantial enzyme interactions.

SELECTED SYNDROME-SPECIFIC ADVANCES

Prescription cannabidiol has United States indications for seizures associated with Dravet syndrome, Lennox–Gastaut syndrome and tuberous sclerosis complex. This does not validate unregulated cannabis products or establish a psychiatric indication.

Ganaxolone is a neuroactive steroid modulator of gamma-aminobutyric acid type A receptors, approved there for seizures associated with cyclin-dependent kinase-like 5 deficiency disorder from age two years; sedation and interactions require monitoring.

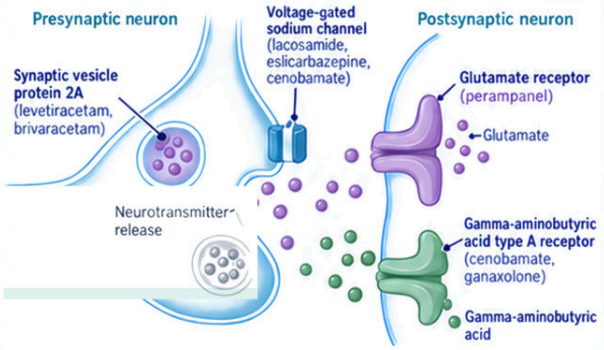
SEIZURE CONTROL IS NOT A MOOD-STABILISER LICENCE

Lamotrigine is an established older comparator with a bipolar I maintenance indication; it is not an acute antimanic treatment. Guidelines also support a role in bipolar depression, distinct from its regulatory maintenance indication. Slow titration and rash counselling are essential.

Pregabalin binds the alpha-2-delta calcium-channel subunit; it is authorised in Europe for adult generalised anxiety disorder as well as neurological indications. It can cause sedation, dependence and withdrawal; it is not a proven bipolar mood stabiliser.

Topiramate and gabapentin should not be assumed effective for bipolar mood episodes merely because they treat seizures.

CONCEPTUAL TARGETS ACROSS SYNAPSES



KEY MEDICINES AT A GLANCE

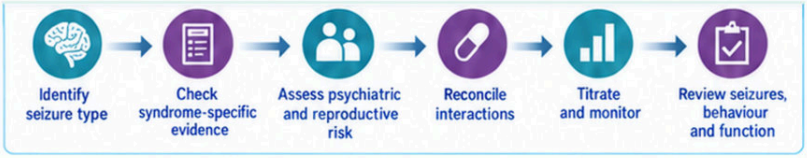
Drug	Main mechanism	Seizure role (licensed/established)	Key psychiatric or safety issue
Levetiracetam	Binds synaptic vesicle protein 2A	Broad seizure applications	Irritability, aggression, depression or psychotic symptoms
Brivaracetam	Binds synaptic vesicle protein 2A	Focal-onset seizures	Irritability, aggression, depression or psychotic symptoms; not behaviourally risk-free
Lacosamide	Enhances slow inactivation of voltage-gated sodium channels	Focal seizures; adjunctive for primary generalised tonic-clonic seizures in relevant jurisdictions	Dizziness, ataxia and cardiac conduction slowing (prolongation of the interval from atrial activation to ventricular activation)
Eslicarbazepine	Stabilises inactivated sodium channels	Focal seizures	Dizziness, rash and low sodium; confusion may reflect hyponatraemia; check renal dosing and contraceptive interactions
Perampanel	Non-competitively blocks the alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid glutamate receptor	Focal seizures; adjunctive for primary generalised tonic-clonic seizures	Serious aggression, hostility and other behavioural reactions; avoid alcohol
Cenobamate	Effects on persistent sodium currents and gamma-aminobutyric acid type A receptor signalling (exact mechanism not fully established)	Adult focal seizures	Drug reaction with eosinophilia and systemic symptoms (risk reduced by slow titration); shortens the electrocardiographic Q-to-T interval; review sedation and substantial enzyme interactions

INTERACTIONS AND MONITORING THAT CHANGE DECISIONS

Cenobamate inhibits cytochrome P450 2C19, increasing exposure to substrates including clobazam's active metabolite; sedation may require dose adjustment. It induces cytochrome P450 3A and can lower susceptible medicine and hormonal contraceptive exposure. Enzyme-inducing antiseizure medicines lower perampanel concentrations. Eslicarbazepine and higher-dose perampanel can compromise relevant hormonal contraceptives. Verify the particular preparation and interaction rather than applying one rule to the class.

Record baseline mood, suicidality, aggression and cognition; ask carers about changes after initiation or titration. Screen for sedation, falls and adherence. Review pregnancy risks and contraception before treatment; do not abruptly stop an effective antiseizure medicine without a supervised plan.

SELECTION FLOW AND MEMORY AID



IMPORTANT BOUNDARIES

- Some medicines can worsen absence or myoclonic seizures; focal-seizure efficacy cannot be extrapolated to every generalised epilepsy.
- Lamotrigine can occasionally worsen myoclonus.

Seizure fit → Behaviour → Interactions → Follow-up.

Newer antiseizure medicines and psychiatry. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Epilepsy and psychiatry.

Credits and references: retained within the illustration.

AREA 12 OF 15 · P4-A06

Nutritional, toxic and metabolic encephalopathies

5	4.2%	IV-82	IV-86
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

HIGH 3 SINGLE 2

REVISION ORDER

Thiamine deficiency and the alcohol complications slot together, they overlap almost completely and one is the general case of the other. Hepatic encephalopathy next. Then the SIADH slot, and read its correction rate carefully rather than quickly. Wilson disease last, and it is self-contained.

WHAT IS IN THIS AREA

Wilson disease with its management, the neuropsychiatric manifestations of thiamine deficiency, hepatic encephalopathy, and the neurological complications of alcohol dependence syndrome. The fifth slot is the syndrome of inappropriate antidiuretic hormone secretion, which is an endocrine question sitting in a nutritional and toxic area, and it holds the most consequential number in this book.

IV - 82

Neuropsychiatric manifestation of Wilson disease and its management. [5+5]

5 + 5 | NUTRITIONAL, TOXIC AND METABOLIC ENCEPHALOPATHIES

DNB, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

1	The disease	Copper, one gene, and the age window it declares itself in
4	Neuropsychiatric picture	Movement, psychiatric and cognitive, in that order
4	Management	Confirm, decopper, maintain, then treat the psychiatry
1	The family rule	Screen every sibling, because it is treatable and inherited

THE DEFINITION

An autosomal recessive disorder of copper transport caused by mutation in the ATP7B gene on chromosome 13. Biliary copper excretion fails, copper accumulates in liver then brain, and the basal ganglia take the damage. It is one of the few reversible dementias, which is why it is asked.

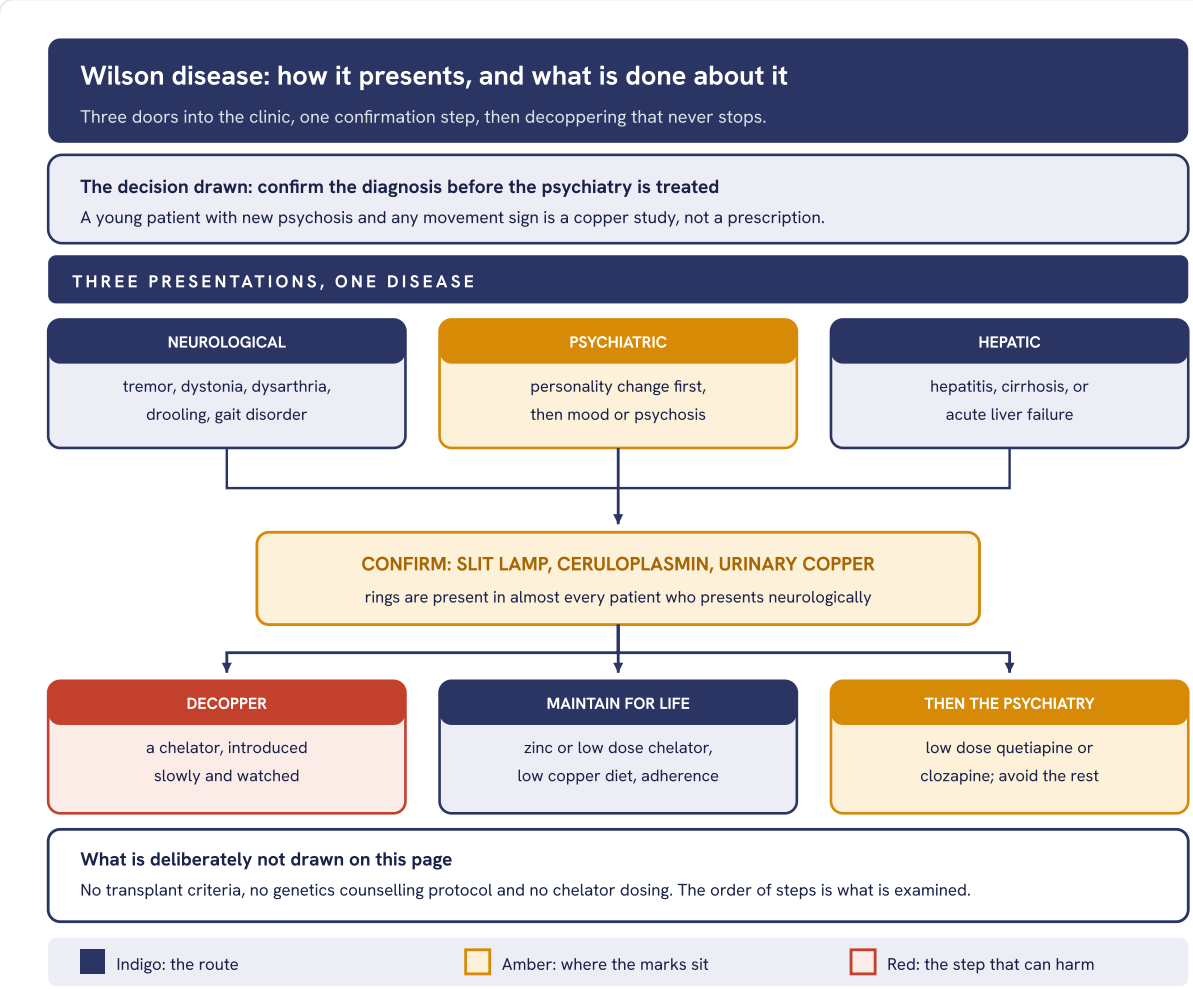


Fig 082.1 Three routes into the clinic, one confirmation step that must precede treatment, and the three arms of management that follow it.

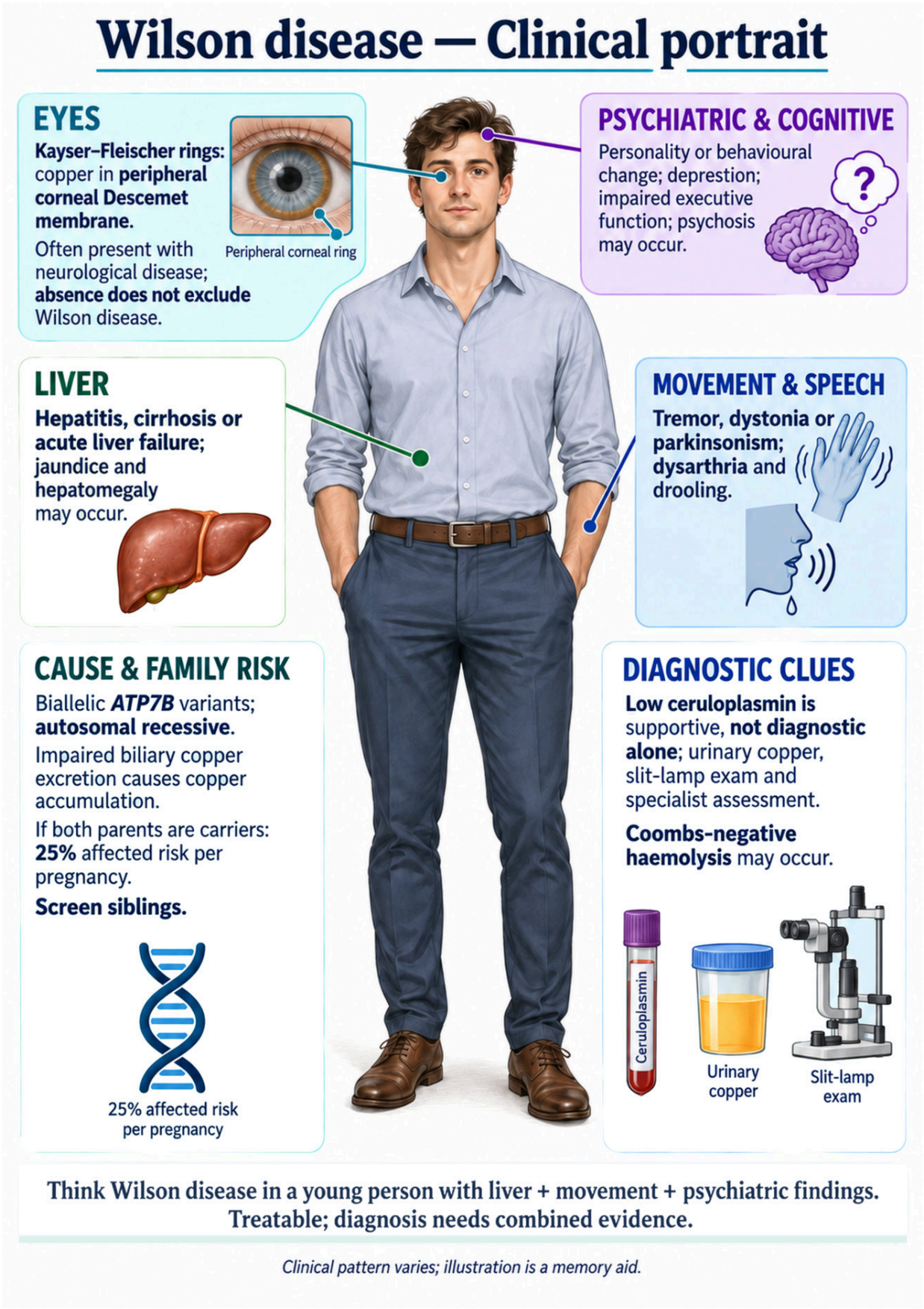
- **The psychiatric picture.** Personality change and falling school or work performance come first, and are usually read as adolescence. Depression is the commonest syndrome, with real self harm risk; psychosis occurs but is rarer than textbooks imply. Cognitive decline is subcortical: slow, dysexecutive, memory relatively spared.
- **Confirming it.** Kayser-Fleischer rings on slit lamp, low serum ceruloplasmin, raised twenty four hour urinary copper. Rings are present in nearly all neurological presentations but may be absent in purely hepatic ones, so their absence never excludes the disease.
- **The trap.** Chelation can worsen neurological signs in the first weeks, so it is introduced slowly and the patient is warned. Never give an antipsychotic with high extrapyramidal liability to an untreated patient.

WHAT EARNS THE MARKS

- **Personality change named as the first psychiatric sign.** Most answers open with psychosis and lose the natural history.
- **The confirmation step written before any treatment.** Rings, ceruloplasmin, urinary copper.
- **Lifelong maintenance, not a course.** Stopping is why a treated patient deteriorates.
- **Screening the siblings.** One line, and it separates a good answer from a competent one.

SEE ALSO Slot IV - 48 Huntington's disease Slot IV - 84 hepatic encephalopathy **SPRINT** Day 33, Endocrine & metabolic psychiatry

WEAVE 100 · PAPER IV · D4.14



Wilson disease - Clinical portrait risks and features. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-82.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · F25

COPPER METABOLISM AND WILSON DISEASE — ESSENTIAL METAL, FAILED DISPOSAL

Foundation 25 • Normal function → mechanism → clinical recognition → management

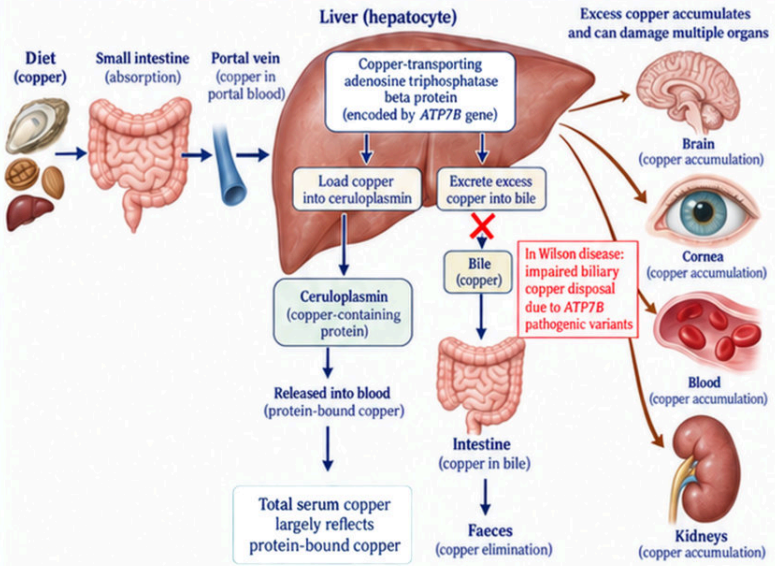
NORMAL PHYSIOLOGY

Normal: copper is a trace element needed for energy production, iron handling, connective tissue and antioxidant enzymes. Dietary copper is absorbed in the small intestine and carried through portal blood to the liver. Hepatocytes use the copper-transporting adenosine triphosphatase beta protein, encoded by the *ATP7B* gene, to load copper into ceruloplasmin and excrete excess copper into bile. Bile carries it into the intestine for faecal elimination. Ceruloplasmin is a circulating copper-containing protein; total serum copper largely reflects protein-bound copper.

WHAT GOES WRONG

What goes wrong: pathogenic variants in both copies of *ATP7B* impair biliary copper disposal and incorporation into ceruloplasmin. Copper first accumulates in the liver; toxic circulating copper can then damage brain, cornea, kidneys and blood cells. Inheritance is autosomal recessive; when both parents are carriers, each pregnancy has a 25% chance of an affected child, 50% of a carrier child and 25% of a child with neither familial variant. Age at presentation varies; do not exclude the diagnosis solely because someone is older than the usual adolescent/young-adult presentation.

COPPER METABOLISM AND PATHOGENESIS IN WILSON DISEASE



MECHANISM → RECOGNITION

Mechanism → recognition: copper-mediated hepatocyte injury → hepatitis, jaundice, cirrhosis or acute liver failure. Basal ganglia injury → tremor, dystonia, slowness, dysarthria, drooling and gait disorder. Corneal copper deposition → Kayser–Fleischer rings, best assessed by slit-lamp examination. Oxidative red-cell injury → direct-antiglobulin-test-negative haemolysis, particularly with acute liver failure. Kidney tubular injury can cause urinary abnormalities. Cognitive or behavioural change may accompany neurological disease; these symptoms alone are not specific. Rings may be absent, especially in hepatic presentations.

INVESTIGATIONS

Investigate a pattern, not one number: liver tests, albumin, clotting function, complete blood count, haemolysis assessment when appropriate, serum ceruloplasmin, basal 24-hour urinary copper, slit-lamp examination and neurological examination. Brain magnetic resonance imaging assesses neurological involvement. Molecular testing can confirm pathogenic variants and guide family screening. Hepatic copper quantification or a validated diagnostic scoring system helps when the initial assessment is inconclusive.

REFERENCE VALUES AND TRAPS

Reference values and traps: many adult laboratories use ceruloplasmin approximately 20–40 milligrams per decilitre; use the local range. Values below 14 milligrams per decilitre support suspicion but low levels have other causes, and normal levels do not exclude disease. Untreated symptomatic Wilson disease often gives urinary copper above 100 micrograms per 24 hours; above 40 micrograms per 24 hours can warrant investigation in children or asymptomatic individuals. Collection quality matters. Total serum copper may be low despite tissue copper excess because ceruloplasmin is low; a low total value does not prove copper deficiency. Treatment changes urinary copper, so untreated diagnostic thresholds are not interchangeable with monitoring targets.

MANAGEMENT

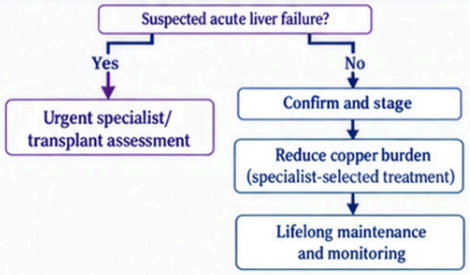
Management flow: suspected acute liver failure → urgent specialist/transplant assessment; otherwise confirm and stage → remove excess copper → lifelong maintenance and monitoring.

Chelators such as penicillamine or trientine bind copper and promote urinary excretion. Start and monitor under specialist care because neurological worsening can occur, particularly early. Penicillamine can cause rash, cytopenias or proteinuria; monitor blood count, urine and renal function.

Zinc reduces intestinal copper absorption and is used in selected asymptomatic or maintenance settings; it is not automatically interchangeable with chelation for significant active hepatic disease. Coordinate timing away from food and interacting medicines according to the prescribed regimen. Dietary moderation of high-copper foods supports treatment but cannot replace medication.

Follow clinical status, liver tests, copper indices, adherence and evidence of overtreatment. Screen first-degree relatives. Stopping effective therapy can cause severe relapse; successful liver transplantation corrects the hepatic transport defect in appropriately selected patients.

MANAGEMENT FLOWCHART



MEMORY AID | **Copper builds up when export fails; interpret the whole pattern.**

Foundation 25 • Medical foundations • Source-checked supplementary teaching • September 2026.
Laboratory ranges and treatment protocols require local context.

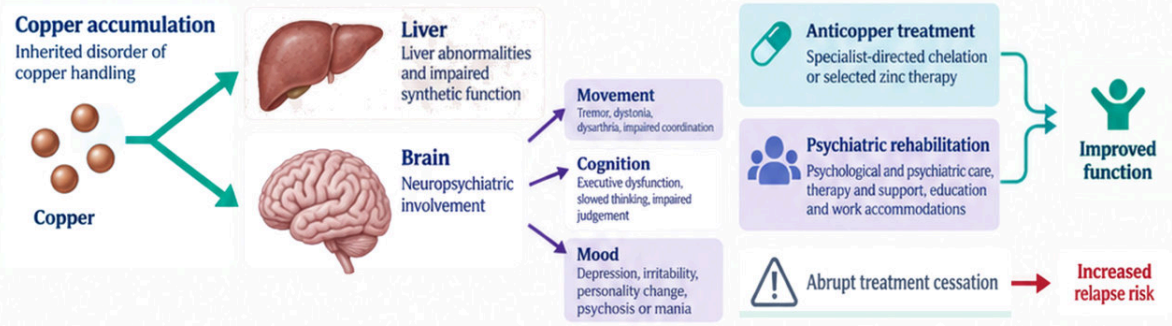
Copper metabolism and Wilson disease. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-82.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P17

WILSON DISEASE: PSYCHIATRIC PRESENTATION AND COORDINATED CARE

Psychiatric companion



Wilson disease is an inherited disorder of copper handling that can affect liver, movement, cognition and behaviour. Psychiatric symptoms may precede recognition of hepatic or neurological disease. Depression, irritability, personality change, impaired executive function and occasionally psychosis or mania require assessment in their own right. A psychiatric presentation does not establish Wilson disease, and a confirmed diagnosis does not mean every later symptom is caused by copper.

1 Recognize the pattern.

Consider Wilson disease when behavioural change accompanies tremor, dystonia, dysarthria, drooling, impaired coordination, unexplained liver abnormalities or a compatible family history. Presentation is often in younger people but age alone must not exclude it. Ask about school or work decline, slowed thinking, loss of judgement and difficulty completing tasks. Obtain collateral history with consent. Kayser–Fleischer rings may be absent, especially in predominaatly hepatic disease; an ordinary glance at the eyes cannot replace slit-lamp examination.

2 Assess the psychiatric syndrome.

Establish onset, progression, premorbid personality, substances, medicines and recent treatment changes. Examine mood, psychosis, cognition, sleep and suicide risk. Distinguish fluctuating inattention from stable cognitive impairment: delirium or hepatic encephalopathy requires medical investigation. Check whether apparent agitation reflects pain, communication difficulty or involuntary movements. Severe dysarthria does not automatically imply poor comprehension or incapacity. Support communication before judging a decision.

3 Investigate with the medical team.

Assessment combines liver function and synthetic function, blood count, serum ceruloplasmin, correctly collected basal 24-hour urinary copper, slit-lamp examination and neurological findings. Genetic testing and further specialist investigations resolve uncertain cases. Low ceruloplasmin alone is not diagnostic, and normal ceruloplasmin does not exclude disease. Total serum copper can be low despite tissue copper excess because much circulating copper is protein-bound. Do not diagnose copper deficiency or prescribe supplements from that result alone. Brain magnetic resonance imaging helps assess neurological involvement but does not independently diagnose a psychiatric syndrome.

4 Treat copper disease and distress together.

Specialist-directed chelation or selected zinc therapy addresses the underlying disorder; psychological and psychiatric care address current disability and risk. Effective treatment generally continues lifelong. Explain why improvement is gradual and why stopping treatment can precipitate serious deterioration. If neurological or psychiatric symptoms worsen after treatment starts, review promptly for paradoxical neurological worsening, adverse effects, adherence problems and alternative causes. Do not advise the patient to abandon anticopper treatment without specialist coordination.

5 Choose psychotropics cautiously.

Liver impairment, swallowing difficulty, movement disorders, renal function and drug interactions influence selection and dose. Antipsychotics can worsen extrapyramidal symptoms; use the lowest effective individualized regimen when necessary, with neurological monitoring. There is no universal rule that only two antipsychotics are permissible. Clozapine requires its usual blood monitoring and careful consideration of seizures and other adverse effects. Mood stabilizers require particular attention to hepatic or renal risks. An antidepressant may be appropriate for a diagnosed depressive disorder; assess past mania and monitor activation, suicidality and tolerability. Acute dangerous agitation, severe depression or inability to maintain nutrition may require inpatient multidisciplinary care.

6 Monitor function, not copper values alone.

Track mood, suicidal thinking, cognition, movement, daily living, adherence and caregiver strain alongside specialist laboratory monitoring. Document baseline motor findings before dopamine-blocking medicines. Provide memory aids, speech or occupational therapy and realistic education or work accommodations. Offer genetic counselling and family screening through the treating service. Support autonomy and reassess capacity for specific decisions when cognition or acute illness changes.

Memory aid: "Copper, cognition, coordination: assess all three."

This is a clinical reminder, not a diagnostic criterion.

P17 • Psychiatric companion • **Supplementary teaching.**

Wilson disease psychiatry. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-82.

Credits and references: retained within the illustration.

IV - 83

Neuropsychiatric manifestations of thiamine deficiency. [10]

10 NUTRITIONAL, TOXIC AND METABOLIC ENCEPHALOPATHIES

KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2 **The deficiency**	What thiamine does, and who runs out of it
3 **Wernicke encephalopathy**	The triad, and how it actually presents
3 **Korsakoff syndrome**	The amnestic picture that follows an untreated attack
2 **Treatment**	Parenteral, high dose, and before any carbohydrate

THE DEFINITION

Thiamine is a cofactor for transketolase and for the pyruvate and alpha-ketoglutarate dehydrogenase complexes, so deficiency starves the brain of usable glucose. Body stores last only weeks. The damage falls on structures around the third and fourth ventricles: mammillary bodies, medial thalamus and periaqueductal grey.

- **Wernicke encephalopathy.** The classical triad is confusion, ophthalmoplegia and ataxia, but all three are present in only a minority; most patients show confusion alone, and the diagnosis is missed for that reason. At risk are alcohol dependence, hyperemesis, bariatric surgery, prolonged vomiting, malnutrition and refeeding.
- **Treatment.** Parenteral thiamine, in doses far above the nutritional requirement, given on suspicion rather than on proof. The commonly taught regimen is intravenous thiamine three times daily for the first two to three days, then a lower daily dose continued orally. Oral thiamine alone is inadequate in the acute phase because absorption is saturable.
- **Korsakoff syndrome.** Dense anterograde amnesia with disorientation for time, confabulation and apathy, on a background of clear consciousness and preserved immediate recall and procedural memory. It follows most untreated attacks of Wernicke encephalopathy and is only partly reversible.

PITFALL

Glucose given before thiamine can precipitate or deepen Wernicke encephalopathy, because the last thiamine is consumed driving the glucose load. In any malnourished or alcohol dependent patient thiamine goes in first. A normal blood thiamine level never excludes the diagnosis.

WHAT EARNS THE MARKS

- **The triad named, and then contradicted.** Saying that it is complete in a minority is the line that shows clinical reading.
- **Thiamine before glucose, written as an order of actions.**
- **Korsakoff described as a distinct amnestic syndrome,** not as a later stage of the same confusion.
- **The parenteral route, with the reason oral fails.**

SEE ALSO Slot IV - 86 neurological complications of alcohol Slot IV - 93 amnestic disorder SPRINT Day 33, Endocrine & metabolic psychiatry

WEAVE 100 · PAPER IV · F26

THIAMINE — FUEL HANDLING AND DEFICIENCY SYNDROMES

Foundation 26 • Normal function → mechanism → clinical recognition → management

Normal

Thiamine, vitamin B 1, is a water-soluble vitamin obtained from food. Its active form, **thiamine diphosphate**, assists enzymes that convert carbohydrate-derived fuel into usable cellular energy. **Pyruvate dehydrogenase** links pyruvate to **acetyl coenzyme A; alpha-ketoglutarate dehydrogenase** operates in the citric acid cycle; **transketolase** supports the pentose phosphate pathway. **Magnesium** is needed for thiamine-dependent enzyme function. The **brain, peripheral nerves and heart** are particularly vulnerable when energy metabolism fails.

What goes wrong

Limited body stores are depleted by poor intake, prolonged vomiting, malabsorption, bariatric surgery or inadequate supplementation during nutritional support. Alcohol dependence can combine poor intake, reduced absorption and impaired utilisation. Increased metabolic demand during refeeding can expose an existing deficiency. **Deficiency is not confined to alcohol use.** Failure of oxidative metabolism reduces energy availability and can contribute to lactate accumulation and neuronal injury.

Mechanism → syndromes

Peripheral nerve injury produces **dry beriberi**, a typically symmetrical neuropathy with sensory symptoms, weakness and reduced reflexes.

Cardiovascular involvement produces **wet beriberi**: vasodilatation, tachycardia, oedema and heart failure, sometimes with severe circulatory collapse.

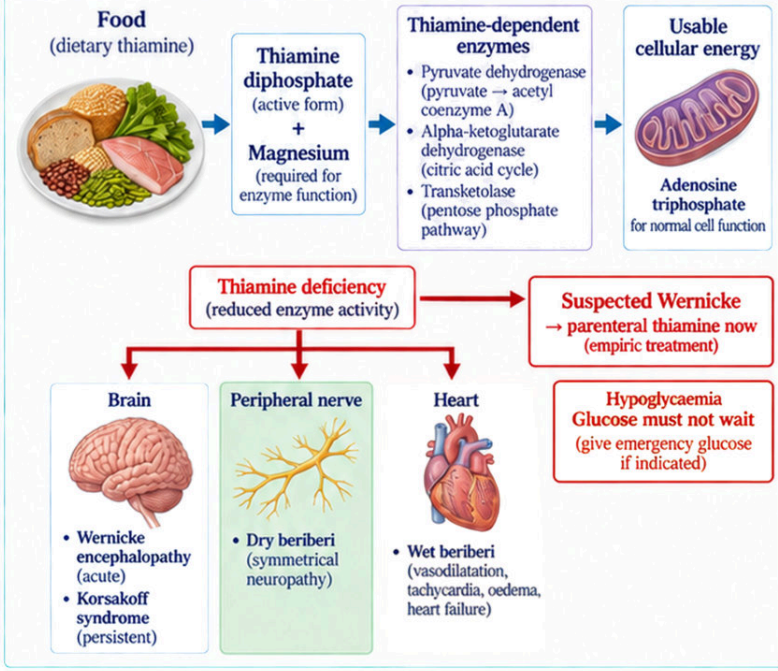
Wernicke encephalopathy is an acute brain syndrome with altered mental state, gait/coordination problems and ocular signs such as nystagmus or ophthalmoplegia. The complete classical triad is often absent: do not wait for all three. Injury around the mammillary bodies, thalamus and periaqueductal regions explains memory, gait and eye-movement abnormalities.

Korsakoff syndrome is a persistent amnestic disorder, especially difficulty forming new memories, sometimes with confabulation; it is not simply ongoing delirium.

MEMORY AID

Suspect Wernicke, give parenteral thiamine; emergency glucose must not wait.

From food to cellular energy — and the consequences of deficiency



Recognition and investigations

Assess diet, weight loss, vomiting, surgery, alcohol history, eye movements, gait, cognition, cardiovascular status and refeeding risk.

Clinical suspicion is the treatment trigger.

Caine criteria support recognition in appropriate settings: at least two of dietary deficiency, eye signs, cerebellar dysfunction, or altered mental state/memory impairment.

Check bedside glucose immediately; obtain blood count, electrolytes, kidney/liver function, magnesium and phosphate according to presentation. Magnetic resonance imaging may support the diagnosis but normal imaging does not exclude it.

Blood thiamine assays can help document deficiency, but treatment must not await the result and a normal result does not reliably exclude acute Wernicke encephalopathy.

Reference context

Adult dietary requirements are approximately 1.2 milligrams daily for men and 1.1 milligrams daily for women; pregnancy/lactation generally require 1.4 milligrams daily. These nutritional amounts are not treatment doses.

Whole-blood thiamine diphosphate reference intervals are assay-dependent; interpret the local laboratory range rather than a universal cut-off.

Refeeding assessment follows serial phosphate, potassium and magnesium, not a thiamine level alone.

Management flow

Suspected Wernicke encephalopathy → acute hospital treatment → immediate parenteral thiamine → review response and alternative diagnoses daily. One current United Kingdom guideline regimen is intravenous thiamine 300–500 milligrams three times daily for 3–5 days, followed, when symptoms persist, by 300–500 milligrams once daily for a further 3–5 days and while improvement continues; then oral replacement and nutritional follow-up. Regimens vary by jurisdiction and local protocol. This treatment schedule must not be confused with lower-dose prophylaxis for an asymptomatic at-risk patient. Oral treatment alone is inadequate for suspected acute disease because absorptuen may be insufficient. Administer in a setting equipped for rare serious allergic reactions.

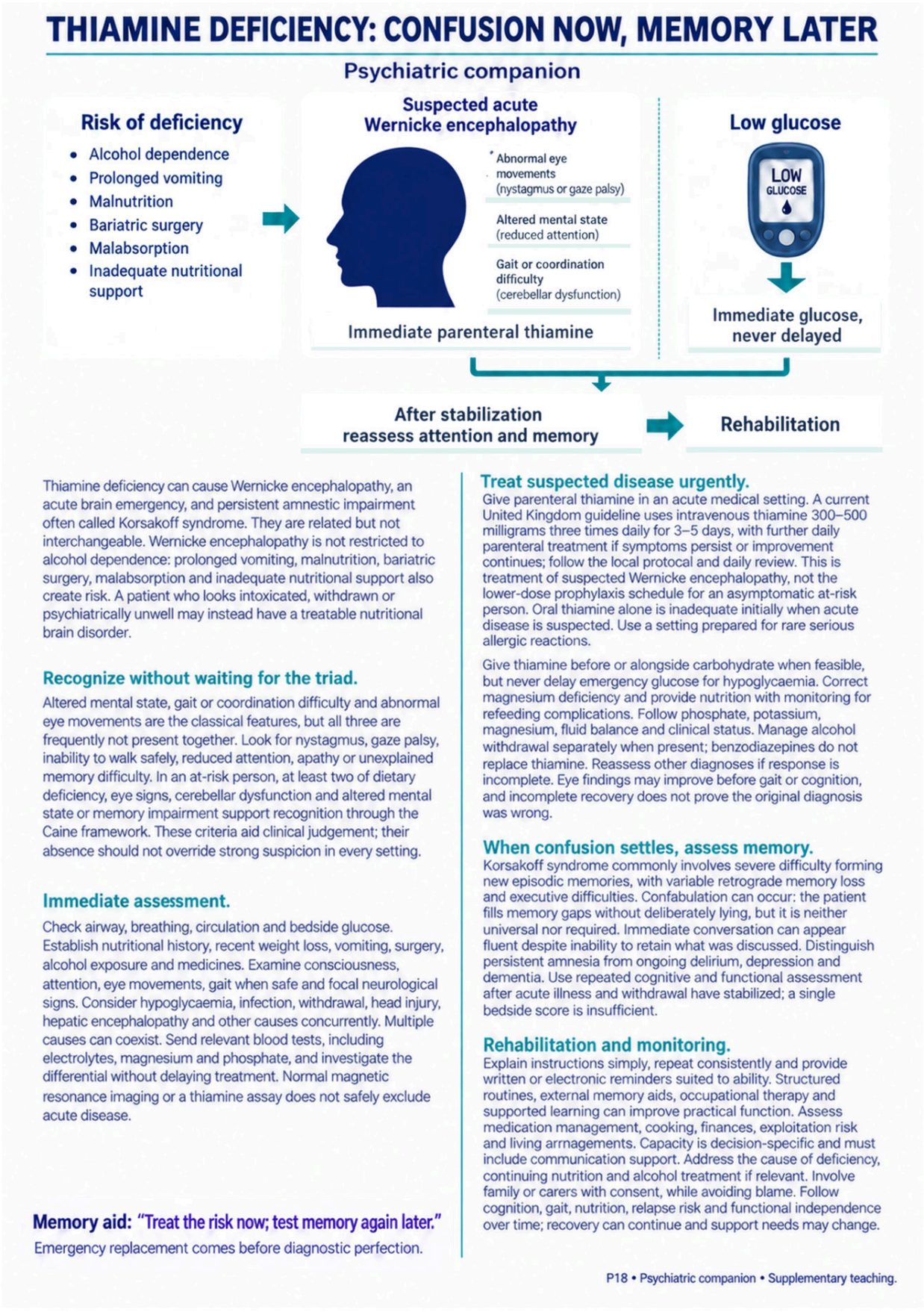
Give thiamine promptly before or alongside carbohydrate when feasible, but never delay emergency glucose for hypoglycaemia. Correct magnesium deficiency, provide balanced nutrition with monitored refeeding, treat the underlying cause and address alcohol withdrawal safely when present. Physiotherapy, memory rehabilitation and long-term nutritional support may be required. Improvement in ocular signs can precede recovery of gait or cognition; residual memory impairment is possible.

Foundation 26 • Medical foundations • Source-checked supplementary teaching • September 2026.
Laboratory ranges and treatment protocols require local context.

Thiamine function and deficiency. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-83.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P18



Thiamine deficiency psychiatric syndromes. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-83.

Credits and references: retained within the illustration.

IV - 84

Hepatic encephalopathy. [10]

10 NUTRITIONAL, TOXIC AND METABOLIC ENCEPHALOPATHIES

KNRUHS, KUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	Definition and mechanism	Ammonia, the astrocyte, and the shunt
2	Grading	West Haven, and the minimal grade above it
3	Clinical picture	The psychiatric presentations it is mistaken for
3	Management	Find the precipitant first, then lactulose and rifaximin

DEFINITION, AND HOW IT PRESENTS

A reversible neuropsychiatric syndrome of liver failure or portosystemic shunting. Gut derived nitrogenous substances, chiefly ammonia, reach the brain, where astrocytes swell and GABAergic tone rises. It presents as delirium, but also as depression, apathy or personality change, and in the minimal grade only as impaired driving and falling work performance. Sleep-wake reversal is often the earliest change a family reports.

GRADE	CONSCIOUSNESS	BEHAVIOUR AND COGNITION	BEDSIDE SIGN
Minimal	Normal	Normal on examination; deficits only on testing	None
One	Sleep-wake reversal	Euphoria or anxiety, short attention span	**Mild asterixis or tremor**
Two	Lethargy, apathy	Disorientation for time, personality change	**Obvious asterixis, slurred speech**
Three	Somnolent but rousable	Gross disorientation, confusion, bizarre behaviour	**Asterixis often lost, rigidity**
Four	Coma	Unresponsive	Decerebrate posturing

Amber cells are the bedside discriminator. Asterixis appears early and is lost again as the patient sinks, so its absence means either grade zero or grade four, never the middle.

- **Precipitants carry the marks.** Gastrointestinal bleeding, infection including spontaneous bacterial peritonitis, constipation, dehydration and diuretics, electrolyte disturbance, a high protein load, and sedative drugs. Finding and correcting the precipitant is the treatment; lactulose and rifaximin support it. Blood ammonia correlates poorly with grade, and a normal value does not exclude the diagnosis.

WHAT EARNS THE MARKS

- **The precipitant searched for before any drug is named.** That single line is the difference between treating and reciting.
- **A grade written with its bedside sign,** not as an unqualified word like confusion.
- **Minimal hepatic encephalopathy mentioned.** Most answers begin at grade one and lose the mark.
- **Benzodiazepines named as harmful here,** with the short acting exception if sedation cannot be avoided.

SEE ALSO Slot IV - 92 delirium Slot IV - 86 neurological complications of alcohol SPRINT

Day 33, Endocrine & metabolic psychiatry

WEAVE 100 · PAPER IV · F15

LIVER FUNCTION, LIVER FAILURE AND HEPATIC ENCEPHALOPATHY

Foundation 15 • Normal function → mechanism → clinical recognition → management

NORMAL LIVER FUNCTIONS

Portal venous blood brings absorbed nutrients and intestinal products to the liver; hepatic arterial blood supplies additional oxygen. Hepatocytes process carbohydrates, lipids and proteins, store glycogen and metabolise many medicines. They synthesise albumin and most coagulation factors. Bilirubin from haem breakdown is conjugated and excreted into bile. Ammonia generated partly by intestinal nitrogen metabolism is converted into urea for kidney excretion. Bile supports fat absorption. The liver therefore links metabolism, detoxification, synthesis and digestion; an enzyme level measures injury rather than all these functions.

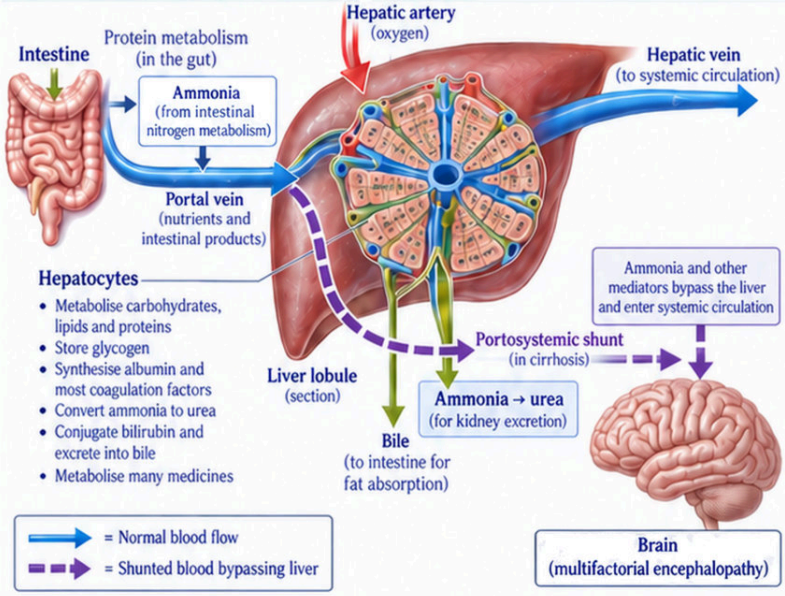
INJURY → FAILURE

Viral hepatitis, alcohol-related disease, metabolic dysfunction-associated steatotic liver disease, autoimmune disease, toxins and other disorders can injure hepatocytes. Chronic injury → fibrosis → cirrhosis, with nodules and distorted architecture. Increased resistance to portal flow produces portal hypertension. Varices are dilated collateral veins that may bleed; fluid retention and portal pressure cause ascites. Loss of synthesis and clearance causes low albumin, impaired coagulation, jaundice and drug accumulation. Acute liver failure develops rapidly with impaired coagulation and encephalopathy in someone without pre-existing cirrhosis; it requires urgent specialist and transplant-centre assessment.

MECHANISM → RECOGNITION

Bilirubin accumulation → jaundice. Portal hypertension and renal sodium retention → ascites and oedema. Variceal rupture → haematemesis or melaena. Reduced detoxification plus portosystemic shunting → brain exposure to ammonia and other mediators. Inflammation and altered neurotransmission contribute to hepatic encephalopathy; it is not simply one blood ammonia number. Impaired attention, sleep reversal, disorientation and reduced consciousness may progress to coma. Asterixis is a brief lapse of sustained posture, commonly seen as hand flapping; it is neither specific nor obligatory.

LIVER FUNCTION AND HEPATIC ENCEPHALOPATHY: KEY MECHANISMS



INVESTIGATIONS AND VALUES

Typical adult reference intervals: albumin 35–50 grams/litre; total bilirubin about 5–21 micromoles/litre; international normalised ratio about 0.8–1.2 without anticoagulation. Ranges vary by laboratory, population and method. Alanine and aspartate aminotransferases indicate hepatocellular injury; alkaline phosphatase and gamma-glutamyl transferase help assess cholestatic patterns. Markedly abnormal enzymes do not directly measure residual function, and advanced cirrhosis may have modest enzyme elevations. Check blood count, kidney function, sodium, glucose and infection markers; use ultrasound and appropriate aetiological tests. Ascites often requires diagnostic sampling. Hepatic encephalopathy is a clinical diagnosis after evaluating alternatives; high ammonia alone does not confirm or grade it. A normal ammonia should prompt diagnostic reconsideration, not automatic closure.

MEMORY AID

Injury enzymes are not liver function; confusion needs a cause.

TREAT THE CAUSE AND COMPLICATION

Suspected acute failure, bleeding, sepsis or severe encephalopathy → urgent hospital care. Protect airway when consciousness is markedly reduced. Treat the underlying disease and review hepatotoxic or accumulating medicines. Cirrhosis care includes nutrition, vaccination, surveillance for hepatocellular carcinoma and portal-hypertension management. Ascites may require sodium moderation, diuretics or therapeutic paracentesis with albumin according to the clinical setting; avoid dehydration and kidney injury.

ENCEPHALOPATHY FLOW

Identify and correct precipitant → infection, gastrointestinal bleeding, constipation, dehydration, electrolyte disturbance or sedating medicine → exclude hypoglycaemia, stroke and other delirium causes → lactulose for overt episodes in cirrhosis, titrated usually to two or three soft stools daily. Excess diarrhoea can worsen dehydration and encephalopathy. Rifaximin is added for recurrent episodes in appropriate patients. Maintain adequate nutrition and protein; routine protein restriction worsens malnutrition. Persistent or recurrent decompensation warrants transplant assessment. Monitor cognition, bowel pattern, hydration and caregiver understanding. These cirrhosis measures must not replace the distinct emergency management of acute liver failure.

Foundation 15 • Medical foundations • Source-checked supplementary teaching • September 2026.
Laboratory ranges and treatment protocols require local context.

Liver function liver failure and hepatic encephalopathy. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-84.

Credits and references: retained within the illustration.

IV - 85

Syndrome of Inappropriate Anti Diuretic Hormone Secretion (SIADH). [10]

10 | ASKED AT 7 MARKS | NUTRITIONAL, TOXIC AND METABOLIC ENCEPHALOPATHIES

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	Definition and criteria	Euvolaemic hyponatraemia with inappropriately concentrated urine
2	Causes	The psychotropics first, since they are ours
2	Clinical picture	What a falling sodium looks like on a ward round
3	Management	Stop the drug, restrict fluid, respect the correction rate

THE DEFINITION

Antidiuretic hormone secretion that continues despite plasma hypo-osmolality, producing dilutional hyponatraemia in a clinically euvolaemic patient. The diagnosis needs all of: low serum sodium with low plasma osmolality, urine osmolality inappropriately high, raised urine sodium, clinical euvolaemia, and normal thyroid, adrenal and renal function in a patient not taking diuretics.

- **Why this belongs to psychiatry.** Selective serotonin reuptake inhibitors are the commonest psychotropic cause, typically in older patients within the first weeks of treatment. Carbamazepine and oxcarbazepine, antipsychotics and tricyclics all cause it. Other causes are small cell lung carcinoma, pneumonia and tuberculosis, stroke and head injury, and pain or nausea.
- **The picture.** Mild falls give nausea, headache, poor concentration and unsteadiness; moderate falls give confusion, delirium and falls themselves; severe falls give seizures and coma. Every one of these is read at first as the illness being treated, or as dementia in an older patient.
- **Two things it is not.** In psychogenic polydipsia the urine is dilute, not concentrated, because the kidney is behaving correctly. In cerebral salt wasting the patient is volume depleted rather than euvolaemic, and needs salt and water rather than restriction.

PITFALL

Correct slowly. Raising serum sodium faster than roughly eight to ten millimoles per litre in twenty four hours risks osmotic demyelination. The patient who improves for a day and then deteriorates with dysarthria, quadriparesis or a locked-in picture has been corrected too fast, and that damage is not reversible.

WHAT EARNS THE MARKS

- **The full criterion set, including euvolaemia** and the normal endocrine and renal function that must be shown.
- **Psychotropic causes named first.** The examiner is asking a psychiatrist.
- **The correction rate, with the harm it prevents.** Most answers stop at fluid restriction.

SEE ALSO Slot IV - 92 delirium Slot IV - 59 psychiatric disorders of thyroid and parathyroid disease SPRINT Day 33, Endocrine & metabolic psychiatry

WEAVE 100 · PAPER IV · F10

WATER AND SODIUM: CONCENTRATION IS NOT TOTAL CONTENT

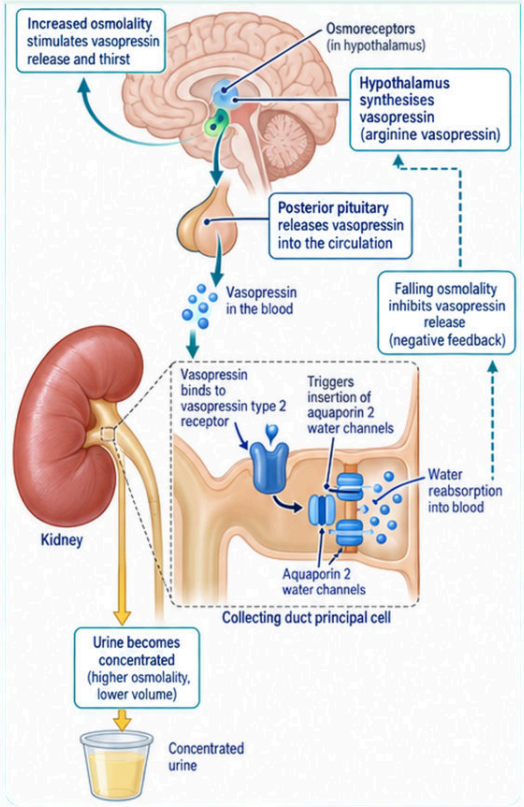
Foundation 10 • Vasopressin → water balance → hyponatraemia

NORMAL PHYSIOLOGY

Sodium is the main extracellular cation. Serum sodium mainly reflects the balance between body water and exchangeable sodium/potassium, not the amount of sodium eaten that day. Total-body sodium helps determine extracellular volume; water balance strongly determines its concentration.

Hypothalamic osmoreceptors sense concentration. Increased osmolality stimulates thirst and release of arginine vasopressin, also called antidiuretic hormone. Vasopressin is made in the hypothalamus and released from the posterior pituitary. It acts at kidney collecting-duct vasopressin type 2 receptors, increasing aquaporin 2 water channels and water reabsorption. Urine becomes concentrated. With excess water, normal suppression permits dilute urine. Low effective circulating volume can stimulate vasopressin despite low osmolality.

VASOPRESSIN MECHANISM AND FEEDBACK



EXCESS WATER (NORMAL RESPONSE)

Lower osmolality suppresses vasopressin release
↓
Reduced water reabsorption in kidney collecting ducts
↓
Dilute urine (low osmolality, higher volume)
↓
Excretes excess water maintains sodium concentration

INAPPROPRIATE ANTIDIURESIS

Persistent vasopressin release despite low osmolality
↓
Continued water reabsorption
↓
Retained water expands body water
↓
Diluted sodium (hyponatraemia)
↓
Mild volume expansion usually clinically euvolaemic (rather than visibly oedematous)

NORMAL VALUES, DIFFERENT MEANINGS

- Typical adult serum sodium: 135–145 mmol/L; laboratory intervals vary.
- Serum osmolality commonly 275–295 mOsm/kg.
- Sodium below 135 mmol/L is hyponatraemia; above 145 mmol/L is hypernatraemia.
- Urine osmolality must be interpreted against the clinical state; there is no single 'normal' concentration.
- A kidney should dilute urine when excess water needs excretion and concentrate it when water must be conserved.

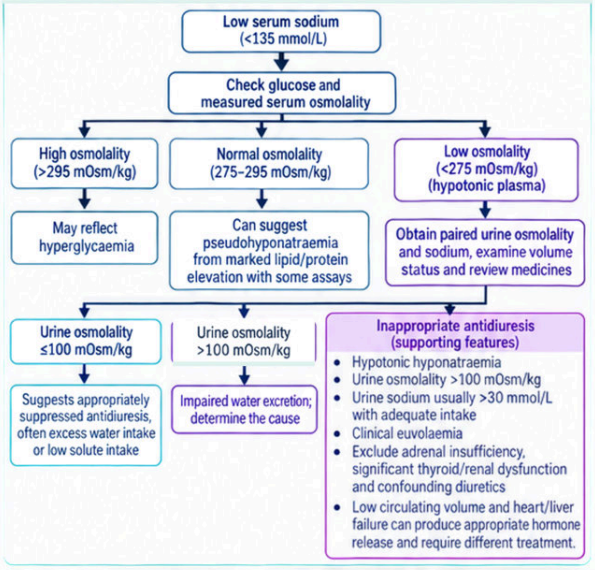
WHAT INAPPROPRIATE ANTIDIURESIS DOES

- Persistent antidiuretic action despite hypotonicity → retained water → diluted sodium.
- Mild volume expansion promotes sodium excretion, so the patient is usually clinically euvolaemic rather than visibly oedematous. 'Euvolaemic' does not mean no extra body water.
- Causes include pulmonary or central nervous system disease, some malignancies, medicines, pain and nausea.
- The term syndrome of inappropriate antidiuresis is broader than assuming every case has a measured high hormone level.

WHY SYMPTOMS OCCUR

- Low extracellular tonicity moves water into cells. Brain swelling can cause headache, nausea, impaired attention, confusion, seizures or reduced consciousness.
- Severity depends on speed of change as well as sodium concentration; one number cannot predict the examination.
- Over time the brain loses osmoles to adapt. Rapid correction of chronic hyponatraemia then removes water from adapted cells and risks osmotic demyelination, with delayed neurological deterioration.
- Both untreated cerebral oedema and excessive correction are dangerous.

DIAGNOSTIC FLOW



BASIC MANAGEMENT: SEVERITY FIRST, CAUSE NEXT

- Seizures or major neurological symptoms require urgent monitored hypertonic saline under an experienced clinician, with an initial small rise to relieve cerebral oedema—not immediate normalisation.
- Society for Endocrinology 2022 aims for about 5 mmol/L initially, limiting total rise to 10 mmol/L in the first 24 hours and 8 mmol/L per 24 hours thereafter. These are ceilings, not targets; high-risk patients require more conservative plans.
- Monitor sodium and urine output closely; sudden water diuresis can overcorrect.
- Overcorrection needs urgent specialist action and sometimes controlled re-lowering.
- For stable inappropriate antidiuresis, treat the cause, review offending drugs and use individualised fluid restriction/solute strategies. Restriction is not the treatment for hypovolaemic hyponatraemia.
- Vasopressin deficiency/resistance causes dilute polyuria and possible hypernatraemia—the opposite water-handling problem. It requires a separate supervised pathway.

Check tonicity, urine and cause; correct with care.

Sources: European hyponatraemia guideline; Society for Endocrinology 2022; National Institute of Diabetes and Digestive and Kidney Diseases; Gloucestershire Hospitals laboratory.

Water-sodium balance and inappropriate antidiuretic hormone secretion. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-85.

Credits and references: retained within the illustration.

IV - 86

Neurological complications of Alcohol dependence syndrome. [10]

10 NUTRITIONAL, TOXIC AND METABOLIC ENCEPHALOPATHIES

RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The four mechanisms	Withdrawal, nutritional, direct toxicity, and the indirect routes
3 Acute complications	Seizures, delirium tremens, Wernicke encephalopathy
3 Chronic complications	Amnestic, cerebellar, peripheral, cognitive
2 The named rarities	The two syndromes that separate a top answer

MECHANISM	SYNDROME	THE RECOGNISING FEATURE	TIMING
Withdrawal	Seizures, delirium tremens	**Generalised fits within two days; delirium with tremor and autonomic storm**	Hours to days
Thiamine loss	Wernicke, then Korsakoff	**Confusion with eye signs and ataxia, then dense anterograde amnesia**	Days, then lasting
Direct toxicity	Cerebellar degeneration	Wide based gait and truncal ataxia, arms and speech spared	Years
Direct toxicity	Peripheral neuropathy	Symmetrical, distal, sensory first, painful burning feet	Years
Direct toxicity	Alcohol related dementia	Dysexecutive, with relative sparing of recognition memory	Years
Indirect, hepatic	Hepatic encephalopathy	Asterixis, sleep reversal, a known cirrhotic liver	Variable
Indirect, traumatic	Subdural haematoma	Fluctuating consciousness after a fall that may not be recalled	Weeks
Osmotic and rare	Central pontine myelinolysis; Marchiafava-Bignami	Over-fast sodium correction; corpus callosum demyelination	Days to weeks

Amber rows are the emergencies. Organising the answer by mechanism rather than by an alphabetical list is itself worth marks, because it shows why each complication happens.

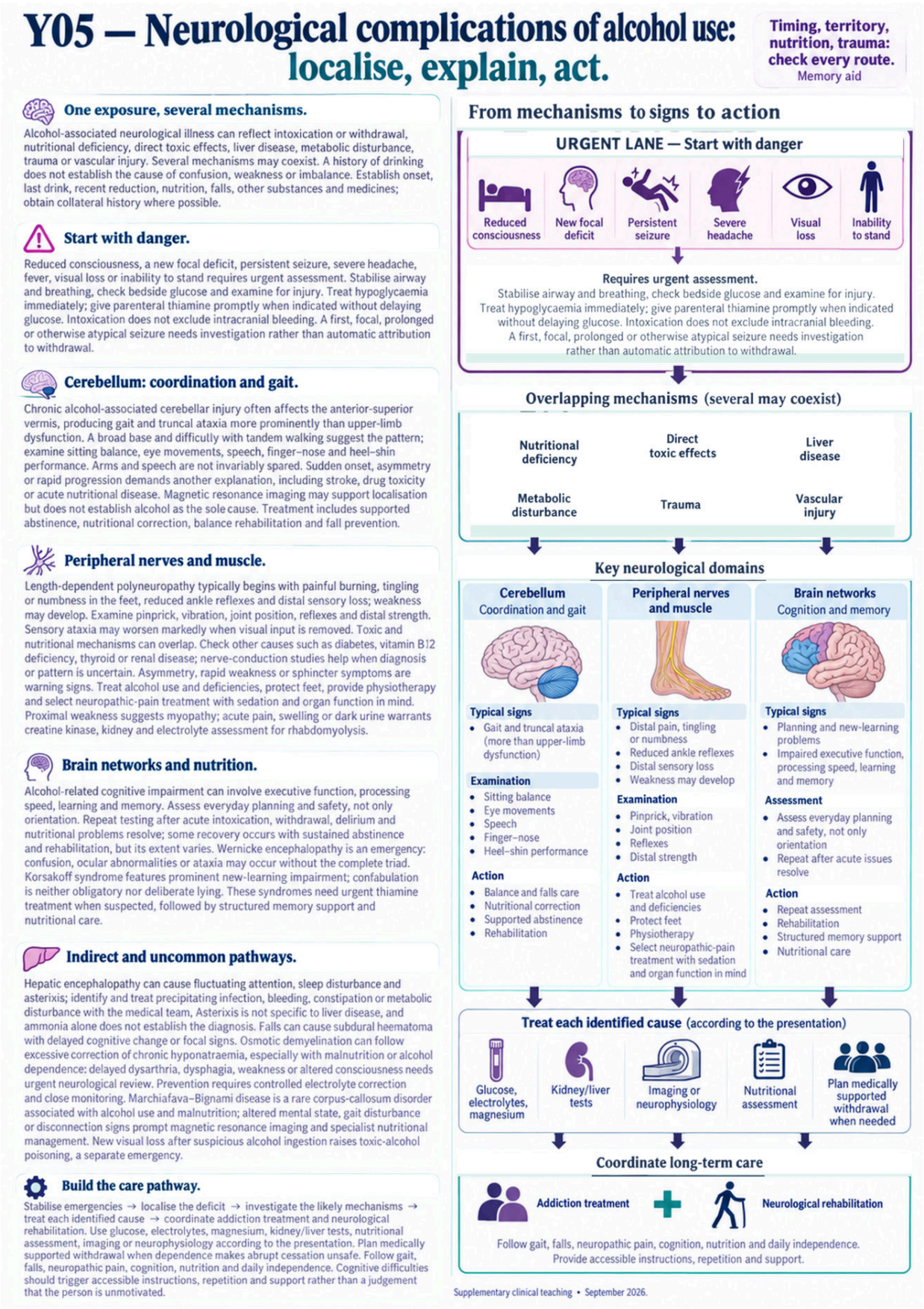
- **The two that separate a top answer.** Marchiafava-Bignami disease is demyelination of the corpus callosum, giving dementia, gait disturbance and interhemispheric disconnection signs. Central pontine myelinolysis follows too rapid correction of hyponatraemia and gives dysarthria, quadriparesis and a locked-in state.
- **The overlap trap.** These rarely arrive alone. A dependent patient who is confused may be withdrawing, thiamine depleted, hepatic, post-ictal and bleeding under the dura at once, so each mechanism is excluded rather than assumed.

WHAT EARNS THE MARKS

- **The answer organised by mechanism,** not as an undifferentiated list of names.
- **Acute separated from chronic,** because the two demand different actions on the day.
- **The two rare named syndromes.** They are the cheapest distinguishing marks on this page.

SEE ALSO Slot IV - 83 thiamine deficiency Slot IV - 43 subdural haematoma SPRINT Day 33, Endocrine & metabolic psychiatry

WEAVE 100 · PAPER IV · Y05



Neurological complications of alcohol use. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-86.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P08

WATER, SODIUM AND PSYCHOTROPIC MEDICINES

Psychiatric companion 08 • Retained water, excess drinking or impaired concentration?

SODIUM DESCRIBES A WATER BALANCE

Serum sodium usually lies around 135–145 millimoles/litre. A low concentration often reflects excess water relative to exchangeable sodium/potassium, not simply dietary salt deficiency. Vasopressin promotes renal water retention. Medicines, illnesses and drinking patterns can disturb the balance through different mechanisms, requiring different treatments.

RECOGNISE BRAIN SYMPTOMS EARLY



- New headache
- Nausea
- Unsteadiness
- Confusion
- Lethargy
- Seizures

Severe symptoms or reduced consciousness require emergency care regardless of a remembered previous result.

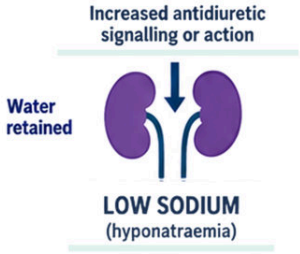
New neurological symptoms after a medication change should trigger sodium and medical assessment. Both the degree and speed of sodium change matter. Do not attribute new confusion to psychiatric relapse or respond only by increasing sedatives.

Review the timeline, dose changes, all prescribed/nonprescribed drugs, fluid intake, vomiting/diarrhoea, pain, nausea and comorbidity. Older age, low body mass, prior hyponatraemia and concurrent sodium-lowering medicines increase concern.

THREE PATTERNS TO SEPARATE

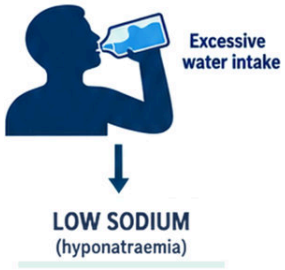
Inappropriate antidiuresis

Selective serotonin reuptake inhibitors, serotonin–noradrenaline reuptake inhibitors, antipsychotics, carbamazepine and oxcarbazepine are associated with hyponatraemia. Mechanisms include enhanced antidiuretic signalling or action. Water is retained despite low tonicity; urine is inappropriately concentrated. Drug association alone does not establish this diagnosis.



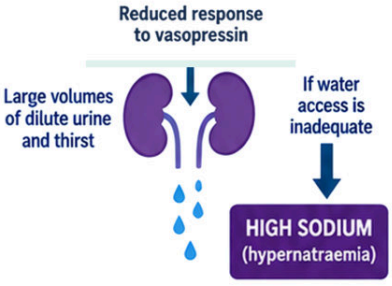
Primary polydipsia

Water intake exceeds excretory capacity. It may accompany psychosis but also occurs in other settings. Urine is often very dilute when vasopressin is suppressed; low solute intake or concurrent illness/drugs can complicate the pattern. Ask about actual intake without assuming motive.



Lithium-associated renal concentrating impairment

The kidney may respond poorly to vasopressin, producing large volumes of dilute urine and thirst. If water access is inadequate, dehydration and hypernatraemia can develop. This is physiologically different from inappropriate water retention.



INVESTIGATE BEFORE NAMING THE SYNDROME

Confirm sodium and assess glucose, measured serum osmolality, urine osmolality, urine sodium, renal function and clinical volume status. Evaluate adrenal and thyroid causes where indicated. Hyperglycaemia-related low sodium and laboratory pseudohyponatraemia are not equivalent to hypotonic hyponatraemia.

Hypotonic serum with urine osmolality above 100 milliosmoles/kilogram indicates impaired water excretion but is not specific to inappropriate antidiuresis. Urine sodium, intake, diuretics and volume status refine interpretation. Obtain appropriately timed samples where feasible without delaying emergency treatment.

MEDICATION DECISIONS FOLLOW SEVERITY

Suspected drug-associated symptomatic or marked hyponatraemia generally requires stopping the implicated medicine and urgent medical assessment. Mild stable cases may permit carefully supervised reduction or substitution, but psychiatric relapse risk and competing causes must be weighed. Avoid routine rechallenge after a convincing serious reaction when alternatives exist. Mirtazapine or bupropion may have lower reported risk in selected antidepressant situations, but neither is risk-free or suitable for every patient.

Baseline sodium is prudent in higher-risk patients; plan early repeat measurement after initiation or increases and whenever symptoms occur. Exact intervals depend on risk, drug and local protocol. The psychotropic-specific monitoring algorithm is expert consensus, not proof that one schedule prevents all events. Oxcarbazepine reactions can occur late as well as early.

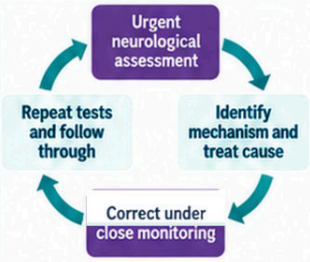
CORRECT SAFELY AND FOLLOW THROUGH

Severe symptomatic hypotonic hyponatraemia may need monitored hypertonic saline. Overcorrection can cause osmotic demyelination; limits and rescue treatment belong to a supervised protocol. Stopping a causative drug or restricting intake can provoke rapid water diuresis, so monitor sodium and urine output.

Fluid restriction is not universal: it may suit selected stable inappropriate-antidiuresis cases but can harm hypovolaemia or lithium-related water loss. Lithium-related polyuria requires renal assessment, lithium concentrations and a coordinated treatment plan. Document the likely cause, medication changes, repeat-test timing and responsible clinician.

MEMORY AID

Check symptoms, measure tonicity, identify the mechanism, correct under monitoring.



Sources: Association of Medicine and Psychiatry expert consensus; European hyponatraemia guideline; Society for Endocrinology; Specialist Pharmacy Service; oxcarbazepine prescribing information.

Water-sodium and psychotropic effects. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Nutritional, toxic and metabolic encephalopathies.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P11

LIVER DISEASE: MENTAL STATE AND SAFE PRESCRIBING

Psychiatric companion 11 • Recognise encephalopathy → identify contributors → adapt treatment

SEPARATE THREE CLINICAL QUESTIONS

A patient with liver disease may have hepatic encephalopathy, a separate psychiatric disorder, medication toxicity, or several together. Portosystemic shunting and impaired hepatic clearance allow gut-derived substances to affect the brain; inflammation and other metabolic disturbances also contribute. Slowed thinking, sleep reversal, apathy and altered behaviour can precede obvious disorientation. Their presence does not establish depression or encephalopathy without assessment.

Ask whether attention and awareness fluctuate, whether symptoms developed acutely, and how functioning differs from baseline. Obtain collateral information about sleep, driving, falls, adherence and personality change. Examine for disorientation and asterixis, a brief failure to maintain posture. Asterixis is neither specific nor obligatory; its absence cannot assign an encephalopathy grade.

LOOK FOR REVERSIBLE CAUSES FIRST

New confusion requires urgent medical assessment. Search for infection, gastrointestinal bleeding, constipation, dehydration, renal injury, electrolyte disturbance and sedating medicines. Also consider hypoglycaemia, alcohol withdrawal, thiamine deficiency, intoxication, intracranial disease and other causes of delirium. Do not anchor on cirrhosis and miss a second illness.

Ammonia is not a standalone diagnostic test or treatment target in chronic hepatic encephalopathy. Levels are affected by collection and processing; a normal result in suspected overt encephalopathy should prompt diagnostic reconsideration. Assess clinical course and the full differential. Laboratory reference ranges vary, so interpret the local report rather than applying a universal cutoff.

TREAT ENCEPHALOPATHY WITH THE MEDICAL TEAM

Correct the precipitant and protect the airway when consciousness is severely reduced. Lactulose is usually first-line therapy for overt episodes; adjust to approximately two or three soft bowel motions daily after initial treatment, avoiding excessive diarrhoea and dehydration. Rifaximin is commonly added to prevent recurrent episodes. Nutrition needs active support; routine protein restriction can worsen malnutrition and is not the solution.

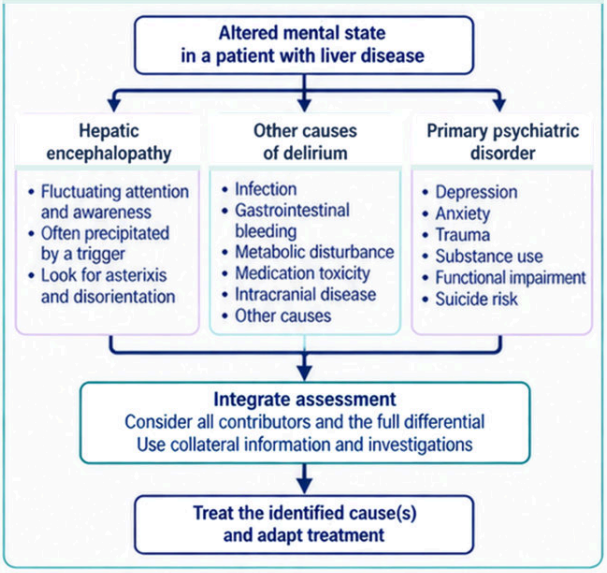
Use orientation, a quiet environment, sleep support and familiar assistance. Review driving and other safety-sensitive activities. Sedatives can worsen cognition and falls; avoid treating confusion reflexively with a sleeping tablet. Severe agitation threatening immediate safety requires individual specialist management alongside treatment of its cause.

ASSESS PERSISTENT PSYCHIATRIC SYMPTOMS

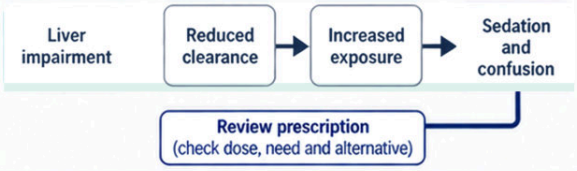
After acute contributors are addressed, assess depression, anxiety, alcohol use, trauma, suicide risk and functional impairment. Distinguish persistent low mood and loss of pleasure from fluctuating encephalopathy or sedation. Psychological treatment can address stigma, avoidance and the burden of chronic illness without adding metabolic drug load. Agree realistic goals and involve carers with consent.

Alcohol withdrawal can itself be life-threatening. Benzodiazepines may be necessary in a monitored withdrawal protocol despite their encephalopathy risk; specialist choice and dosing matter. Lorazepam or oxazepam may be favoured because they rely less on oxidative hepatic metabolism, but neither is harmless in advanced disease. This exception does not justify routine benzodiazepine prescribing for insomnia or anxiety.

DIFFERENTIAL DIAGNOSIS AND ASSESSMENT FLOW



THE EFFECT OF LIVER IMPAIRMENT ON DRUG EXPOSURE



PRESCRIBE FOR FUNCTION, NOT ENZYMES ALONE

Reduced clearance, low albumin and shunting can increase exposure to psychotropics. Establish liver-disease severity, prior encephalopathy, renal function, electrolytes and the full drug list. Normal aminotransferases do not prove normal hepatic function. Check product-specific advice; 'start low and go slowly' cannot replace a contraindication.

For example, sertraline labelling recommends half the usual starting and therapeutic doses in mild hepatic impairment and does not recommend use in moderate or severe impairment. Duloxetine should be avoided in chronic liver disease or substantial alcohol use. Lithium avoids hepatic metabolism but may become toxic with renal dysfunction, dehydration or interacting diuretics; it is not automatically the safer choice.

MONITOR AND REASSESS

Track alertness, falls, bowel pattern, mood, adherence and adverse effects after changes. Repeat relevant liver, renal and electrolyte tests according to the medicine and clinical state. New jaundice, marked sedation or confusion requires prompt review. Coordinate prescribing so one clinician knows the complete plan.

MEMORY AID

Check attention, find the trigger, check clearance, review function.

Sources: American Association for the Study of Liver Diseases; sertraline and duloxetine prescribing information.

Liver disease psychiatry. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Nutritional, toxic and metabolic encephalopathies.

Credits and references: retained within the illustration.

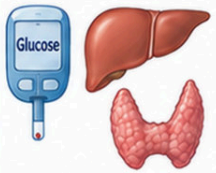
ENCEPHALOPATHY

Six cause clusters • recognise brain dysfunction, reverse threats, identify the cause

What the term means

Encephalopathy describes brain dysfunction from diverse causes. Acute encephalopathy is the underlying rapidly developing brain process; its clinical expression may include subsyndromal delirium, delirium or coma. Delirium involves acute, fluctuating disturbance of attention and awareness with cognitive change. Encephalitis specifically implies brain inflammation. These six clusters organies assessment; they overlap and are not six mutually exclusive diseases.

1 METABOLIC, ENDOCRINE AND NUTRITIONAL



Causes: abnormal glucose, sodium or calcium; renal or hepatic failure; thyroid or adrenal crisis; thiamine deficiency.

Clues: systemic illness, dehydration, asterixis or malnutrition. Asterixis is not specific to hepatic disease. Wernicke encephalopathy can occur without the complete confusion–ataxia–eye-sign triad.

Action: correct the identified disturbance with monitored, cause-specific treatment. Suspected Wernicke encephalopathy needs prompt parenteral thiamine. Hepatic encephalopathy requires precipitant treatment and usually lactulose; renal replacement may be needed for uraemic encephalopathy.

2 TOXIC AND DRUG RELATED

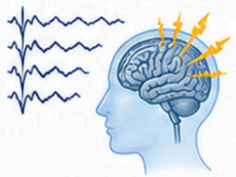


Causes: sedatives, opioids, anticholinergic medicines, lithium, valproate, carbon monoxide; alcohol or sedative withdrawal.

Clues: exposure or dose change, impaired drug clearance, characteristic pupils, respiratory depression, autonomic changes or tremor.

Action: reconcile prescriptions, non-prescribed substances and recent withdrawal; stop or adjust implicated treatment safely. Use relevant drug levels, electrocardiography and toxicology advice. Give indicated antidotes, such as naloxone for opioid-induced respiratory depression; treat dangerous withdrawal with a monitored protocol.

6 SEIZURE RELATED AND ELECTRICAL



Causes: ongoing convulsive seizures, non-convulsive status epilepticus or a postictal state.

Clues: witnessed seizure, subtle repetitive movements, unexplained fluctuation or failure to recover as expected.

Action: convulsive seizures lasting five minutes or more require emergency status treatment. Obtain urgent electroencephalography when non-convulsive status is possible. Do not assume persistent unresponsiveness is simply postictal; reassess metabolic, toxic and structural causes.

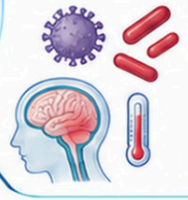


ENCEPHALOPATHY

Altered attention • cognition • behaviour • arousal

One presentation, often several contributors

3 INFECTIOUS

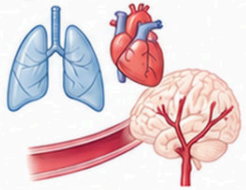


Causes: meningitis or encephalitis from direct central nervous system infection; sepsis-associated brain dysfunction without direct brain infection.

Clues: fever, headache, meningism, seizures or immunosuppression; absence of fever or neck stiffness does not exclude infection.

Action: obtain appropriate cultures and cerebrospinal fluid when safe. Start indicated empirical antimicrobials promptly; suspected herpes simplex encephalitis needs intravenous aciclovir without waiting for delayed investigations. Sepsis also requires source control and circulatory support; metabolic and hypoxic mechanisms may coexist.

5 HYPOXIC, ISCHAEMIC AND VASCULAR

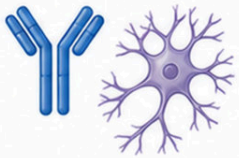


Causes: respiratory failure, shock or cardiac arrest; stroke, haemorrhage or hypertensive encephalopathy, including posterior reversible encephalopathy syndrome.

Clues: low oxygenation, hypotension, abrupt focal deficit, severe headache, visual symptoms or markedly raised blood pressure.

Action: restore oxygenation, ventilation and perfusion; activate urgent stroke or haemorrhage assessment when indicated. Treat hypertensive emergencies with controlled reduction under monitoring. Vascular disease may produce focal deficits as well as global dysfunction; the word "reversible" does not guarantee recovery.

4 IMMUNE AND INFLAMMATORY



Causes: autoimmune encephalitis, inflammatory demyelination or systemic autoimmune disease affecting the brain.

Clues: subacute cognitive or behavioural change with seizures, movement disorder, focal signs or autonomic instability.

Action: assess magnetic resonance imaging, cerebrospinal fluid and clinically directed antibody tests. Normal imaging does not exclude autoimmune encephalitis. A serum antibody alone does not establish the diagnosis. Neurology guides immunotherapy after infection is adequately excluded; investigate associated malignancy when indicated.

INITIAL MANAGEMENT — REAL CLINICAL FLOW

New altered mental state → Stabilise airway, breathing and circulation; check bedside glucose, oxygenation, temperature and seizures → **Treat immediate threats in parallel:** hypoglycaemia, hypoxia, shock, ongoing seizures and suspected serious infection → Obtain collateral history, baseline function, medication/exposure history and focused neurological examination → **Select investigations and cause-directed treatment** → Reassess consciousness, physiology and response; escalate deterioration to critical care.

Do not delay emergency glucose for thiamine; give thiamine promptly when indicated.

TARGETED INVESTIGATIONS

Initial blood tests commonly include blood count, glucose, electrolytes, calcium, kidney and liver function; add blood gas, cultures, drug levels, thyroid or adrenal tests as indicated. Urgent brain imaging is especially important with focal signs, trauma or suspected intracranial pathology. Lumbar puncture requires a safety assessment; electroencephalography detects electrical causes. Ammonia alone neither confirms nor grades hepatic encephalopathy.

REASSESSMENT AND MEMORY AID

Record arousal and attention repeatedly; support orientation, hearing, vision, sleep, hydration and safe mobilisation. Treat distress individually; sedation does not treat the underlying cause.

Stabilise → Search → Treat → Reassess.

Mixed causes are common; a plausible first explanation must not stop the search.

Encephalopathy - six cause groups and urgent assessment. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Nutritional, toxic and metabolic encephalopathies.

Credits and references: retained within the illustration.

AREA 13 OF 15 · P4-A09

Neuro-infective and autoimmune neuropsychiatry

5	4.0%	IV-87	IV-91
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 SINGLE 4

REVISION ORDER

The two HIV slots together, the second sits inside the first. Anti-NMDA receptor encephalitis next, it is the longest answer here and the only one asked across five limbs. Tuberculous meningitis after that. PANDAS last, and it is the shortest map in the area.

WHAT IS IN THIS AREA

HIV in two slots, the neuropsychiatric manifestations of the disease and the AIDS dementia complex. Then the psychiatric manifestations of tuberculous meningitis, anti-NMDA receptor encephalitis asked from aetiology through to management, and PANDAS.

IV - 87

Describe the neuropsychiatric manifestations of HIV disease. [10]

10 NEURO-INFECTIVE AND AUTOIMMUNE NEUROPSYCHIATRY

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

SKELETON

2	The framework	Five routes by which HIV reaches the mind
3	Direct effects	The neurocognitive spectrum, and its subcortical shape
3	Indirect effects	Opportunistic infection, tumour, and the drugs
2	Management	Treat what is treatable, and prescribe around the antiretrovirals

THE FRAMEWORK

Answer by route, not by symptom. HIV reaches the brain directly, permits opportunistic infection and tumour, is treated with drugs that carry neuropsychiatric effects, and brings stigma and loss of its own. Naming the routes before listing anything is what organises this answer.

ROUTE	WHAT IT PRODUCES	THE CLUE
Direct viral	Neurocognitive impairment, from asymptomatic through mild disorder to HIV-associated dementia	Subcortical profile, tracks viral load
Opportunistic and neoplastic	Cerebral toxoplasmosis, cryptococcal meningitis, tuberculous meningitis, progressive multifocal leukoencephalopathy, primary central nervous system lymphoma	**Focal signs, fever, headache; low CD4 count**
Iatrogenic	Efavirenz causing vivid dreams, mood change and rarely psychosis; steroids; interferon	**Temporal link to starting a drug**
Psychosocial	Depression, adjustment disorder, substance use, suicide risk	May precede or follow the diagnosis

Amber marks the two clues that change management on the day: a low CD4 count reframes any new neurological sign as an opportunistic infection until imaged, and a recent drug change is the cheapest explanation to exclude.

- **The cognitive profile.** Subcortical: psychomotor slowing, poor attention and executive function, apathy and withdrawal, with motor clumsiness and tremor. Memory is relatively spared early, and aphasia and agnosia are absent, which is what separates it from a cortical dementia. In India tuberculous meningitis is the opportunistic infection to think of first.
- **Prescribing.** Depression is the commonest psychiatric diagnosis here and is under-treated, though it responds normally. Watch enzyme interactions between antiretrovirals and psychotropics, and combined effects on the QT interval. Never stop antiretroviral therapy to manage a psychiatric side effect without the treating physician.

WHAT EARNS THE MARKS

- **The five routes named before any list.** It converts recall into a structure.
- **The cognitive picture identified as subcortical,** with what is spared.
- **Drug interactions mentioned.** Most answers stop at naming the syndromes.

SEE ALSO Slot IV - 91 AIDS dementia complex Slot IV - 88 tuberculous meningitis SPRINT

Day 35, Neuropsychiatry of systemic & neurological disease

IV - 88

Describe Psychiatric manifestations of Tuberculous Meningitis. [10]

10 NEURO-INFECTIVE AND AUTOIMMUNE NEUROPSYCHIATRY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Why psychiatrists see it	A subacute basal meningitis whose first changes are behavioural
3	The psychiatric picture	Prodrome, delirium, and the syndromes it imitates
3	Diagnosis	The fluid, the images, and why treatment does not wait
2	The drugs	The treatment carries psychiatric hazards of its own

WHY THIS REACHES A PSYCHIATRIST

Tuberculous meningitis is a subacute meningitis centred on the base of the brain, with exudate, hydrocephalus and infarction rather than the abrupt prostration of pyogenic disease. It remains common in India. Its prodrome is measured in weeks and is behavioural before it is neurological, so the first doctor consulted is often a psychiatrist.

- **The presentation.** Weeks of malaise, low grade fever, night sweats, weight loss and headache, with apathy, irritability and personality change that families describe as depression. Then confusion and delirium, cranial nerve palsies with the sixth and third most affected, meningism, seizures and the papilloedema of hydrocephalus.
- **The syndromes it imitates.** Depression with marked apathy, an acute or subacute psychosis, delirium of unclear cause, and occasionally catatonia. Later, after treatment, a residual amnestic or dysexecutive picture from infarction and hydrocephalus, which is the part that does not recover.
- **Diagnosis, and the drugs.** Cerebrospinal fluid shows a lymphocytic pleocytosis with high protein and low glucose; culture is slow, so nucleic acid amplification and imaging of basal exudates carry the early diagnosis, and treatment is never delayed for confirmation. Isoniazid causes psychosis and neuropathy, prevented by pyridoxine; cycloserine causes depression, psychosis and suicidal ideation; steroids alter mood; and rifampicin induces liver enzymes, lowering levels of many psychotropics.

PITFALL

A young adult with weeks of headache, low fever and a change in personality is imaged and tapped before an antipsychotic is started. Treating this as a first episode of psychosis costs weeks, and the harm done by hydrocephalus and infarction in that time does not reverse.

WHAT EARNS THE MARKS

- **The behavioural prodrome named as the reason for the referral.**
- **The cerebrospinal fluid picture, all three values.**
- **Antitubercular drugs named as psychiatric causes,** with pyridoxine for isoniazid.
- **The residual deficit acknowledged.** Recovery is not always complete, and saying so is honest.

SEE ALSO Slot IV - 87 neuropsychiatric manifestations of HIV Slot IV - 92 delirium SPRINT
Day 35, Neuropsychiatry of systemic & neurological disease

WEAVE 100 · PAPER IV · F27

TUBERCULOSIS AND TUBERCULOUS MENINGITIS — FROM LUNG DEFENCE TO BASAL INFLAMMATION

Foundation 27 • Normal function → mechanism → clinical recognition → management

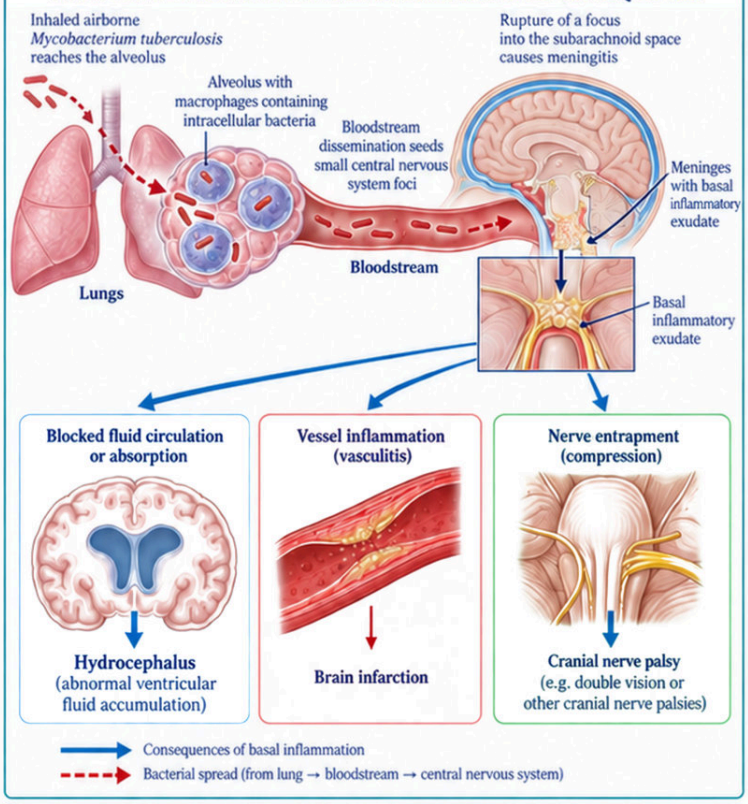
1. NORMAL FUNCTION

- Inhaled particles meet airway clearance and alveolar macrophages.
- T lymphocytes help macrophages contain intracellular organisms.
- A granuloma is an organised collection of immune cells that can limit spread but does not guarantee sterilisation.
- The meninges protect the brain and spinal cord; cerebrospinal fluid circulates through ventricles and subarachnoid spaces and is reabsorbed into the circulation.

2. WHAT GOES WRONG

- *Mycobacterium tuberculosis* spreads through airborne particles from infectious pulmonary or laryngeal disease.
- Inhaled bacteria survive within macrophages.
- Infection can remain contained without symptoms or progress to active disease; impaired immunity increases progression risk.
- Bloodstream dissemination can seed small central nervous system foci.
- Rupture of a focus into the subarachnoid space causes meningitis.
- Basal inflammatory exudate obstructs cerebrospinal fluid flow or absorption, entraps cranial nerves and inflames vessels.
- Hydrocephalus means abnormal ventricular fluid accumulation; vasculitis can cause brain infarction.
- A tuberculoma is a focal granulomatous mass and is distinct from meningitis, although both can coexist.

FROM LUNG INFECTION TO BASAL MENINGITIS AND ITS CONSEQUENCES



3. MECHANISM → RECOGNITION

- **Pulmonary disease** → persistent cough, fever, night sweats, weight loss and sometimes haemoptysis.
- Meningitis often evolves over days to weeks with headache, fever, reduced appetite, irritability or apathy, then neck stiffness, vomiting, altered consciousness, seizures or focal deficits.
- Basal nerve involvement can cause double vision or other cranial nerve palsies.
- Raised intracranial pressure produces worsening headache, vomiting, papilloedema or reduced consciousness.
- Absence of obvious lung disease, neck stiffness or a classic full syndrome does not exclude meningitis.

4. ASSESSMENT IS URGENT

- Stabilise airway, breathing and circulation, check glucose and neurological status, assess seizures and raised-pressure signs, obtain blood tests and evaluate for pulmonary or disseminated infection.
- Brain imaging assesses hydrocephalus, basal enhancement, infarcts and tuberculomas.
- Lumbar puncture is performed when safe; investigate contraindications such as a dangerous mass effect rather than insisting everyone must be tapped immediately.
- Collect cerebrospinal fluid for cell count, protein, paired blood/fluid glucose, rapid molecular testing and mycobacterial culture with resistance assessment.
- Rapid molecular tests such as Xpert Ultra improve early detection, but a negative result does not reliably exclude disease.
- Culture remains useful but is slow.

5. FLUID INTERPRETATION

- A typical adult cerebrospinal fluid sample contains 0–5 white cells per microlitre and protein roughly 15–45 milligrams per decilitre; laboratory and age-specific ranges vary.
- Glucose is interpreted with simultaneous blood glucose, normally about 60% of the blood value.
- Tuberculous meningitis often has lymphocyte-predominant cells, raised protein and low glucose; early disease can be neutrophilic.
- None of these findings alone is diagnostic.
- Tuberculin skin or interferon-gamma release tests indicate immune sensitisation, not proof of active meningitis; negative results do not exclude it.

6. MANAGEMENT FLOW

- High clinical suspicion → obtain samples promptly when safe → start specialist-directed multidrug treatment without waiting for culture → control inflammation and complications.
- Initial drug-susceptible treatment generally includes isoniazid, rifampicin, pyrazinamide and ethambutol, followed by a continuation phase; central nervous system regimens and duration require age-, resistance- and national-programme-specific selection.
- Do not substitute a short pulmonary regimen automatically.
- World Health Organization guidance recommends adjunctive dexamethosone or prednisolone tapered over 6–8 weeks.
- Provide pyridoxine with isoniazid when indicated, and monitor hepatic toxicity, neuropathy, visual toxicity, interactions and adherence.
- Rifampicin strongly induces drug metabolism.
- Neurosurgical management may be needed for hydrocephalus.
- Treat seizures, fluid/electrolyte disturbances and nutrition; evaluate human immunodeficiency virus and coordinate antiretroviral timing with specialists.
- Arrange rehabilitation and cognitive/hearing/vision follow-up because residual disability may persist.

MEMORY AID

Basal inflammation can block fluid, injure vessels and trap nerves.

Foundation 27 • Medical foundations • Source-checked supplementary teaching • September 2026.

Laboratory ranges and treatment protocols require local context.

Tuberculosis and tuberculous meningitis. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-88.

Credits and references: retained within the illustration.

IV - 89

PANDAS. [10]

10 NEURO-INFECTIVE AND AUTOIMMUNE NEUROPSYCHIATRY

KNRUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition and criteria	The five, named, with the mechanism proposed
3	Clinical picture	What the abrupt onset actually looks like
2	Investigation	What supports it, and what has to be excluded
3	Management and status	Three arms, and an honest word on the evidence

THE FIVE CRITERIA

Paediatric autoimmune neuropsychiatric disorders associated with streptococcal infection. Obsessive compulsive disorder or a tic disorder; onset before puberty; abrupt onset with an episodic course of dramatic relapse and remission; a temporal association with group A streptococcal infection; and associated neurological signs, particularly choreiform movements. The proposed mechanism is molecular mimicry, with antibodies to streptococcus cross-reacting with basal ganglia.

- **The picture.** A previously well child develops obsessions, compulsions or tics almost overnight, and parents can usually name the day. Around it come separation anxiety, urinary frequency, deteriorating handwriting, emotional lability, irritability and restricted eating. The abruptness, not the content, is what is distinctive.
- **Investigation.** A throat swab and antistreptococcal antibody titres support recent infection but never confirm the diagnosis, since raised titres are common in healthy school-age children. There is no diagnostic test. Exclude Sydenham chorea, Wilson disease, autoimmune encephalitis, and an ordinary tic or obsessive compulsive disorder with a coincidental infection.
- **Management.** Treat proven streptococcal infection with antibiotics. Treat the psychiatric disorder on its own terms: cognitive behavioural therapy with exposure and response prevention, and a selective serotonin reuptake inhibitor started at a low dose, because these children are often sensitive. Immunomodulation is confined to severe cases in specialist hands.

THE HONEST POSITION

The entity is contested. PANS is the broader construct, which drops the streptococcal requirement and accepts any abrupt onset presentation. Saying that the diagnosis is disputed, and giving the reason, earns more than presenting it as settled fact; long term antibiotic prophylaxis in particular is not established.

WHAT EARNS THE MARKS

- **All five criteria, with abruptness and the episodic course.**
- **Molecular mimicry named as the proposed mechanism.**
- **Titres described as supportive but not diagnostic.**
- **The controversy stated, with PANS distinguished.**

SEE ALSO Slot IV - 90 anti-NMDA receptor encephalitis Slot IV - 82 Wilson disease **SPRINT** Day 35, Neuropsychiatry of systemic & neurological disease

IV - 90

Discuss in detail about the etiology, clinical features, differential diagnosis, assessment and management of Anti-NMDA receptor encephalitis. [10]

10 | ASKED AT 15 MARKS | NEURO-INFECTIVE AND AUTOIMMUNE NEUROPSYCHIATRY

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	**Aetiology**	The antibody, the tumour, and the post-viral route
3	**Clinical features**	The stages, in the order they arrive
2	**Differential and assessment**	What it imitates, and what confirms it
3	**Management**	Immunotherapy, the tumour, and supportive care

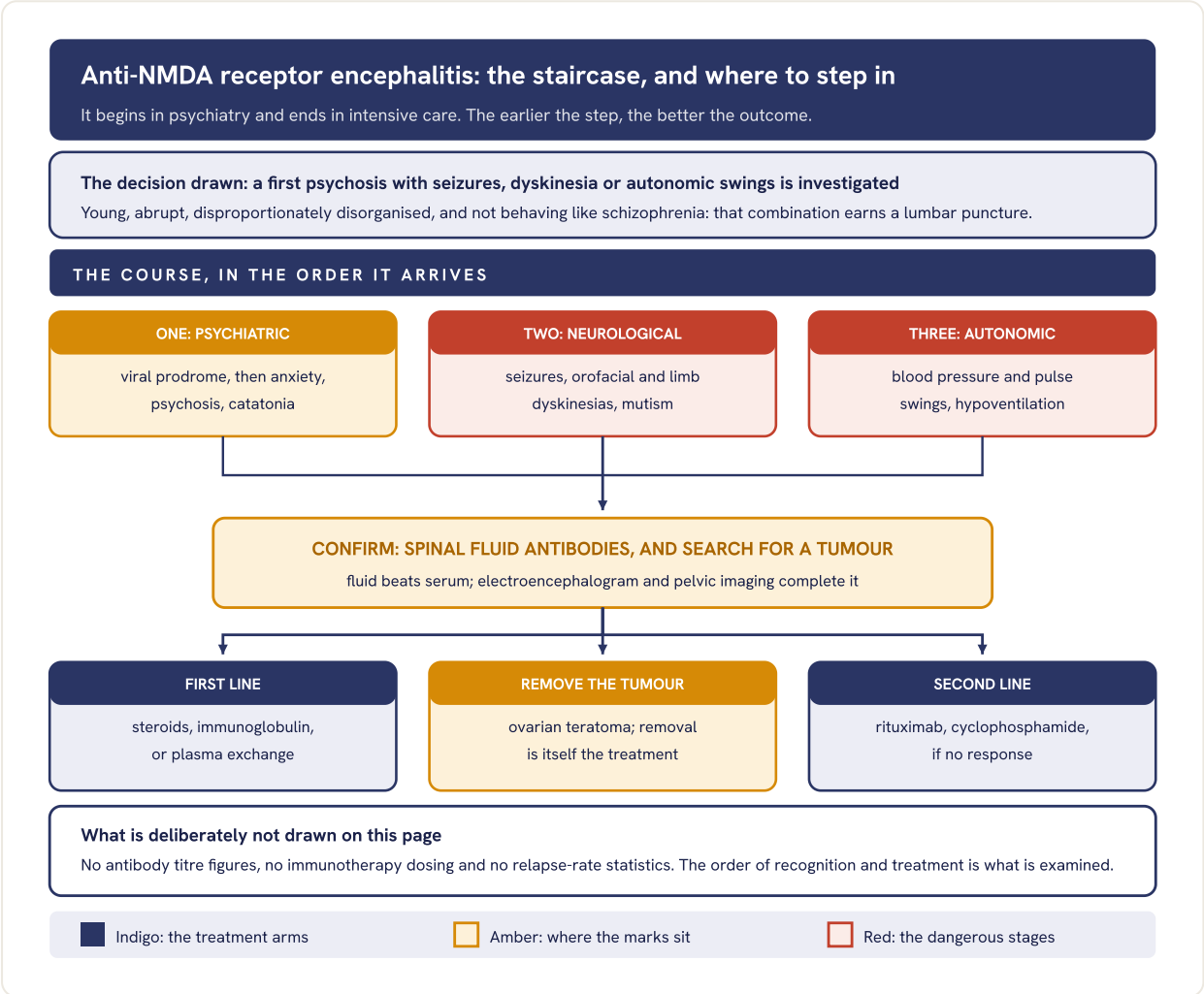


Fig 090.1 The staged course from psychiatric presentation to autonomic instability, the confirmatory step, and the three arms of treatment that follow it.

- **Aetiology and differential.** An antibody against the NR1 subunit of the NMDA receptor. Some cases are paraneoplastic, classically an ovarian teratoma in a young woman; others follow herpes simplex encephalitis; many have no trigger found. It is mistaken for a first episode of schizophrenia, for a drug induced psychosis, for catatonia, and, once rigid and febrile on antipsychotics, for neuroleptic malignant syndrome.

- **What confirms it.** Antibodies in cerebrospinal fluid, which is more sensitive than serum and may be positive when serum is negative, so a negative blood test never excludes it. The electroencephalogram is diffusely slow and may show the extreme delta brush pattern; magnetic resonance imaging is often normal, which misleads. Tumour search includes pelvic imaging.

WHAT EARNS THE MARKS

- **The stages given in order,** which is what makes the diagnosis recognisable in a psychiatric ward.
- **Spinal fluid preferred over serum,** stated as a rule with its reason.
- **Tumour search named as treatment,** not merely as investigation.
- **Normal imaging noted as common,** so a clean scan does not close the question.

SEE ALSO Slot IV - 89 PANDAS Slot IV - 92 delirium **SPRINT**
Day 35, Neuropsychiatry of systemic & neurological disease

WEAVE 100 · PAPER IV · F28

AUTOIMMUNE ENCEPHALITIS — WHEN IMMUNE RESPONSES DISRUPT BRAIN SIGNALLING

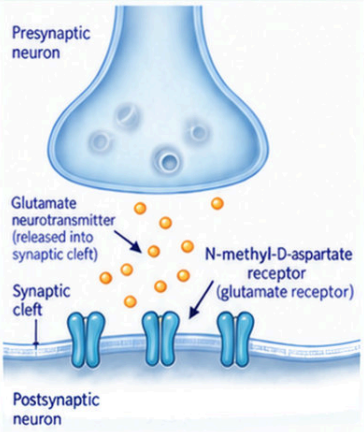
Foundation 28 • Normal function → mechanism → clinical recognition → management

1. NORMAL FUNCTION

Normal: neurons communicate across synapses through neurotransmitters and receptors; excitatory and inhibitory signals are balanced within networks. The N-methyl-D-aspartate receptor is a glutamate receptor involved in synaptic plasticity, learning and memory. Immune tolerance usually prevents damaging responses against self-proteins, while the blood-brain interfaces regulate immune access. These barriers are selective, not absolute walls.

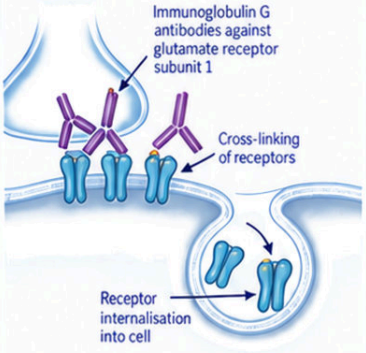
From normal synaptic signalling to antibody-mediated receptor internalisation

A. Healthy glutamate synapse



Normal receptor signalling supports synaptic plasticity, learning and memory.

B. Antibody-linked receptor internalisation



Reduced surface receptor availability leads to impaired signalling (not neuronal destruction in every case).

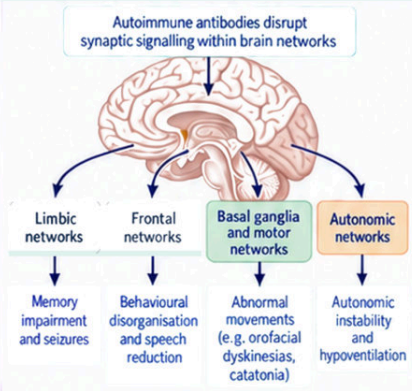
2. WHAT GOES WRONG

What goes wrong: autoimmune encephalitis is inflammatory brain dysfunction caused by an immune response directed against neural targets. Antibodies to neuronal surface or synaptic proteins can alter signalling; immune responses to intracellular targets are often linked to cytotoxic T-lymphocyte injury and malignancy. These categories differ in mechanism, prognosis and treatment response. In anti-N-methyl-D-aspartate receptor encephalitis, immunoglobulin G antibodies against the glutamate receptor subunit 1 reduce surface receptor availability through cross-linking and internalisation. A tumour, notably ovarian teratoma, or preceding herpes simplex encephalitis can be associated; many patients have neither. "All cases are paraneoplastic" is false.

3. MECHANISM → RECOGNITION

Mechanism → recognition: limbic network dysfunction → new memory impairment and seizures; wider synaptic dysfunction → behavioural disorganisation, speech reduction, abnormal movements or reduced consciousness. Anti-N-methyl-D-aspartate receptor disease can include prominent orofacial dyskinesias, catatonia, autonomic instability and hypoventilation. Symptoms may overlap and need not follow a fixed staircase. Leucine-rich glioma-inactivated 1 antibody disease often presents with memory change, seizures and hyponatraemia; brief frequent face–arm dystonic seizures are a useful clue. These associations guide testing but do not diagnose by themselves.

Effects on brain networks and clinical features



4. DIAGNOSTIC CRITERIA

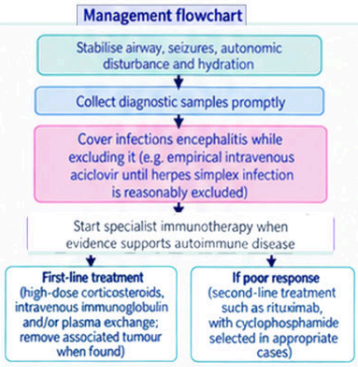
Possible autoimmune encephalitis, consensus framework: subacute progression over less than three months of working-memory deficit, altered mental state or psychiatric symptoms; plus at least one of new focal central nervous system findings, unexplained new seizures, cerebrospinal fluid white cells above 5 per microlitre, or suggestive magnetic resonance imaging; and reasonable exclusion of alternative causes. This is an entry-level clinical category, not confirmed antibody disease. Some antibody-defined syndromes need their own criteria. Isolated chronic psychiatric symptoms or a weak serum antibody result do not establish encephalitis.

5. INVESTIGATIONS

Investigations: urgent neurological assessment; magnetic resonance imaging; electroencephalography to detect slowing, epileptiform activity or nonconvulsive seizures; lumbar puncture for inflammatory indices, infection tests and neural antibodies. Send paired serum and cerebrospinal fluid where appropriate because sensitivity and specificity depend on the antibody. Cerebrospinal fluid is particularly important for anti-N-methyl-D-aspartate receptor antibodies; a negative serum sample cannot exclude the disease. Conversely, normal routine fluid or imaging does not completely exclude autoimmune encephalitis. Extreme delta brush is a supportive electroencephalographic pattern, not a required or universally present finding. Review drugs, metabolic causes, infection and malignancy; tumour screening follows age, sex, syndrome and antibody association. There is no universal diagnostic antibody titre or normal value across laboratories.

6. MANAGEMENT FLOW

Management flow: stabilise airway, seizures, autonomic disturbance and hydration → collect diagnostic samples promptly → cover infectious encephalitis while excluding it → start specialist immunotherapy when evidence supports autoimmune disease. Empirical intravenous aciclovir is commonly required until herpes simplex infection is reasonably excluded. First-line immune treatment generally uses high-dose corticosteroids, intravenous immunoglobulin and/or plasma exchange; remove an associated tumour when found. Poor response may require second-line treatment such as rituximab, with cyclophosphamide selected in appropriate cases. Choices depend on phenotype, infection risk and treatment toxicity, not one rigid universal sequence.



7. SUPPORTIVE TREATMENT

Supportive treatment includes seizure control, thrombosis prevention, nutrition, respiratory support when needed, sleep and rehabilitation. Use sedating or dopamine-blocking medicines cautiously because movement disorders, autonomic instability and severe adverse reactions can complicate care. Monitor infection, blood counts, organ function and treatment-specific risks. Recovery often takes months; repeated cognitive and functional assessment is more meaningful than antibody titres alone. Plan relapse surveillance and coordinated neurological follow-up.

MEMORY AID — **New brain dysfunction needs evidence, exclusions and timely treatment.**

Foundation 28 • Medical foundations • Source-checked supplementary teaching • September 2026.
Laboratory ranges and treatment protocols require local context.

Autoimmune encephalitis. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-90.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P20

P20 — Autoimmune encephalitis: psychiatric symptoms with neurological urgency

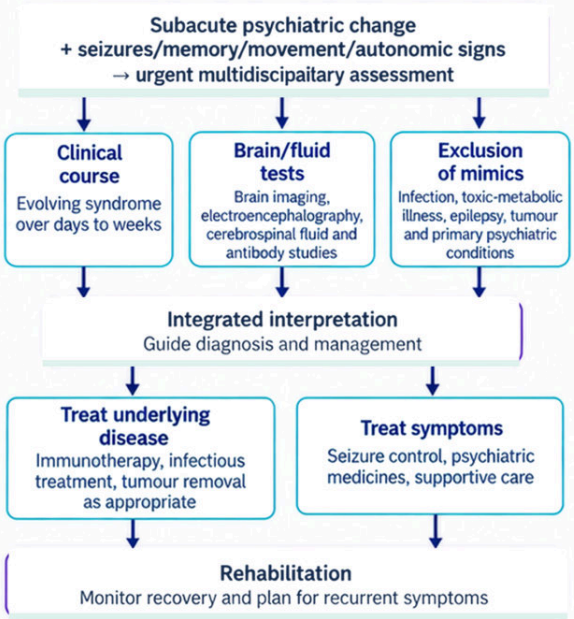
Autoimmune encephalitis can begin with behavioural change, insomnia, anxiety, mood symptoms or psychosis. Recognition depends on the whole evolving syndrome, not on unusual psychiatric content alone. Rapid progression, memory impairment, seizures, movement abnormalities or autonomic disturbance should prompt neurological assessment. Most people with isolated psychiatric symptoms do not have autoimmune encephalitis; indiscriminate antibody testing can produce misleading results.

Identify the red-flag pattern.

Ask about change over days to weeks, impaired short-term memory, fluctuating awareness, speech reduction, catatonia, new seizures, abnormal movements and unexplained fever. Anti-N-methyl-D-aspartate receptor encephalitis may combine psychiatric symptoms with orofacial dyskinesias, reduced consciousness, autonomic instability or hypoventilation. The sequence is variable, and no fixed progression is required. Frequent brief face–arm dystonic events and low sodium can suggest leucine-rich glioma-inactivated 1 antibody disease. These clues guide investigation; they are not individually diagnostic.

Assess safely.

Record vital signs, respiratory status, hydration, nutrition, level of consciousness, attention, memory and neurological findings. Obtain collateral history and a timeline of medicines, infections, substances and previous psychiatric episodes. Distinguish agitation from delirium, pain, seizures or involuntary movement. Catatonia requires active assessment of immobility, mutism, posturing and other features; it can coexist with medical instability. Fever, rigidity and autonomic change after antipsychotic exposure raise concern for a severe drug reaction as well as progression of the underlying illness.



Use criteria without overclaiming.

The consensus possible-autoimmune-encephalitis framework includes subacute psychiatric symptoms, altered mental state or working-memory deficit, plus supportive neurological, cerebrospinal fluid or imaging findings and reasonable exclusion of alternatives. “Possible” is not equivalent to confirmed antibody disease. Infection, toxic-metabolic illness, epilepsy, tumour and primary psychiatric conditions remain part of the differential. Normal routine imaging or cerebrospinal fluid does not exclude every autoimmune syndrome, but a weak serum antibody result does not override an incompatible clinical picture.

Investigations need coordination.

Brain magnetic resonance imaging, electroencephalography and lumbar puncture assess inflammation, seizures and alternative causes. Send paired serum and cerebrospinal fluid antibody studies where appropriate; cerebrospinal fluid is particularly important for anti-N-methyl-D-aspartate receptor testing. Interpret the antibody type, assay, specimen and clinical syndrome together. There is no universal diagnostic titre. Tumour screening follows age, sex, syndrome and antibody association. Urgent empirical treatment for infectious encephalitis, commonly including intravenous aciclovir while herpes simplex infection is evaluated, may be necessary.

Treat the brain disease and the symptoms.

Stabilization, seizure control and specialist immunotherapy are central. Corticosteroids, intravenous immunoglobulin or plasma exchange are common first-line options; further immune treatment and tumour removal depend on the individual case. Psychiatric medicines support safety and comfort but do not replace immune or infectious treatment. Use antipsychotics cautiously because movement disorders, rigidity, sedation and cardiac or autonomic vulnerability can complicate care. Monitor the electrocardiogram when the selected medicine and clinical risks warrant it; the QT interval measures ventricular electrical activation and recovery.

Ongoing care and recovery.

For catatonia, specialist-supervised benzodiazepines or electroconvulsive therapy may be considered within coordinated medical care, with attention to respiratory and autonomic risks. Maintain hydration, nutrition, pressure-area care and thrombosis prevention when immobility is prolonged. Offer orientation, low-stimulation surroundings and family communication. Follow cognition, sleep, mood, seizures and daily function during recovery, which may take months. Antibody titres alone should not determine recovery or relapse. Arrange rehabilitation and a clear plan for recurrent symptoms.

Memory aid: “Rapid change plus brain signs: assess urgently.”
Neither psychosis alone nor an antibody alone establishes the diagnosis.

Supplementary psychiatric teaching • September 2026

Autoimmune encephalitis psychiatric presentation. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-90.

Credits and references: retained within the illustration.

IV - 91

AIDS Dementia Complex. [10]

10 NEURO-INFECTIVE AND AUTOIMMUNE NEUROPSYCHIATRY

KUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2	Definition and naming	The older term, and where it sits in the current scheme
3	Clinical picture	The subcortical triad: cognitive, motor, behavioural
2	Investigation	A diagnosis of exclusion, and what must be excluded
3	Management and course	What antiretroviral therapy changed, and what it did not

THE DEFINITION

A subcortical dementia caused by HIV itself rather than by opportunistic infection. The older name AIDS dementia complex now sits as the most severe tier of HIV-associated neurocognitive disorder, and using the current three-tier scheme while naming the old term is the safer answer.

TIER	COGNITIVE TESTING	EFFECT ON DAILY FUNCTION
Asymptomatic neurocognitive impairment	Impairment in two or more domains on formal testing	None reported or observed
Mild neurocognitive disorder	Impairment in two or more domains	Mild interference with work or daily activity
HIV-associated dementia	**Marked impairment in two or more domains**	**Marked interference; this is the old AIDS dementia complex**

Amber marks the severe tier the question asks about. The three tiers differ by severity of testing impairment and by functional effect, and quoting both axes is what the definition marks are for.

- **The picture is subcortical.** Cognitive: psychomotor slowing, poor attention and concentration, impaired executive function, with memory affected through retrieval rather than storage. Motor: clumsiness, tremor, unsteady gait, hyperreflexia, and later frontal release signs. Behavioural: apathy, social withdrawal and loss of drive, often mistaken for depression. Aphasia and agnosia are absent, which separates it from a cortical dementia.
- **Exclusion, then treatment.** Imaging shows atrophy and diffuse white matter change; spinal fluid is examined mainly to exclude cryptococcal disease, toxoplasmosis, tuberculosis, lymphoma and neurosyphilis. Depression is the commonest and most treatable mimic and is excluded before dementia is diagnosed. Antiretroviral therapy is the treatment: severe dementia has become much less common since it, though milder impairment persists in treated patients.

WHAT EARNS THE MARKS

- **The current three-tier scheme, with the old name placed inside it.**
- **Subcortical named, with the cortical features that are absent.**
- **Depression excluded explicitly** before the dementia is diagnosed.
- **Antiretroviral therapy given as the treatment,** with what it did and did not change.

SEE ALSO Slot IV - 87 neuropsychiatric manifestations of HIV Slot IV - 19 subcortical dementias SPRINT

Day 35, Neuropsychiatry of systemic & neurological disease

WEAVE 100 · PAPER IV · F03

HUMAN IMMUNODEFICIENCY VIRUS: LOSS OF IMMUNE COORDINATION

Foundation 03 • Virus replication → immune damage → clinical consequences

NORMAL DEFENCE

Skin and mucosal barriers, phagocytes, complement and other innate mechanisms provide early defence. Adaptive immunity adds specific recognition and memory.
B lymphocytes make antibodies; cytotoxic T lymphocytes kill infected cells. **Helper T lymphocytes** expressing cluster of differenitation 4 (CD4) coordinate responses through cell interactions and cytokines. A normal immune response needs regulation as well as strength: uncontrolled activation can also injure tissue.

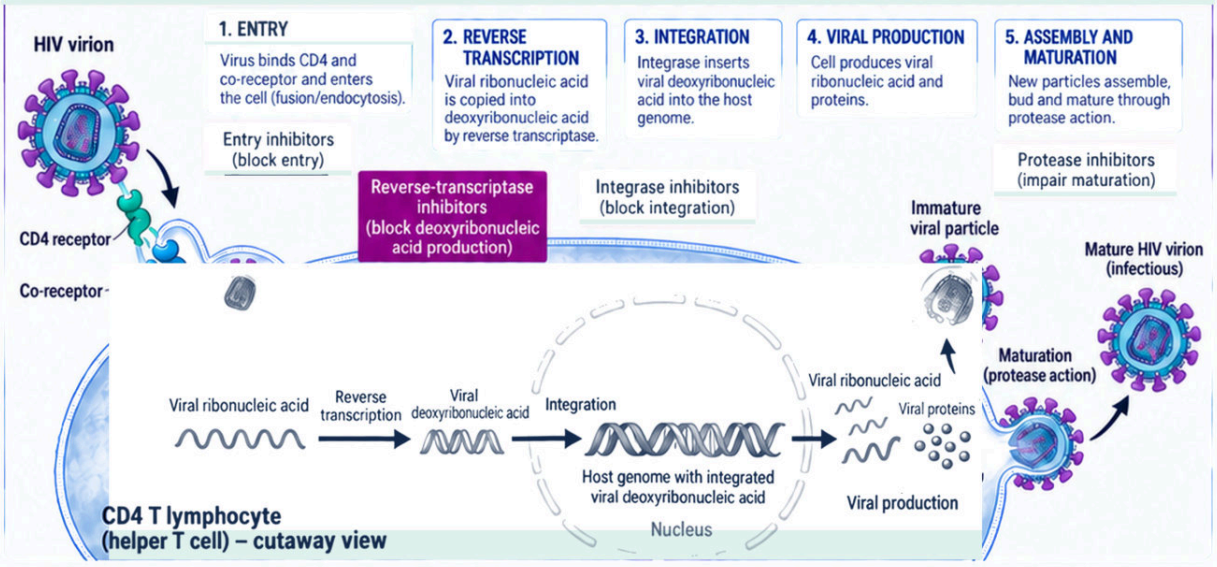
WHAT THE VIRUS DOES

Human immunodeficiency virus (HIV) is a retrovirus. It binds CD4 and a co-receptor, enters the cell and uses reverse transcriptase to copy viral ribonucleic acid into deoxyribonucleic acid. Integrase inserts this deoxyribonucleic acid into the host genome. The cell produces viral components; new particles assemble, bud and mature through protease action. Integrated virus can persist in long-lived reservoirs.

Viral replication, cell loss and chronic immune activation damage the immune system. Falling CD4-cell numbers and function weaken defence against particular infections and cancers. Treatment suppresses replication, permits immune recovery and improves survival; it does not routinely eliminate the reservoir.

DIAGRAM: THE TREATMENT TARGETS

Entry → reverse transcription → integration → viral production → assembly and maturation. Combination treatment attacks more than one step and reduces emergence of resistance.



FROM VIRUS REPLICATION TO IMMUNE DAMAGE

CD4-cell damage
- Direct viral killing
- Immune-mediated injury
- Chronic immune activation
- Apoptosis and loss of function

→

Weakened coordinated immunity
- Falling CD4-cell numbers and function
- Impaired cytokine responses
- Reduced help for B lymphocytes and cytotoxic T lymphocytes

→

Opportunistic infection and cancer
- Increased risk of particular infections (e.g. *Pneumocystis* pneumonia, tuberculosis, fungal and viral infections)
- Higher risk of certain cancers (e.g. Kaposi sarcoma, lymphomas)

THE CLINICAL COURSE

Acute infection may cause fever, sore throat, rash and lymph-node enlargement as viraemia and immune activation rise; it may also be unrecognised. Untreated chronic infection can remain clinically quiet while immune damage continues. Advanced immunodeficiency permits opportunistic disease.

Persistent fever, weight loss, diarrhoea or oral thrush may signal progressive disease. Pneumocystis pneumonia causes breathlessness and hypoxaemia; tuberculosis may occur at any CD4 count. Severe headache, focal neurological signs or altered consciousness demands assessment for central nervous system infection or other pathology. These symptoms do not diagnose HIV by themselves.

TWO NUMBERS, DIFFERENT QUESTIONS

CD4 count: immune reserve, reported as cells/microlitre; one microlitre equals one cubic millimiitre. Adult reference intervals are laboratory-dependent, often roughly 500–1,500 cells/microlitre. Trends and clinical context matter.

Viral load: circulating HIV ribonucleic acid, reported as copies/mL; measures replication and treatment response. Assay detection limits vary. 'Undetectable' is not absence of all virus.

An acquired immunodeficiency syndrome diagnosis follows an acquired immunodeficiency syndrome–defining illness or severe immune suppression, commonly CD4 below 200 cells/microlitre. This is not the threshold for starting treatment: everyone with confirmed HIV should be offered antiretroviral therapy.

DIAGNOSE AND ASSESS

Use a validated national testing algorithm with appropriate confirmatory testing; one reactive screening result is not the whole diagnosis. Antigen–antibody testing detects infection earlier than antibody-only testing. Very recent infection can fall within a test's window period, suspected acute infection may require HIV ribonucleic acid testing.

At diagnosis assess viral load, CD4 count, tuberculosis and hepatitis, other infections, pregnancy where relevant, renal/liver function, medicines and resistance as indicated. Choose infection prophylaxis according to CD4 count, exposure, serology and current protocols—not one rule for every infection.

BASIC MANAGEMENT AND PREVENTION

Start an appropriate combination regimen promptly and support sustained adherence. Review interactions, toxicity and response. Timing needs specialist adjustment with certain central nervous system opportunistic infections. Treat infections, provide indicated vaccines and prevention, and address access barriers.

Sustained viral suppression below 200 copies/mL prevents sexual transmission: undetectable equals untransmittable for sex. It does not imply cure or identical risk for every transmission route. Safe injecting practices and pre-/post-exposure prophylaxis are separate prevention tools.

MEMORY AID

Cell count = immune reserve; viral load = replication.

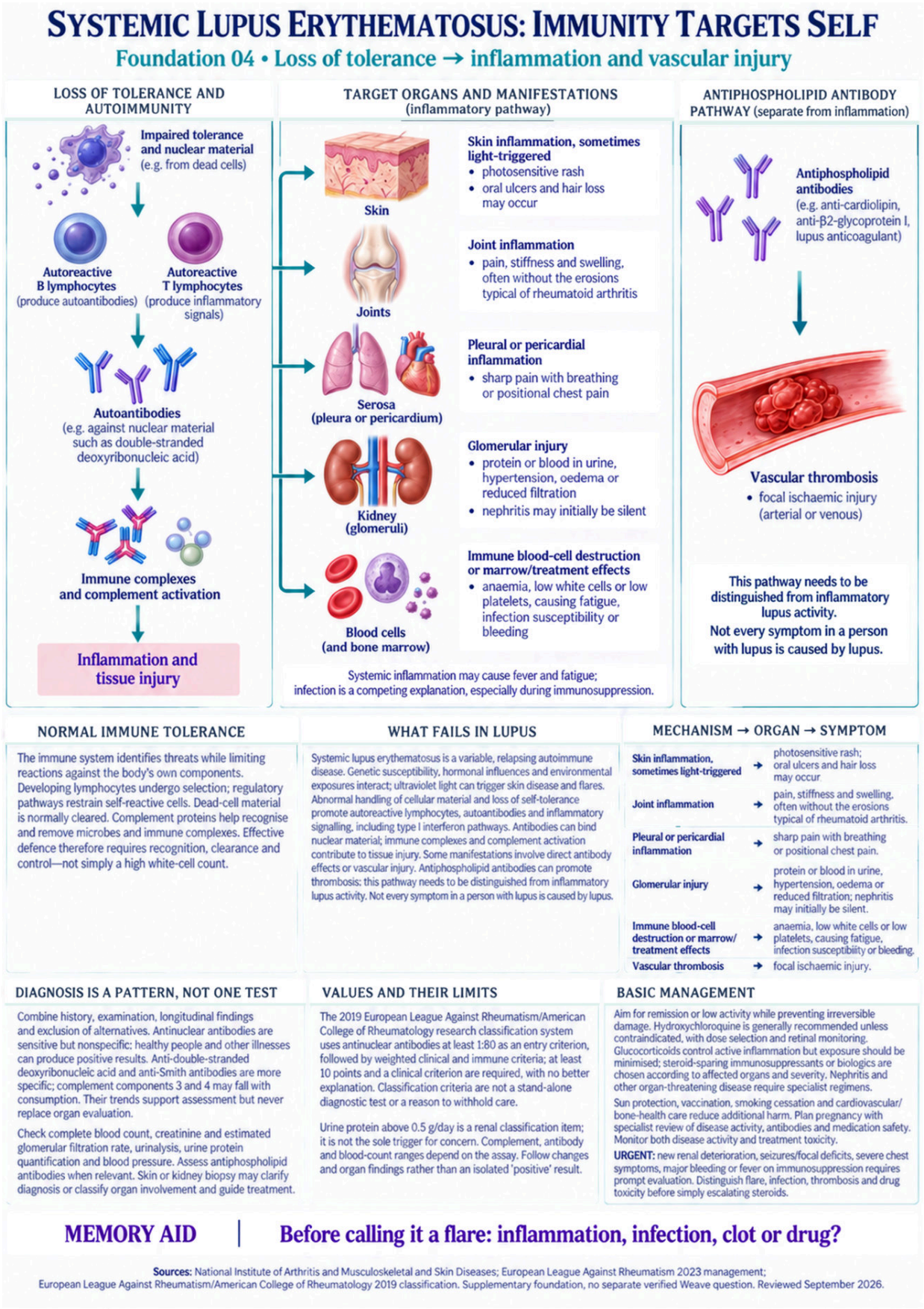
Sources: National Institutes of Health—life cycle, treatment and monitoring; Centers for Disease Control and Prevention—testing and sexual transmission; World Health Organization—immune cell ranges. Supplementary foundation linked to original Paper IV Question 87, page 121.

Reviewed September 2026. **Foundation03.**

Human immunodeficiency virus and immune dysfunction. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neuro-infective and autoimmune neuropsychiatry.

Credits and references: retained within the illustration.

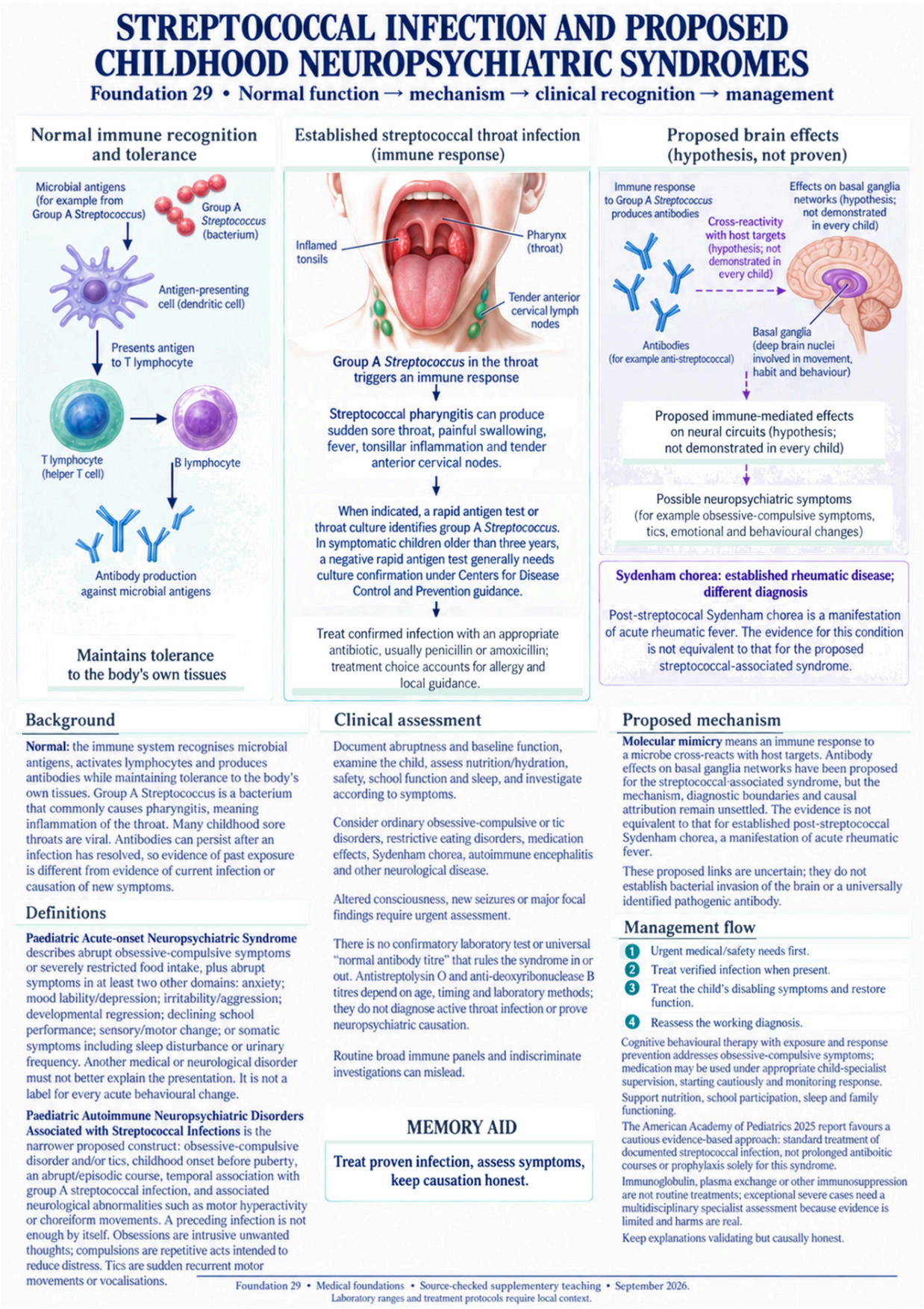
WEAVE 100 · PAPER IV · F04



Systemic lupus erythematosus. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neuro-infective and autoimmune neuropsychiatry.

Credits and references: retained within the illustration.

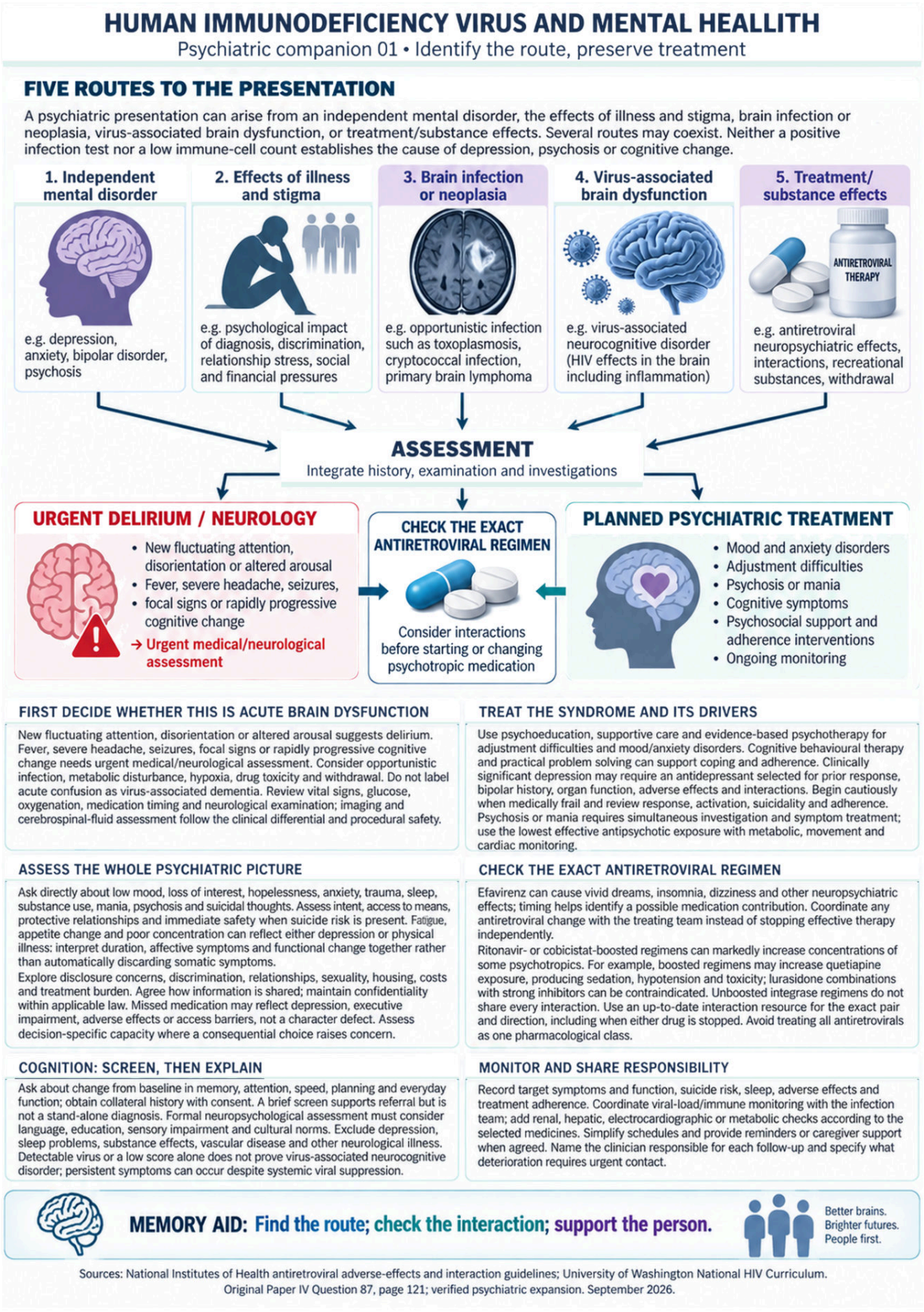
WEAVE 100 · PAPER IV · F29



Streptococcal infection and proposed childhood neuropsychiatric syndromes. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neuro-infective and autoimmune neuropsychiatry.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P01



Human immunodeficiency virus psychiatric companion. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neuro-infective and autoimmune neuropsychiatry.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P02

LUPUS AND PSYCHIATRIC PRESENTATIONS

Psychiatric companion 02 • Attribution determines treatment

Brain minds lives in context

ONE DISEASE, SEVERAL EXPLANATIONS

Depression, anxiety, cognitive complaints, psychosis and acute confusion can occur in someone with systemic lupus erythematosus. They may reflect an independent psychiatric disorder, distress and disability, inflammatory nervous-system disease, vascular injury, infection or treatment toxicity. **Neuropsychiatric lupus is an attribution decision after assessment, not a synonym for every symptom in a person with lupus.**

START AS YOU WOULD WITHOUT LUPUS

Clarify onset, fluctuation, prior episodes, baseline cognition and functional change. Examine attention, arousal, orientation, mood, psychosis, neurological signs and suicide risk. Obtain collateral information and a complete timeline of lupus activity, infections, glucocorticoid exposure and other medicines. Acute confusion, seizures, focal deficits, meningism, severe headache or reduced consciousness needs urgent medical/neurological evaluation.

Assess glucose, oxygenation, electrolytes, kidney/liver function and blood count as indicated. Review pain, sleep, anaemia, substance use and medication effects. Infection is particularly important during immunosuppression. A familiar psychiatric diagnosis must not prevent assessment of a new neurological syndrome.

ASSESSMENT FLOW FROM NEW SYMPTOMS TO JOINT CARE

New psychiatric or neurological symptoms
Depression, anxiety, cognitive complaints, psychosis or acute confusion

Inflammation



- Selected acute confusional, psychotic, seizure or other neurological syndromes may reflect immune-mediated injury.

Vascular injury



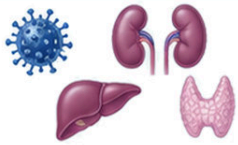
- Focal vascular events related to antiphospholipid-associated thrombosis require mechanism-specific antithrombotic assessment.

Treatment effect



- Glucocorticoids and other medicines can cause psychiatric or cognitive symptoms.

Other illness



- Independent depression, anxiety, infection, metabolic illness and medication adverse effects require their own treatments.

Exclude infection and metabolic causes
Assess for infection, glucose, oxygenation, electrolytes, kidney/liver function, blood count, substance use and medication effects.

Rheumatology Neurology Psychiatry

Reconvene for joint care and attribution
Integrate findings from all pathways. More than one explanation can be active simultaneously.

Individualised treatment and follow-up

DISTINGUISH THREE TREATMENT PATHWAYS

Inflammatory disease

- Selected acute confusional, psychotic, seizure or other neurological syndromes may reflect immune-mediated injury.
- Rheumatology/neurology assesses the clinical pattern, systemic activity and alternatives; glucocorticoids and immunosuppressive treatment may be indicated when inflammatory attribution is convincing.

Thrombotic disease

- Focal vascular events related to antiphospholipid-associated thrombosis require mechanism-specific antithrombotic assessment.
- Do not treat a clot as if it were solely inflammation.
- Antibody positivity without a relevant clinical event does not automatically require the same treatment as established thrombosis.

Other causes

- Independent depression, anxiety, infection, metabolic illness and medication adverse effects require their own treatments.
- More than one pathway can be active simultaneously.

TESTS SUPPORT, BUT DO NOT SETTLE, ATTRIBUTION

- Brain magnetic resonance imaging may identify vascular, inflammatory or alternative pathology; a normal scan does not exclude every neuropsychiatric syndrome.
- Electroencephalography helps assess seizures or unexplained altered awareness.
- Cerebrospinal-fluid testing is often directed toward excluding infection and other diagnoses.
- Neuropsychological assessment characterises persistent cognitive impairment with attention to language, education, mood and fatigue.
- Complement and anti-double-stranded deoxyribonucleic acid antibody trends can support systemic disease assessment but cannot alone prove that a psychiatric symptom is inflammatory lupus.
- There is no single universally diagnostic blood or cerebrospinal-fluid marker for all neuropsychiatric lupus.

GLUCOCORTICOIDS: TREATMENT AND POƎHIIBLE CAUSE



- Glucocorticoids can produce insomnia, activation, mood change, mania, depression, psychosis or delirium.
- A recent initiation or increase strengthens suspicion but does not exclude simultaneous inflammatory disease.
- Review dose and timing with the treating team.
- When feasible, coordinated dose reduction or a steroid-sparing strategy may help; abrupt unsupervised cessation risks adrenal and disease complications.
- Severe symptoms may need short-term psychiatric medication and a monitored setting while causation is clarified.

PSYCHIATRIC CARE CONTINUES ALONGSIDE DISEASE CARE

- Use clear explanation, supportive therapy and cognitive behavioural approaches for distress, depression or anxiety, with rehabilitation and pacing for cognitive/fatigue difficulites.
- Treat syndromal depression or psychosis according to severity, prior response and medical context.
- Screen for bipolar disorder before antidepressant treatment.
- Consider kidney involvement, thrombocytopenia, anticoagulants, seizure risk and interacting medicines when prescribing.
- Avoid unnecessary anticholinergic or sedative burden in cognitive impairment.

MONITOR BENEFIT AND HARM

- Track mood, cognition, sleep, function, suicidality and adverse effects rather than laboratory results alone.
- Antipsychotic treatment may require electrocardiography, electrolyte, movement and metabolic checks, especially with other cardiac-risk medicines.
- Coordinate infection, blood-count and organ monitoring for immunosuppression.
- Review symptoms after treatment changes and name follow-up responsibility.
- Improvement after immunotherapy can support a formulation but is not proof by itself.

MEMORY AID: Consider inflammation, clot, medicine and independent illness.

Sources: European League Against Rheumatism neuropsychiatric recommendations and 2023 lupus update; National Institute of Arthritis and Meuculoskkeltal and Skin Diseases; Medicines and Healthcare products Regulatory Agency corticosteroid safety. Supplementary companion, September 2026.

Systemic lupus neuropsychiatry. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neuro-infective and autoimmune neuropsychiatry.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · P19

P19 — Tuberculosis: mood, brain infection and treatment interactions

Psychiatric symptoms during tuberculosis have several possible pathways: the burden of illness and stigma, a coexisting mental disorder, direct central nervous system disease, metabolic disturbance or treatment adverse effects. These pathways can overlap. Depression is not an inevitable reaction to infection, and new psychosis should not automatically be attributed to an antibiotic. Establish the timing and examine the patient before deciding which explanation fits.

Start with function and safety.

Ask about low mood, loss of interest, hopelessness, anxiety, sleep, substance use and suicidal thinking. Distinguish persistent depressive symptoms from fatigue, poor appetite and weight loss due to infection. Assess housing, food security, financial strain, isolation, family support and barriers to attending care. Confidential, nonjudgemental conversations can uncover why doses are missed. Avoid labelling all adherence difficulty as lack of insight: cost, nausea, cognitive problems or fear may be the immediate cause.

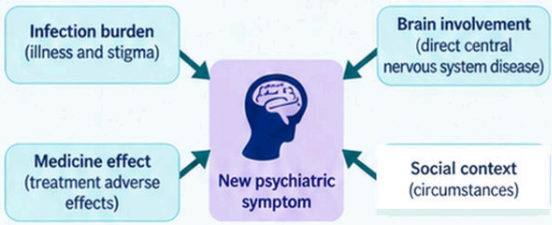
Recognize meningitis and delirium.

Subacute headache, fever, irritability or apathy followed by confusion, vomiting, cranial nerve abnormalities, seizures or reduced consciousness raises concern for tuberculous meningitis. Neck stiffness may be absent. Fluctuating attention suggests delirium and requires urgent investigation for infection, hypoxia, glucose or electrolyte disturbance, organ dysfunction, withdrawal and medicines. New focal deficits or deteriorating consciousness need emergency neurological assessment. A psychiatric ward label must not delay medical transfer when these signs appear.

Investigate the suspected mechanism.

Review the exact tuberculosis regimen, resistance status, dates of initiation and all prescribed, nonprescribed and herbal medicines. Examine cognition and neurological function. Medical testing may include glucose, oxygenation, electrolytes, liver and kidney function, brain imaging and cerebrospinal fluid assessment when safe. Negative rapid molecular testing does not reliably exclude tuberculous meningitis. If suspicion is high, specialist treatment should not wait for slow culture results. Do not use a positive skin test or interferon-gamma release assay as proof of brain infection.

Pathways to new psychiatric symptoms



Rifampicin interaction (drug-dependent)



Know the interaction directions.

Rifampicin induces drug-metabolizing pathways and can lower exposure to several antidepressants, antipsychotics, anticonvulsants and methadone. Loss of psychiatric control may follow introduction; toxicity can emerge after rifampicin is stopped if an increased psychotropic dose is not reviewed. The effect is drug-specific and changes over time. Check a current interaction reference and monitor clinically, with drug levels when appropriate, rather than applying one dose multiplier to every medicine.

Isoniazid can cause neurotoxicity, including neuropathy and uncommon psychiatric symptoms; pyridoxine supplementation follows risk and regimen guidance. Cycloserine, used in selected drug-resistant regimens, can cause depression, psychosis or seizures. Fluoroquinolones and corticosteroids can also contribute to psychiatric symptoms. Linezolid inhibits monoamine oxidase and can interact with serotonergic medicines; review the combination urgently with the tuberculosis team and pharmacist. Serotonin toxicity may include agitation, sweating, diarrhoea, tremor, clonus and fever. Do not independently stop essential tuberculosis therapy or abruptly change psychiatric medicines without a coordinated plan.

Treat and monitor together.

Provide evidence-based depression or anxiety care, adapted psychological support and practical adherence assistance. Choose medicines considering hepatic injury, renal function, sedation, seizure risk and interactions. For acute psychosis or suicidal risk, arrange prompt specialist care and reassess possible drug toxicity. Record a baseline mental state before neurotoxic regimens when feasible, then ask about symptoms at follow-up. Include family with consent and provide clear instructions for urgent deterioration. After meningitis, assess cognition, hearing, vision and rehabilitation needs. Successful care includes infection control and restoration of daily functioning.

Memory aid: "Illness, brain, medicines, circumstances: check each pathway."

Supplementary psychiatric teaching • September2026.

Tuberculosis neuropsychiatry. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neuro-infective and autoimmune neuropsychiatry.

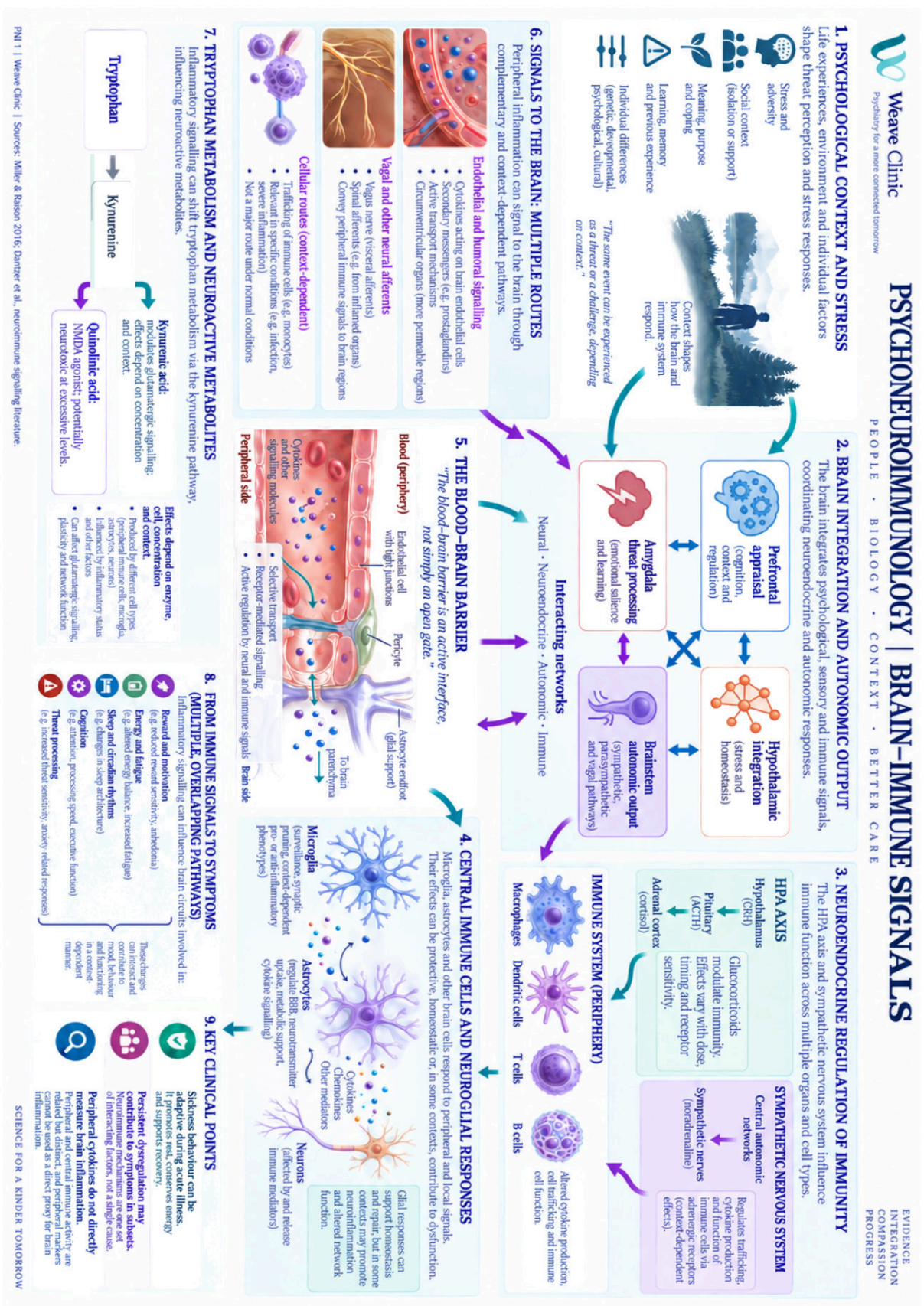
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WEAVE 100 · PAPER IV · P21



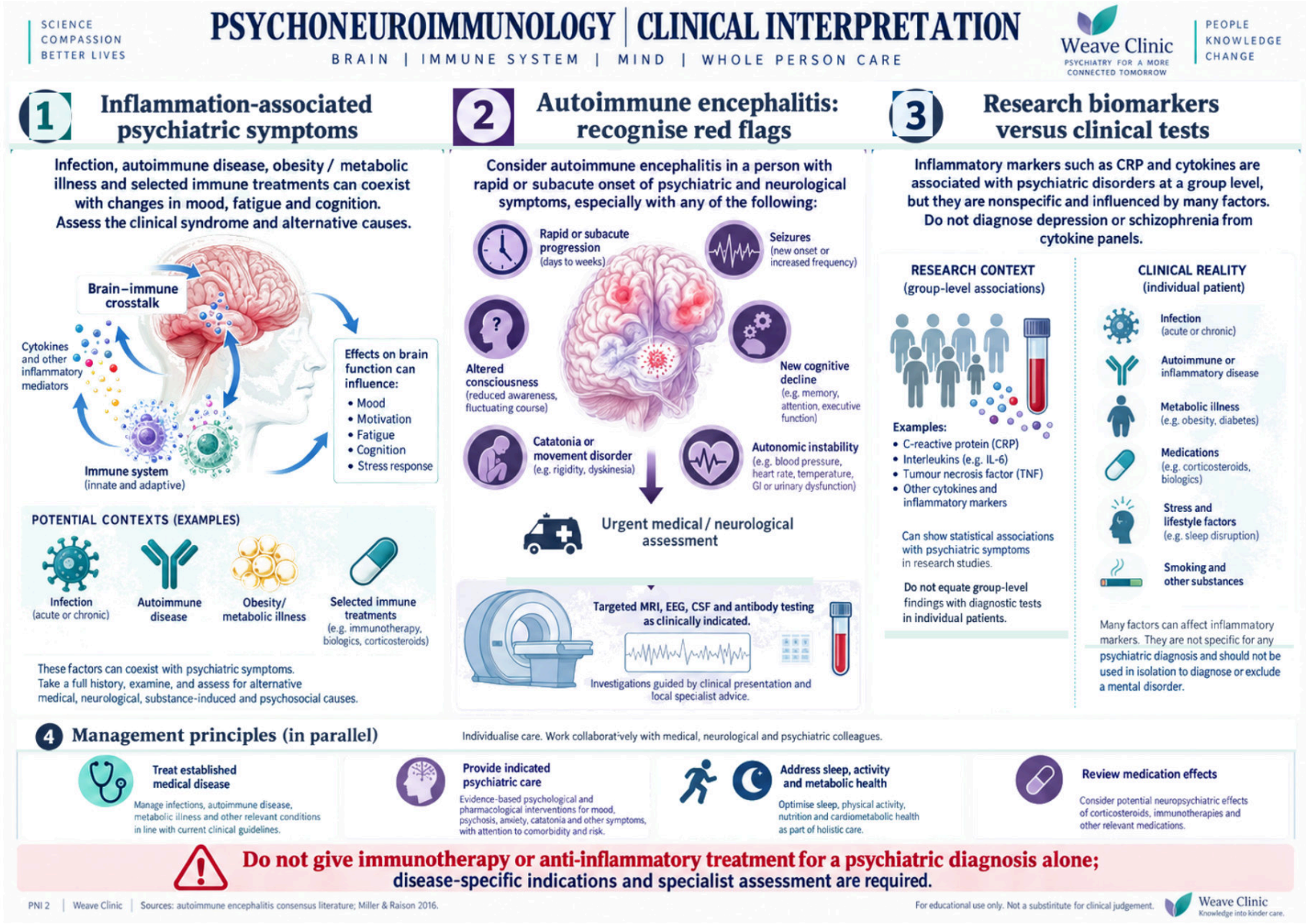
Streptococcal-associated childhood neuropsychiatric care. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neuro-infective and autoimmune neuropsychiatry.

Credits and references: retained within the illustration.



Psychoneuroimmunology - PNI-1. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Neuro-infective and autoimmune neuropsychiatry.

Credits and references: retained within the illustration.



Psychoneuroimmunology - PNI-2. Use the visual organisation to recall the key distinctions and structure your answer.
Section companion: Neuro-infective and autoimmune neuropsychiatry.

Credits and references: retained within the illustration.

AREA 14 OF 15 · P4-A08

Delirium and amnestic syndromes

5	3.9%	IV-92	IV-96
SLOTS IN THIS BOOK	SHARE OF THE HUNDRED	FIRST SLOT	LAST SLOT

ANCHOR 1 RECURRENT 1 SINGLE 3

REVISION ORDER

The three delirium slots together and in that order: define it, screen for it, then examine. Inattention is the core feature on all three and all three stems require it. The two amnestic slots next, they are one answer split by axis and both fragments declare the split.

WHAT IS IN THIS AREA

Delirium in three slots: the definition with the causes and the management, the screening instruments, and the examination of cognitive function at the bedside. The amnestic syndromes in two, one asking for the disorder itself and one for the criteria and the causes sorted by mechanism.

IV - 92

Define delirium. What are the common causes of delirium? Discuss the management of delirium. [2+2+6]

2 + 2 + 6 | DELIRIUM AND AMNESTIC SYNDROMES

DNB, KUHS, TN-MGRMU - 4 APPEARANCES, 4 OF 78 SESSIONS

SKELETON

2 Definition	Attention first, then the acute onset and the fluctuation
2 Causes	Grouped so that they can actually be searched
5 Management	Cause, then environment, then a drug if forced
1 Outcome	Why it is never a benign episode

THE DEFINITION

An acute, fluctuating disturbance of attention and awareness, with an additional disturbance of cognition, developing over hours to days, and caused by a medical condition, a substance or withdrawal. Inattention is the core feature; the disturbed conscious level is what separates it from dementia.

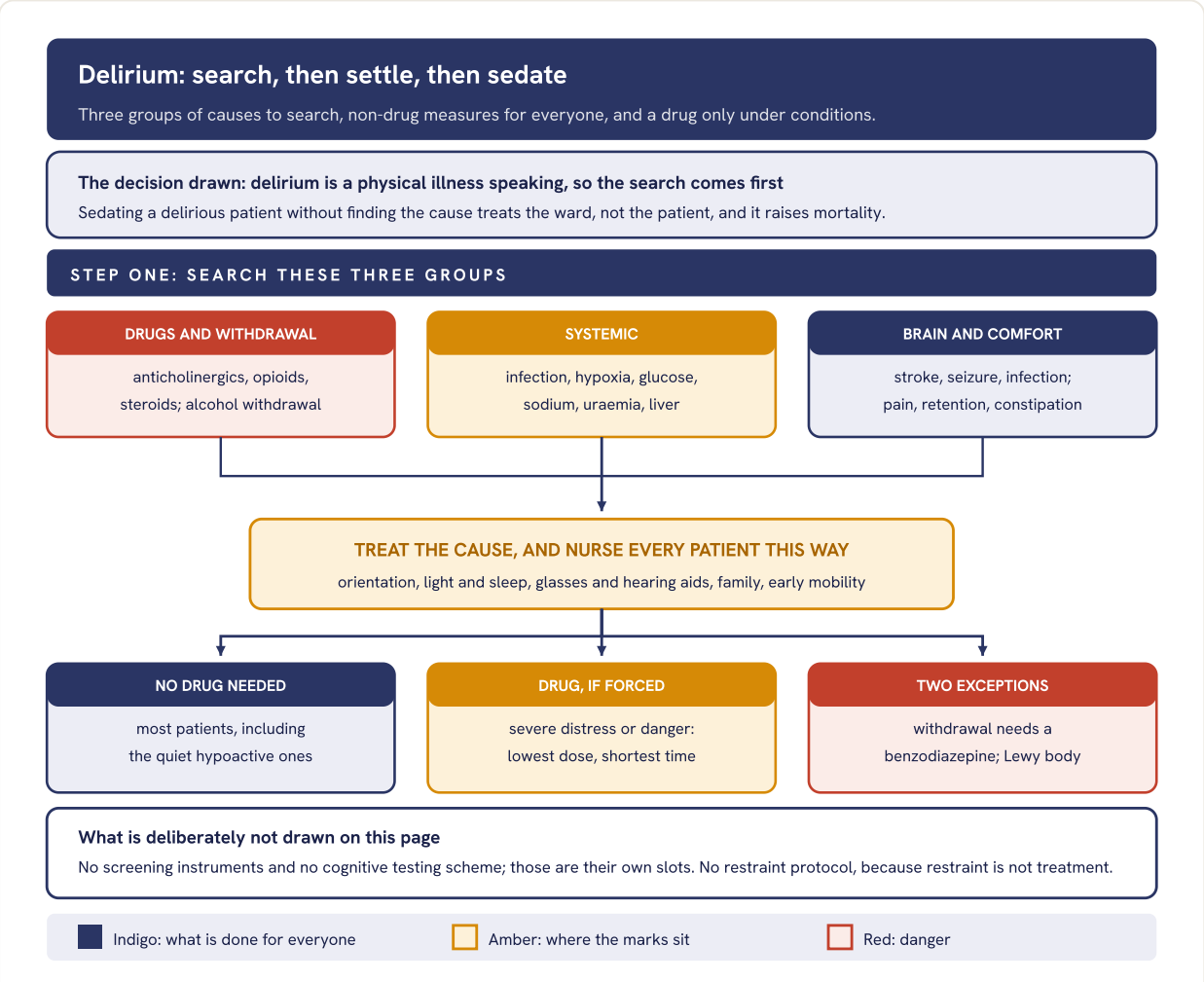


Fig 092.1 Three groups of causes to search, the non-drug care that every delirious patient receives, and the narrow conditions under which a drug is added.

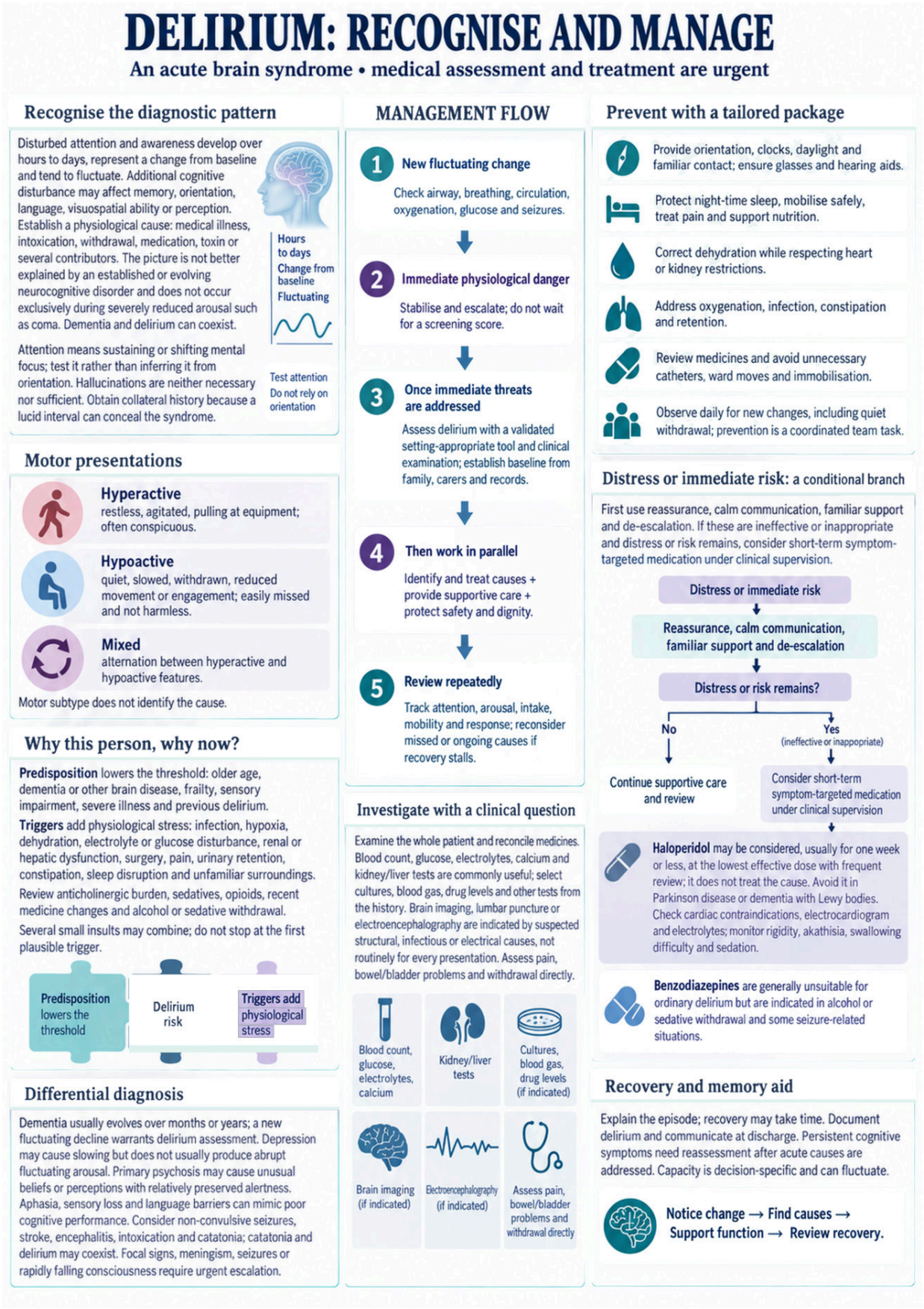
- **If a drug is unavoidable.** A low dose antipsychotic, for the shortest possible time, reviewed daily. Haloperidol in small doses is the usual choice. In Parkinson disease and Lewy body dementia antipsychotics can be dangerous, and quetiapine or clozapine are preferred. Benzodiazepines are first line only in alcohol or sedative withdrawal.
- **The one most often missed.** Hypoactive delirium is commoner than the agitated form, is mistaken for depression or for tiredness, and carries the worse outcome. A quiet patient who is inattentive is delirious until proven otherwise.

WHAT EARNS THE MARKS

- **Inattention named as the core feature** in the opening definition.
- **Causes grouped, not listed,** so the answer reads as a search strategy.
- **Non-drug measures written before any drug,** and given to every patient.
- **The hypoactive subtype named,** with the fact that it does worse.

SEE ALSO Slot IV - 94 screening assessment in delirium Slot IV - 95 cognitive assessment in delirium **SPRINT** Day 34, Stroke, TBI, delirium & focal brain syndromes

WEAVE 100 · PAPER IV · Y02



Delirium recognition and management. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-92.

Credits and references: retained within the illustration.

IV - 93

Amnestic disorder. [10]

10 DELIRIUM AND AMNESTIC SYNDROMES RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	Definition	One domain lost, everything else standing
2	The anatomy	Two circuits, and why either one produces it
4	Clinical picture	What is lost, what is spared, and confabulation
2	Management	Treat the cause, then rehabilitate around the deficit

THE DEFINITION

An acquired impairment of memory severe enough to disrupt daily function, occurring in clear consciousness and without the general cognitive decline of dementia. Anterograde memory carries the damage; the isolation of the deficit is what defines the syndrome.

- **The anatomy.** Two circuits, and damage to either produces the same syndrome. The medial temporal route through hippocampus, fornix and mammillary bodies, and the diencephalic route through the mammillary bodies and dorsomedial thalamus. Both are links in the Papez circuit, which is why bilateral damage anywhere along it is enough.
- **Lost and spared.** Lost: new learning, so the patient cannot lay down day to day memory, with retrograde amnesia that is worst for the period nearest the injury. Spared: immediate recall and digit span, procedural and skill memory, language, perception and, at least early, social manner. That preserved surface is why families and wards underestimate the disability.
- **Confabulation.** Filling gaps with plausible or fantastic material, not deliberately, and it is most marked early and where frontal damage accompanies the amnesia. Correcting the patient repeatedly is futile and distressing; it fades as the syndrome stabilises.

PITFALL

Clear consciousness separates this from delirium, and the intact non-memory domains separate it from dementia. Test attention explicitly before concluding amnesia: an inattentive patient cannot register anything, so their apparent memory failure is delirium wearing an amnestic mask.

WHAT EARNS THE MARKS

- **The deficit named as isolated,** with the spared domains listed as evidence.
- **Both anatomical routes,** not the hippocampus alone.
- **Confabulation described without moral language.**
- **Attention tested first,** which is the line that rules out delirium.

SEE ALSO Slot IV - 96 criteria and causes of amnesic syndromes Slot IV - 83 thiamine deficiency SPRINT Day 34, Stroke, TBI, delirium & focal brain syndromes

WEAVE 100 · PAPER IV · Y04

AMNESTIC SYNDROMES: WHEN NEW EXPERIENCE DOES NOT STAY

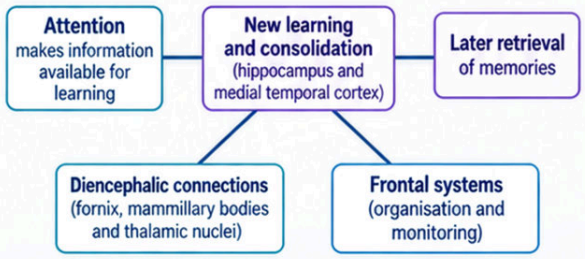
Identify the memory profile, time course and treatable cause

MEMORY IS A NETWORK, NOT A SINGLE STORE

Attention makes information available for learning. Medial temporal structures, including the hippocampus and surrounding cortex, help establish new declarative memories: events and facts. Connections through the fornix, mammillary bodies and thalamic nuclei form part of an extended memory network. Frontal systems help organise retrieval and check whether a remembered account fits its context. Damage to different components can produce overlapping profiles; a bedside pattern does not precisely locate a lesion.

Working memory holds information briefly; procedural learning supports skills. These can be relatively preserved despite severe difficulty retaining new events. Fluent conversation or intact digit span therefore does not establish intact everyday memory.

A CONCEPTUAL MEMORY NETWORK



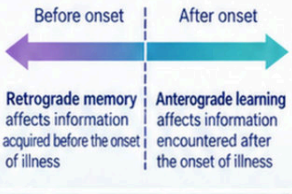
Functional relationships, not a complete anatomical circuit

DEFINE WHAT IS LOST

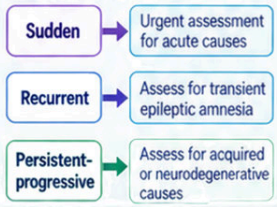
Anterograde amnesia is impaired learning or retention of information encountered after the onset of illness. **Retrograde amnesia** affects information acquired before it. Recent past events may be more vulnerable than remote memories, but the pattern varies with cause. Ask separately about personal events, factual knowledge and learned skills.

An ammestic syndrome describes disproportionate memory impairment affecting function, often with relatively preserved alertness and other abilities. It is not necessarily a completely isolated deficit: executive dysfunction and other impairments may coexist. Establish whether attention is sufficiently stable to interpret memory testing. Delirium can prevent effective registration and imitate severe amnesia.

TIME RELATIVE TO ONSET



CLINICAL FORK AND FOCUS



RECOGNISE THE TIME COURSE

Sudden amnesia requires urgent assessment for stroke, seizures, hypoglycaemia, intoxication, head injury or encephalitis. Fever, seizures, reduced consciousness or focal neurological signs increase urgency. Hypoxic injury and damage vulnerable hippocampal networks after cardiac arrest or respiratory compromise.

Transient global amnesia causes abrupt, prominent difficulty retaining new information with repetitive questioning, preserved personal identity and no explanatory focal deficit. It resolves within 24 hours. Diagnose clinically after excluding concerning alternatives; persistent symptoms or atypical features require another explanation. Small hippocampal diffusion lesions may be most apparent on magnetic resonance imaging after 24–72 hours, but urgent assessment must not wait for that window.

Recurrent brief amnestic spells, often on waking, suggest transient epileptic amnesia, especially with olfactory experiences, automatisms or other seizure evidence. Electroencephalography, sometimes including sleep, and specialist assessment are appropriate; a normal routine recording does not exclude it.

PERSISTENT SYNDROMES AND CONFABULATION

Thiamine deficiency may leave a Korsakoff amnestic syndrome after acute Wernicke encephalopathy. Other causes include temporal or thalamic vascular injury, herpes simplex or autoimmune encephalitis, trauma and medication or substance effects. Progressive decline raises neurodegeneration rather than a static acquired lesion. Review sedatives, alcohol exposure, nutritional risk and the exact onset relative to treatment.

Confabulation is an inaccurate memory account produced without deliberate deception. It may reflect faulty retrieval, source monitoring or temporal context. It is neither universal nor required for an amnestic syndrome. Verify important information with collateral history and records, respond calmly and use orientation supports; repeated confrontational correction may increase distress.

ASSESS MEMORY IN CONTEXT

Document baseline function and collateral examples of missed appointments, repeated conversations, medication errors or getting lost. Test attention, initial registration, learning across trials, delayed recall and recognition; examine language, executive and visuospatial abilities. Compare verbal and visual material where appropriate. Cueing patterns help describe the profile but are not a stand-alone diagnosis. Account for hearing, language, education, mood, fatigue and effort.

Use targeted metabolic and nutritional tests, brain imaging and seizure or inflammatory investigations according to presentation. Neuropsychological assessment clarifies persistent impairment and rehabilitation needs. Depression and functional or dissociative symptoms can impair memory; dissociative amnesia often emphasises autobiographical gaps, but diagnosis requires positive clinical assessment, not simply normal imaging.

TREAT CAUSES AND SUPPORT FUNCTION

Give urgent parenteral thiamine when Wernicke encephalopathy is suspected; do not await a blood level or delay emergency glucose.
Treat seizures, infection, inflammation or vascular disease through the appropriate pathway. Reduce contributory medicines when safe.
Use consistent routines, external reminders, supported learning and occupational rehabilitation. Assess decision-specific capacity, home safety and caregiver needs, then review actual daily function over time.

MEMORY AID | **Attention first; new learning, old memories, cause and daily function.**

Y04 • Supplementary clinical teaching.

Amnestic syndromes. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-93.

Credits and references: retained within the illustration.

IV - 94

Screening assessment in Delirium. [10]

10 DELIRIUM AND AMNESTIC SYNDROMES RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Why screen at all	It is missed in most cases unless it is looked for
2 Who, and when	The at-risk groups, and the daily rhythm of screening
4 The instruments	What each one is for, and who can administer it
2 After a positive screen	A screen is not a diagnosis; what follows it

INSTRUMENT	WHAT IT DOES	WHO USES IT	SETTING
Confusion Assessment Method	Four features: acute fluctuating course, inattention, disorganised thinking, altered consciousness	Trained clinician	General ward, the reference standard
4AT	Alertness, orientation, attention by months backward, acute change; needs no training	Any staff member	Rapid bedside and admission screening
CAM-ICU	The same four features adapted for the ventilated, non-speaking patient	Intensive care staff	Critical care
Nursing Delirium Screening Scale	Observational, scored across a nursing shift	Nursing staff	Repeated observation over time
Delirium Rating Scale, revised	Grades severity rather than presence	Clinician	Monitoring response, and research

Amber marks the two that answer most questions. The Confusion Assessment Method requires features one and two to be present, plus either three or four; the 4AT is the practical instrument where no one has been trained.

- **Who and when.** Screen everyone over sixty five, everyone with dementia or prior delirium, everyone admitted with hip fracture or acute severe illness, and all intensive care patients. Screen on admission and then at least daily, because the disorder fluctuates and a single normal screen means very little.
- **A screen is not a diagnosis.** A positive screen triggers a diagnostic assessment against the full criteria, a search for the cause, and a review of the drug chart. A negative screen in a patient the nurses say is not himself is a reason to screen again, not a reason to stop.

WHAT EARNS THE MARKS

- **The instruments named with what each one is for,** rather than as a list of acronyms.
- **The rule for the Confusion Assessment Method,** which features must be present.
- **Daily repetition justified by fluctuation.**
- **The line separating a screen from a diagnosis.**

SEE ALSO Slot IV - 92 definition, causes and management of delirium Slot IV - 95 cognitive assessment in delirium

SPRINT Day 34, Stroke, TBI, delirium & focal brain syndromes

WEAVE 100 · PAPER IV · Y03

DELIRIUM: BEDSIDE ASSESSMENT

Establish change • test attention • interpret in context • repeat



RECOGNISE
ASSESS
TREAT
IMPROVE OUTCOMES

PREPARE BEFORE SCORING

Check physiological stability and arousal first. Obtain baseline function and timing from records, nurses, family or carers. Provide hearing aids, glasses, an interpreter and a quiet approach; record sedation, aphasia and motor limitations. A tool supports clinical diagnosis; no score identifies the cause. Reassess because delirium fluctuates.

Glasses · Hearing aids · Interpreter · Quiet approach

PRACTICAL ATTENTION EXAMINATION

Ask for months backwards from December; observe losing the task, distractibility and need for repeated prompting. Digit span and a simple target-letter response can add information, but are not interchangeable scoring systems. Orientation questions test a different domain: knowing the date does not establish intact attention. Record the task, actual performance and barriers rather than writing only "confused".

4AT: FOUR-ITEM ASSESSMENT TEST

Total range: zero to twelve. Use the official form and validated language version.

Item	How to assess	Points
Alertness	Normal throughout, including brief sleepiness under ten seconds after waking	0
	Clearly abnormal drowsiness or agitation	4
Abbreviated Mental Test–4	Ask age, birth date, current location and current year: no errors / one error / at least two errors or untestable	0 / 1 / 2
Attention	Months backwards from December: at least seven correct / starts but fewer than seven, or declines / cannot engage because too unwell, drowsy or inattentive	0 / 1 / 2
Acute change or fluctuation	Significant recent change in alertness, cognition or other mental function arising within two weeks, with evidence during the last twenty-four hours: absent / present	0 / 4

Interpretation:

- at least four suggests possible delirium, with or without cognitive impairment.
- One to three suggests cognitive impairment; investigate clinically.
- Zero makes delirium less likely but does not exclude it.

Untestable cognitive items receive their specified points, not zero. Communication barriers and severe dementia may raise scores; establish baseline and seek a clinical explanation. Missing collateral information is uncertainty, not proof of no change.

NONVERBAL DOES NOT MEAN UNASSESSABLE

Use a reliable squeeze, nod or other validated response, accounting for weakness and sensory loss. Deep sedation, deafness, aphasia or paralysis can invalidate performance; document the barrier rather than automatically diagnosing delirium. In critical care or postoperative recovery, use the intensive-care method or Intensive Care Delirium Screening Checklist according to local practice.



Hand squeeze (if able) · Nod or other validated response · Account for sensory loss (e.g. deafness) · Use intensive-care method when appropriate

STANDARD CONFUSION ASSESSMENT METHOD

Perform a suitable cognitive interview and observation; training improves reliability. Rate:

1. Acute onset and fluctuating course.
2. Inattention.
3. Disorganised thinking: illogical, incoherent or unpredictably shifting ideas.
4. Altered consciousness: anything other than normal alertness.

Positive algorithm = features one AND two AND either three OR four.

This is a feature-based decision, not an additive total. Do not score from casual conversation alone or confuse quiet behaviour with normal attention.

CONFUSION ASSESSMENT METHOD FOR THE INTENSIVE CARE UNIT

Designed for patients who may be unable to speak, including intubated patients who can respond. First assess the Richmond Agitation–Sedation Scale: minus four or minus five means too deeply sedated/unresponsive for this delirium assessment; record unable to assess and reassess later. At minus three or above, proceed.

1. Acute baseline change OR fluctuation within twenty-four hours.
2. Inattention. Read the ten-letter sequence S–A–V–E–A–H–A–A–R–T, about three seconds apart; request a hand squeeze for each A. Count missed target responses and responses to other letters. More than two errors makes this feature positive. Use the validated picture alternative when appropriate.
3. Altered arousal, meaning the current Richmond Agitation–Sedation Scale is not zero.
4. Disorganised thinking, assessed with the official four yes/no questions and two-part finger command; more than one combined error is positive. Follow the official motor-adaptation instructions.

Positive algorithm = features one AND two AND either three OR four.

Note: features three and four are numbered differently from the standard method.

ACTION FLOW



Positive or clinically concerning result → clinician confirms the syndrome → urgent cause assessment and treatment → repeat and document trajectory

Baseline · Arousal · Attention · Change · Clinical judgement

Educational summary; use official forms and training.

Intensive-care method: E. Wesley Ely and Vanderbilt University, 2002.

Delirium bedside assessment and validated tools. Use the visual organisation to recall the key distinctions and structure your answer. Read with IV-94.

Credits and references: retained within the illustration.

IV - 95

Assessment of Cognitive functions in Delirium. [10]

10 | DELIRIUM AND AMNESTIC SYNDROMES | KUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 The principle	Attention is tested first and everything is read through it
4 The domains	Each one, and how it is tested at the bedside
2 Instruments and limits	What the standard scales cannot do in a fluctuating patient
2 The discrimination	Delirium against dementia and against depression

- **Attention first.** Digit span forwards, months of the year backwards, serial sevens, or a simple vigilance task. If attention is impaired, every other score becomes a floor rather than a measurement, because a patient who cannot attend cannot register, and cannot then recall.
- **Then the domains.** Orientation to time before place; registration and delayed recall; language and naming; visuospatial function by clock drawing or intersecting pentagons; thought process for disorganisation; perception for illusions and visual hallucinations; and level of consciousness, which is observed rather than asked about.
- **The limits of the scales.** The mini mental state examination and the Montreal Cognitive Assessment were built for stable patients. In delirium they fluctuate hour to hour, they cannot separate inattention from amnesia, and a low score records how impaired the patient is, never why. Retesting at a different time of day is worth more than one score.

FEATURE	DELIRIUM	DEMENTIA	DEPRESSION
Onset	Hours to days	Months to years	Weeks
Course over a day	Fluctuates, worse at night	Stable, slowly progressive	Stable, often worse in the morning
Attention	Impaired, the core feature	Preserved until late	Variable, effort dependent
Consciousness	Altered, hyper or hypoalert	Clear until very late	Clear
Perception	Visual hallucinations, illusions	Later, less prominent	Uncommon, mood congruent
Memory failure	Registration fails, through inattention	Recall fails despite registration	Answers do not know, improves with cues

Amber cells are the discriminators. Fluctuation, inattention and a clouded conscious level settle delirium; the pattern of memory failure is what separates dementia from depression.

WHAT EARNS THE MARKS

- **Attention tested first, with a named test.** Saying assess cognition earns nothing.
- **The limits of the standard scales stated plainly.**
- **The three-way discrimination,** with depression included rather than only dementia.

SEE ALSO Slot IV - 94 screening assessment in delirium Slot IV - 92 definition, causes and management of delirium

SPRINT Day 34, Stroke, TBI, delirium & focal brain syndromes

IV - 96

Describe the criteria and causes of Amnesic Syndromes. [10]

10 | DELIRIUM AND AMNESTIC SYNDROMES | RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

3	The criteria	What must be present, and what must be absent
4	Causes by mechanism	Six groups, each with the clue that points to it
2	Investigation	How the cause is actually established
1	The functional differential	What dissociative amnesia looks like instead

THE CRITERIA

Present: impairment of the ability to learn new material, or to recall previously learned material, severe enough to impair function. Absent: the clouded consciousness of delirium, the multi-domain decline of dementia, and impairment better explained by intoxication. Required: evidence of a cause capable of producing it.

MECHANISM	EXAMPLES	THE CLUE THAT POINTS TO IT
Nutritional	Korsakoff syndrome from thiamine deficiency	**Alcohol history, or a preceding Wernicke episode**
Vascular and hypoxic	Posterior cerebral artery infarction, thalamic stroke, subarachnoid haemorrhage, cardiac arrest, carbon monoxide	Abrupt onset with focal signs, or a documented hypoxic event
Infective	Herpes simplex encephalitis	**Fever and seizures, temporal changes on imaging**
Traumatic	Head injury, particularly with temporal contusion	The injury, and the length of post-traumatic amnesia
Surgical and epileptic	Bilateral temporal lobectomy, transient epileptic amnesia	The operation, or brief recurrent episodes on waking
Toxic and iatrogenic	Benzodiazepines, electroconvulsive therapy, alcohol blackouts	A clear temporal link to the agent, and reversibility

Amber marks the two that are both common and treatable, and are therefore the two an examiner most wants named early.

- **Investigation.** Informant history, imaging of the medial temporal and diencephalic structures, thiamine status, a metabolic screen, electroencephalography where epilepsy is possible, and neuropsychological testing to confirm the deficit is isolated.
- **The functional differential.** Dissociative amnesia typically loses autobiographical and personal identity information while new learning stays intact, which is the reverse of the organic pattern. Transient global amnesia is distinct again: an isolated episode of dense anterograde amnesia with repetitive questioning, resolving within a day.

WHAT EARNS THE MARKS

- **Criteria written as present, absent and required.** The exclusions carry as much credit as the inclusions.
- **Causes grouped by mechanism,** which makes the list defensible rather than remembered.
- **The reversed pattern in dissociative amnesia.** One sentence, and it separates the top band.

SEE ALSO Slot IV - 93 amnestic disorder Slot IV - 83 thiamine deficiency SPRINT
Day 34, Stroke, TBI, delirium & focal brain syndromes

AREA 15 OF 15 · P4-A04

Cognitive assessment and dementia management

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

HIGH 1 SINGLE 3

REVISION ORDER

The two cognitive rehabilitation slots together, they are one answer under two names and what the examiner wants is where each is used. Memantine next, a single-drug answer and the best-formed dose in this book. Successful ageing last, and it is the one slot here that is not written about a deficit.

WHAT IS IN THIS AREA

Cognitive rehabilitation and cognitive remedial therapy in two slots that ask for nearly the same body of material, the mechanisms and the interventions that support successful ageing, and memantine on its own.

IV - 97

Cognitive rehabilitation [10]

10 COGNITIVE ASSESSMENT AND DEMENTIA MANAGEMENT

KNRUHS, RGUHS - 2 APPEARANCES, 2 OF 78 SESSIONS

SKELETON

2	**Definition**	Individual, goal-oriented, and aimed at function
3	**The three that get confused**	Stimulation, training, rehabilitation, kept apart
3	**How it is done**	Named learning methods, aids, and the environment
2	**Evidence and place**	What it changes, and what it honestly does not

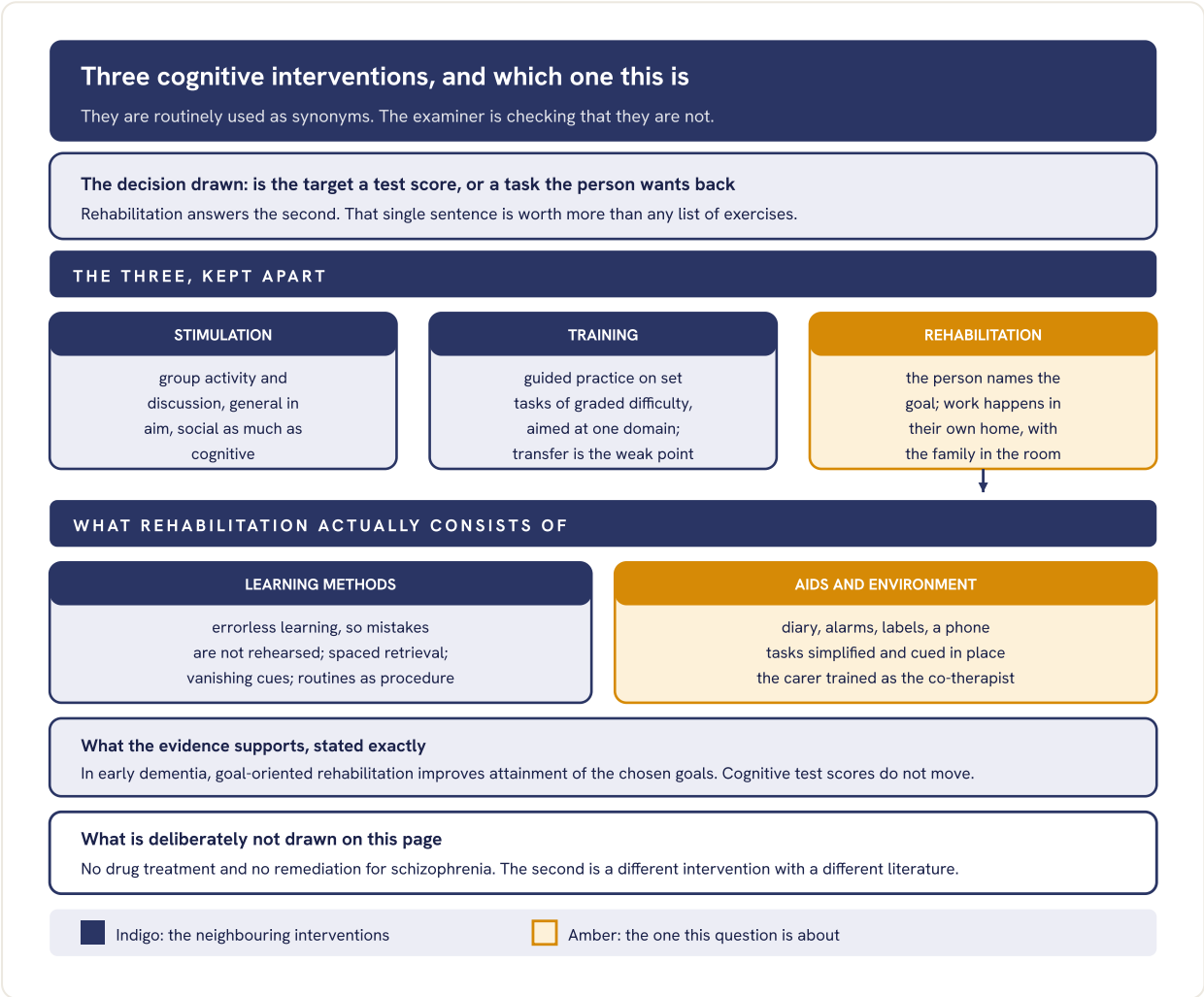


Fig 097.1 The three interventions separated at the top, then the content of rehabilitation itself below, with the evidence statement carried as a strip so the claim and its limit are read together.

- **The definition to write first.** An individualised, goal-oriented intervention in which the person and family choose everyday goals that matter to them, and strategies are built to reach those goals in the person's own setting.
- **Beyond dementia.** The same methods carry the strongest evidence after traumatic brain injury and stroke, where the deficit is stable rather than progressive.

PITFALL

Describing brain-training exercises. That is cognitive training, and its weakness is exactly the thing rehabilitation is built to solve: practice improves the practised task and rarely the life around it.

WHAT EARNS THE MARKS

- **Function named as the target,** not cognitive score, in the opening line.
- **The three interventions separated,** which is the discrimination the question exists to test.
- **Two named learning methods,** errorless learning and spaced retrieval among them.
- **The honest evidence statement,** goals improve and test scores do not.

SEE ALSO Slot IV - 99 cognitive remedial therapy Slot IV - 12 mild cognitive impairment **SPRINT** Day 32, Dementias & neurocognitive disorders

IV - 98

Explain the mechanism and interventions to enhance successful aging. [10]

10 | ASKED AT 15 MARKS | COGNITIVE ASSESSMENT AND DEMENTIA MANAGEMENT

TN-MGRMU - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 **Define it**	The three-component model, named and attributed
3 **Mechanisms**	Reserve, plasticity, vascular and inflammatory ageing
3 **Interventions**	Ordered by the strength of the evidence behind them
2 **The critique**	Who a model built on absence of disease leaves out

THE DEFINITION, AND WHERE IT COMES FROM

Successful ageing is defined by three components together: low probability of disease and of disease-related disability, high physical and cognitive functional capacity, and active engagement with life through relationships and productive activity. Two related models sit alongside it: selective optimisation with compensation, and the shift toward emotionally meaningful goals as time horizons shorten.

MECHANISM	WHAT IT DOES	WHAT ACTS ON IT	EVIDENCE
Cognitive reserve	Buffers the same pathology behind better function	Education, occupation, lifelong learning, bilingualism	Observational
Vascular health	Protects white matter and perfusion	Blood pressure, glucose, lipids, smoking cessation	**Randomised support**
Neuroplasticity and muscle	Maintains function and slows sarcopenia	Aerobic and resistance exercise, protein intake	**Strongest single lever**
Sensory input	Keeps cognition and social contact fed	Hearing aids, cataract surgery	**Modifiable and neglected**
Engagement and role	Sustains mood, purpose and identity	Family role, work, volunteering, community	Consistent, observational

Amber cells are the discriminators. Naming the strength of evidence beside each lever is what separates a plan from a wish list.

- **Two clinical additions.** Treat depression, pain and insomnia, since each masquerades as ageing, and review the drug chart for anticholinergic and sedative burden that is quietly costing function.
- **The critique worth writing.** A model built on the absence of disease excludes most older people who have one, is culturally narrow, and ignores income and access. Adaptation within illness is a fairer standard.

WHAT EARNS THE MARKS

- **All three components of the model,** stated as required together rather than as alternatives.
- **Mechanisms before interventions,** because the stem asks for mechanism first.
- **Exercise and sensory correction named,** the two most often left out of the intervention list.
- **One honest criticism of the model.**

SEE ALSO Slot IV - 16 prevention of Alzheimer disease Slot IV - 97 cognitive rehabilitation **SPRINT**

Day 29, Consultation-liaison, geriatric, transcultural psychiatry & OSCE

IV - 99

Cognitive Remedial therapy. [10]

10 COGNITIVE ASSESSMENT AND DEMENTIA MANAGEMENT

RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 Definition	Behavioural training, with transfer as the stated aim
2 The active ingredients	Four, and all four are needed for the name to apply
3 Where and how	Populations, the two approaches, and delivery
3 Evidence and limits	What improves, and what has to be added for it to count

THE DEFINITION, AND THE FOUR INGREDIENTS

A behavioural training intervention aimed at improving attention, memory, executive function, social cognition or metacognition, with durability and transfer to everyday functioning as explicit goals. The agreed ingredients are a trained therapist, repeated practice, the teaching of cognitive strategies, and procedures that engineer transfer into daily life.

DIMENSION	DRILL AND PRACTICE	STRATEGY BASED	IN PRACTICE
What it does	Repeated graded exercises to restore the process	Teaches ways around the deficit, then rehearses them	Most programmes combine both
Delivery	Often computer assisted, individual	Therapist led, individual or group	A therapist is required either way
Populations	Schizophrenia is the main one	Also anorexia nervosa, brain injury, mood disorder	Rigidity is the target in anorexia
Weak point	Gains stay near the trained task	Needs motivation and a therapist	Transfer is engineered, never assumed

Amber cells are the discriminators. The transfer problem is what the whole intervention is organised around.

- **The evidence, stated carefully.** In schizophrenia, meta-analyses show small to moderate gains in global cognition and smaller gains in functioning. Functional gain is substantially larger when remediation is delivered alongside psychiatric rehabilitation such as supported employment.
- **What it does not do.** It has little effect on positive symptoms, needs trained staff and repeated sessions, and does not replace medication or psychosocial care.

WHAT EARNS THE MARKS

- **Transfer named in the definition,** which is what distinguishes this from ordinary brain training.
- **All four ingredients listed,** the therapist among them.
- **The combination finding,** that remediation plus rehabilitation beats remediation alone.

SEE ALSO Slot IV - 97 cognitive rehabilitation Slot IV - 12 mild cognitive impairment

IV - 100

Memantine. [10]

10 COGNITIVE ASSESSMENT AND DEMENTIA MANAGEMENT

RGUHS - 1 APPEARANCES, 1 OF 78 SESSIONS

SKELETON

2 What it is	The receptor, and the kind of block it applies
2 Why that matters	Noise blocked, the signal for learning left alone
3 Where it belongs	Indications, the titration, and the combination
3 Safety and honest size	Adverse effects, the renal route, and the real benefit

THE MECHANISM, IN THE FORM THE MARKS ARE WRITTEN FOR

Memantine is an uncompetitive, voltage-dependent antagonist of the NMDA glutamate receptor with moderate affinity and a fast off-rate. It blocks the low-level tonic activation that drives excitotoxic noise, while still allowing the brief high-amplitude signal that learning depends on. That selectivity, not simple blockade, is the whole argument for the drug.

QUESTION	ANSWER	REASON	NOTE
Who is it for	Moderate to severe Alzheimer disease	Where the trial benefit sits	Not for mild disease
Who is it not for	Mild cognitive impairment, frontotemporal dementia	No demonstrated benefit	A common misprescription
How is it started	5 mg daily, raised weekly in 5 mg steps to 20 mg daily	Titration limits dizziness	Once daily forms exist
With what	May be combined with a cholinesterase inhibitor	Different mechanisms	No cholinergic overlap
How is it cleared	Largely renal, and not a cytochrome P450 substrate	Few drug interactions	Reduce dose in renal failure

Amber cells are the discriminators. Where a drug does not belong is as markable as where it does.

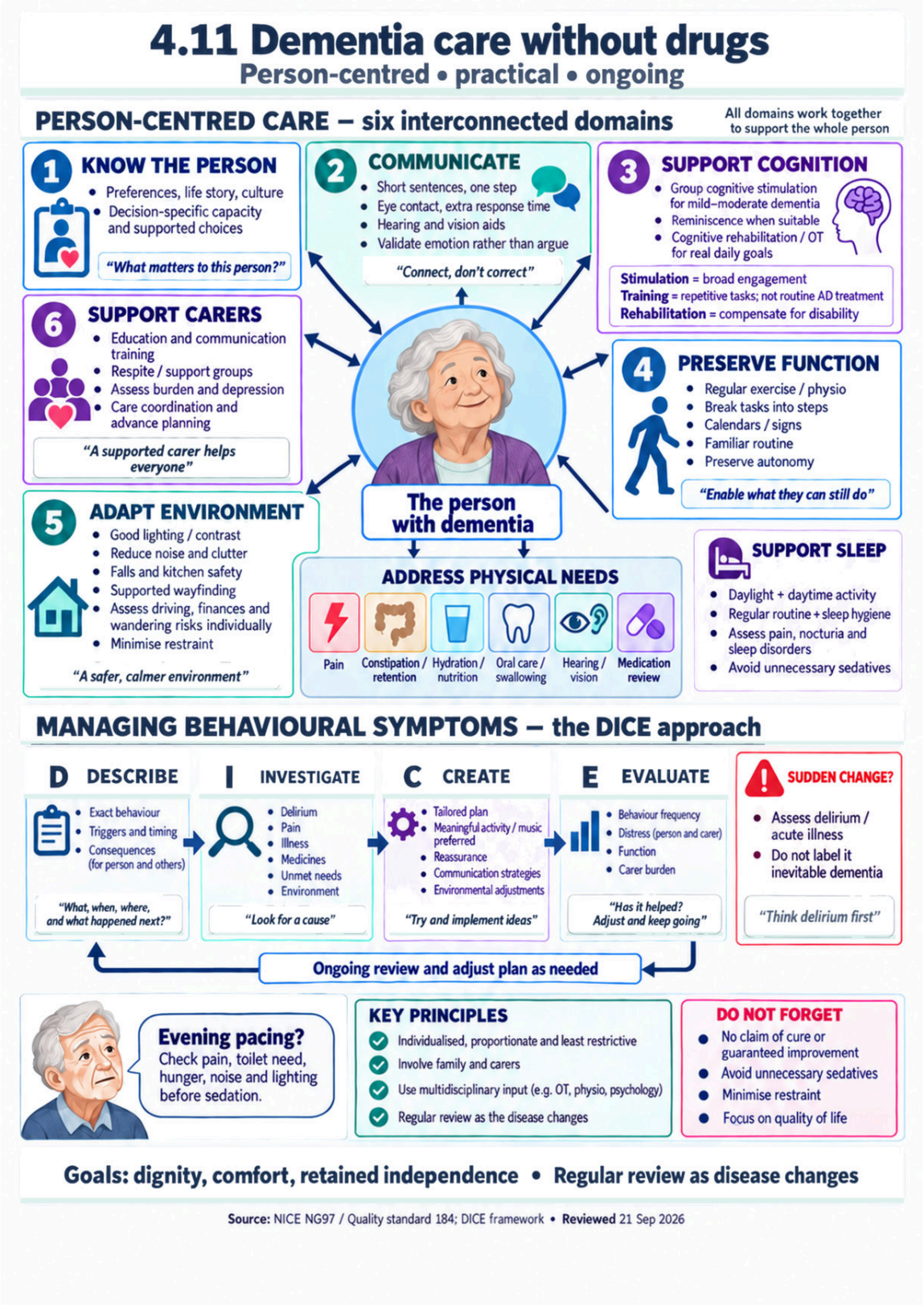
- **Tolerability.** Dizziness, headache, constipation, somnolence and confusion, occasionally hallucinations. It has none of the cholinergic gastrointestinal effects and none of the bradycardia, which is why it suits patients who cannot take a cholinesterase inhibitor.
- **The honest size of the benefit.** Symptomatic and modest: cognition, daily function and agitation shift a little. It does not modify the disease, and saying so is worth a mark rather than costing one.

WHAT EARNS THE MARKS

- **Uncompetitive and voltage-dependent stated,** not simply NMDA antagonist.
- **The tonic and phasic distinction,** which explains why blocking the receptor does not abolish learning.
- **Severity band named,** with the dementias it does not help.
- **Benefit described as symptomatic and modest.**

SEE ALSO Slot IV - 17 cholinesterase inhibitors Slot IV - 13 frontotemporal dementia SPRINT Day 32, Dementias & neurocognitive disorders

WEAVE 100 · PAPER IV · D4.11



Dementia care without drugs - Function behaviour and caregiver support. Use the visual organisation to recall the key distinctions and structure your answer. Section companion: Cognitive assessment and dementia management.

Credits and references: retained within the illustration.

WEAVE 100 · PAPER IV · Y06

Y06 — Cognitive rehabilitation and remediation: improve performance where life happens.

Related terms, different emphasis.

Cognitive rehabilitation is an individualised process that helps someone achieve meaningful activities despite cognitive impairment. It can combine restoration of abilities, compensatory strategies, environmental adaptation and family or workplace support.

Cognitive remediation uses behavioural training to improve cognitive processes, with the aim of durable benefit and transfer to everyday functioning. It is often used in schizophreina rehabilitation.

The terms overlap: remediation is not restricted to computer games, and rehabilitation is not restricted to compensation.

Start with a functional assessment.

Identify attention, processing speed, memory, executive, language and social-cognitive difficulties alongside strengths.

Assess hearing, vision, literacy, preferred language, motor ability, sleep, pain, fatigue, mood, medication effects and substance use.

An acute change may require investigation for delirium rather than training.

Combine appropriate cognitive testing with observation of a real task and the person's account; a global screening score does not explain every failure.

With consent, ask family or staff about performance across settings.

Agree a goal that can be observed.

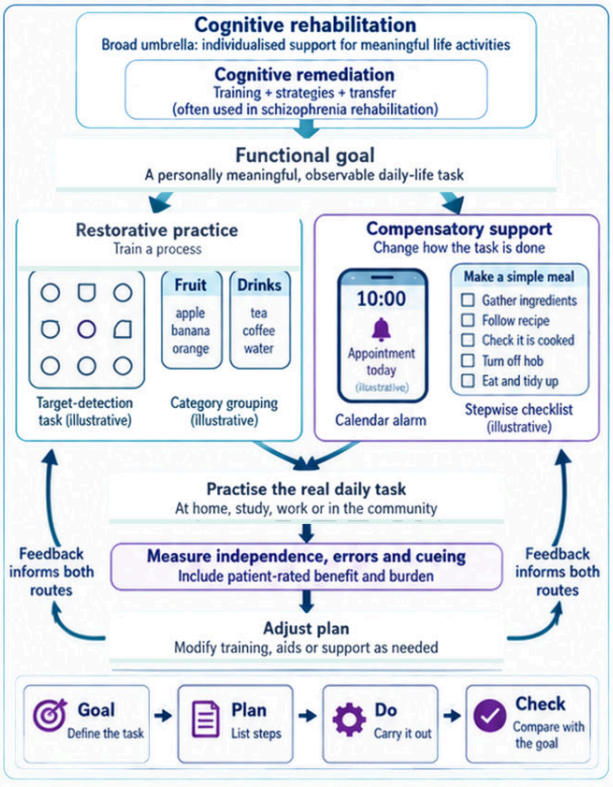
Replace "improve memory" with a personally important task, such as arriving at a weekly appointment using a calendar reminder, or completing a simple meal safely with a checklist.

Record baseline success, errors, time, cues and distress.

Ask what matters to the person and what level of independence is realistic.

Goals must respect fatigue, safety and available support; unaided performance is not the only valid outcome.

Memory aid:
"Choose a goal, practise a strategy, use it in life, measure the difference."



Restorative practice: train a process.

Attention training may begin with detecting a target in a short quiet task, then vary duration or distraction as tolerated.

Working-memory practice might involve retaining a brief instruction while carrying out one step.

Learning strategies can include grouping shopping items into categories and explaining the strategy aloud.

Adjust difficulty to sustain useful practice and feedback.

Improvement on the practised task does not guarantee improvement in an untrained activity; deliberately rehearse the target daily-life task as well.

Compensation: change how the task is done.

External aids include a single calendar, phone alarms, labelled storage and a stepwise checklist.

Teach the person to notice the cue, open the aid, perform the action and check completion; handing over a notebook is not training.

Reduce distracting noise and unnecessary choices, and establish predictable routines.

For marked memory difficulty, errorless learning provides the correct response or sufficient cues before guessing; gradually reduce support.

Spaced retrieval practises successful recall over increasing intervals, shortening the interval if unsuccessful.

These methods are chosen for the individual and task, not prescribed universally.

Executive strategies and real-world transfer.

Use "Goal → Plan → Do → Check": define the task, list steps, carry them out, then compare the result with the goal.

Practise with a meaningful activity such as preparing for a journey.

The therapist models the strategy, prompts self-monitoring and gradually fades cues.

A bridging discussion asks where the same strategy could help at home, study or work; practise there with appropriate support.

Social-cognitive exercises may explore facial emotion, context and alternative interpretations without assuming one facial expression proves another person's intentions.

Remediation within psychiatric rehabilitation.

Core elements include an active trained therapist, repeated cognitive exercises, development of strategies and methods that support transfer.

Programmes can be individual or group-based and use paper, computers or ordinary activities.

Cognitive remediation can improve cognition in schizophrenia; effects on everyday functioning vary and are stronger when connected to broader rehabilitation.

Consider linking it with supported employment, education or independent-living practice.

It complements clinical treatment and does not replace antipsychotic care, psychological therapy or social support.

Evidence in one disorder should not automatically be generalised to every diagnosis.

Measure what changed and adapt.

Reassess the chosen daily task, cueing required, participation, patient-rated benefit and burden.

Use validated cognitive measures where appropriate, accounting for repeat-testing effects, language and education.

A better test score without functional benefit requires revisiting transfer, task choice or environmental barriers.

If progress stalls, assess fatigue, sensory limitations, distress, adherence barriers and treatment fit before increasing drill intensity.

Document the plan, train supporters with consent, review durability and modify aids as life demands change.

Supplementary clinical teaching • September 2026.

Cognitive rehabilitation versus remediation. Read across the labelled contrasts to distinguish the related concepts. Section companion: Cognitive assessment and dementia management.

Credits and references: retained within the illustration.

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
IV-S1	IV-12 p 41	IV-60 p 184	IV-05 p 15	IV-20 p 57	IV-66 p 198	IV-30 p 93	IV-44 p 134	IV-74 p 209	IV-52 p 161	IV-96 p 270
IV-S2	IV-13 p 43	IV-82 p 225	IV-06 p 17	IV-07 p 18	IV-09 p 20	IV-42 p 132	IV-45 p 135	IV-57 p 173	IV-71 p 204	IV-95 p 269
IV-S3	IV-01 p 11	IV-72 p 206	IV-26 p 77	IV-87 p 243	IV-34 p 112	IV-77 p 214	IV-64 p 189	IV-56 p 172	IV-58 p 175	IV-99 p 275
IV-S4	IV-14 p 45	IV-48 p 150	IV-31 p 105	IV-19 p 56	IV-67 p 199	IV-29 p 90	IV-65 p 190	IV-89 p 246	IV-93 p 265	IV-98 p 274
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IV-S6	IV-39 p 124	IV-16 p 50	IV-18 p 55	IV-32 p 106	IV-11 p 22	IV-88 p 244	IV-68 p 200	IV-69 p 202	IV-59 p 178	IV-81 p 222
IV-S7	IV-02 p 12	IV-04 p 14	IV-24 p 73	IV-33 p 110	IV-35 p 113	IV-63 p 188	IV-78 p 217	IV-90 p 247	IV-38 p 116	IV-80 p 221
IV-S8	IV-40 p 126	IV-23 p 71	IV-25 p 75	IV-84 p 233	IV-41 p 130	IV-21 p 60	IV-79 p 218	IV-50 p 157	IV-85 p 235	IV-86 p 237
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IV-S10	IV-22 p 69	IV-03 p 13	IV-62 p 187	IV-83 p 230	IV-49 p 155	IV-73 p 207	IV-43 p 133	IV-75 p 210	IV-76 p 211	IV-91 p 251

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
IV-S1	100	0.422	9	3	Dementias: types 2; Recent advances 1; Community 1
IV-S2	100	0.418	7	2	Community 3; Stroke 2; Dementias: types 1
IV-S3	100	0.415	9	3	Endocrine 2; Community 1; Psychopharmacology: principles 1
IV-S4	100	0.402	9	3	Dementias: types 2; Movement 1; Clinical neurology 1
IV-S5	100	0.395	8	5	Dementias: types 2; Movement 2; Cognitive assessment 1
IV-S6	100	0.394	8	2	Dementias: types 2; Drug emergencies 2; Stroke 1
IV-S7	100	0.394	6	3	Clinical neurology 3; Community 2; Epilepsy 2
IV-S8	100	0.399	6	5	Nutritional 3; Stroke 2; Consultation-liaison 2
IV-S9	100	0.392	10	3	Delirium 1; Movement 1; Recent advances 1
IV-S10	100	0.390	8	4	Psychopharmacology: principles 3; Consultation-liaison 1; Community 1

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.

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Dr. Niharika Reddy	Clinical review of the community and public mental health area, the consultation-liaison maps, and the dementia and delirium management pages. Co-author.

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