WEAVE SPRINT . PAPER IV

Medical and systemic-disease psychiatry

The consultation-liaison chapter at the medicine interface, written short on purpose: six of its sixteen constructs are cross-references and are mapped once on a routing plate rather than argued six times, so that what remains can be taught properly. The heart that fails and the device that shocks, breathlessness told apart from panic, the transplanted patient and the drugs that keep the graft, the operation and the cognition that follows it, the red flags that raise the question of an occult malignancy, six systemic autoimmune diseases on one comparator because their value is telling them apart, and burn injury, taught here because it is taught nowhere else in these papers.

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WEAVE . CENTRE FOR INTEGRATIVE PSYCHIATRY

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Concept / rule Key number Trap

PAPER IV

Medical and systemic-disease psychiatry

Four bands . one complete notes document

Cardiac, respiratory and critical-illness psychiatry

1 · The organ interface: what this chapter owns, and what it deliberately points to

At the interface with medicine the psychiatrist is rarely asked what the diagnosis is, and is almost always asked which of three things is producing the picture. The medical illness may be generating the psychiatric symptoms directly. The treatment given for that illness may be generating them. Or the patient may have a psychiatric disorder of their own, arriving in a body that happens to be unwell. Those three answers oblige three different actions, and this chapter is built to make the choice between them quicker and more defensible. This orientation installs that **triage**, adds the fourth question the triage forces, which is who teaches a construct once it has been placed, and draws the map of what follows.

One feature of this chapter should be stated at the outset rather than discovered halfway through. It is deliberately short for the number of constructs it names, because several of them are named here and taught elsewhere in the series. That is a design decision with a defence, and the defence is set out below in full, because a reader who does not understand why a chapter points will read the pointing as a shortfall.

1.1 The three-way question, and what each answer obliges you to do

The first limb is the illness acting on the brain. A systemic disease can produce psychiatric symptoms through mechanisms that require no psychological step at all: inflammation reaching the central nervous system, an immune response raised against a tumour that also recognises neural tissue, hypoxia and carbon dioxide retention, the accumulated products of failing kidneys, endocrine derangement, small-vessel injury, a surge of circulating catecholamines. When this limb is the answer, the psychiatric task is largely to establish it and to hand the patient back for treatment of the disease, because no psychiatric intervention will hold while the process continues.

The second limb is the treatment acting on the brain. Modern medical treatment is potent and it is neuroactive, and the psychiatric consequences of it are not rare accidents but predictable properties of certain agents and certain procedures. Immunosuppression, cytotoxic treatment, interferon, corticosteroids, anaesthesia and surgery itself, oxygen and ventilatory support and implanted devices all change the mental state, each with its own syndrome and its own interval. When this limb is the answer, the decisive evidence usually sits in the drug chart and the procedure record rather than in the mental state examination, and the action is a conversation with the treating team about the exposure.

The third limb is a psychiatric disorder in its own right, occurring in a person who is medically unwell. This limb has two internal shapes that behave alike for management purposes. The

disorder may be one the patient would have developed anyway, whose timing merely coincides with the admission. Or it may be a response to the illness, to what the illness has taken, and to what it will take next. Both need treating as psychiatric conditions, and both are vulnerable to the same argument, which is that a person in this situation would reasonably feel that way. When this limb is the answer, the psychiatric task is the largest and the least likely to be delegated.

Two properties of the triage matter more than the categories themselves. The limbs are not exclusive, and in a medically unwell patient more than one is usually operating at once, so the working question is not which limb is true but which limb is doing the most damage now and which, if missed, is dangerous. And the limbs are answered by different evidence: the first by the disease course and its physiology, the second by exposure and timing, the third by the patient's history, baseline and account of themselves.

Illness	Treatment	Independent disorder	Owner
READ FROM THE DISEASE COURSE	READ FROM EXPOSURE AND TIMING	READ FROM BASELINE AND HISTORY	WHO TEACHES WHAT YOU HAND ON

RULE **State which limb you are in, and say what evidence put you there.** An answer at this interface that names a psychiatric syndrome without placing it against illness, treatment or independent disorder has skipped the step the chapter examines. The examiner's follow-up is almost always the same question in different words: what else could be producing this, and what would separate them?

1.2 The examination technique the triage runs on

The triage is decided by history far more often than by investigation, and by a small number of manoeuvres that are cheap and frequently skipped. Learning them as a fixed order is worth more than learning any single association in this chapter, because the order transfers to every section in it.

Establish the baseline first, from someone other than the patient. What was this person like a month ago, at work, at home, in conversation? A medically unwell patient is observed by staff who never met the well version, so the comparison that would make the change visible does not exist on the ward unless a psychiatrist goes and gets it. Without a baseline, a description of the present state is the only thing available, and a present state cannot establish a change.

Then establish **tempo**. Every psychiatric illness has a characteristic time signature, and the useful reading is the mismatch: a presentation moving faster than the condition it resembles, or continuing to move while adequate treatment is being given, or following a slope rather than an episode. Tempo costs nothing but a careful timeline with corroboration, and it is the reading most often left vague.

Then lay two timelines side by side. The first is the medical course, with its events dated: the acute episode, the operation, the transfer, the deterioration. The second is the drug chart, read for start dates rather than for the current list. Laying the psychiatric change against those two timelines is the single highest-yield act at this interface, and it is what separates the first limb

from the second. A change that begins with the illness and tracks its severity points one way. A change that begins after an agent was introduced, and in the interval characteristic of that agent, points the other.

Ask what was stopped as well as what was started. An admission removes access to alcohol, to nicotine and to whatever else the patient used at home, at a predictable hour, and it frequently interrupts the patient's own long-term psychotropic treatment on the ward round of a team that does not prescribe it. A withdrawal syndrome or an abrupt discontinuation, declaring itself on a medical ward, is read by people looking for a medical explanation as a complication of the illness.

Attend to consciousness and attention before anything else in the mental state. A fluctuating disturbance of attention and awareness changes the entire answer and moves the problem into territory this chapter does not teach, and the whole of that assessment, including any bedside instrument, rating scale or drug choice for it, belongs to the stroke, traumatic brain injury, delirium and focal brain syndromes notes. Recognising the shape and handing it on is the examinable act here.

TRAP **Settling for understandable.** The commonest error at this interface is not a wrong diagnosis but an abandoned one: the low mood is judged a reasonable response to a serious illness, and the assessment stops. Comprehensibility is not a diagnostic exclusion and would not be accepted for any other comorbidity. It costs more than usual here, because the psychiatric state is often what removes the patient from the treatment, the rehabilitation and the adherence that would change the medical trajectory.

1.3 Why a chapter that points beats a chapter that teaches everything thinly

The alternative to routing a topic away is not depth, it is a thin second copy, and a thin copy is worse than an absence because it does not feel like one. A paragraph read on a topic taught properly elsewhere leaves the reader recognising every term, able to defend none of them, and unable to tell which of their own facts are the thin ones. A half-learned fact sits in memory looking exactly like a fact learned well. It survives the first question and fails the follow-up, which is where the marks are, and it fails at the bedside for the same reason.

An explicit omission behaves differently. A reader who has been told that a construct is taught in a named chapter knows precisely what they do not yet have, and can act on it. A reader who met a thin paragraph knows nothing of the kind. That difference is also what makes routing checkable rather than merely tidy: whether a chapter mentioned a topic is a weak test that a chapter can pass while leaving its reader unable to answer anything, whereas whether the reader can name the owner is a test with a right answer.

There is a second argument from the reader's side, and in the last weeks before an examination it is the stronger one. Revision time is fixed, and a chapter that duplicates an adjacent chapter spends that time twice on one topic and not at all on the topics only it can teach. The material this chapter alone carries, which is the systemic and inflammatory disease that presents psychiatrically before anyone knows it is present, is exactly the material that goes unrevised when a chapter pads itself out to look complete. Shortness here buys depth where depth is scarce.

A third argument concerns the text rather than the reader, and this chapter's routing section makes it in full: two accounts of one topic, written by different hands, drift apart, and one owner with one account is the only arrangement in which two pages of the same book cannot disagree.

The fourth argument is the one that matters clinically, and it is why the routing question is not bookkeeping. Liaison work is **routing**. A referral arrives from another team, written by someone unsure what to ask, and the psychiatrist's first act is to decide whose question it is: the treating team's, the psychiatrist's, or a third specialty's altogether. A resident who has practised deciding where a construct belongs has been practising the actual skill, and one who has memorised a paragraph about each has not. The map that follows is therefore content, not furniture.

RULE **A declared gap is a correct answer, and a supplied specific is not.** Where this chapter does not hold a figure, a threshold or a management sequence, it says so and names the kind of source such a statement would rest on. In an examination the equivalent move is to state the direction confidently, state that the magnitude is not something you can quote, and go on. That answer is stronger than a number produced from recollection, which is unverifiable and frequently wrong.

The map of routed constructs is drawn once, on the plate below, and the routing section that follows gives each one an exact address and a single boundary sentence stating what this chapter withholds about it.

1.4 The map of the chapter, and the question each section answers

The chapter is organised into four bands. The first covers cardiac, respiratory and critical-illness psychiatry. The second covers renal, transplant and perioperative psychiatry. The third covers oncology, palliative and paraneoplastic psychiatry. The fourth covers autoimmune, rheumatological and inflammatory systemic disease. A section carrying the psychiatric care of the severely burned patient sits alongside them, and a routing section carries the constructs taught elsewhere. Each section below is named by the question it answers, because a question is easier to hold in mind than a title and it is what an examiner is actually setting.

SECTION	THE QUESTION IT ANSWERS	LIMB OF THE TRIAGE IT MOSTLY TRAINS
Heart failure, arrhythmia and cardiac devices	Which of the somatic symptoms belong to the failing circulation, how do rhythm presentations separate from panic, and what does an implanted device do to a person and a household?	Illness, and independent disorder
Obstructive airways disease, asthma and chronic respiratory failure	Is this breathlessness or is it panic, and which behavioural levers change the trajectory?	Illness, and independent disorder
The stress-heart interface	Can an acute psychological event produce a structural cardiac lesion, and why is the presentation mistaken for a coronary event?	The arrow reversed, running from the psychological event to the organ
Transplant psychiatry and immunosuppressant neurotoxicity	What must a pre-transplant psychiatric opinion contain, and which new neuropsychiatric states belong to the drugs that keep the graft?	Treatment
Perioperative psychiatry and postoperative neurocognition	In what order do the perioperative questions arrive, and which cognitive construct is being named after the operation?	Treatment
Cancer-drug neuropsychiatry	Which agent, which syndrome, and over what interval does that syndrome appear and get looked for?	Treatment
Paraneoplastic neuropsychiatric syndromes	When should a psychiatric presentation start a directed search for a malignancy nobody has found yet?	Illness, before the illness is known
Neuropsychiatric lupus	Does the syndrome in front of you belong to the disease at all, and how is that attribution reasoned when no test settles it?	Illness, with attribution as the whole difficulty
Neurosarcoidosis, cerebral vasculitis and antiphospholipid syndrome	How do you answer on a rare systemic cause without inventing a phenotype for it?	Illness, and evidential discipline
Coeliac disease, inflammatory bowel disease and the gut-brain axis	What do the association and bidirectionality findings actually state, and what will their design bear?	Illness, and independent disorder

SECTION	THE QUESTION IT ANSWERS	LIMB OF THE TRIAGE IT MOSTLY TRAINS
Burns psychiatry	What is the psychiatric task at each stage of the burn timeline, and what question does the injury itself raise about what preceded it?	Independent disorder, and response to injury
Organ-interface routing	Who teaches the constructs this chapter names and does not teach, and what exactly is withheld here?	The fourth question

Read the third column across and the lens becomes visible in the shape of the chapter. The sections on paraneoplastic syndromes, lupus and the rare systemic autoimmune causes are extended training in the first limb, and specifically in its hardest form, where the illness has not yet been identified and the psychiatrist is the person who must raise the possibility. The sections on transplantation, the perioperative period and cancer drugs are training in the second limb, where the answer is in the exposure and the interval rather than in the mental state. The cardiac, respiratory, bowel-disease and burns sections spend most of their length on the third limb, on psychiatric disorder in a person who is genuinely and often permanently unwell, and on the reasoning that keeps it treatable rather than merely understandable.

One section sits outside the scheme on purpose. The stress-heart interface runs the arrow the other way, from an acute psychological event to a demonstrable cardiac lesion, and it is in the chapter because it is the clearest available demonstration that the traffic at this interface is not one-directional. Holding that section next to the triage is useful precisely because it is the exception.

1.5 What this chapter does not teach, and where each of those lives

Five boundaries are enforced throughout, and knowing them in advance saves a reader from searching this chapter for material it has deliberately excluded.

- Prescribing at the interface, meaning organ-adjusted dosing, interaction rankings and the choice of agent in cardiac, hepatic, renal or transplant disease, belongs to the psychopharmacology notes. This chapter teaches everything that precedes a prescription and adds no second version of the prescription itself.
- The assessment and management of the acute confusional state belongs to the stroke, traumatic brain injury, delirium and focal brain syndromes notes, including every bedside instrument, rating scale and drug choice for it. Several sections here reach that boundary, at the end of life, after an operation, in critical illness and in the early burn injury, and each marks the hand-off rather than crossing it.
- Capacity belongs to the mental health legislation notes. This chapter says only that capacity is decision-specific and time-specific, which is enough to reason with, and it states no statutory test.

- The generic immune-brain framework, the workup of autoimmune encephalitis and the neuropsychiatry of interferon-beta belong to the neurology and psychiatry interface notes. What this chapter owns on the immune side is the disease-specific presentation and the decision to suspect, not the general model and not the investigative protocol.
- The constructs defined by a clinical setting rather than by an organ, being the coronary care unit, the intensive care unit, the dialysis unit, the oncology ward and the palliative care unit, belong to the consultation-liaison, geriatric and transcultural psychiatry notes, and the psychiatric disturbance that follows a corticosteroid belongs to the endocrine and metabolic psychiatry notes. Those two owners take the constructs this chapter routes, and the routing section names each one with its boundary.

None of this is a shortfall being managed politely. What those constructs share is a referral pathway and a liaison relationship, not a pathophysiology, so they belong with the chapter that teaches liaison work. The one that follows a drug follows the drug. The classification is working, and stating it out loud is the difference between a boundary and an omission.

1.6 How to use this chapter in the weeks before the examination

Read the routing plate before anything else, so that the constructs this chapter will not teach are settled and stop feeling missing. Then read each section for its discriminators rather than for its lists, because every section here is built around a small number of separations that decide the answer, and the lists around them are recognisable at the paper and not defensible under questioning.

Practise the triage out loud on whichever medical patient you happen to be asked about, and practise it as a four-step answer rather than as three categories. The four cues below are a teaching scaffold for that sequence rather than a validated instrument, and the sequence itself is the answer structure this whole chapter installs. It works on a construct you have never met, which is more than can be said for a memorised list.

MNEMONIC

Limb, Evidence, Shift, Owner

Limb: which of the three the picture mostly sits in, the illness acting on the brain, the treatment acting on the brain, or a psychiatric disorder in its own right in a person who is unwell. **Evidence**: what placed it there, which is the disease course and its physiology for the first limb, exposure and timing for the second, and the patient's baseline, history and account of themselves for the third. **Shift**: what would move you to another limb, said out loud, because the limbs are not exclusive and the working question is which one is doing the most damage now. **Owner**: which chapter takes whatever you have just handed on, named rather than rebuilt from memory.

IN INDIA

No India-specific epidemiology is held for this orientation and none has been invented. The Indian point here is one of service structure. In most Indian general hospitals the psychiatrist arrives at the medical bedside on request, usually late, usually to a referral question written by a team under pressure, and often without the previous notes, the collateral history or the drug chart's start dates to hand. Every manoeuvre in the triage above depends on information the referring team holds and the referral letter rarely contains, so the practical skill is asking for it explicitly and early rather than working from the mental state alone. That is also the argument for reading the consultation-liaison material before a liaison posting begins.

GOOD TO REMEMBER

Three questions at the bedside and one afterwards. Is the disease doing this? Is the treatment doing this? Is this a psychiatric disorder in a person who is unwell? And then, before writing: whose chapter answers the part I am handing on?

This chapter teaches a triage and a map, and it teaches them together, because at the medicine interface deciding where a problem belongs is most of deciding what to do about it.

2 · Depression and anxiety after myocardial infarction and in coronary artery disease: prognosis and safe antidepressant choice

Six of the topics in this chapter are addresses rather than lessons, and saying so plainly is the most useful thing this section can do. Depression and anxiety after myocardial infarction and in coronary artery disease, intensive care psychiatry and the psychological sequelae of critical illness, psychiatric assessment in end-stage renal disease and on dialysis, depression and demoralisation in cancer, palliative care psychiatry, and steroid-induced psychiatric disturbance are each named in this chapter and taught in none of it. Each has an owner elsewhere in the series. This section hands the reader that owner, states what is withheld here, and explains why withholding it is honest rather than lazy.

2.1 Why a routed chapter is more honest than a complete-looking one

The alternative to routing is not depth. It is another thin copy. A chapter that gives every adjacent topic a paragraph produces a reader who recognises every term on the paper, can defend none, and cannot tell which of their facts are the thin ones. That is the harm, and it is not obvious: a half-taught fact does not feel half-taught. It sits in memory looking like a fact learned properly, and it fails at the point of use rather than the point of reading.

The second harm is duplication, and it is invisible while writing. Two accounts of one topic, written by different hands against different sources, do not stay identical. They drift, and a reader who has met both cannot tell which is authoritative. One owner and one account is the only arrangement in which two pages of the same book cannot disagree.

The third point is about how completeness gets checked, which is why routing must be declared out loud rather than practised quietly. Asking whether a chapter mentions a topic is an existence check, and a chapter can pass it while leaving the reader unable to answer anything. Asking whether the reader can name the owner is the better test, because it is checkable. A **routed construct** turns an implicit omission into an explicit one, and an explicit omission can be acted on. Silence cannot.

A fourth reason is particular to this chapter. Five of the routed topics are not defined by an organ at all. They are defined by a setting: the coronary care unit, the intensive care unit, the dialysis unit, the oncology ward, the palliative care unit. What they share is a referral pathway and a liaison relationship, not a pathophysiology, so they belong together in the chapter that teaches liaison work. The remaining topic is defined by a drug and follows the drug. Routing here is the classification working correctly, not a shortfall being managed politely.

RULE **Each routed construct gets one exact cross-reference and one boundary sentence here, and nothing else.** This section supplies no incidence, no prognosis, no management sequence and no dose for any of them, and that restriction is its design, not its limitation. A sentence here that supplied one would be the second copy this arrangement exists to prevent.

2.2 The routing plate, and how to read it

The plate below is this section's content, and each row holds three things.

- The construct, worded as the examination will name it.
- The chapter that owns it, which is what the reader is meant to leave with.
- The single sentence stating what this chapter will not say about it, so that the omission is explicit rather than silent.

The rows are addresses with a warning label attached; they will not survive being revised from.

The owning-chapter column is repetitive, because most of these are liaison presentations pointing at the same place. Some rows carry a split, because a construct can have its assessment owned by one chapter and a single limb owned by another, and the plate says so.

Fig 40.1 The routing map, which is the whole visible content of the organ-interface routing section. Six of this chapter's constructs have owners in earlier chapters, and drawing that once is what makes a short chapter honest rather than evasive. Read the shape before the words: five rows converge on a single destination panel, so the reader sees at a glance that consultation-liaison psychiatry, not this chapter, holds post-infarction mood, critical-illness sequelae, dialysis, cancer and palliative care. The sixth row breaks away because steroid psychiatry belongs to the endocrine and metabolic chapter, and that break is the only thing the second colour means. What each row prints in amber is the entire permitted output: a naming sentence, the exact cross-reference, and a full stop. From memory you should be able to say, for any of the six, which chapter owns it and that this one adds no figure, no threshold and no ladder of its own.

2.3 What this section cannot establish, and how to answer without it

Two things a fuller account would supply are declared here as gaps, because a **declared gap** is a correct answer and an invented figure is not.

The first concerns anxiety rather than depression after infarction. A statement of its prognostic effect would rest on a meta-analysis in the cardiology literature, identified for this series but not opened in quotable form. The consequence is stated rather than concealed: this chapter attaches no prognostic figure to post-infarct anxiety, and a candidate should attach none from memory. What can still be said is that anxiety and depression after infarction are separate constructs on separate evidence, and that an answer treating them as interchangeable makes a claim it cannot support.

The second concerns the desire for hastened death in advanced illness. No current guideline recommendation, and no primary intervention study, could be opened for this series with text usable on its own terms on assessing or managing it; such a claim would rest on an oncology

clinical-practice guideline group, or on investigators of primary palliative-care psychotherapy trials. The absence of a quotable recommendation licenses no inaction: a statement of that kind is met as a communication carrying meaning rather than a symptom to be corrected, and decision-making capacity, which is decision-specific and time-specific, is doctrine owned by the Mental health legislation and forensic psychiatry notes. What is withheld here is a protocol, not the obligation to respond.

2.4 The rows in words, with the boundary stated for each

Depression and anxiety after myocardial infarction and in coronary artery disease is owned by the Consultation-liaison, geriatric and transcultural psychiatry notes, and the choice of antidepressant in established cardiac disease is owned by the Psychopharmacology II: emergencies, resistance, monitoring and interactions notes. This chapter states neither the size of the prognostic effect nor the name of any preferred agent.

3 · ICU psychiatry, post-intensive-care syndrome and psychological sequelae of critical illness

Intensive care psychiatry and the psychological sequelae of critical illness are owned by the Consultation-liaison, geriatric and transcultural psychiatry notes, and delirium in the critically ill patient is owned by the Stroke, TBI, delirium and focal brain syndromes notes. The route follows from the shape of the construct, since post-intensive care syndrome encompasses physical, cognitive, and psychological impairments that persist in patients after discharge from an intensive care unit. Only one of those domains is psychiatric, so a chapter organised by organ could own only a slice of it. Its assessment, its course and its treatment appear here in no form.

4 · Psychiatric assessment and management in end-stage renal disease and on dialysis

Psychiatric assessment and management in end-stage renal disease and on dialysis is owned by the Consultation-liaison, geriatric and transcultural psychiatry notes, and dose adjustment in renal impairment is owned by the Psychopharmacology II: emergencies, resistance, monitoring and interactions notes. The routing follows the service design rather than the diagnosis, since psychological support and interventions should be delivered within the kidney care setting. That makes it a liaison topic before a nephrology one, and no screening approach, treatment sequence or dosing rule appears here.

5 · Depression, demoralisation and adjustment in cancer: screening and treatment

Depression, demoralisation and adjustment in cancer is owned by the Consultation-liaison, geriatric and transcultural psychiatry notes. That chapter carries the distinction between demoralisation and depressive disorder, and the treatment sequence; this chapter carries neither, so a candidate revising how demoralisation differs from depression should not look here. A screening question or brief instrument for depression in advanced disease is stated by neither chapter, and that is a gap in the series rather than a section you have failed to find, so supply none from memory.

6 · Palliative care psychiatry: delirium at end of life, desire for hastened death and existential distress

Palliative care psychiatry is owned by the Consultation-liaison, geriatric and transcultural psychiatry notes, with delirium as a syndrome owned by the Stroke, TBI, delirium and focal brain syndromes notes and the doctrine of capacity owned by the Mental health legislation and forensic psychiatry notes. This chapter says only that capacity is decision-specific and time-specific, and states no approach to sedation, existential distress or a request for hastened death. What changes about delirium once treatment intent has changed, including how far to investigate and how antipsychotic choice differs in the final phase, is stated by neither owner and is a gap in the series rather than a section you have failed to find.

7 · Steroid-induced psychiatric disturbance: mania, depression, psychosis and management

Steroid-induced psychiatric disturbance is owned by the Endocrine and metabolic psychiatry notes. It is the row that follows a drug rather than a setting, so its dose relationship, its onset window and its management sequence all sit in that chapter and none is stated here. The chemotherapy and interferon section of this chapter carries no steroid limb either, by owner ruling, so there is exactly one place to look.

TRAP **Revising from the routing line as though it were an abstract.** A cross-reference is an address, written to be too short to revise from. The failure mode is a candidate who says depression after infarction matters to prognosis but cannot say what it does to it, or who knows corticosteroids cause psychiatric disturbance but cannot say when. Both collapse on the follow-up.

IN INDIA

No India-specific row is held for this section, and none has been invented. The Indian point is one of study order rather than epidemiology. Each of these constructs is by definition a liaison presentation: the patient is admitted under another team, and the psychiatrist arrives on request, often late and with a referral question written by somebody unsure what to ask. The chapters that own them also carry the referral, capacity and communication material, so read them before a liaison posting starts.

GOOD TO REMEMBER

At the bedside the question is not what you remember but which chapter answers the referral in front of you. Cardiac ward, intensive care, dialysis unit, oncology ward and palliative care unit all route to the liaison chapter. A psychiatric disturbance that began after a corticosteroid was started routes to the endocrine and metabolic chapter. Knowing that split in advance beats a half-remembered paragraph about any of them.

These constructs are named here, routed here and taught nowhere in this chapter, so that each is taught once, in full, where it is owned.

8 · Psychiatric aspects of heart failure, arrhythmia and cardiac devices

The failing heart produces most of the somatic symptoms a depression scale counts, so cardiac psychiatry is decided by history and by stage of illness rather than by a symptom total. Drug treatment is deliberately absent: prognosis and antidepressant choice in cardiac disease are taught with depression and anxiety after myocardial infarction and in coronary artery disease, and organ-adjusted dosing with the psychopharmacology notes. What is examinable here precedes any prescription.

8.1 The overlap that defeats a symptom count

Advanced heart failure and a depressive episode share almost the entire somatic side of the syndrome. Breathlessness on exertion, fatigue, disturbed sleep, poor appetite, weight change, psychomotor slowing and loss of sexual interest each follow from a failing circulation, from the treatment burden it imposes, or from a mood disorder, and none of them separates the possibilities alone. A scale that counts them inflates where accuracy matters most; a clinician who discounts them all misses the depression that is present.

Weight the **cognitive and affective items** and interrogate the somatic ones instead of scoring them. Anhedonia in activities that make no physical demand is the most useful single question on a cardiology ward: a person too breathless to walk to the gate can still say whether the radio or a visitor gives any pleasure. Guilt, worthlessness, hopelessness out of proportion to the prognosis actually given, and the thought that others would be better off without them are produced by mood.

Timing is the strongest discriminator on history. If breathlessness and oedema improved with cardiac treatment while mood, interest and outlook stayed flat, the mood state has separated from the haemodynamic state and is behaving as an illness in its own right. If low mood arrived with a decompensation and lifted as it settled, an adjustment reaction reads better. Nocturnal waking divides the same way: waking breathless and needing to sit upright points at the circulation, while waking in the early hours, alert, with the day at its worst on rising, points at mood.

In adults with confirmed heart failure, **people with heart failure and depressive symptoms had poor health-related quality of life**, so the depressive syndrome tracks with how the person actually lives rather than with a score alone. That finding carries a direction and a population and no magnitude, so its use is to justify looking, and to justify treating what is found.

RULE **Attribute by timing and by course, never by the symptom itself.** Any single somatic symptom in heart failure is uninformative. What discriminates is whether it moved with the cardiac state or independently of it, and only a sequential history answers that. Put the cardiac events, the treatment changes and the mood changes on one timeline and the attribution usually declares itself.

8.2 Stage of illness as the organising question

Heart failure is not one psychiatric situation, and the commonest error in a viva is to describe it as though it were. The task, the assessment priority and the realistic intervention all change with the stage.

STAGE	THE PSYCHOLOGICAL TASK	WHAT THE ASSESSMENT MUST REACH
New diagnosis	Absorbing a prognosis and a changed identity, often while still physically well	What they were told, what they believe it means, and who else heard it
Stable on treatment	A daily regimen, fluid and salt restriction, weighing, symptom diaries	Adherence, the cost of the regimen, and whether the effort has become hopeless rather than tiresome
Recurrent decompensation	Repeated admission, unpredictability, lost confidence in the body	Demoralisation, fear of dying in an episode, sleep, the carer's own state
Advanced illness	Anticipated dying, dependence, the meaning of continued treatment	Depression as distinct from a considered response to a real prognosis

The hand-offs are explicit. Demoralisation as a construct, and the screening and treatment of depression and adjustment in a life-limiting diagnosis, are taught in the organ-interface routing section, which also owns palliative care psychiatry and the desire for hastened death. Delirium in the decompensated or dying cardiac patient belongs to the stroke, traumatic brain injury and focal brain syndromes notes, including the bedside instruments.

Cognition is examined rarely and matters constantly. Where attention and processing speed are slowed in advanced disease, a crowded regimen and a consent conversation are at risk, and the risk peaks during a decompensation. Since **heart failure with depressive symptoms ran with poor health-related quality of life**, the stage review is a functional review as much as a diagnostic one.

8.3 Arrhythmia in the psychiatric clinic, and the separation from panic

Arrhythmia reaches psychiatry along several routes: a paroxysmal rhythm disturbance already called panic, an anxiety syndrome that grew out of a documented arrhythmia, a rhythm disturbance and an anxiety disorder present together, and the causes shared by each, which include alcohol, stimulants, thyroid disease and sleep deprivation. The separation is made on history.

Onset and offset carry most of the information. A paroxysmal rhythm disturbance tends to start and stop abruptly, sometimes with a trigger such as a change of posture, and the racing heart precedes the fear. A panic attack builds to a peak over minutes and settles more gradually, fear and bodily symptoms rise together, and catastrophic thinking about the heart is present at the peak. Episodes that wake a person from sleep, that carry syncope, or that occur on exertion rather than at rest shift the balance towards a cardiac cause.

TRAP **Diagnosing panic disorder in a patient whose rhythm has never been recorded during an episode.** They overlap almost completely at the level of reported symptoms, and the label is sticky: once panic is written, every later episode is read through it and the rhythm is never captured. The requirement is an ambulatory or event recording during a typical attack, and the formulation should state whether one exists.

The reverse error is commoner in cardiology. A person with a documented rhythm disturbance and an anxiety disorder is often told the palpitations are anxiety, which is heard as an accusation and produces exactly the hypervigilant self-monitoring that amplifies the next episode. The workable formulation is that the rhythm is real, the fear is real, and each raises the salience of the other.

8.4 Rhythm procedures and the peri-procedural window

Atrial fibrillation is an episodic, bodily felt and unpredictable illness, and unpredictability is the property that generates anticipatory anxiety in any organ system. The load in the group referred for rhythm procedures is substantial: **depressive and anxiety disorders are highly prevalent among patients with atrial fibrillation (AF) undergoing pulmonary vein isolation (PVI)**.

Because **depressive and anxiety disorders are highly prevalent in the group referred for pulmonary vein isolation**, screening belongs before the procedure rather than after a disappointing result. A person anxious before ablation who remains symptomatic after it is easily written off as a treatment failure when the rhythm record is clean. Naming it in advance changes how a residual symptom is read: as an expected part of recovery rather than as proof the procedure failed.

Symptom-rhythm concordance is the idea underneath this. Do the symptoms the patient reports coincide with the rhythm the record shows? Concordance supports a rhythm intervention; discordance says something other than the rhythm is generating the experience, usually interoceptive vigilance, which responds to psychological work rather than to further ablation.

8.5 Living with an implanted defibrillator

An implanted defibrillator is unlike any other treatment a psychiatrist meets, because its therapeutic action is an aversive event delivered without warning from inside the body. The psychopathology follows from that: the therapy hurts, its timing is unpredictable, and it cannot be escaped by leaving the situation. A sudden painful event paired with whatever the person happened to be doing is all ordinary conditioning needs.

Shock-related anxiety is anticipatory: continuous apprehension about the next discharge, with hypervigilant monitoring of the pulse, the chest and any sensation that preceded a previous one. **Avoidance** is the disabling part and the part most often missed, because the person rarely volunteers it. Exertion, sexual activity and driving are avoided in case they provoke the device, being alone in case a discharge occurs with nobody present, and public places because a discharge witnessed by strangers humiliates as well as frightens. After repeated discharges in a short period, intrusive recollection, hyperarousal and phobic avoidance can take a post-traumatic form. Some

patients describe a **phantom shock**, the vivid experience of a discharge the device record shows did not happen.

The household is part of the presentation. Partners commonly witness the discharge, and witnessing what looks like a resuscitation produces its own distress, its own hypervigilance and its own protective restriction of the patient's activity, so a couple can arrive at a shared avoidance that neither of them has named. Hence the recommendation that **psychological assessment, monitoring, and therapy should be offered to ICD patients and their partners as part of routine care**. Each noun does work: assessment at implantation and after any discharge, monitoring because a person well at the device clinic one month may be housebound the next, therapy because detection alone is not a service, and partners because the couple is the unit of care.

RULE **Ask what the person has stopped doing since the device was implanted.** Anxiety is reported readily and avoidance is volunteered almost never, yet avoidance is what removes exercise capacity, work, driving, intimacy and social life. Put the question to the patient and to the partner separately.

TRAP **Reading shock anxiety as simple reassurance-seeking and answering it with reassurance.** Reassurance relieves for minutes and teaches the person that the fear requires an external answer, so the checking increases. The device is genuinely capable of firing, so honest information plus a rehearsed plan for what to do when it fires converts an unbounded threat into an event with a script.

8.6 The device at the end of life, and the deactivation conversation

As cardiac disease advances, the shock function can fire during the dying process, with no prospect of restoring a life the person is already leaving. Deactivating the shock therapy is therefore a planned conversation, held while the person is well enough to hold it and recorded where the treating teams can find it. Psychiatry is asked in for three reasons: capacity, whether depression is colouring the decision, and how to hold the discussion with a family who may hear deactivation as abandonment.

Capacity here is decision-specific and time-specific, so the question is whether this person can make this decision now, and the answer can change with the delirium, the hypoxia or the sedation of an acute episode. The doctrine and the statutory test belong to the mental health legislation notes. What psychiatry adds is the mood assessment underneath: a wish to stop a burdensome therapy late in an illness can be a considered choice, whereas the same wish carried on pervasive hopelessness and a conviction of being a burden requires that the depression be treated and the question asked again.

Families frequently hear deactivation as switching the device off altogether, or as an act that causes the death. What deactivation covers is for the cardiology and device team to answer explicitly, in the family's own language; the psychiatric contribution is to ensure the question is asked and the answer understood.

8.7 Non-drug management, and who does what

Everything here is deliverable without a prescription, which matters where the cardiac regimen is already crowded.

- A written plan for what to do if a discharge occurs or an episode starts: rest, position, who to telephone, when to attend, and when the device record will be checked.
- A graded return to activity, agreed with the cardiologist in writing and shared with the partner. Avoidance is reversed by activity that has been sanctioned; encouragement without sanction moves the conflict into the household.
- Cognitive-behavioural work aimed at the maintaining cycle: catastrophic interpretation of a bodily sensation, checking, avoidance, and the relief that reinforces it. Interoceptive exposure needs explicit cardiology agreement first.
- Paced breathing and applied relaxation, taught as skills for the episode rather than offered as a cure.
- Couple-inclusive sessions as standard rather than as an extra, since **psychological assessment, monitoring and therapy are recommended for defibrillator patients and their partners as part of routine care**.
- Coordination with cardiac rehabilitation where it exists, and with the device clinic in every case, including a question about remote monitoring: it reassures some patients and feeds the vigilance of others.

Sleep is where the cardiac and psychiatric arms of the plan meet. Orthopnoea, nocturia from the regimen, fear of dying in sleep and anticipatory fear of a night-time discharge each fragment the night and each has a different remedy, and asking what wakes the person, and what they do next, separates them faster than any sleep questionnaire.

What belongs elsewhere, named: antidepressant choice, prognostic effect and cardiac safety sit with depression and anxiety after myocardial infarction and in coronary artery disease; organ-adjusted dosing with the psychopharmacology notes; the acutely stress-precipitated cardiomyopathy that mimics infarction with the stress-heart interface section; delirium with the stroke, traumatic brain injury and focal brain syndromes notes; palliative care psychiatry with the organ-interface routing section.

Timing, not the symptom	Stage, not the label	Recorded, not assumed	Avoidance, and the partner
WHAT DECIDES ATTRIBUTION	WHAT ORGANISES THE ASSESSMENT	WHAT THE PANIC LABEL NEEDS FIRST	WHAT THE DEVICE INTERVIEW MUST REACH

Each tile carries a decision rather than a fact, and not one of them can be settled from the symptom in front of you: attribution comes from the timeline, priority from the stage, the panic label from a recording, and the device interview from the partner as well as the patient.

IN INDIA

Device therapy is largely an out-of-pocket purchase, the device clinic may be a day's travel away, and the family rather than the individual usually carries the decision and the follow-up. The couple-inclusive element of the recommendation is therefore easier to deliver here than in services built around the individual patient, while longitudinal monitoring is harder, because the person is seen when they can travel rather than when they are due. A written plan the family can act on at home does more work here than a scheduled review. What this section does not hold is worth stating: no Indian prevalence figure for depression in heart failure, no Indian count of implanted devices, no Indian cost figure for defibrillator therapy.

GOOD TO REMEMBER

A short set of questions does most of the ward work: what has changed since the last cardiac event and in what order, what the person has stopped doing since then, and who else was in the room when the prognosis or the device was explained. Attribution, avoidance, and the partner who is carrying the other part of the distress.

The plate lays the limbs of the assessment side by side: the shared somatic symptoms with the questions that separate them, the arrhythmia presentations and where each routes, the psychopathology of living with a device, and the non-drug management that follows. Prescribing is absent from it by design.

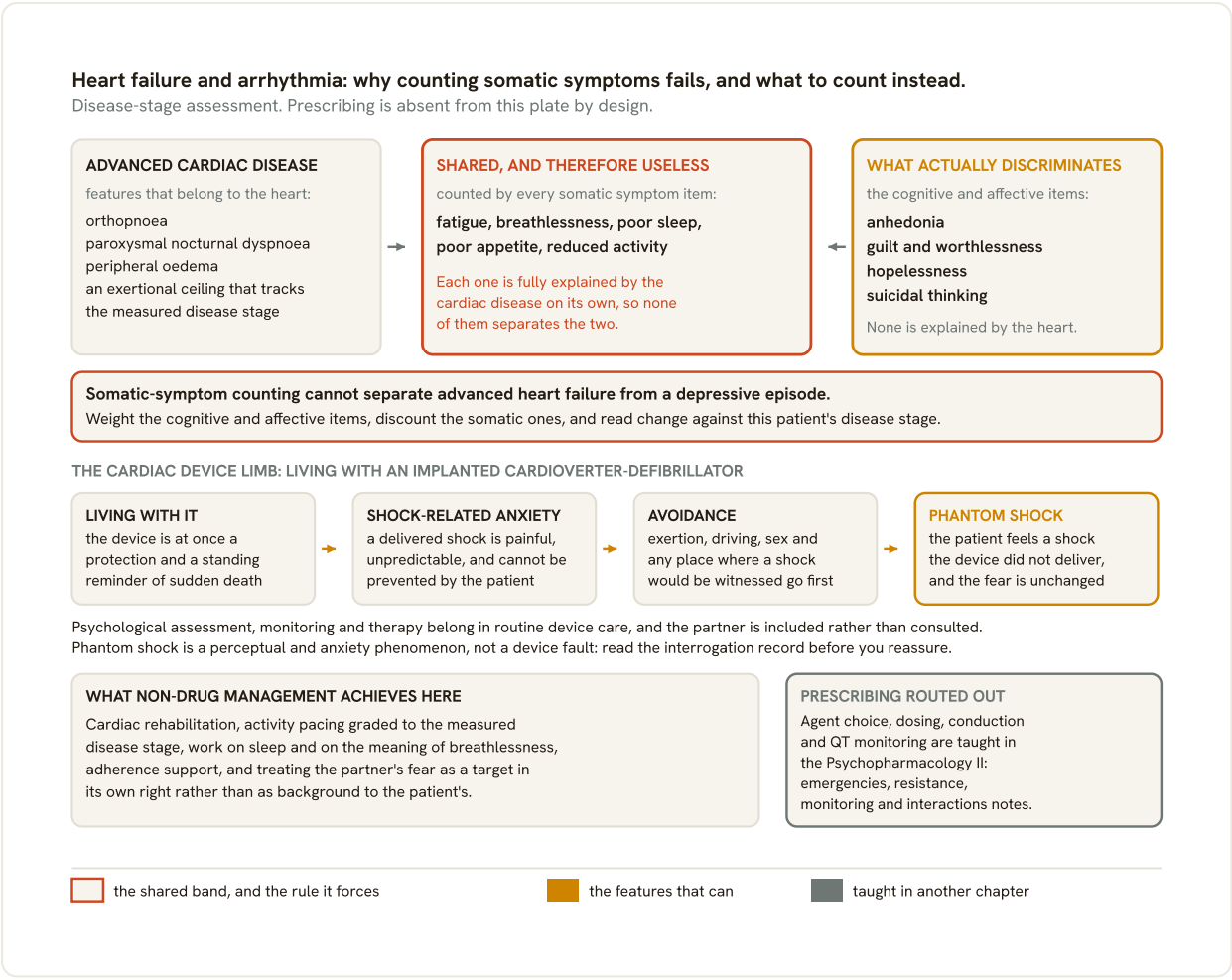

Fig 40.2 Disease-stage assessment in heart failure and arrhythmia, built around the symptom-overlap problem. The centre panel holds the five features a somatic symptom count would score, and every one of them is fully produced by advanced cardiac disease on its own, which is why an instrument weighted toward somatic items inflates in this population and a candidate who counts them reaches the wrong answer confidently. The discrimination lives in the right-hand panel, in items the heart does not generate. The device limb runs on its own logic: the implanted cardioverter-defibrillator protects and threatens at the same time, a delivered shock is painful and cannot be prevented by the patient, avoidance follows, and phantom shock closes the loop by producing the fear without the event. Prescribing is deliberately absent. From memory you should be able to name the five shared features, the four discriminating ones, and the four steps of the device limb, and say why the first list cannot settle the diagnosis.

> Attribute by timing and course, ask what the person has stopped doing since the device was implanted, and treat the couple, since psychological assessment, monitoring and therapy should be offered to defibrillator patients and their partners as part of routine care.

9 · Psychiatric aspects of COPD, asthma and chronic respiratory failure

Breathlessness and panic recruit the same body, and the patient living through either one cannot reliably tell you which of them started. Respiratory psychiatry is examined on that ambiguity and on what a clinician does about it at the bedside. This section installs four things: a working discrimination between the dyspnoea of obstructive airways disease and the dyspnoea of a panic attack; the two specialty-guideline positions this chapter can anchor to a respiratory source; the behavioural levers that move outcome, which are referral to rehabilitation and inhaler adherence; and an honest declaration about chronic respiratory failure, where a specialty

statement was sought and not obtained. Cardiac material is not taught here. Heart failure, arrhythmia and cardiac devices have their own section, and the psychiatric sequelae of critical illness, including post-intensive-care syndrome, sit in the merged organ-interface routing section.

9.1 The discrimination this section exists for

A patient who is breathless, frightened, tachycardic, tremulous and unable to complete a sentence has produced a presentation that two very different processes can generate. Airflow limitation produces breathlessness, and breathlessness produces fear; fear produces hyperventilation, and hyperventilation produces breathlessness. The loop closes in both directions, which is why the question is rarely which one the patient has, and almost always which one is driving the episode in front of you.

Several features shift the probability without settling it. The temporal shape is the most useful single discriminator: a panic attack crescendos and then subsides over a bounded period, whereas an exacerbation of airways disease climbs and stays. The relation to exertion matters, because bronchoconstriction and deconditioning both load onto effort while a panic attack characteristically arrives at rest and out of context. Objective airflow limitation, audible wheeze, a fall in oxygen saturation and a response to a reliever inhaler point one way; circumoral and peripheral paraesthesiae, carpopedal spasm and the light-headedness of hypocapnia point the other, since an obstructed patient usually cannot blow off enough carbon dioxide to produce them. The content of the thought is informative and is often the most accessible part of the history: the catastrophic cognition in panic is about dying or losing control now, and it recurs in the same form.

None of this licenses a clean split, and the examinable position is that it does not need to. A patient with obstructive lung disease who develops recurrent panic attacks has both conditions, each worsening the other, and treating only one leaves the disability where it was. **Anxiety sensitivity**, the tendency to read bodily sensations as dangerous, is the variable that couples them, and it is the one a psychiatrist can treat.

RULE **The dangerous direction of error is treating airflow limitation as anxiety, not the reverse.** Calling a panic attack an exacerbation costs an unnecessary course of treatment. Calling an exacerbation a panic attack can cost a life. Where the two cannot be separated in the room, treat the airway first, interrogate the psychiatry afterwards, and record why.

9.2 Why mood and anxiety cluster in obstructive lung disease, and what they cost

The association is not a coincidence of two common conditions sharing a population, and a candidate who says only that will be pressed. Several distinct pathways converge, and naming them separately earns the marks.

The behavioural pathway is the strongest and the most modifiable. Breathlessness on exertion is aversive, so the patient reduces exertion; reduced exertion produces deconditioning, and deconditioning lowers the threshold at which the next effort produces breathlessness. The spiral narrows the patient's world by degrees and delivers the ingredients of depression, which are loss

of activity, loss of role and loss of contact. From the anxiety side it is avoidance learning: exertion is paired with a frightening sensation, and the patient stops.

Beyond that sit contributions this chapter can name but does not quantify. Tobacco use is a shared risk factor for the lung disease and for several psychiatric conditions, and it means a proportion of these patients carry a nicotine dependence any plan must address. Chronic hypoxaemia and hypercapnia affect attention, processing speed and daytime alertness, and disrupted nocturnal breathing fragments sleep, which independently degrades mood. Systemic corticosteroid exposure is common here and carries its own psychiatric consequences; that construct is owned elsewhere and is named only as a hand-off. Finally, the disease is progressive, visible and frightening with a foreseeable trajectory, and demoralisation in that setting is a reasonable response as well as a clinical problem.

The cost is not only distress. Comorbid mood and anxiety symptoms track with poorer adherence, more frequent presentation to acute services and worse functional status at any given level of measured airflow limitation. No prevalence figure, effect estimate or readmission proportion is held by any claim row available here, so none is stated: carry the direction, and say openly that the magnitude was not obtained.

TRAP **Deciding that low mood in advanced lung disease is understandable, and therefore leaving it alone.** Comprehensibility is not a diagnostic exclusion, and this reasoning would not be accepted for any other comorbidity. It matters more than usual here because the depressive component is what removes the patient from the activity, the rehabilitation and the adherence that would change the trajectory. An understandable depression and a treatable depression are the same depression.

9.3 Pulmonary rehabilitation, and the referral statement to memorise

Pulmonary rehabilitation is a structured programme of supervised exercise training combined with education and self-management support for patients with chronic respiratory disease. It belongs in a psychiatry chapter because it acts on the behavioural spiral above: it is the intervention most likely to reverse the deconditioning, avoidance and social withdrawal carrying much of the psychiatric burden here.

The single most examinable statement this section owns concerns who gets referred. The specialty guideline position is that **coexistent symptoms of anxiety and/or depression in patients with COPD should not preclude referral to pulmonary rehabilitation**. That sentence is worth learning in its shape as well as its content, because it is written as a prohibition on exclusion rather than as an encouragement to refer.

It is written that way because the pressure in real services runs the other way. A patient who appears low, unmotivated or unlikely to complete a course is exactly the patient a stretched programme is tempted to screen out, and the screening is usually informal, done by the referrer, on a judgement about probable engagement rather than by any stated criterion. The guideline blocks that move, and the logic is worth stating in a viva: the psychiatric symptoms are in

substantial part a product of the very deactivation the programme treats, so using them to withhold it removes the intervention from the group with most to gain.

Two boundaries keep the statement honest. It concerns referral and not completion, so supporting attendance, which is where anxious and depressed patients genuinely fall away, remains a clinical task. And no effect size, completion rate or symptom change accompanies the recommendation in the row this chapter holds, so no magnitude is claimed. What a candidate can say without qualification is that coexistent anxiety and depressive symptoms are not a reason to withhold referral to pulmonary rehabilitation, and that the psychiatrist's contribution is often to say so to the referring team.

RULE **Psychiatric comorbidity is an indication to support the referral, not a filter applied before it.** When a respiratory team asks whether a low or anxious patient is suitable for rehabilitation, the guideline-anchored answer is that coexistent symptoms of anxiety and/or depression should not preclude referral. The useful follow-up question is not whether to refer, but what must be arranged so the patient reaches the second session.

9.4 Adherence and technique, which is the behavioural half of disease control

Inhaled treatment is the one therapeutic area where a psychiatrist's ordinary skills change a respiratory outcome, because almost every failure of inhaled treatment is behavioural rather than pharmacological. Two distinctions organise the assessment. The first is between **unintentional non-adherence**, where the patient intends to take the treatment and does not manage it, and **intentional non-adherence**, where the patient has decided against it. The second is between not taking the drug and not delivering it, since a patient with poor inhaler technique is fully adherent and receiving very little.

Psychiatric symptoms map onto these categories specifically rather than generally. Depressive symptoms produce forgetting, loss of routine and the hopeless reasoning that treatment will not help anyway. Anxiety produces overuse of the reliever inhaler and a dependence on it that masks deteriorating control and intermittently reinforces using it again. Beliefs about harm, particularly the fear that inhaled steroid preparations are dangerous or habit-forming, produce quiet intentional non-adherence the patient will not volunteer. Cognitive impairment and manual difficulty produce technique failure independent of all of this.

FAILURE MODE	WHAT IT LOOKS LIKE IN CLINIC	WHAT ACTUALLY CHANGES IT
Technique failure	Correct prescription, exhausted canister, poor control, patient reports full adherence	Watching the patient use the device, then re-teaching or changing the device
Forgetting and lost routine	Erratic use, worse in the morning, tracks with mood and with disorganised days	Anchoring the dose to an existing fixed daily act, plus treating the mood state
Reliever overuse	Frequent reliever use, escalating anxiety, preventer untouched	Naming the cycle explicitly, then treating the anxiety alongside the airway
Belief-driven refusal	Articulate reasons, often about steroids, offered only when asked directly	Eliciting the belief without correcting it first, then addressing the specific fear

The instruction that follows is single and easy to examine: ask the patient to demonstrate, do not ask the patient to report. A demonstration takes less time than the conversation about escalating treatment that follows from assuming the technique was adequate.

Dyspnoea against panic: six discriminators, and the band where they genuinely overlap.
Neither outer column settles the question alone. The middle column is the reason it stays hard.

FEATURE	DYSPNOEA OF COPD OR ASTHMA	WHERE THEY OVERLAP	PANIC ATTACK
ONSET	over minutes to hours, from a baseline that was already limited	both begin with breathlessness and fear, in the same body	abrupt, peaking within minutes from an unremarkable baseline
TRIGGER	exertion, cold air, allergen, infection or a missed preventer	exertion can start either, and fear can follow either	often at rest and out of context, or cued by fear of an attack
TIME COURSE	climbs, and stays up until the airway is treated	a long attack and a short exacerbation look alike	crescendos, then subsides over a bounded period
WHAT RELIEVES IT	a reliever inhaler, and treating the airway itself	reliever overuse eases both for a while, and masks both	slowed breathing and time: the attack is self-limiting
WHAT THE PATIENT DOES	drops exertion, then drops the activities that needed it	avoidance narrows the life the same way from either cause	escapes the situation, carries the inhaler as a safety object
WHAT THE OBSERVER SEES	wheeze, accessory muscle use, falling oxygen saturation	tachypnoea, tachycardia, tremor and visible fear in both	circumoral and peripheral paraesthesiae, carpopedal spasm

The dangerous direction of error is treating airflow limitation as anxiety, never the reverse.
Where the two cannot be separated in the room, treat the airway first, interrogate the psychiatry after, and record why.

LEVER ONE: PULMONARY REHABILITATION
Coexistent symptoms of anxiety and/or depression in patients with COPD should not preclude referral to pulmonary rehabilitation. It is written as a prohibition on exclusion, not an encouragement.

LEVER TWO: INHALER ADHERENCE AND TECHNIQUE
Separate not taking the drug from not delivering it, and unintentional from intentional non-adherence. Then ask the patient to demonstrate the device rather than to report on it.

ROUTED OUT Steroid-induced psychiatric disturbance is taught in the Endocrine and metabolic psychiatry notes.
ROUTED OUT Agent choice, dosing and monitoring are taught in the Psychopharmacology II: emergencies, resistance, monitoring and interactions notes. This plate names no agent and prints no dose.

Fig 40.3 Separating the dyspnoea of obstructive airways disease from a panic attack, with the overlap drawn rather than glossed. The two outer columns are the features that shift probability, read across six axes rather than argued one at a time. The coral column is the honest part: breathlessness and fear recruit the same body, the loop closes in both directions, and a patient with obstructive lung disease who also has panic attacks has both conditions, each worsening the other. That is why the examinable question is almost never which one the patient has but which is driving this episode and in what proportion. The two levers below are where a psychiatrist changes a respiratory outcome, and both are behavioural rather than pharmacological. From memory you should be able to run the six axes in either direction, state the asymmetry of risk in the rule box, and give the referral sentence and the demonstration instruction verbatim.

9.5 Asthma in adolescence, and the association that has to be stated carefully

Asthma carries a second and separate examinable position, and it is age-specific. The specialty guideline records that **asthma in adolescence is associated with an increased likelihood of major depression, panic attacks and anxiety disorder**. Three things about that sentence are worth holding. It names adolescence rather than asthma at any age. It names three outcomes rather than a general elevation in distress. And it states an association, so a candidate who converts it into a causal claim in either direction has overstated the source.

The direction genuinely is uncertain and saying so is the stronger answer. A chronic, visible, sometimes frightening condition that constrains sport, sleep and social life at the stage where all three matter most is a plausible cause of low mood. Anxiety, equally, worsens the perception of breathlessness and the behaviours that control it. Shared factors sit underneath both, including disrupted sleep, corticosteroid exposure and family stress.

The consequence for practice is concrete. Because adolescent asthma is associated with an increased likelihood of major depression, panic attacks and anxiety disorder, mood and panic symptoms are worth asking about routinely in this group rather than waiting for them to be raised, and mood and anxiety are candidate contributors to poor asthma control rather than a separate matter for later. The overlap with panic is awkward because both under-perception and over-perception of airflow limitation occur: the adolescent reporting severe symptoms may be obstructed, panicking, or both, and the one reporting little may be seriously obstructed.

TRAP **Labelling an adolescent's attack as panic and thereby delaying respiratory treatment.** The association above makes panic more likely in this group, which is exactly why the label becomes available and exactly why it is dangerous. An adolescent with asthma presenting with an acute frightening episode is treated for the airway on clinical grounds, and the psychiatric formulation is completed once the episode has settled. Offering reassurance as the first move in an acute presentation answers the question badly, however well the comorbidity is discussed afterwards.

9.6 Chronic respiratory failure, and the gap this chapter declares

The third condition in the section's title is the one for which no guideline statement was obtained, and the honest position has to be stated rather than papered over. A specialty-guideline statement specifically linking chronic respiratory failure with psychiatric sequelae, with psychiatric assessment, or with psychiatric management was sought for this chapter from the respiratory bodies whose statements would carry such a recommendation, being the national thoracic guideline body and the international obstructive lung disease initiative. It was not obtained in a form this chapter can bind. That absence describes what this chapter opened. It is not evidence that no such statement exists, and it must not be converted into a claim that the specialty is silent.

What a candidate does without it is reason from general principles and say that this is what they are doing. Chronic hypercapnia produces daytime somnolence, headache, impaired attention and, as it advances, confusion, any of which can present first as an apparent psychiatric change. Nocturnal hypoventilation fragments sleep, which degrades mood independently. Long-term oxygen therapy and domiciliary ventilation impose visible dependence on a machine and reorganise family life around the equipment. The loss of control is the psychological core, and interventions that return any control to the patient tend to help.

Two boundaries apply. Acute confusional states here are assessed and managed under the delirium material in the stroke, traumatic brain injury and focal brain syndromes notes, and no bedside delirium instrument, rating scale or antipsychotic choice is taught in this section. And a patient declining domiciliary ventilation raises a capacity question; capacity is decision-specific and time-specific, and everything beyond that, including the statutory test, belongs to the mental health legislation notes.

IN INDIA

No figure for the availability, distribution or coverage of pulmonary rehabilitation programmes in India is held by any claim row available to this chapter, so none is offered. What can be said is what the referral position implies for a service that has a programme and for one that does not. Where a programme exists, coexistent anxiety and depressive symptoms are not grounds to withhold referral to pulmonary rehabilitation, and a psychiatrist asked to assess a low patient before referral has a guideline-anchored answer for the respiratory team. Where no programme exists, which is the position across much of the country outside larger centres, the deliverable residue is still the active ingredient: a graded, written, supervised increase in activity that a physiotherapy service, a nursing team or an informed family member can sustain, with education about the breathlessness cycle. The gap in a district service is usually the structure and the supervision, not the exercise.

9.7 What this section hands off, and does not teach

Corticosteroid-induced psychiatric disturbance is common in this population and is owned elsewhere. It is named here as a differential whenever mood, sleep or mental state changes after a course of systemic steroid; the mania, depression, psychosis and management content sits with the corticosteroid owner in the endocrine and metabolic material and on the chapter's routing plate.

Prescribing in respiratory disease routes to the psychopharmacology notes without exception. Sedative burden and respiratory depressant risk are why a psychiatrist is cautious here, and the drug-by-drug reasoning, the comparative rankings and every dose belong there, so none is restated. The same applies to the effect of tobacco smoke on the metabolism of certain psychotropics: it is named as a hand-off, and neither its direction nor its magnitude is stated here.

Non-pharmacological treatment of the anxiety component follows ordinary principles with one adaptation. Interventions that deliberately provoke the feared bodily sensation require care when the airways are genuinely obstructed, because the sensation is not false. The workable version treats the interpretation and the avoidance rather than trying to show the sensation is harmless, and it runs alongside optimised inhaled treatment.

GOOD TO REMEMBER

On the ward, three moves cover most of the value a psychiatrist adds to a breathless patient. Watch the inhaler being used before believing anything about adherence. Ask what the patient thinks is happening during an attack, because the answer separates the catastrophic cognition of panic from the accurate fear of someone obstructed before. And ask whether the patient has been referred for rehabilitation, because anxiety and depressive symptoms are not a reason to preclude that referral and a psychiatric opinion is often what is used to justify not making it.

9.8 The answer, assembled

An examiner asking about psychiatric aspects of respiratory disease wants a structure rather than a list. The following sequence carries this section.

- State the discrimination first: breathlessness and panic share a physiology, the loop runs both ways, and the common answer in this population is both rather than either.
- Give the safe rule of error: treat the airway first when the two cannot be separated, because the costly mistake is calling obstruction anxiety.
- Explain the behavioural spiral of breathlessness, avoidance and deconditioning, since it accounts for much of the presentation and is what treatment targets.
- Deliver the referral position verbatim: coexistent symptoms of anxiety and/or depression in patients with COPD should not preclude referral to pulmonary rehabilitation.
- Deliver the asthma position with its age qualifier and its association wording: asthma in adolescence is associated with an increased likelihood of major depression, panic attacks and anxiety disorder, and say the direction was not established.
- Cover adherence and technique as a behavioural target, and demonstrate rather than ask.
- Declare the chronic respiratory failure gap openly: reason from hypercapnia, sleep, dependence and loss of control, and say that a specialty statement binding psychiatric management to chronic respiratory failure was sought and not obtained.

Both, not either	**Refer, not exclude**	**Watched, not reported**	**Declared, not inferred**
WHAT DYSPNOEA PLUS PANIC USUALLY MEANS	WHAT COEXISTENT LOW MOOD CHANGES	HOW INHALER ADHERENCE IS ASSESSED	WHAT TO SAY ON RESPIRATORY FAILURE

Each tile names a default that ordinary practice sets the other way, which makes the strip an audit of one's own clinic behaviour rather than a summary of the text.

PITFALL

Answering this item as a general essay on the psychological impact of chronic illness. The material is true and it earns very little, because it could have been written about any organ. What makes an answer respiratory is the dyspnoea and panic discrimination, the referral position on rehabilitation, the age-specific asthma association and the behavioural analysis of inhaled treatment.

Breathlessness and panic are usually both present; treat the airway first, refer for rehabilitation regardless of coexistent anxiety or depression, watch the inhaler being used, and ask every adolescent with asthma about mood and panic.

10 · Takotsubo cardiomyopathy and the stress-heart interface

Takotsubo syndrome is the plainest demonstration in medicine that an acute psychological event can produce a structural cardiac lesion, which is why it belongs in a psychiatry paper and not only in a cardiology one. An emotional or physical stressor is followed, usually within hours, by chest pain, breathlessness or collapse, by an electrocardiogram reading as an evolving anterior infarct, by rising biomarkers of myocyte injury, and by a left ventricle gone akinetic over

a territory larger than any single coronary artery supplies. The arteries are then found unobstructed, and the wall-motion abnormality resolves, which infarcted muscle never does. This section teaches the syndrome by mechanism rather than by table: the epidemiological specifics a resident would ordinarily quote are absent from this build's rows, and are declared rather than supplied from memory.

Several neighbouring topics are named here and taught elsewhere, and knowing the boundary is itself examinable. Depression and anxiety after myocardial infarction, and safe antidepressant choice in established coronary disease, belong to the organ-interface routing section. Heart failure, arrhythmia and implanted cardiac devices have their own section. Delirium on the coronary care unit sits with the notes that own delirium, and the psychological sequelae of critical illness with intensive-care psychiatry. Organ-adjusted dosing is a psychopharmacology question. What remains is the mechanism, the differential and the liaison contribution.

10.1 The shape of the syndrome, and why a psychiatrist is called at all

The syndrome is defined by a pattern rather than by any single test, and each element carries weight. There is an acute regional abnormality of left ventricular contraction, extending beyond the distribution of one coronary artery, which makes an occlusive explanation implausible on anatomical grounds alone. There is no culprit obstruction able to account for it. And there is recovery, so the ventricle akinetic on admission contracts normally later. Transience is not a happy outcome appended to the definition, it is part of the definition, and why the diagnosis is partly retrospective.

The name describes the shape the ventricle takes at end systole in the commonest form, a rounded akinetic apex above a narrow hypercontractile base, resembling a narrow-necked pot. That image encodes the paradox the mechanism has to explain: the segment furthest from the base fails while the base works harder than usual.

A psychiatrist is called into this picture for a small number of reasons, and knowing them in advance determines what a useful consultation note contains. The antecedent was psychological and somebody wants it characterised. A psychotropic drug, a substance or a withdrawal state is suspected. The patient is distressed or agitated on a cardiac unit. Or the team, having found clean arteries, is uncertain what to tell the patient and what to arrange on discharge. Each is answerable. Reciting a cardiology description back to a cardiologist is not.

10.2 The catecholamine-surge account, taken in the order the account itself runs

The dominant mechanistic account is a **catecholamine surge** acting on myocardium, and it is best learned as a chain, because each link is separately testable and separately arguable.

The chain begins centrally. A stressor is appraised as threatening, or the body is placed under acute physiological load, and the central autonomic network drives the sympatho-adrenal axis. The adrenal medulla releases adrenaline into the circulation; cardiac sympathetic terminals release noradrenaline into the myocardial interstitium. These are separate deliveries with different distributions, which matters later: circulating adrenaline reaches the whole ventricle, whereas neural noradrenaline concentrates wherever innervation is densest.

The next link is the effect of that load on the myocyte. High catecholamine exposure drives calcium entry and calcium overload, and calcium-overloaded myocytes contract in a disordered, sustained way. The histological correlate described in this setting is **contraction band necrosis**, a pattern distinct from the coagulative necrosis of coronary occlusion, accompanied by oxidative stress, mitochondrial dysfunction and falling cellular energy supply. This is a toxic injury, not an ischaemic one, and the distinction is why the muscle can recover.

A parallel link is vascular. Catecholamine excess is associated with epicardial coronary spasm and, more importantly here, with **microvascular dysfunction**: impaired dilatation of the small vessels that determine actual myocardial perfusion. Perfusion can therefore fall while the large arteries remain widely patent, which reconciles a genuinely injured myocardium with a normal angiogram.

The most interesting link, and the one that best explains why the apex, is receptor level. Adrenoceptor density is described as higher towards the apex while sympathetic innervation is sparser there, leaving the apex disproportionately exposed to circulating adrenaline. The subtype predominating apically can also switch its intracellular coupling: at ordinary concentrations it signals through the stimulatory G protein and increases contraction, and at the concentrations reached in a surge it couples instead to the inhibitory G protein, reducing contraction. On this account the apex is not merely overwhelmed, it is actively switched off, and the switch is protective, sparing the myocyte a lethal calcium load at the cost of regional akinesia.

The final link is **myocardial stunning**. Stunned myocardium is alive but not contracting: the cells persist, the contractile apparatus is temporarily disabled, and function returns when the insult is withdrawn. Stunning is why a ventricle that looks infarcted on admission can look normal later, and it is the single concept that makes the whole syndrome coherent.

RULE **Learn the chain, not the label.** Central appraisal, sympatho-adrenal outflow, adrenal and neural catecholamine delivery, calcium overload with contraction band necrosis, microvascular impairment, apical receptor coupling switch, regional stunning, then recovery. A candidate who can walk that chain can answer any question asked about this syndrome, including those on variants and differential diagnosis, because each attacks a specific link.

10.3 What the account explains well, and where it strains

Good teaching of a mechanism includes its limits, and this one has clear limits. The apical receptor argument explains the commonest morphology elegantly. It explains it too well, because the syndrome also occurs with the akinetic segment midventricular, basal with a hypercontractile apex, and focal. A gradient anchored at the apex predicts apical disease; it predicts basal disease poorly. The honest reading is that the receptor gradient is one contributor within a more general catecholamine-mediated regional vulnerability, and that regional variation reflects unresolved individual differences in innervation, receptor distribution and microvascular reactivity.

The account also under-determines who develops the syndrome. Acute catecholamine surges are common in human life and this presentation is not, so a susceptibility term is required: something about vascular reactivity, hormonal environment, autonomic set point or genetic

variation separating the person who stuns from the far larger number who do not. Naming that term, and saying it is unresolved, is a better answer than asserting a single cause.

This is where a conventional account would supply a demographic profile, since the demographic skew of this syndrome is the specific most often reproduced about it. That figure is exactly what this build cannot supply: the rows available here carry no distribution by sex, no age distribution, and no proportion of acute coronary presentations accounted for by the syndrome. What can be taught instead is what such a figure would require: a consecutive unselected series taken for angiography with suspected acute coronary syndrome, an explicit diagnostic definition applied prospectively, and a stated catchment and period, so that numerator and denominator each mean something. Any figure quoted without those is a number without a population.

TRAP **Treating the receptor gradient as the whole explanation.** It is an attractive and memorable argument, and it accounts for the classical morphology only. Candidates who commit to it are then unable to explain the basal and midventricular variants, and an examiner who asks about variants is usually asking precisely this question.

10.4 Why the presentation is mistaken for an acute coronary syndrome

It is mistaken for an acute coronary syndrome because at first contact it is genuinely indistinguishable from one, which is a property of the syndrome rather than a failure of the clinician. The symptom is the same: chest pain, breathlessness, syncope or shock. The electrocardiogram carries ischaemic change in a territory, commonly ST-segment elevation anteriorly, later T-wave inversion and prolongation of the corrected QT interval. The biomarkers rise, because myocytes really are injured. The ventricle really is akinetic. Every element that ordinarily signals an occluded artery is present, and the artery is not occluded.

What separates them is therefore investigation and time, not acumen at the bedside. Angiography shows no culprit lesion. Imaging shows a wall-motion abnormality crossing coronary territories. Follow-up imaging shows recovery. None of that is available in the first minutes, which is why the correct initial management is the acute coronary syndrome pathway, and why a psychiatrist confident at the bedside that this is stress-related is confident about something they cannot yet know.

The psychiatric traps here run in opposite directions and each is serious. The first is re-labelling: the angiogram is reported clean, the word psychological enters the notes, and the presentation is reinterpreted as anxiety, panic or somatisation. Unobstructed arteries are compatible with a severely injured ventricle, with pulmonary oedema, arrhythmia and haemodynamic collapse: a clean angiogram is not a normal heart. The second is the mirror image: a recognisable emotional antecedent leads the team to a stress explanation while an atherothrombotic occlusion is in progress. A psychological trigger has no power to exclude an occlusion.

TRAP **Reading a clean angiogram as a psychiatric diagnosis.** The angiogram excludes an obstruction; it excludes nothing else. The commonest way this syndrome is mismanaged on a psychiatric liaison list is that the cardiac finding is treated as ruled out and the patient is handed over as an anxiety presentation, at which point nobody is monitoring a ventricle that is still stunned.

10.5 The psychiatric antecedents, and what can honestly be said about them

Antecedents fall into recognisable classes, and the classification here is conceptual rather than quantitative. Emotional antecedents include bereavement, acute interpersonal conflict, fear, grave news, an assault on status or reputation, and acute panic. Physical antecedents include surgery, acute severe illness, sepsis, seizure, respiratory failure and metabolic disturbance, engaging the same sympatho-adrenal pathway with no psychological content. A proportion of cases have no identifiable antecedent of either kind, which matters, because demanding one before entertaining the diagnosis will miss those patients.

A subtler point is valence. The mechanism is driven by arousal and catecholamine load, and arousal is not the exclusive property of aversive emotion, so intensely positive events have also been described as antecedents. This follows from the chain above and is not an anomaly needing separate explanation. What proportion of antecedents is emotional against physical, and what proportion is aversive against pleasurable, are quantitative questions with no grounded answer here.

Comorbidity is a different question from causation, and here this section does hold a grounded statement. **Psychiatric disorders, depression and anxiety among them, are common in patients with this syndrome**, and that is worth stating precisely for what it is: a statement of co-occurrence in the affected population. It establishes that the psychiatrist has a population-level reason to be involved rather than an incidental one.

What it leaves open is the direction of the relationship, and an examiner rewards a candidate who says so. Depression and anxiety may be an antecedent vulnerability, plausibly through sustained autonomic and hypothalamic-pituitary-adrenal dysregulation that alters the response to a surge. They may be a consequence, since surviving a frightening cardiac event with an uncertain explanation generates fear and low mood. They may reflect a shared substrate, with autonomic dysregulation producing each. Co-occurrence is compatible with all of those, and the proportion affected, with any temporal ordering, is a specific this build does not carry.

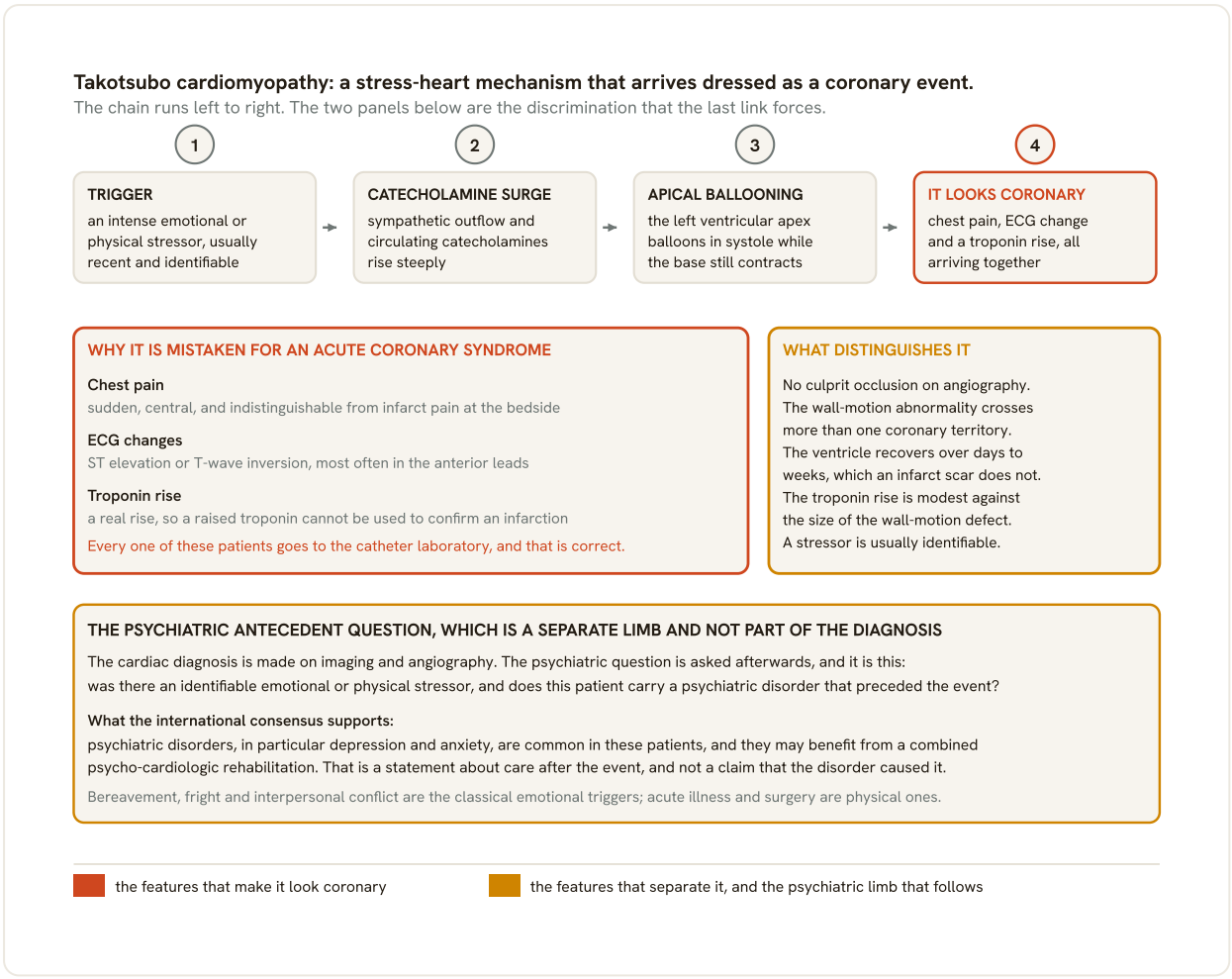

Fig 40.4 The stress-heart interface as a four-link chain, with the mimicry and the discrimination drawn side by side. The mechanism is the examinable spine: a trigger, a catecholamine surge, an apex that balloons in systole while the base still contracts, and a presentation that carries all three cardinal features of an acute coronary syndrome at once. That is why the mistake is not carelessness, and why the correct first move is still the catheter laboratory. What separates the two is the pattern rather than any single test: the wall-motion abnormality does not respect a coronary territory, no culprit occlusion is found, and the ventricle recovers in a way an infarct scar does not. The psychiatric limb is deliberately drawn apart from the diagnosis, because it is asked after the cardiac question is settled. From memory you should be able to give the four links in order, the three mimicking features, at least three discriminating ones, and the reason the antecedent question is a separate limb rather than a diagnostic criterion.

10.6 The differential of the surge, which is largely a psychiatric differential

Because the mechanism is catecholamine load, the differential of the antecedent is a list of states psychiatrists meet constantly, and this is where the liaison psychiatrist supplies information nobody else can obtain.

- **Exogenous catecholamine or sympathomimetic load.** Adrenaline given therapeutically, local anaesthetic containing adrenaline, and sympathomimetic substance use including stimulants and cocaine. The substance history establishes these.
- **Withdrawal states.** Alcohol and sedative-hypnotic withdrawal produce sustained autonomic arousal, and opioid withdrawal a lesser but real adrenergic load. A withdrawal syndrome is an endogenous catecholamine infusion the patient is unaware of receiving.
- **Iatrogenic surges in psychiatric treatment.** Electroconvulsive therapy produces a transient, deliberate and monitored autonomic surge, a recognised context in which this presentation

has been described. Management of that surge belongs to the treatment's own protocol.

- **Hyperadrenergic psychiatric emergencies.** Neuroleptic malignant syndrome, serotonin toxicity and catatonia with autonomic instability sustain adrenergic arousal for hours or days, a longer exposure than a discrete emotional trigger delivers.
- **Acute severe panic and acute stress reactions.** These are most likely to be dismissed as merely psychological on a medical ward, and the mechanism gives a reason to take them seriously as physiological events.
- **Organic causes with psychiatric presentations.** Phaeochromocytoma delivers precisely the surge the mechanism requires and may present with paroxysmal anxiety. Subarachnoid haemorrhage produces neurogenic stunned myocardium by an overlapping pathway, and prolonged seizure activity does the same. Each can reach a psychiatrist first.

Psychotropic drugs with cardiac effects, including corrected QT interval prolongation and the cardiovascular effects of stimulants, are a psychopharmacology topic, cross-referred rather than restated. Their relevance here is that the medication list is part of the surge history.

FIGURE CONVENTIONALLY QUOTED	WHAT WOULD HAVE TO BE MEASURED	WHAT THIS BUILD HOLDS
Frequency among suspected acute coronary presentations	Consecutive unselected patients angiographed for suspected acute coronary syndrome, prospective definition, stated catchment and period	Not grounded here
Sex and age distribution	Composition of a consecutive diagnosed series, read against the composition of the source population	Not grounded here
Emotional against physical antecedents	Antecedent classified prospectively by stated criteria in every case, including cases with none identified	Not grounded here
Time to recovery of wall motion	Protocol-defined repeat imaging at fixed intervals, with a stated definition of normalisation	Not grounded here
In-hospital complications and mortality	Pre-specified complication set, admission to discharge, on a consecutive series	Not grounded here
Recurrence	Cumulative recurrence over a stated follow-up period, with defined ascertainment	Not grounded here
Depression and anxiety in this population	Case ascertainment by a stated diagnostic method, at a stated point after the acute event	**Qualitative statement of commonness only**

10.7 What the psychiatrist on the liaison call actually contributes

The consultation has a small number of jobs, and doing them well is the difference between a note that changes management and a note that restates the referral.

The first is to characterise the antecedent properly. A cardiology record says stress. A psychiatric record says what happened, at what hour, with whom, what it meant to this person, and how long the arousal lasted before the chest pain began. That timed narrative is the only evidence anyone will have about the trigger, it can be obtained on the day and rarely afterwards, and no other member of the team is trained to take it.

The second is to establish the exogenous and withdrawal load, using the differential above. This is a diagnostic contribution, because a withdrawal syndrome or a stimulant exposure changes what the cardiac team monitors and treats.

The third is to separate acute distress proportionate to a frightening event from a psychiatric disorder requiring treatment. The responses differ, and conflating them either medicalises normal fear or misses treatable illness.

The fourth is explanation, and it is underrated. The patient has been told they had something resembling a heart attack and also that their arteries are clean, and those messages conflict unless somebody supplies the mechanism reconciling them. A coherent, non-technical account of a surge, a stunned muscle and a recovering ventricle does real therapeutic work, replacing an incoherent threat with a comprehensible one.

The fifth is treatment and follow-up. **Where depression or anxiety is present, it is treatable in its own right, and a combined psycho-cardiologic rehabilitation approach is the model these patients might benefit from.** The obstacle is structural: a patient discharged with a normal angiogram is likely to leave with follow-up from neither service, and arranging it before discharge is the most useful line in the consultation note. Choice of agent in cardiac disease is a psychopharmacology matter and is handed over. Where consent arises, the operating principle is that capacity is decision-specific and time-specific, and the doctrine belongs to the mental health legislation notes.

RULE **Write the timed antecedent narrative on the day you are called.** Everything else in the consultation can be reconstructed later from records and from the patient. The hour-by-hour account of what happened before onset degrades within days, and it is the one datum that makes the case interpretable to anyone reading the notes afterwards.

IN INDIA

This build located no Indian series, registry entry, service description or cost datum for this syndrome, and none is supplied. What can be said structurally, without inventing a figure, is that recognition depends on early angiography, on repeat ventricular imaging to demonstrate recovery, and on a liaison service that receives the referral before discharge, and that the availability of each varies widely across Indian settings. A resident asked about the Indian picture is on firmer ground naming those dependencies and the absent data than quoting a prevalence figure with no Indian denominator.

GOOD TO REMEMBER

On the ward, hold these points. A clean angiogram excludes an obstruction and excludes nothing else. A psychological trigger never excludes a coronary occlusion. And the muscle is stunned rather than dead, which is why the story ends in recovery, and why the psychiatric work of characterising the antecedent, treating comorbid illness and arranging follow-up is done during an admission everyone else treats as resolved.

MNEMONIC

Surge, Switch, Stun, Settle

Surge is the catecholamine load from adrenal and neural sources. Switch is the apical receptor coupling change from stimulatory to inhibitory. Stun is living but non-contractile myocardium. Settle is the recovery that makes the diagnosis retrospective. The psychiatric work attaches at the start, on the antecedent, and at the end, on comorbidity and follow-up.

Beyond one territory	Stunned	Common	Timed narrative
THE WALL-MOTION FEATURE THAT MAKES OCCLUSION IMPLAUSIBLE	NOT INFARCTED, WHICH IS WHY IT RECOVERS	DEPRESSION AND ANXIETY IN THIS POPULATION, STATED QUALITATIVELY	THE DATUM ONLY THE PSYCHIATRIST CAN OBTAIN

A surge of catecholamines stuns living myocardium, the presentation copies an acute coronary syndrome exactly because the muscle really is injured, and the diagnosis is completed by recovery rather than by the angiogram. Take the timed antecedent on the day, look for exogenous and withdrawal catecholamine load, treat the comorbid depression and anxiety, and arrange the follow-up a normal angiogram will otherwise cancel.

Renal, transplant and perioperative psychiatry

11 · Pre-transplant psychiatric evaluation and post-transplant neuropsychiatry of immunosuppressants (steroids, tacrolimus, ciclosporin)

A transplant psychiatric opinion is read by a committee allocating a scarce organ, which makes it the one psychiatric assessment whose reasoning has to be legible to people outside

psychiatry. The topic splits cleanly and the sides are examined differently. Before transplantation, the question is what the psychosocial assessment must contain and how its conclusion should be written. Afterwards, it is which neuropsychiatric syndromes the immunosuppressants produce, and how to keep that possibility open while a recipient with several plausible causes of a new confusional state is worked up. The routing is strict, and one part of it is a gap rather than a destination. The general machinery by which a co-prescribed agent alters exposure, the inhibitor and inducer ladders and the cytochrome pathways, belongs to the psychopharmacology notes; that chapter names no immunosuppressant, so the interaction rankings and dose changes specific to these drugs are stated nowhere in this series and must not be reconstructed from memory. The confusional state itself belongs to the stroke, traumatic brain injury, delirium and focal brain syndromes notes. The steroid-induced psychiatric syndrome is routed on the plate below and taught in the endocrine and metabolic notes, while dialysis and the intensive-care sequelae around a transplant admission sit in the organ-interface routing section.

11.1 Where the opinion's form is owned, and what this section adds

Two limbs of this assessment are owned elsewhere and are named here rather than taught. How the opinion itself should be written, and the dual agency the interview runs under, belong to the Consultation-liaison, geriatric and transcultural psychiatry notes; so does the separate assessment of a living donor, where the question is whether the offer is voluntary and free of inducement, and where the psychiatrist works for the donor alone. The statutory framework governing donor authorisation belongs to the mental health and medical legislation notes. What this section adds is the limb those chapters do not carry: what the psychosocial assessment must contain, and how a capacity finding made during organ failure should be scoped, dated and re-asked.

11.2 The psychosocial assessment, and the limb this packet grounds

Guidance on evaluating candidates for kidney transplantation specifies the content of the psychosocial assessment, and the limb concerning psychiatry is stated explicitly. The elements of that assessment should include **a mental-status examination**, together with cognitive evaluation to ensure valid decision-making capacity and the ability to provide informed consent for transplantation.

Read that carefully, because the source is narrower than the way the topic is usually taught. It attaches the requirement to the psychosocial assessment as such, and attaches no timing qualifier of its own. This fragment therefore states it at that scope, and treats "pre-transplant" as a description of the setting in which the assessment is customarily performed rather than as part of what the guidance recommends.

The cognitive limb is the one candidates under-weight and the one most likely to change the answer. Organ failure impairs cognition by several routes at once, and the assessment often happens at the point of greatest impairment: uraemia, hepatic encephalopathy, hypoxia, poor cerebral perfusion and disrupted sleep. A patient examined in that state may appear unable to hold a decision they could hold comfortably once dialysis, oxygenation or ammonia handling

improved. So date the finding, name the reversible contributors, and set a point at which the question is asked again.

That licenses the one doctrinal point this section may state. Capacity is **decision-specific and time-specific**: a person may hold capacity for one decision and lack it for another on the same afternoon, and may regain it as their physiology improves. The statutory test, and the machinery that follows a finding of incapacity, are taught in the mental health legislation notes and named here rather than reproduced.

TRAP **Recording a capacity finding as a property of the patient rather than of a decision at a time.** In a population whose cognition tracks a treatable metabolic state hour by hour, writing "lacks capacity" as though it were a diagnosis removes a patient from consideration on a measurement taken at their worst, and the harm is invisible in the notes.

11.3 The rest of the interview, and where its evidence lives

Beyond the capacity limb, the assessment covers ground this packet does not ground with a source, so the honest move is to teach the structure of the interview rather than thresholds nobody here can cite. The domains a unit expects an opinion to address are the psychiatric history and current mental state; substance use and what any stated abstinence rests on; the adherence record; the caring arrangements after discharge; the patient's own account of the regimen and its permanence; and the financial arrangements that will keep immunosuppression running once the admission ends.

One interviewing discipline deserves stating outright, because it is where weak assessments go wrong. Ask about behaviour already on the record in preference to intention offered in the room. The record can be checked: dialysis attendance, missed appointments, prescription collection, what happened the last time a regimen became inconvenient. Why a stated intention is the weaker evidence here, given who the opinion is written for, is argued in the Consultation-liaison, geriatric and transcultural psychiatry notes. That is a point about where evidence lives, and this packet holds no figure licensing any claim that a given behaviour predicts a graft outcome.

Mental state and cognition	Record, not intention	Decision and time specific	Dated, then re-asked
WHAT THE ASSESSMENT MUST CONTAIN	WHERE ADHERENCE EVIDENCE LIVES	HOW CAPACITY IS SCOPED	WHAT A COGNITIVE FINDING IS

11.4 Tacrolimus, and the shape of calcineurin-inhibitor neurotoxicity

The calcineurin inhibitors are what make post-transplant care a psychiatric topic rather than a purely medical one. Their neurotoxicity is prominent enough in the labelling to belong on the differential of every neuropsychiatric presentation in a recipient. The tacrolimus prescribing information states the shape directly: tacrolimus may cause a spectrum of neurotoxicities, and the most severe of these include **posterior reversible encephalopathy syndrome (PRES), delirium, seizure and coma**.

Treat the word spectrum as the examinable part. A label describing a spectrum and then naming only its severe end is telling you a milder end exists while leaving it undefined. This packet holds

no row describing that milder end, so this fragment names the severe end the source names and goes no further.

What the statement licenses is a low threshold, and not a diagnosis. A recipient on tacrolimus who develops a seizure, a fluctuating confusional state or a depressed level of consciousness has a drug on the differential from the first minute, alongside infection, metabolic derangement, graft dysfunction and cerebrovascular events. The observation raising the drug's rank is temporal: whether the change followed an alteration in the regimen, a change of formulation, or the addition of another agent. Which agents do that, in which direction, and what dose response is then required are prescribing questions owned by the psychopharmacology notes.

Note the boundary inside that list: delirium appears as a manifestation of the drug's toxicity, which puts the drug into the delirium differential without putting delirium assessment into this section.

11.5 Ciclosporin, and a signal the label attaches to a combination

Ciclosporin is the other calcineurin inhibitor in routine use, and its labelling carries a neurotoxicity statement structured differently from the tacrolimus one, which is why it is worth learning separately. The statement is conditional on a co-prescription: convulsions have been reported in adult and paediatric patients receiving ciclosporin, particularly in combination with high-dose methylprednisolone.

Several features of that sentence repay attention.

- The signal is attached to a combination. A convulsion in the days after pulsed corticosteroid for suspected rejection is a combination event, and the history must establish what else was given and in what order, rather than settling on whichever agent the patient has taken longest.
- The labelling names adult and paediatric patients alike, so the statement spans the age range and a child or adolescent recipient attracts the same consideration.
- The wording reports occurrence rather than a rate. It licenses vigilance and a position on the differential; it licenses no statement about how often this happens.
- The named partner is a corticosteroid, so the same episode sits inside the corticosteroid material routed below. Naming the combination is this section's job; the steroid syndrome is not.

The plate below arranges the direct neuropsychiatric toxicity of the immunosuppressants by agent class rather than by syndrome, which is the arrangement answering the ward question: you know what the patient is taking and you want to know what it can do. The calcineurin inhibitors and the mTOR inhibitors are marked as interaction-sensitive, and that mark is a pointer rather than a teaching, since the interaction rankings and dose changes they oblige sit in the psychopharmacology notes. This packet holds no row describing an mTOR-inhibitor neuropsychiatric syndrome, so the prose asserts nothing about that class.

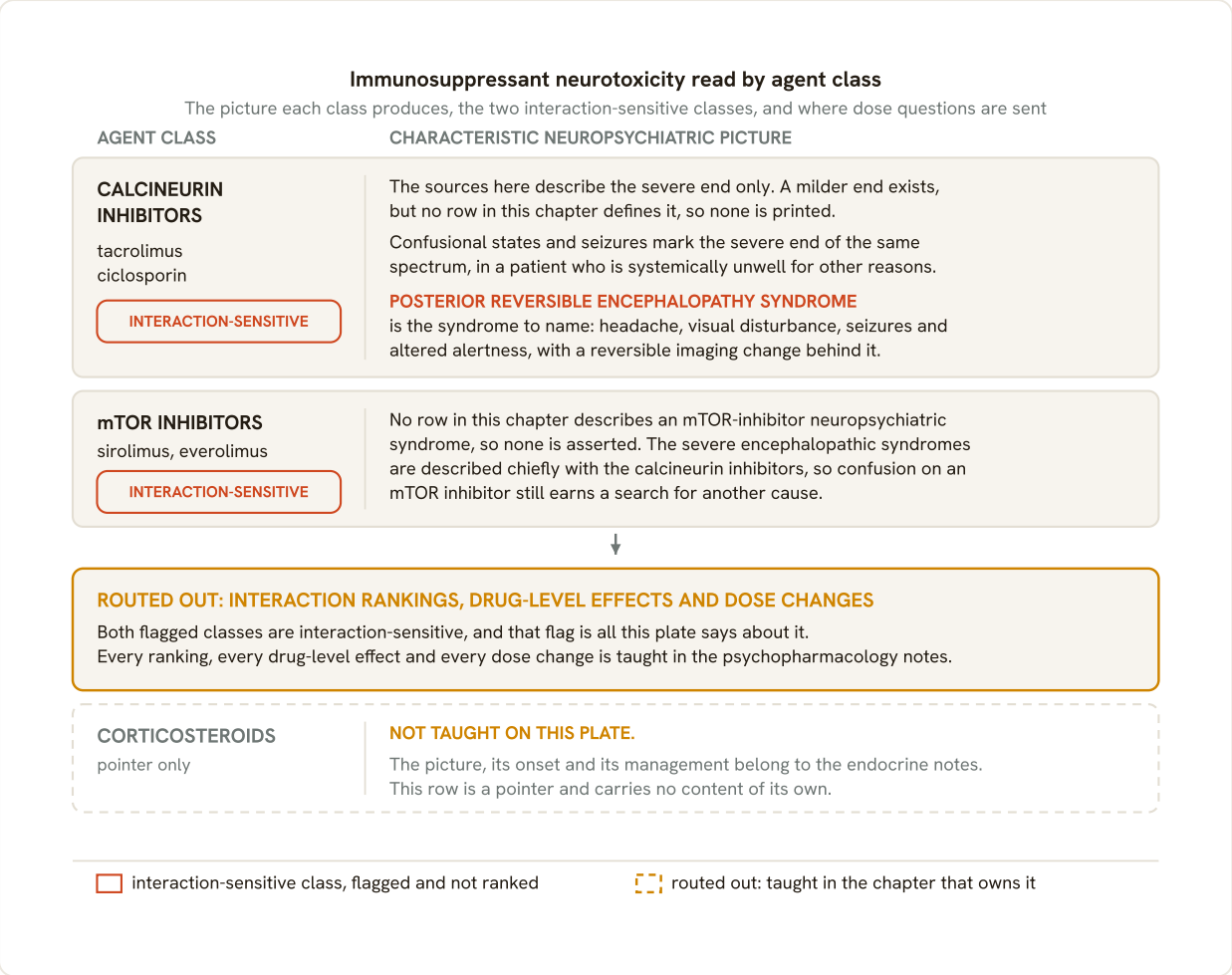

Fig 40.5 Direct neuropsychiatric toxicity of the immunosuppressants, read by agent class rather than by symptom. The calcineurin inhibitors carry the fullest picture: confusional states and seizures at the severe end, and posterior reversible encephalopathy syndrome as a named syndrome rather than a loose description of a confused transplant recipient. The milder end of that spectrum is left undefined here, deliberately, because no source in this chapter's evidence defines it, and naming a spectrum while supplying only one end of it from memory is how a plate acquires content its text does not hold. Nothing is asserted about an mTOR-inhibitor neuropsychiatric syndrome either, for the same reason; what the plate does say is that the severe encephalopathic syndromes are described chiefly with the calcineurin inhibitors, so confusion in a patient taking an mTOR inhibitor still earns a search for another cause before the drug is blamed. Three limbs are deliberately absent from the plate. Interaction rankings, drug-level effects and every dose change are taught in the Psychopharmacology II: emergencies, resistance, monitoring and interactions notes; the corticosteroid picture is taught in the Endocrine and metabolic psychiatry notes; and the form of the pre-transplant opinion, together with the separate living-donor interview, belongs to the Consultation-liaison, geriatric and transcultural psychiatry notes. What that chapter records as a declared gap, the contents of the psychosocial assessment, is taught here. No Indian incidence figure is printed here, because the record was not located. From memory you should be able to name the class, give its severe picture, say the one syndrome out loud, and say which limbs this plate deliberately does not carry.

11.6 Corticosteroids, named here and taught elsewhere

Corticosteroids belong to the transplant regimen and therefore to this chapter, but the psychiatric syndrome they produce is owned elsewhere, so this section states the toxicity at label level and routes the clinical construct. Behavioural and mood changes sit among the common adverse reactions listed for corticosteroids, and that is the level at which this fragment may state them.

Steroid-induced psychiatric disturbance itself, meaning the mania, depression and psychosis that follow corticosteroid exposure and the management of each, belongs to the organ-interface

routing section of this chapter and to the endocrine and metabolic notes. An examiner asking about it in a recipient is asking a question whose answer is the same as in any other steroid-exposed patient: the transplant setting adds a co-prescribed calcineurin inhibitor and an immunosuppressed host, and adds no separate steroid syndrome.

11.7 The recipient with new neuropsychiatric signs, and the order of the questions

The characteristic difficulty after transplantation is that several sufficient causes are present at once, so the examinable skill is sequencing rather than selection. An immunosuppressed patient may be septic while barely febrile, metabolically deranged from graft dysfunction, hyponatraemic, hypoxic, and established on a calcineurin inhibitor and a corticosteroid at once, while carrying the psychological load of an operation they may have waited years for. An answer that names the drug and stops has selected the interesting cause rather than the first one.

The sequence that survives scrutiny puts reversible and dangerous causes first, keeps the immunosuppressant permanently on the list rather than at the end of it, and treats the psychological formulation as concurrent rather than as a diagnosis of exclusion. A recipient's distress about the donor, the graft, or a life reorganised around a regimen is real whether or not a metabolic cause is found, and deferring it until everything else is excluded means it is never addressed.

WHAT YOU OBSERVE	WHAT IT OBLIGES YOU TO ASK	WHERE IT IS TAUGHT
Fluctuating confusion or altered arousal	What is the medical cause, and what is the current regimen	Stroke, traumatic brain injury, delirium and focal brain syndromes notes
Seizure on a calcineurin inhibitor	What else was given, in what order, and when relative to the event	Label level here; prescribing in the psychopharmacology notes
Mania, depression or psychosis after corticosteroid exposure	Which agent, over what course, and what is the earlier psychiatric history	Organ-interface routing section; endocrine and metabolic notes
Doubt about a listing decision or an ongoing consent	Which decision, at what time, and what is reversible now	The requirement here; the statutory test in the legislation notes
Low mood on dialysis while awaiting a graft	What is the renal context, and what treatment is safe in it	Organ-interface routing section, which owns dialysis

Read the middle column rather than the first. Observation-to-diagnosis pairs fail on a stem where the observation has several sufficient causes; observation-to-question survives it, because the question stays the same whichever cause is operating.

11.8 The Indian setting, and what the assessment has to ask in it

Indian practice shapes this assessment more than any refinement of technique. Where the graft comes from a living relative, the psychiatrist acquires a second person to assess, with a different question and a different loyalty, inside a family in which pressure on a potential donor may be entirely unspoken. And because maintenance immunosuppression is a lifelong recurring cost, the sustainability of the regimen is a clinical variable rather than an administrative one.

IN INDIA

Ask the funding question in plain words, at the assessment and again before discharge: who will pay for immunosuppression once the admission is forgotten, and what happens if the recipient was the household's earner. Ask the travel question the same way, since a review schedule assuming a nearby clinic quietly excludes families who cannot make the journey. Where the donor is a relative, hold the separate donor interview the Consultation-liaison, geriatric and transcultural psychiatry notes describe, and record what was asked about voluntariness rather than only the conclusion. This packet holds no Indian figure for transplant volumes, living-donor proportion, drug cost, or psychiatric morbidity in Indian cohorts, so this box states the questions and states no magnitude.

GOOD TO REMEMBER

A short set of sentences carries most of the marks. The psychosocial assessment should include a mental-status examination and cognitive evaluation of decision-making capacity and the ability to provide informed consent for transplantation. Tacrolimus produces a spectrum of neurotoxicity whose severe end includes PRES, delirium, seizure and coma. Convulsions have been reported with ciclosporin, particularly in combination with high-dose methylprednisolone, and behavioural and mood changes are listed among the common corticosteroid adverse reactions.

PITFALL

Attributing a post-transplant seizure to ciclosporin alone. The labelled signal is conditional on a combination with high-dose methylprednisolone, so an answer naming the calcineurin inhibitor and omitting the corticosteroid has reported part of the statement and has stopped taking the history where it was about to become informative. The reverse error costs the same marks.

The pre-transplant side this section owns is the content of the psychosocial assessment, a mental-status examination and a dated cognitive evaluation of capacity, with the opinion's own form owned elsewhere; the post-transplant side is a permanently open differential in which tacrolimus carries a spectrum reaching PRES, delirium, seizure and coma, ciclosporin carries a convulsion signal tied to high-dose methylprednisolone, and corticosteroids are named here and taught elsewhere.

12 · Perioperative psychiatric management, capacity for surgery and postoperative cognitive dysfunction

Surgery reliably produces a new psychiatric presentation in patients who had none the week before. Perioperative psychiatry is best learned as a sequence, because the questions arrive in a fixed order and each has a different owner. Before the operation the question is optimisation and measurement. At consent it is capacity. Afterwards it is which cognitive construct is being named. This section installs that sequence and makes the hand-offs a candidate is expected to make rather than answer: delirium assessment and management, taught with the stroke, brain injury and focal syndrome material, and capacity doctrine with its statutory test, taught with the mental health legislation material.

12.1 The perioperative sequence, and who owns each question

A psychiatrist meets a surgical patient in a small number of recognisable ways, and naming them is the fastest route to a structured answer. The first is the planned preoperative review of a patient with established psychiatric illness, asking whether the illness or its treatment changes the operation or the recovery. The second is the urgent review at consent, where the team is unsure whether the patient can make the decision in front of them. The third, by far the commonest, is the postoperative call about a patient who was lucid on admission and is now confused or agitated. The fourth, most often missed, is the late review months afterwards, where the complaint is not confusion but persisting difficulty with memory or work.

Those shapes belong to different literatures and different chapters, and an examiner wants them separated before the answer starts. The characteristic failure is a single undifferentiated account of postoperative confusion, which loses marks in both directions: it imports delirium teaching into a question about cognitive trajectory, and trajectory language into a question about a disturbed patient on the ward tonight. So fix ownership first. The acute confusional presentation is the delirium chapter's, entire; consent and its statutory machinery are the legislation chapter's; agent, dose and organ-adjusted prescribing are the psychopharmacology chapter's. The plate below traces the pathway from preoperative review, through the operation, to the postoperative neurocognitive outcome, marking each exit where it occurs.

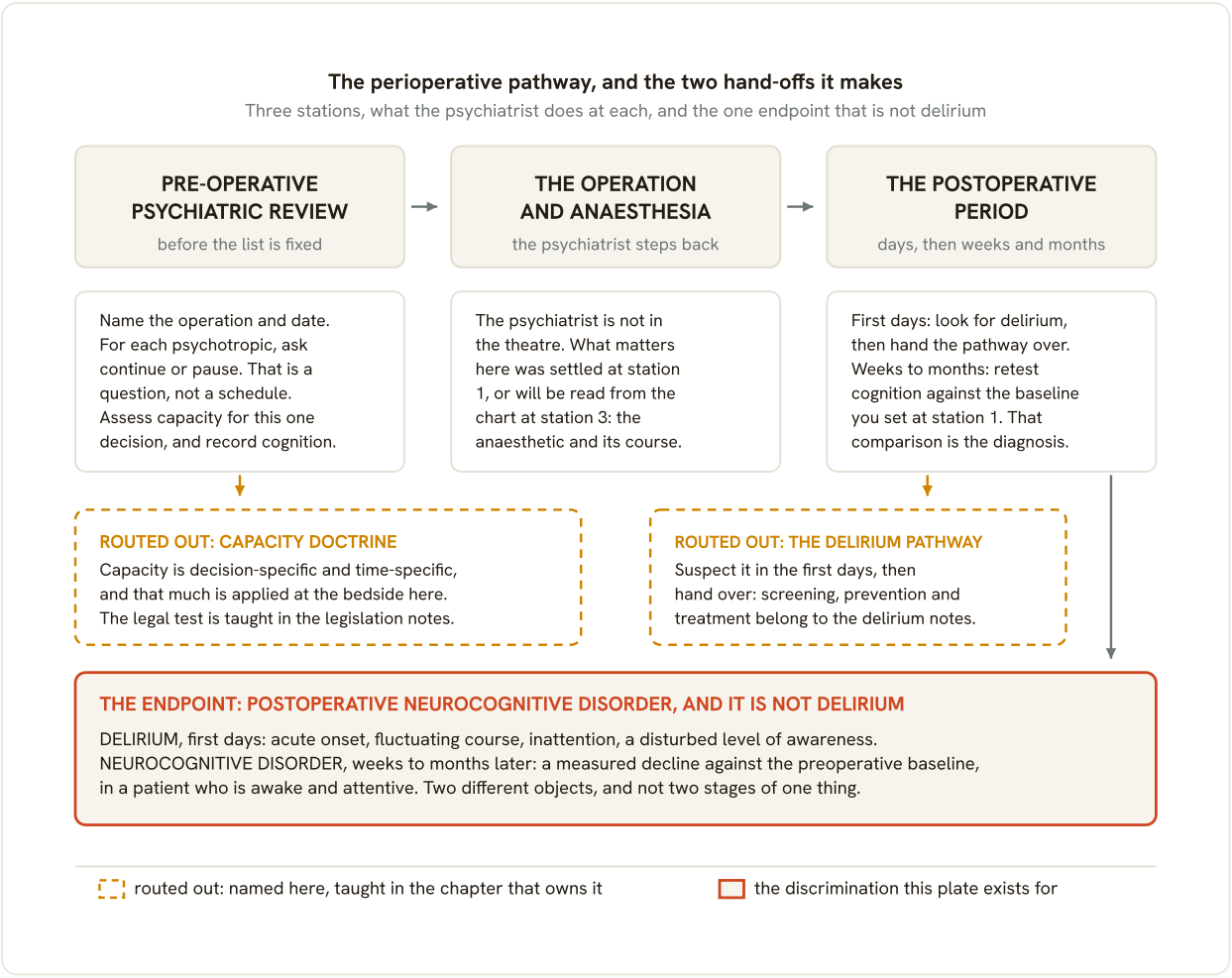

Fig 40.6 The perioperative pathway drawn left to right, with the psychiatrist's task at each station and the two hand-offs marked as branches off the rail. Station 1 is where the work is actually done: medication decisions are framed as questions rather than as a schedule, capacity is assessed for this one decision at this one time, and the cognitive baseline is recorded, because without that baseline the endpoint at station 3 cannot be measured at all. The capacity branch routes to the Mental health legislation and forensic psychiatry notes, which hold the legal test, and consent to the surgical decision is set out in the Forensic ethics, consent and legal procedure notes; the delirium branch routes to the Stroke, TBI, delirium and focal brain syndromes notes, which hold screening, prevention and treatment. The endpoint is postoperative neurocognitive disorder, and a nomenclature working group has recommended perioperative neurocognitive disorders as the overarching term for cognitive impairment identified in the preoperative or postoperative period. From memory you should be able to state what the psychiatrist does at each station, name both hand-offs, and separate delirium from postoperative neurocognitive disorder on onset, attention and course.

12.2 The name of the thing, and why the name was changed

The older vocabulary here was built by researchers rather than clinicians, and it showed. Terms for cognitive change after anaesthesia and surgery accumulated separately, each defined by whichever measurement a study happened to use, so a single patient could be described in incompatible ways depending on which literature the describer had read.

A nomenclature consensus working group addressed this by proposing a single umbrella. **The working group recommends that perioperative neurocognitive disorders be used as an overarching term for cognitive impairment identified in the preoperative or postoperative period.** Read that formulation slowly, because it does more than rename an old idea.

Notice what kind of statement it is: a recommendation about terminology, issued by a working group, and it should be presented as such. An answer promoting it into a diagnostic category with

criteria and thresholds has claimed more than the recommendation says. Notice next where the umbrella reaches. It covers impairment identified in the preoperative period as well as the postoperative period, which is the counter-intuitive limb and the examinable one. That decision converts preoperative cognitive assessment from a chore attached to the construct into part of the construct itself.

RULE **The umbrella reaches backwards, not only forwards.** The overarching term covers cognitive impairment identified before the operation as well as after it. A candidate who describes it as a class of postoperative complication has described half of it, and has lost the reason preoperative assessment belongs in the same discussion.

12.3 The preoperative psychiatric review, and what it is actually for

A preoperative review earns its place by settling a short list of questions where the surgical and anaesthetic teams will find the answers. The first is continuity of psychotropic treatment across the fasting window and any period in which the patient cannot swallow. The review does not choose the agent, which belongs with every dose and interaction ranking to the psychopharmacology material, but it must state, for each agent, whether it continues, is withheld or is given by another route, and which team owns that decision. A plan recording only that the patient takes long-term medication has settled nothing.

The second is withdrawal. An elective admission removes access to alcohol, to nicotine and to anything else the patient used at home, at a predictable hour. A withdrawal syndrome declaring itself on a surgical ward is read, by people looking for a surgical explanation, as postoperative confusion. Establishing what the patient used before admission is preoperative work, and recording it where the postoperative team will see it is the same job.

The third is the illness features that change postoperative risk without changing the operation: patients who communicate pain poorly, who do not seek help when something is wrong, who become agitated in unfamiliar surroundings, or whose treatment history makes an interruption expensive. Each is a practical problem invisible to a clerking that asks about organs. Pre-transplant psychiatric evaluation shares this shape with a different content and a different threshold, and is taught by the transplant section of this chapter.

12.4 The preoperative cognitive baseline, and why it is not optional

The most useful contribution a preoperative review makes to the postoperative period is a measurement taken before the operation. **Older patients should have preoperative cognitive assessment using established screening or diagnostic tools.** The recommendation names a class of instrument rather than a particular one, and this section follows it exactly. Naming a specific instrument here would need a source that attaches a property to it, and none is held, so supplying one from recollection is the error this chapter exists to prevent.

The reasoning is worth stating, because it is what makes the recommendation defensible rather than merely official. Postoperative cognitive change is a change, and a change is a relation between two observations; one postoperative observation cannot establish it. Without a

preoperative measure the team compares the patient against an impression of the patient, formed by relatives under stress and reported after the event.

There is a quieter second yield. Impairment found at the preoperative assessment is informative before any operation happens: it marks a patient whose recovery needs more support, it bears on how the consent conversation is conducted, and it frequently identifies impairment nobody had recognised.

TRAP **Reporting a low postoperative score as a postoperative deficit.** Candidates do this because a score looks like evidence and because the postoperative score is the one they have. Without a preoperative measure it establishes the patient's current level and nothing about change, and a patient with unrecognised pre-existing impairment generates the same result. The examinable point is not the score, it is the comparison the score was supposed to enter.

12.5 Capacity for surgery, and the part of it that belongs here

Almost all of capacity doctrine sits elsewhere. The statutory test and its limbs, the presumption the law starts from, the safeguards and the procedure that follows a finding of incapacity are taught with the mental health legislation material, and none of it is restated here or cited by provision. One property does belong in this section, because the perioperative period is where it does most of its work. **Capacity is described as decision-specific and time-specific.**

Decision-specific means the question is never whether the patient has capacity as a standing attribute, but whether this patient can make this decision. A patient may be unable to manage a complex financial arrangement and entirely able to decide about an operation, and the reverse holds equally. An opinion recorded for one purpose therefore does not answer a different question asked later.

Time-specific means the answer attaches to a moment rather than to a person, and the perioperative period supplies an unusual density of moments in which it can move: pain, disturbed sleep, premedication and the hours immediately after anaesthesia all sit inside the window in which consent is sought. An opinion formed on the morning ward round is not transferable to a consent conversation held that evening. What the psychiatrist supplies is a description of the patient's functional abilities for the specified decision at the specified time, in the terms the legislation sets out; the decision itself belongs to the treating team.

PITFALL

Recording a global verdict. A note stating that the patient lacks capacity, with no decision named and no time attached, is unusable by the team receiving it and indefensible if it is later examined. It also travels: written once, it is quoted for months, across decisions it was never about. Name the decision, name the time, confine the opinion to both.

12.6 Postoperative neurocognitive disorder distinguished from delirium

The two constructs are distinguished by what kind of thing each one is, not by comparing symptom lists. Delirium names an acute disturbance of attention and awareness with a characteristically fluctuating course. It is a condition in its own right, with its own screening

instruments, precipitant list and management, all taught with the stroke, traumatic brain injury and focal brain syndrome material and none of it repeated here. Postoperative neurocognitive disorder names something structurally different: a change in cognitive performance relative to a preoperative baseline, established by comparison rather than by observation at the bedside.

That difference in kind produces every other difference an examiner will want. One is recognised by looking at the patient now; the other cannot be, because a single observation carries no baseline. One fluctuates, which is why repeated observation detects it; the other is a persisting shift, which comparison rather than repetition finds. One declares itself on the ward; the other declares itself after discharge, and reaches a clinician only if somebody thinks to ask.

THE QUESTION	DELIRIUM	POSTOPERATIVE NEUROCOGNITIVE DISORDER
What the term names	An acute disturbance of attention and awareness	A change in cognitive performance against a preoperative baseline
How it is established	Observation of the patient's current state	Comparison of a later measure against an earlier one
Characteristic course	Fluctuating, often worse at night	Persisting, without the same fluctuation
Where it declares itself	On the ward, to the clinical team	After discharge, to the patient and family
Who teaches assessment and management	The stroke, brain injury and focal syndrome material	This section for the construct; psychopharmacology for any agent

RULE **Delirium is observed; a perioperative neurocognitive disorder is computed.** One is a state visible in the room, and the whole of its assessment lives in the chapter that owns it. The other is a difference between two measurements, which is why a service that never takes the first measurement can never make the diagnosis, however carefully it examines the patient afterwards.

The recommendation governing the acute limb is stated here only as a boundary. **Postoperative delirium screening should use a validated delirium score.** Which score, how it is administered and what follows a positive result are the delirium chapter's, and a candidate who reproduces an instrument here has answered another chapter's question while believing they were thorough. The perioperative point is narrower: screening is systematic and instrumented rather than impressionistic, which is a statement about how a surgical service is organised.

The constructs are not alternatives to choose between, and the same patient may pass through both, in sequence, after one operation. Whether an episode of postoperative delirium predicts the later cognitive trajectory is a question no source held for this section answers, and it is recorded below as a gap rather than taught.

MNEMONIC

Measure before, ask at, name after

Measure cognition before the operation, because a change needs an earlier observation. Ask the capacity question at the decision and at the moment. Name the construct afterwards, because delirium and a perioperative neurocognitive disorder are different kinds of thing with different owners.

12.7 Answering the postoperative call without importing the wrong chapter

The postoperative call is where most candidates meet this material, and the discipline that rescues an answer is to establish the shape of the problem before naming it. Establish whether a preoperative cognitive measure exists, because that determines what can be concluded at all. Establish the timeline, separating a disturbance that arrived over hours from a difficulty the family has watched for weeks. Establish whether the disturbance is of arousal and attention now, or of performance over time. Establish what else on the chart could produce it, since a surgical patient carries pain, immobility, infection risk and an interrupted medication list at once.

A postoperative note worth reading later states each of the following.

- Whether a preoperative cognitive assessment exists, where it is recorded, and what it showed.
- The timeline of the change, expressed against the operation rather than against the referral.
- Which construct is being described, and which is being excluded.
- The medication position: what was withheld, what was resumed, what is outstanding.
- Who reviews the patient after discharge, since the trajectory question cannot be answered on the ward.

Routing follows the same discipline. Psychological sequelae of a critical-illness stay, and delirium at the end of life, are taught by the organ-interface section of this chapter. Every question about agent, dose and organ failure belongs to the psychopharmacology material. Naming the destination answers that part of a question completely.

Before it, and after it	Measured, not recalled	This decision, this moment	Observed, or computed
WHAT THE UMBRELLA TERM COVERS	WHAT A LATER SCORE IS COMPARED WITH	HOW THE CAPACITY OPINION IS SCOPED	WHICH CONSTRUCT IS BEING NAMED

Every tile scopes something rather than reporting it: the reach of the term, the comparison a later score must enter, the boundaries of a capacity opinion, and which of the two constructs is in play.

12.8 The Indian perioperative reality, and what it changes

The perioperative recommendations held here come from a European perioperative safety guide, and the nomenclature recommendation from an international consensus working group. No Indian perioperative cognitive-assessment standard, and no Indian figure for how often preoperative cognitive assessment is performed, is held by any source behind this section. That absence is stated rather than filled, and it should be stated in an answer too.

What transfers is clear enough to reason about. The preoperative cognitive baseline is among the cheapest recommendations in perioperative medicine: a brief structured assessment, a person to administer it, and a place in the record the postoperative team will find. The barrier in most Indian surgical services is not cost but ownership, because preoperative clerking belongs to the surgical and anaesthetic teams, and the cognitive item is the first dropped when the list is long. Language and literacy add a second consideration: an instrument administered in a language the patient does not use, or whose items assume schooling the patient did not have, produces a low score that is a finding about the instrument rather than the patient.

IN INDIA

The practical move for an Indian unit is to attach the cognitive item to the anaesthetic preoperative assessment, where older surgical patients are already seen and documented, rather than to build a separate psychiatric pathway that depends on a referral being made. The psychiatric contribution is then to specify which instrument the unit uses, in which languages, administered by whom and recorded where, and to accept that a brief assessment applied consistently beats an ideal one done occasionally. No Indian coverage or outcome figure for such a pathway is held here.

GOOD TO REMEMBER

When the surgical team telephones about a confused postoperative patient, the first question is not what is wrong but whether anybody measured them before. If yes, a genuine comparison is available and a trajectory question can be answered. If no, say so plainly in the note, manage the acute presentation through the chapter that owns it, and arrange the review after discharge.

Delirium is seen at the bedside and belongs to another chapter; a perioperative neurocognitive disorder is a comparison against a preoperative baseline, which is why the measurement taken before the operation is this section's whole practical yield.

Oncology, palliative and paraneoplastic psychiatry

13 · Psychiatric effects of corticosteroids, chemotherapy and interferon (steroid psychosis, chemobrain)

The examinable content in cancer-drug neuropsychiatry is not a list of side-effects but a mapping: which agent, which syndrome, and over what interval that syndrome appears and is looked for. A candidate who has memorised that cytotoxic chemotherapy is associated with cognitive complaints, and that interferon-alpha is associated with depression, has learned the two least discriminating facts available. Marks sit on the rest: where in the course a syndrome belongs, what else produces the same picture at that point, whether the complaint has an objective correlate, and what the evidence licenses in advance rather than in response. This section installs that mapping for two agent families and deliberately routes a third away.

13.1 The boundaries this section keeps

Three boundaries govern what follows. Each exists because the material sits in full elsewhere in these notes, and a second version written here would drift from the first without anyone noticing.

The corticosteroid limb of this topic is not taught here. Systemic corticosteroids and steroid-induced psychiatric disturbance are routed on the chapter's opening plate to the endocrine and metabolic psychiatry material, and the merged organ-interface routing section of these notes carries the clinical construct. This section names steroids and goes no further with them.

Organ-adjusted prescribing is elsewhere too. Doses, dose adjustments and interaction rankings between psychotropics and cytotoxic agents belong to the psychopharmacology notes, and no figure of that kind appears below. The commonest way a candidate loses control of this topic is to answer a question about syndrome recognition with a half-remembered interaction table.

The third boundary is interferon. **Interferon-alpha** is taught here. **Interferon-beta** is taught in the Neuropsychiatry of systemic and neurological disease notes. That is a boundary rather than an accident of filing, and a later subtopic gives the reason.

RULE **Name the agent, name the syndrome, name the interval.** An answer that pairs an agent with a syndrome and stops has given half of what a ward needs. The interval tells the clinician when to look and which explanations compete at that moment.

13.2 Cancer-related cognitive impairment, and why the patient's word is the wrong construct

Patients say **chemobrain**. The construct a clinician should hold is **cancer-related cognitive impairment**, and the change of name is not fastidiousness. A term that asserts its own cause stops the clinician looking, and the value of this topic lies in what is still worth looking for after the complaint has been taken seriously.

The presenting complaint is characteristically about effortful cognition rather than frank forgetting: holding several things in mind, sustaining attention through a task, retrieving an available word, working at the old speed. Patients describe the change against their own baseline rather than any norm, which is both why the complaint is genuine and why it is hard to demonstrate.

Note what the study design in the next subtopic concedes before a single result is read. Its comparison groups are patients with the same cancers, not healthy volunteers. A design that needs disease controls is admitting that the disease, its investigations, its surgery, its other treatments and the fatigue around all of them are candidate causes of the same complaint. The cytotoxic agent is one contributor inside a bundle, and the patient's word names the one easiest to blame.

That study measured cognition with self-reported and performance-based cognitive measures, and it is worth registering that it used both. A complaint and a test result are different observations: the first cannot tell you whether performance has changed, and the second cannot tell you whether the person's life has changed.

13.3 The longitudinal evidence, read for exactly what it licenses

The anchor for the time course here is a longitudinal study of patients receiving chemotherapy for two tumour types, each type carrying its own control group. **225 participants** were assessed. The breast-cancer stratum held **41 patients and 36 controls**; the lymphoma stratum held **73 patients and 75 controls**. The reported conclusion is that, compared with controls, patients with breast cancer and lymphoma showed persistent cognitive worsening still present at **1 year** after treatment.

Read the boundary of that sentence carefully, because it is where the marks are. It is a group-level comparison: it describes two treated groups against two disease-matched groups and predicts nothing about the individual in front of you. It covers two tumour types, so it says nothing about regimens used for other cancers. The strata are modest, and the lymphoma stratum is the larger, so generalise more slowly from the smaller.

TRAP **Converting an observation window into a prognosis.** A finding reported up to a stated point after treatment describes how far the looking reached, not where the problem ended. Candidates translate it into reassurance, that it settles within the year, or into catastrophe, that it is permanent. The evidence supports neither, only that worsening was still detectable at the end of the window examined.

The consequence of persistence is a scheduling consequence. If the complaint outlasts the treatment, the point at which oncology hands the patient back is the point at which nobody is reviewing cognition, and it is exactly then that the patient is trying to return to work. A service that reviews cognition only while chemotherapy runs has arranged to stop looking just before the problem becomes disabling.

13.4 Assessing the complaint without reaching for an instrument

The task is not to confirm the patient's attribution. It is to characterise the complaint, subtract everything treatable that produces the same picture, and decide whether anything acute or evolving sits underneath it. The order matters: the treatable contributors are common and the acute ones are dangerous, and both are missed by a clinician who has already accepted the diagnosis the patient offered.

THE QUESTION TO ASK	WHY IT DISCRIMINATES	WHERE THE ANSWER ROUTES
Did the difficulty predate the first cycle?	Separates a complaint attributed to treatment from one already present at diagnosis	Keeps the differential open instead of closing it on the drug
Is the picture stable across the day, or fluctuating with clouded attention?	Fluctuating inattention is an acute-state question, not a chronic-impairment one	The stroke, traumatic brain injury, delirium and focal brain syndromes notes
Is mood, sleep, pain or fatigue carrying the complaint?	Effortful cognition fails when drive and sleep fail, and those are addressable now	The depression, demoralisation and adjustment material in the merged organ-interface routing section
Is the picture static, or evolving between reviews?	An evolving encephalopathy is a different class of problem from a stable deficit	The paraneoplastic neuropsychiatric syndromes section; the workup sits in the neurology-psychiatry interface notes
Does the complaint have an objective correlate on testing?	Self-report and performance are separate observations answering different questions	Decides whether the conversation is about workplace accommodation or further assessment

Two further routings belong in the same breath. Capacity, where a treatment decision turns on it, is decision-specific and time-specific, and the statutory test is taught in the mental health legislation notes. Where the complaint sits alongside a request to stop treatment, the palliative and existential material in the merged organ-interface routing section owns that conversation.

GOOD TO REMEMBER

The most useful thing a liaison psychiatrist adds here is a timeline. Ask when the difficulty began relative to diagnosis, to surgery, to the first cycle and to the end of treatment, and ask which task first went wrong. A complaint that predates any cytotoxic agent, one that fluctuates within a day, and one still worsening after treatment finished are three different problems wearing the same words.

13.5 Interferon-alpha, and the one thing this section can state as evidence

A spelling point first, because it costs marks in reading rather than in writing. The oncological preparation is written **interferon alfa** in drug nomenclature and in much of the trial literature, and interferon-alpha in general prose. They are the same agent.

The evidence this section can state concerns prevention rather than treatment. In patients with malignant melanoma who were eligible for high-dose interferon alfa therapy, pretreatment with paroxetine appears to be an effective strategy for minimising depression induced by interferon alfa. That was tested in a randomised comparison in which **40 patients were randomised, 20 to paroxetine and 20 to placebo**, with symptoms consistent with major depression during interferon alfa therapy as the outcome of interest.

Retain the qualification exactly as the source states it. Pretreatment appears to be an effective strategy, which is weaker than a claim that it prevents the syndrome, and an answer that upgrades

it has misreported the source. The population is equally load-bearing: melanoma, high-dose interferon alfa, one antidepressant, and pretreatment given before the interferon rather than treatment started once mood has changed. None of those boundaries transfers on its own to another cancer, another interferon, another antidepressant or a reactive prescription.

One thing can be inferred honestly from the design, and it carries the time course. A placebo-controlled prevention trial is only designable if the syndrome it prevents is expected often enough, and soon enough after therapy starts, to be counted inside the treatment course. The trial's shape therefore tells you that interferon-alfa depression is foreseeable and time-linked rather than idiosyncratic. That is an inference about the design, and this section publishes no rate.

RULE **Prophylaxis is a different claim from treatment, and a population is part of a claim.** The bound evidence supports pre-emptive antidepressant cover in a specified oncology population starting a specified interferon regimen. It says nothing about what to do once the mood syndrome has declared itself, and it does not extend to everyone about to receive an interferon. Quoting a prevention result as a treatment result is an invisible error unless you state the population every time you state the finding.

13.6 Interferon-alpha is not interferon-beta, and the separation is clinical

These are distinct preparations, given to distinct populations for distinct reasons, and their psychiatric literatures are separate. Interferon-alpha reaches the psychiatrist through oncology and through infectious disease, in courses that are intensive and time-limited. Interferon-beta reaches the psychiatrist through a chronic neurological illness carrying its own mood burden independently of any drug, which is why the Neuropsychiatry of systemic and neurological disease notes hold it and this section does not.

The separation matters for three reasons an examiner can ask about directly. First, the background rate of psychiatric disorder differs between the populations, so a figure pooled across both would describe no real patient and mislead in both directions at once. Second, the confounders differ: in the alpha setting the competing explanation is the cancer or the infection and its treatment, in the beta setting the neurological illness itself. Third, the clinical decision differs. In the alpha setting the live question is whether to cover a foreseeable, time-linked mood syndrome before a defined course begins; in the beta setting it is embedded in long-term management of a chronic disease, and taken up where that disease is taught.

TRAP **Answering "the neuropsychiatric effects of interferon" as a single topic.** A stem that says interferon without saying which preparation is testing whether the candidate knows the distinction exists. The answer that scores separates alpha from beta, states which one it will discuss, and says why pooling them would be uninformative. The answer that fails treats the word as one drug and produces a paragraph true of nobody.

13.7 Agent to syndrome to time course, which is what the plate carries

The plate below is the compressed form of everything above, built on three columns rather than two. The first names the agent family. The second names the syndrome, in the vocabulary a

clinician would use in a note. The third, which does the work, places that syndrome on the treatment timeline: before treatment, during the course, at handover, and after treatment has finished. Read the third column first when revising, because it converts a list of associations into something usable at the bedside.

Two features of the plate are deliberate rather than accidental. The corticosteroid limb is absent, because steroids are routed on the chapter's opening plate to the material that owns them. And interferon-alpha and interferon-beta are drawn apart rather than sharing a row, for the reasons given above.

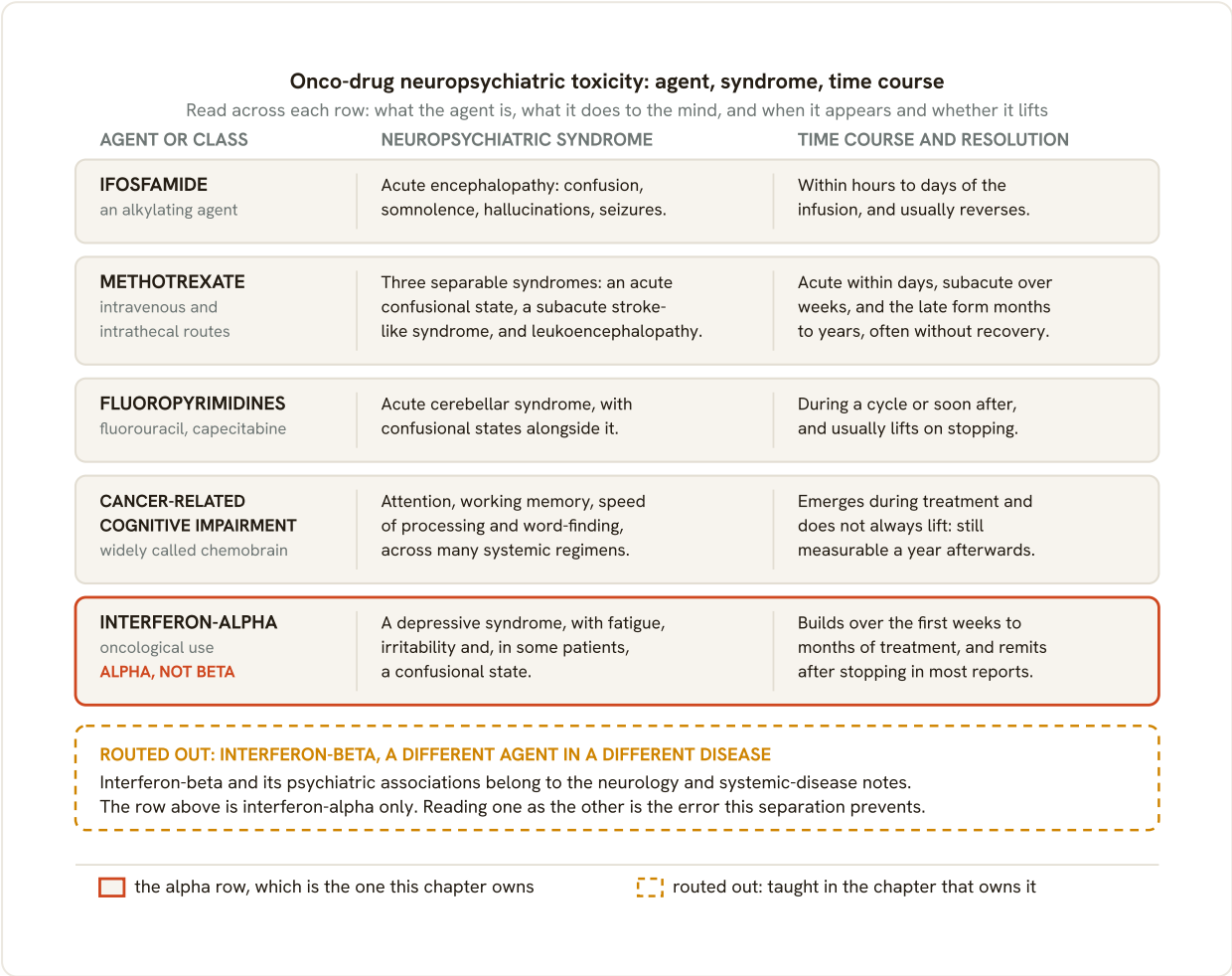

Fig 40.7 Chemotherapy and interferon neuropsychiatric toxicity read as agent, then syndrome, then time course, because at the bedside it is the time course that usually settles the differential. The acute encephalopathies declare themselves within hours to days and usually reverse on withdrawal, which is why ifosfamide, the fluoropyrimidines and the acute methotrexate syndrome sit together; methotrexate is the agent that also carries a subacute stroke-like syndrome and a late leukoencephalopathy that may never recover. Cancer-related cognitive impairment, widely called chemobrain, is the slow limb: it emerges during treatment, spares alertness, and has been measured as still present a year after treatment ends. Interferon-alpha produces a depressive syndrome that builds over the first weeks to months of treatment. The plate separates interferon-alpha from interferon-beta deliberately, because they are different agents used in different diseases and beta belongs to the Neuropsychiatry of systemic and neurological disease notes; the third drug group named in this topic is carried by the routing plate earlier in this chapter and by the Endocrine and metabolic psychiatry notes, so it is not drawn here at all. From memory you should be able to place any of these agents on the three columns and say whether the syndrome is expected to lift.

IN INDIA

No Indian figure for the frequency of cancer-related cognitive impairment, and none for interferon-alfa depression in Indian oncology practice, was located for this section, and none is asserted. What transfers is the structure. Where oncology and psychiatry sit in different institutions and follow-up is episodic, the practical question is not which cognitive instrument to buy but who reviews the patient once the oncology course closes, and when. Since the bound evidence describes a complaint still present after treatment ended, a service with no review point after discharge from oncology has designed itself to miss the window that matters most.

13.8 The shape of an answer that scores

A written answer here is graded on discrimination rather than length, and the discriminating content is ordered.

- Separate the agent families at the outset. Name the corticosteroid limb only to route it, and separate interferon-alpha from interferon-beta before saying anything about either.
- Use the construct rather than the patient's word, and say why the patient's word closes the differential prematurely.
- State every finding with its population attached. A finding without its population is not a shorter answer, it is a weaker and different one.
- Place each syndrome on the treatment timeline, and keep the observation window distinct from the prognosis.
- Route the neighbouring problems by name: delirium, paraneoplastic syndromes, depression and adjustment in cancer, capacity, and organ-adjusted prescribing.
- Say what is not known. The honest gaps here are large, and a candidate who names them is more convincing than one who fills them.

PITFALL

Treating the cognitive complaint as the end of the assessment because the patient has a plausible reason for it. A patient receiving chemotherapy has an explanation available for almost any neuropsychiatric presentation, and that availability is what makes delirium, an evolving encephalopathy and an untreated depression easy to miss. The plausible cause is the one to test last, not the one to accept first.

Agent, syndrome, interval
THE THREE THINGS AN ANSWER MUST NAME

Cancer-related cognitive impairment
THE CONSTRUCT THAT REPLACES THE PATIENT'S WORD

Prevention, not treatment
WHAT THE PRETREATMENT FINDING LICENSES

Alpha is not beta
THE SEPARATION A ONE-WORD STEM IS TESTING

Interferon-alpha is separated from interferon-beta and corticosteroids are routed away; the cognitive syndrome is cancer-related, not chemotherapy-proven; and every finding is stated with its population and its observation window attached.

14 · Paraneoplastic neuropsychiatric syndromes and onconeural antibodies: when to suspect occult malignancy

A paraneoplastic neuropsychiatric syndrome is a psychiatric presentation driven by the immune response to a tumour that has not yet been found. The final clause carries the whole examinable weight. This is not a complication of a cancer already sitting in the case notes; it is the event that makes someone go looking for the cancer in the first place. This section installs the reasoning that converts an ordinary-looking psychiatric presentation into a directed search for occult malignancy: what the term **paraneoplastic** actually claims, why the psychiatric clinic meets these patients ahead of the oncology clinic, what raises suspicion, what a psychiatrist owns and what is handed on, and which antibody has been reported to point where.

14.1 What the term claims, and the sequence that defines it

The prefix does the work. **Para** plus **neoplastic** names a disorder that accompanies a neoplasm while being made of something else. The damaged tissue is nervous tissue, and what damages it is an immune response the tumour provoked. The word **onconeural** names why such a response reaches the brain at all: the antigen belongs to the tumour and to neural tissue alike, so an attack aimed at one compartment lands in the other. Everything examinable here follows from that idea, including why the syndrome can be severe while the tumour stays small enough to have escaped detection.

The defining feature, for examination purposes, is a sequence rather than a severity. **The neurological or psychiatric syndrome comes first, and the malignancy is detected afterwards.** The task-force report that grounds this section states it plainly: **paraneoplastic neurological syndromes almost invariably predate detection of the malignancy**. Hold that sentence in mind through everything that follows, because it is what separates this topic from every other cancer topic on the day, and it is the reason the topic belongs to a psychiatry paper at all.

It is worth being precise about what the label has to displace before it is earned, because a neuropsychiatric picture in someone who turns out to have cancer has several ordinary explanations. It may be a direct effect of tumour mass or of secondary deposits. It may be metabolic or endocrine derangement produced by the tumour. It may be treatment: the cytotoxic agents, the interferon and the corticosteroids, each taught in its own place, in the sibling section on chemotherapy and interferon effects and in the routing section that owns steroid-induced disturbance. It may be adjustment, demoralisation or depression in someone absorbing a serious illness, which the routing section on cancer owns. Cross-refer each by name; this section restates none of their content.

Notice the asymmetry the sequence creates. In the ordinary cancer psychiatry of a known malignancy, those rival explanations are all available on the ward round, because the tumour, the metabolic panel and the drug chart are already in front of you. In the paraneoplastic case they are unavailable at presentation, because nobody has yet established that there is a tumour. The

clinician holds a syndrome and a history, and the question of malignancy has to be raised rather than handed over.

RULE **The defining feature is the sequence, not the severity.** **A paraneoplastic syndrome characteristically declares itself before the malignancy is detected**, which is why the phrase in the bank item is "when to suspect occult malignancy" and why an answer that describes a florid syndrome in a known cancer patient has answered a different question.

14.2 Why the psychiatric clinic meets these patients first

There are honest structural reasons why this presentation lands with a psychiatrist, and a candidate who can state them is well placed to answer the item. The first is the sequence itself. Because **the syndrome usually arrives before the malignancy has been detected**, the first clinician to see the patient is whichever clinician the presenting complaint routes them to. Where that complaint is behavioural, meaning altered mood, altered personality, psychosis, failing memory, reversed sleep, agitation or apathy, that clinician is a psychiatrist. The referral letter says nothing about cancer, because at that point there is nothing about cancer to say.

The second reason is that psychiatric formulations are generous, and their generosity is usually a strength. A subacute behavioural change in an adult can be absorbed comfortably into a first episode of psychosis, a late-onset depressive episode, a mood disorder cycling faster than usual, a dissociative presentation or a functional cognitive disorder. Each is internally coherent and each generates a plausible treatment plan. None of them obliges the clinician to ask what else could be producing this picture, so the question of occult malignancy is one the reasoning process will never raise unless the clinician raises it deliberately.

The third reason is what the examination is actually testing. The bank item asks when to suspect, and suspicion is a skill with a location in time: it belongs at first contact, alongside the initial formulation, and it loses value the longer it is deferred. A suspicion raised after several failed treatment trials is a lesser clinical act than the same suspicion raised at intake, because in the interval the syndrome has progressed while **the malignancy has continued to sit undetected**.

TRAP **Treating the difficulty as a laboratory problem.** Candidates picture the failure as a missed antibody result, so they write about panels and assays. The failure that actually happens is upstream and cheaper to fix: the question of an underlying tumour was never opened, so nothing was ever sent. An answer that begins with the test has skipped the step the item is examining.

14.3 Reading the presentation: tempo, company and fit

Suspicion is a discipline rather than a mood, and it should not be taught as vigilance in general. It is built from a small set of readings that a competent psychiatric history already produces, and the work is to read them deliberately rather than to hope they announce themselves.

The first reading is **tempo**. Psychiatric illnesses have characteristic time signatures, and a clinician who has taken many histories knows them implicitly. What matters here is the mismatch: a presentation that moves faster than the illness it resembles, that keeps moving while treatment is being delivered adequately, and whose trajectory is a slope rather than an episode.

Tempo costs nothing to record, requiring only a careful timeline with collateral corroboration, and it is the reading most often left vague in a psychiatric clerking.

The second reading is **company**, meaning what the psychiatric syndrome has arrived with. Psychiatric syndromes keep characteristic company, and neurological company is different company. When a behavioural presentation is accompanied by features belonging to the nervous system rather than to the mind, the formulation has acquired something it cannot metabolise, and that is informative in itself. The plate below sets out the clusters that carry this weight, and it is the right place to learn them, because they are the content of the source criteria rather than of this author's recollection.

The third reading is **fit**: whether this person fits the illness being diagnosed, and whether a general history has been taken at all. Psychiatric clerking has a known habit of collecting an exhaustive developmental history alongside a thin systemic one, so constitutional features, general medical risk and the ordinary questions an internist would ask are frequently missing. A history that never asked them cannot be reassuring, and the difference between a negative finding and an unasked question is one this chapter returns to repeatedly.

The plate that follows carries the working set: the onconeural antibody group, the tempo and sign clusters that raise suspicion, and the boundary against the autoimmune-encephalitis workup taught elsewhere. Read the plate as the operational list, and this prose as the reasoning that tells you when to reach for it.

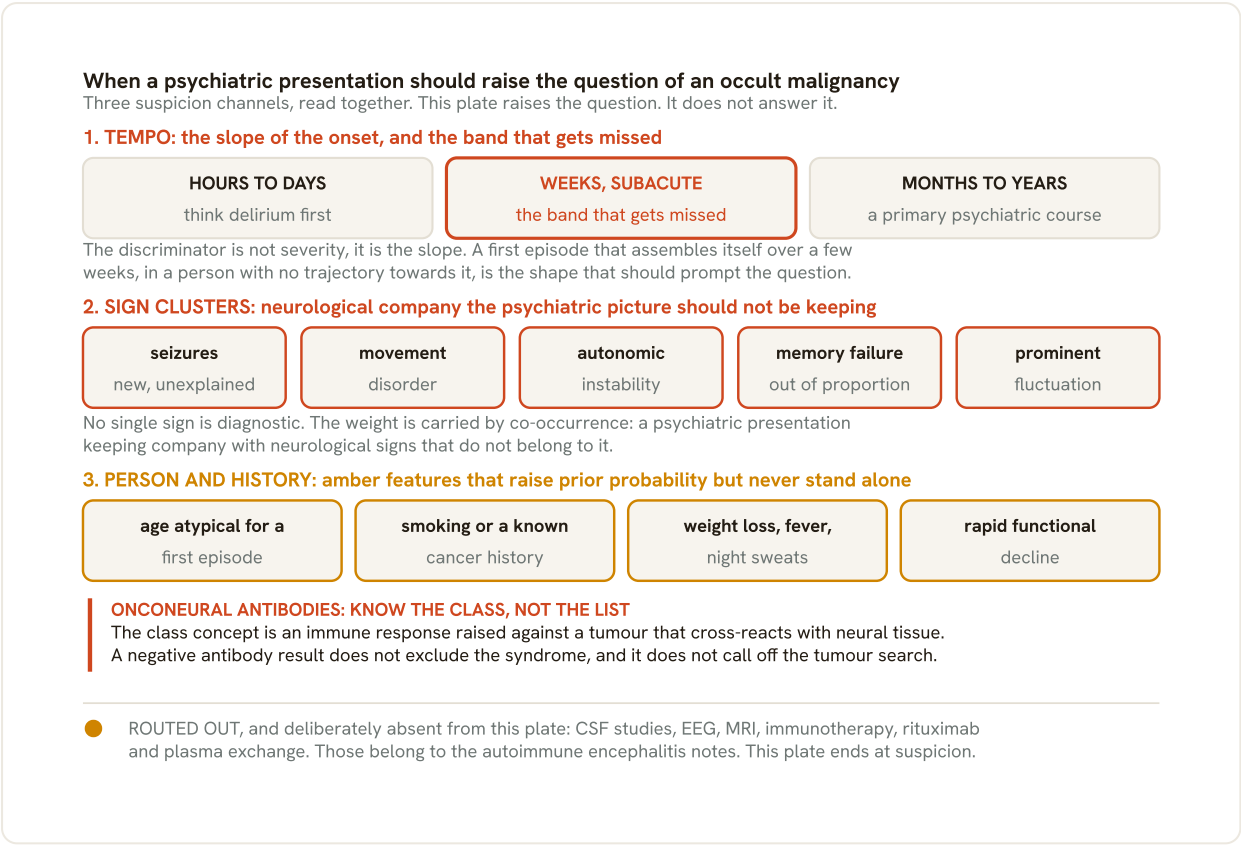

Fig 40.8 The three suspicion channels for an occult malignancy presenting psychiatrically. The plate is deliberately a suspicion instrument and stops there, because the workup and its immunotherapies belong to the autoimmune encephalitis notes and are cross-referenced out rather than duplicated. What it adds to the prose is the tempo rail, which puts the subacute band between two safer neighbours and shows why that band is the one that gets missed: a presentation over hours to days is read as delirium and gets investigated, a presentation over months to years is read as primary psychiatric and gets followed, and the few-week presentation falls between two reflexes. The onconeural antibody idea is carried as a class rather than a list because that is how it is examined and how it behaves, and because a negative result narrows nothing. From memory you should be able to state the tempo band that should worry you, name the sign clusters that a psychiatric picture should not be keeping company with, and say what a negative antibody result does and does not permit you to conclude.

14.4 The boundary: suspicion is yours, the workup is not

This section owns paraneoplastic suspicion. It does not own the autoimmune-encephalitis workup. Cerebrospinal fluid examination, electroencephalography, magnetic resonance imaging, first-line immunotherapy, rituximab, plasma exchange and intravenous immunoglobulin are taught in the Neuropsychiatry of systemic and neurological disease notes, and this text restates no parameter of any of them. The boundary is named explicitly because it is the one an author is most tempted to cross: the workup has protocols and sequences and feels like the substance of the topic, and it is the substance of a different topic with a different owner.

The split is clinical rather than merely editorial. The investigations and the immunotherapy form one coherent body of knowledge, learned and examined together. Suspicion is a separate skill, exercised by a different clinician at an earlier moment, and it is the skill this bank item names.

What the psychiatrist owns is worth stating positively, and it is short enough to hold as a list.

- The timeline, recorded precisely enough that tempo can be read off it later by a clinician who was not in the room.

- The collateral history, often the only source of that tempo.
- The general and systemic history that psychiatric clerking tends to abbreviate.
- The referral itself: one that says "for neurology opinion" transmits only the patient, while one that states the tempo, the company and the explicit reason a tumour is being considered transmits the reasoning.

THE QUESTION IN FRONT OF YOU	WHERE IT IS ANSWERED
Could this psychiatric presentation be driven by a tumour nobody has found yet?	This section.
What do the cerebrospinal fluid, the electroencephalogram and the scan show, and what immunotherapy follows?	Neurology-psychiatry interface notes, autoimmune encephalitis. Never taught here.
Is this delirium, and how is it assessed and managed?	Stroke, traumatic brain injury, delirium and focal brain syndromes notes.
Does this patient have capacity, and what is the statutory test?	Mental health legislation notes. Capacity is decision-specific and time-specific; the test is taught there.
How is the psychotropic adjusted for hepatic or renal impairment?	Psychopharmacology notes. This chapter adds no further version of a dose adjustment.
Is the disturbance caused by the cytotoxic agent, by interferon, or by the corticosteroid?	The sibling section on chemotherapy and interferon effects, and the routing section owning steroid-induced disturbance.
Is this neuropsychiatric lupus, neurosarcoidosis, central nervous system vasculitis or antiphospholipid syndrome?	The sibling sections owning each of those.
Is this depression, demoralisation or adjustment in someone already known to have cancer?	The routing section on cancer.

14.5 The screening imperative, and the guides it names

Once suspicion exists, the criteria are direct about what follows. **Cancer screening should be promptly undertaken when a paraneoplastic neurologic syndrome is suspected, and should be guided by the type of phenotype or antibody.** That single sentence is the operational core of this section, and it repays reading in parts.

The first operative word is **promptly**. It fixes a position in the sequence: the screen belongs at the point of suspicion rather than at the point of diagnostic certainty. The claim carries urgency and carries no interval, so this text supplies none, and a candidate who wants a period in days or weeks should take it from the criteria rather than from a note that would be inventing it.

The second operative phrase is **guided by the type of phenotype or antibody**. The instinct under pressure is to describe a broad sweep, imaging everything and hoping something appears. The criteria describe a search with a direction, where the clinical phenotype narrows the field of plausible primaries and the antibody, when available, narrows it further. An answer that reproduces the urgency while dropping the guidance has reproduced the easier part.

One inference is worth stating, because it is reasoning rather than recollection. If **the syndrome characteristically presents before the malignancy is detected**, the screen is being run in a body where the tumour has so far evaded detection. A first look that finds nothing has therefore not settled the question the way a first look normally settles one, and the posture that follows is continued attention rather than closure.

RULE **Prompt and guided, in that order and with equal weight. The criteria couple the timing of cancer screening to the suspicion of a paraneoplastic neurologic syndrome, and couple its direction to the phenotype or the antibody.** Reproduce the pairing. An answer carrying only urgency describes panic; an answer carrying only direction describes a screen that may arrive too late to matter.

Now the honest limit of this section, narrowed to what the ledger actually withholds. The antibody pairings are cited and taught in the subtopic below, so they have left the list of absences. What is still missing is the machinery around the screen: no row names which imaging modality corresponds to which phenotype, no row gives an interval for repeating a screen that found nothing, and no row states how long surveillance continues. Those three are what a candidate will want next, and they are owed to the source criteria rather than to this note.

14.6 Onconeural antibodies: the risk tier, the phenotype and the reported pairings

The term **onconeural antibody** names an antibody directed against an antigen shared by tumour tissue and nervous tissue. That definition is why the antibody is diagnostically useful at all: because the antigen belongs to each compartment, a serological finding points at a body region that has yet to be searched. Take the term seriously as vocabulary, because it carries the logic of the whole section in a single word.

The antibody earns its place here for the reason the criteria give: **the cancer screen is guided by the type of phenotype or antibody**. The antibody is a laboratory result while the syndrome is a clinical entity, so the result informs the search rather than constituting the diagnosis. And it sits alongside the phenotype in the claim as written rather than replacing it, so a clinician who has a phenotype and awaits a result already holds one of the guides the criteria name.

The criteria then sort the antibodies by the cancer risk each one carries, and that sorting is what makes the guidance operational. **The first group holds the high-risk antibodies, defined as those associated with an underlying cancer in more than 70 per cent of the patients who carry them.** A structural fact travels with the group: **most high-risk antibodies target intracellular antigens**. Put the two together and an inference follows. An antibody raised against an antigen sitting inside the cell is poorly placed to be doing the damage by binding it, so the finding is best used as a pointer towards a hidden tumour rather than as an account of mechanism.

One phenotype belongs to psychiatry more than to any other specialty. **Paraneoplastic limbic encephalitis is characterised by personality change, irritability, depression, seizures, memory loss and sometimes dementia.** Read that list as a psychiatric referral rather than a neurological one. Personality change, irritability and depression are what a psychiatric service is

built to receive; the seizure and the memory failure are what eventually move the formulation, often long after a psychiatric label has been applied and acted upon.

The reported pairings follow, with the evidence grade placed second so that it is read before the pairing it qualifies, and with the two rows that rest on more than one patient placed first. Read the second column before the third. A single reported case establishes that a pairing has been observed in a human being, and nothing beyond it: no frequency, no claim that this tumour is the one the antibody usually points to, and no entitlement to the word "classical".

ANTIBODY	WHAT THE CITED ROW IS	SYNDROME REPORTED WITH IT	TUMOUR REPORTED WITH IT
CRMP5	**The commonest associated diagnoses in a 24-patient cohort.**	**Peripheral neuropathy and cerebellar ataxia**	**Lung cancer**
Anti-Ma2	**Described as a specific marker for that pairing.**	**Limbic and brainstem encephalitis**	**Testicular germ-cell tumour, mainly in adult males**
Anti-Hu	**A single reported case.**	**Adie's pupil with sensorimotor polyneuropathy**	**Primary mediastinal small-cell carcinoma**
Anti-Yo	**A single reported case, arising years after the tumour was resected.**	**Cerebellar degeneration**	**Ovarian cancer**
Anti-Ri	**A single reported case.**	**Opsoclonus-myoclonus**	**Breast carcinoma, in a male patient**
Anti-amphiphysin	**A single reported case.**	**Stiff-limb syndrome**	**Invasive ductal carcinoma of the breast**

The second column is where marks are won and lost, which is why it now sits where the eye reaches it first. Four of these six rows rest on one patient each, and several of those reports were published precisely because they were unusual: an opsoclonus-myoclonus syndrome in a man, a cerebellar syndrome declaring itself years after the ovarian tumour was resected, a mediastinal rather than a pulmonary small-cell primary. A report selected for its rarity is the worst available basis for a statement about what is typical, and an answer that flattens this table into a list of classical associations has manufactured a confidence its own sources never carried.

The criteria also grade how much confidence the label deserves. **The PNS-Care score is a composite model for assigning diagnostic likelihood in a patient with a suspected paraneoplastic neurological syndrome**, and it answers in bands: **definite at 8 or above, probable at 6 to 7, possible at 4 to 5, and a score of 3 or less classed as something other than a paraneoplastic syndrome**. Learn the bands, and learn that a graded answer exists. The items the score weighs are absent from this fragment's ledger, so they are left to the criteria.

Syndrome, then tumour	Prompt and guided	Pointer, not diagnosis	Observed, not typical
THE SEQUENCE THAT DEFINES THE TERM	HOW THE CANCER SCREEN IS RUN	WHAT AN ONCONEURAL ANTIBODY IS FOR	WHAT A SINGLE-CASE PAIRING ROW ESTABLISHES

The four tiles carry the spine of the section: a sequence, a manner of screening, a use for the laboratory result, and a limit on what the pairing table licenses.

IN INDIA

The screening imperative assumes an onconeural antibody panel can be requested, paid for and reported within a useful period, and in much of Indian practice each assumption deserves checking before the plan is written. Panels are sent out to referral laboratories from most district and many teaching hospital settings, the cost is usually met out of pocket, and turnaround runs to the time a courier and a batch run take. The practical consequence is that **the phenotype is frequently the guide available first, with the antibody arriving later to refine the search**, which makes a carefully documented clinical picture more valuable here than where serology returns quickly. The Indian record on availability, pricing and reporting intervals for these panels was never located within this fragment's ledger, so it is described qualitatively rather than quantified.

GOOD TO REMEMBER

On the ward this reduces to a single habit. When a psychiatric presentation moves at the wrong speed, keeps company with neurological features, and sits in a person whose general history was never properly taken, write the timeline down and say the word "paraneoplastic" out loud in the referral. **The syndrome characteristically arrives before the malignancy is found**, so the psychiatrist is often the clinician best positioned to raise the question at the moment it is most useful. What follows, the fluid, the imaging, the electrophysiology and the immunotherapy, belongs to colleagues and to another section. Raising the question well is the part that is yours.

The syndrome almost always arrives before the malignancy is found, so the psychiatrist's part is suspicion at first contact: read tempo, company and fit, write the timeline down, say the word in the referral, and let the cancer screen be prompt and guided while the workup belongs to somebody else.

Autoimmune and inflammatory systemic disease, and burn injury

15 · Neuropsychiatric systemic lupus erythematosus: manifestations, diagnosis and management

The examinable difficulty in neuropsychiatric lupus is not the list of syndromes, it is deciding whether the syndrome in front of you belongs to the disease at all. Lupus reaches the nervous system often enough to produce almost any neurological or psychiatric presentation, and the same patient can also be septic, uraemic, hypertensive, taking drugs with central effects, and

frightened by an illness that has already taken things from them. This section installs the topic in the order the decisions are made: what the term covers, how the manifestations reach a psychiatrist, how attribution is reasoned when no test settles it, and what treatment looks like afterwards.

Several things this presentation touches are owned elsewhere and are named here rather than rebuilt. Delirium assessment and management, including the bedside instruments and the drug choice for an agitated delirious patient, belong to the stroke, traumatic brain injury, delirium and focal brain syndromes notes. Organ-adjusted prescribing belongs to the psychopharmacology notes, and the workup of autoimmune encephalitis to the neurology and psychiatry interface notes. Corticosteroid-induced psychiatric disturbance is taught by the routing section of this chapter that owns it and by the endocrine and metabolic notes, which matters here because the drug class used to treat this disease is itself a recognised cause of psychiatric disturbance. The antiphospholipid syndrome is owned by the sibling section that carries it alongside neurosarcoidosis and central nervous system vasculitis.

15.1 What the term is asked to cover, and why its breadth is the problem

Neuropsychiatric systemic lupus erythematosus, conventionally shortened to **NPSLE**, is a collective label for the neurological and psychiatric manifestations occurring in a person who has lupus. It is a container, not a diagnosis of the kind a psychiatrist is used to making. Standard usage sorts its contents into central nervous system events, peripheral nervous system events and psychiatric syndromes, and those groups share almost nothing at the level of mechanism, severity, investigation or treatment.

That breadth is the source of every difficulty that follows. A label covering both a mild headache and a catatonic psychosis cannot have one test, one mechanism or one treatment, and any sentence beginning "NPSLE is treated with" is suspect before its predicate arrives. The useful move is to handle the label as a question in parts: which syndrome, by which mechanism, in a patient whose disease is at what level of activity, with which alternative explanations excluded.

RULE **Name the syndrome, then the mechanism, then the attribution, then the treatment.** These are separate determinations and each can be right while the next is wrong. A candidate who says "this is neuropsychiatric lupus" has named a container. One who says "this is an acute confusional state, in a patient with active disease, in whom infection and metabolic causes have been excluded, treated as an inflammatory manifestation" can be examined on any of the four.

15.2 How the manifestations reach a psychiatrist

Grouping the manifestations by the route through which they arrive is more useful than grouping them by anatomy, because the route determines what you are being asked to do.

- **Diffuse presentations**, which psychiatry is most often called about: an acute confusional state, mood disturbance of either polarity, psychosis, cognitive impairment across attention, memory and executive function, and anxiety. Here the referral question is usually not "is this lupus" but "is this psychiatric".

- **Focal central events**: seizures, stroke-like episodes, movement disorder, myelopathy, aseptic meningitis, cranial neuropathy and a demyelinating syndrome. These reach neurology or medicine first, and psychiatry is called for the behavioural consequences, for capacity, or because the presentation was read as functional.
- **Peripheral events**: polyneuropathy, mononeuropathy, plexopathy, autonomic disturbance and a neuromuscular junction disorder. These rarely present to psychiatry directly, and their relevance is that pain and disability from them drive mood.

The classes are nomenclature rather than mechanism: they say where the disturbance sits and nothing about why. This chapter also holds no row giving the frequency of any manifestation in any population, so nothing here is called the commonest and no ordering by frequency is printed. Frequency statements circulate widely in this topic and disagree with one another, for the reason the next subtopic makes clear.

15.3 Attribution, which is the actual core of the topic

Everything listed above can be produced by lupus, and every one of them can be produced by something else in a person who happens to have lupus. Diagnosis is therefore **attribution**: a reasoned judgement that this syndrome, in this patient, at this time, belongs to the disease. It is reasoned rather than measured, because **there is no diagnostic gold standard by which the neuropsychiatric syndromes of lupus can be diagnosed**. That fact is the axis of the topic and belongs early in any answer.

Three consequences follow, each worth a sentence. Published frequency estimates disagree, because without a gold standard every study defines its own numerator: a series counting headache and mild mood symptoms and a series counting only severe syndromes are not measuring the same thing, and their percentages are not comparable even when both are correctly reported. No investigation is confirmatory, so every result is evidence to be weighed rather than an answer. And attribution is revisable, since a judgement made on admission may be overturned by the culture result, by the course, or by the response to treatment.

The reasoning runs on a small number of axes worth naming. The first is the temporal relation to disease activity elsewhere: a syndrome appearing while the disease is active in other organs is easier to attribute than one appearing in quiescent disease. The second is the presence or absence of a competing explanation, which the next subtopic takes up because it does the most work. The third is the character of the syndrome, since a course, phenomenology or examination finding atypical for a primary psychiatric disorder points towards a systemic cause. The fourth is response to treatment, and it is retrospective, which is why the attribution has to be made and acted on before the evidence for it is complete.

TRAP **Treating a positive antibody as an attribution.** Serology establishes that the patient has the disease. It does not establish that the syndrome in front of you was produced by it, and a patient with unambiguous lupus can have an unrelated psychiatric illness, an infection or a drug effect. Skipping the attribution step is also how a treatable cause is missed while immunosuppression is escalated.

15.4 What has to be excluded before the disease is credited

Because attribution rests so heavily on the absence of a competing explanation, the exclusion list is the substance of the assessment rather than preliminary housekeeping. Infection comes first and stays first in a patient immunosuppressed by both the disease and its treatment, and it includes meningitis, encephalitis, and the encephalopathy of systemic sepsis without central infection. Metabolic causes follow: uraemia, sodium and glucose disturbance, hepatic and thyroid disturbance. Hypertensive encephalopathy belongs in anyone with renal disease and a new confusional or seizure presentation.

Drug causes are the largest single trap here, because the treatment of the disease and the cause of the psychiatric syndrome can be the same agent. That construct is taught by the routing section of this chapter that owns it; what belongs here is the boundary. A new psychiatric syndrome beginning after a change in immunosuppressive treatment, in a patient whose disease activity elsewhere has improved, pulls in the opposite direction from an attribution to the disease. The distinction changes treatment completely, since one path escalates immunosuppression and the other reduces it.

Primary psychiatric illness stays on the list throughout, supported by a psychiatric history predating the lupus, a family history, and a typical course. So does the ordinary burden of a relapsing systemic illness with a visible skin component and a treatment with visible adverse effects: demoralisation and adjustment disorder in that setting are a person responding to their circumstances rather than the disease reaching the brain, and are treated as such.

Finally, lupus is one member of a family of systemic diseases that reach the nervous system, and the discriminating features are what separate them at the bedside. The plate accompanying this section sets lupus beside the other systemic autoimmune presentations this chapter teaches, neurosarcoidosis, central nervous system vasculitis, the antiphospholipid syndrome, coeliac disease and inflammatory bowel disease, each with its discriminating feature. Each is taught in full by the sibling section that owns it and is named here only to place lupus in its family.

Six systemic autoimmune conditions on one comparator, because the task is telling them apart
One row each. The middle column is the only one that separates them from each other.

CONDITION	THE DISCRIMINATOR, in one feature	WHAT BRINGS IT TO PSYCHIATRIC NOTICE
Neuropsychiatric lupus	multisystem: skin, joints, kidney and blood counts, all in the one patient	psychosis or a mood episode arising inside an already declared illness
Neurosarcoidosis	cranial neuropathy, with basal meningeal or pituitary involvement	apathy and personality change, with hypersomnia and endocrine mood shift
Primary CNS vasculitis	confined to the CNS, with headache and stepwise deficits on a bland screen	no psychiatric presentation is claimed here: a declared gap, see the text
Antiphospholipid syndrome	thrombosis and pregnancy loss are the carrying features, often recurrent	cognitive complaints in a young adult with a thrombotic or obstetric history
Coeliac disease	gluten-driven enteropathy, and the deficiency load does much of the work	depression and apathy that persist after the mood treatment is optimised
Inflammatory bowel disease	relapsing bowel disease, and the burden tracks disease activity	depression and anxiety that flare with the bowel, plus steroid-driven change

READ DOWN THE MIDDLE COLUMN: that is the only column that closes the differential.
Low mood and cognitive change run across these rows, so that column raises the question but cannot answer it.

● ROUTED OUT: the serological and imaging workup for each of these six, and their immunosuppressive treatment, are not taught on this plate. What is taught here is telling the six apart.

Fig 40.9 Six systemic autoimmune conditions laid on a single comparator so that they can be told apart rather than revised one at a time. The plate exists because the right-hand column is nearly identical down all six rows, and that is the teaching point: depression, anxiety and cognitive change are the common currency of systemic inflammation, so the presentation that brings the patient to psychiatric attention almost never identifies which condition it is. The middle column carries the whole differential, and each entry is chosen to be true of that row and false of the other five. One right-hand cell is deliberately empty of a clinical picture: no source explicitly linking primary central nervous system vasculitis to a psychiatric manifestation was located for this chapter, so the gap is printed rather than filled from a neighbouring condition, and the accompanying text says what to do with it. Note the two structural families the table exposes, that the first four reach the brain directly through inflammation, vasculopathy or thrombosis, while the last two reach it indirectly through nutrition, disease activity and treatment exposure. From memory you should be able to give the single discriminating feature for each of the six, and say why the psychiatric column can raise the question but never settle it.

15.5 Investigation, and what each result can and cannot settle

The investigation logic follows from the absence of a gold standard: every test is chosen for what it excludes or supports, and none to confirm. Serology and disease activity markers establish that lupus is present and give some sense of activity elsewhere, which feeds the temporal axis and nothing more. Structural imaging looks for a focal lesion, for ischaemic change and for an alternative structural explanation, and a normal scan is entirely compatible with a severe diffuse syndrome, which is the most misread result in this topic.

Lumbar puncture here is done principally to exclude infection, and its findings in the disease itself are non-specific. Electroencephalography supports or refutes a seizure explanation for an episodic disturbance and characterises an encephalopathy without attributing it. Neuropsychological assessment characterises a cognitive deficit, which is valuable for

rehabilitation and for tracking change, and does not attribute it. Where autoimmune encephalitis is genuinely in question, that workup belongs to the neurology and psychiatry interface notes.

This chapter holds no row carrying an antibody to syndrome association, an imaging finding to syndrome association, or the performance of any investigation in this population, so none is asserted. That is a real gap rather than a stylistic omission, and the antibody associations should be read from a primary source and held separately from what is taught here.

RULE **No investigation closes the question, because no diagnostic gold standard exists for these syndromes.** Order tests to exclude the competing explanations and to characterise the syndrome, and expect to make the attribution on the balance of what you have. An answer promising a confirmatory test is wrong on the central fact of the topic.

15.6 Treatment, and the recommendation this section carries

Once a syndrome has been attributed to the disease, treatment has three limbs, chosen by mechanism rather than by phenomenology. The **inflammatory limb** is immunosuppression, applied when the syndrome is judged to arise from active disease. The **thrombotic limb** is antithrombotic treatment, applied when the mechanism is judged to be vascular occlusion; the antiphospholipid syndrome that most often drives it is owned by the sibling section, so this section names the limb and stops. The **symptomatic limb** is psychiatric and neurological treatment of the syndrome itself, running alongside either of the others rather than after them.

The guideline recommendation this section carries is precise about its population and about its comparison, and both matter more than the drug names. In adult patients with lupus-related severe, acute neuropsychiatric manifestations, the guidance is to **use GCs plus CYC over GCs alone or GCs plus RTX**. The abbreviations are the guideline's own and stand for glucocorticoids, cyclophosphamide and rituximab.

Read the population before using the recommendation, because it carries restrictions a candidate quoting it loosely will drop. It concerns adults, so it says nothing about children. It concerns severe manifestations, so it says nothing about the many mild ones this container also holds. It concerns acute manifestations, so it says nothing about chronic or slowly progressive syndromes, and a chronic cognitive syndrome is precisely what psychiatry is most likely to be asked about. Quoted as though it applied to everyone with this label, it becomes an error of scope.

What this section does not carry is equally worth stating: no dose, no route, no duration, no schedule, no maintenance strategy, no comparison for the mild or chronic presentation, and no statement about what to do when the first choice fails. Organ-adjusted prescribing for renal involvement belongs to the psychopharmacology notes, and remembered regimens in this topic diverge sharply between sources.

15.7 The psychiatric limb, and the boundaries around it

Psychiatric treatment of an attributed syndrome runs alongside the immunosuppression and is not deferred until it works. An acute confusional state is managed as an acute confusional state, by the pathway taught in the delirium notes, while the cause is treated. A psychotic or affective

syndrome is treated symptomatically with two standing cautions in view: renal and haematological involvement constrain the choice of agent, quantified in the psychopharmacology notes rather than here, and the possibility that treatment of the disease is contributing keeps the drug review open.

Two further boundaries belong in an answer. Capacity is assessed as it is anywhere else, and the only things this section says about it are that it is decision-specific and time-specific, which matter unusually often in a fluctuating encephalopathy; the doctrine and the statutory test belong to the mental health legislation notes. Adherence deserves explicit attention, because the psychiatric syndrome sits on top of a demanding medical regimen, and a cognitive syndrome never named will be recorded as non-compliance.

The rehabilitative and psychological work is most often omitted and has the longest reach. A cognitive deficit that has been characterised can be worked with, at home and at work, whether or not it is reversible, and the demoralisation that follows a burdensome illness responds to being named and addressed rather than to being reclassified as a manifestation of the disease.

15.8 Answering the question

The question is almost always some form of "a patient with known lupus presents with a psychiatric syndrome, discuss". Each row below is a decision rather than a heading, so an answer that walks the rows is reasoning aloud rather than reciting.

DECISION	WHAT MOVES IT	WHAT CANNOT SETTLE IT	WHERE THE DETAIL IS TAUGHT
Which syndrome is this	Phenomenology, course, examination, cognitive profile	Serology, which speaks to the disease and not the syndrome	Here
Is it attributable to the disease	Timing against disease activity, absence of a competing explanation, atypical features	Any single investigation, since no confirmatory test exists	Here
Is it infection, metabolic or hypertensive	Cultures, biochemistry, blood pressure, imaging, lumbar puncture	The presence of lupus, which raises infection risk	Here, and the delirium notes
Is it the treatment rather than the disease	Onset after a treatment change, disease quiescent elsewhere, the drug review	Severity, which is uninformative about cause	The routing section owning the corticosteroid construct
Inflammatory or thrombotic mechanism	Focal against diffuse presentation, vascular imaging, antibody profile	Response to a treatment not yet given	Here for the limbs, the sibling section for the antiphospholipid syndrome
What is given, and how is it prescribed	Severity and acuity of the manifestation, and the age group	A remembered dose or regimen	Here for the recommendation, psychopharmacology for prescribing

IN INDIA

No Indian row was located for this item, and the shortfall is declared rather than filled: no Indian cohort, no Indian frequency estimate, no Indian figure for the cost or availability of either named agent, and no Indian guideline statement on this manifestation. The recommendation this section does carry comes from a Latin American clinical practice guideline, so its transferability to an Indian public hospital is a judgement rather than a given, and a candidate who names that is on stronger ground than one who quotes it as though it were local. What is safe to say in an Indian answer is structural: infection carries the most weight of the exclusions where the background burden of chronic infection is high, immunosuppression is decided jointly with rheumatology, and the rehabilitative limb is the one most likely to be dropped where follow-up is difficult.

PITFALL

Answering as though attribution were a formality on the way to treatment. An answer spent on immunosuppression has skipped the contested part, and also the part that protects the patient, since the competing explanations are commoner and more immediately reversible than the manifestation being reached for. Spend the answer where the difficulty is.

GOOD TO REMEMBER

Called to a ward for a patient with lupus and a new psychiatric presentation, give the medical team your view on whether the disease has reached the brain as a judgement rather than a finding, and record what would change it. An attribution offered in that form can be revised without anybody losing face; one asserted as a finding is very hard to withdraw.

MNEMONIC

Syndrome, Sepsis, Salts, Script, Then Attribute

Characterise the syndrome, exclude sepsis and infection generally, check the salts and the rest of the metabolic screen, read the script for drugs that could be doing it, and attribute to the disease last. Everything before the last step is faster, commoner and more reversible than what comes after it.

No gold standard	Attribution	GCs plus CYC
FOR DIAGNOSING THE NEUROPSYCHIATRIC SYNDROMES OF LUPUS	WHAT DIAGNOSIS ACTUALLY CONSISTS OF HERE	PREFERRED OVER GCS ALONE OR GCS PLUS RTX, ADULTS, SEVERE ACUTE MANIFESTATIONS

There is no test that confirms neuropsychiatric lupus, so diagnosis is an attribution built from timing, exclusion and the character of the syndrome, and the exclusions that matter most are infection, metabolic disturbance and the treatment itself. Treatment then splits by mechanism into an inflammatory limb, a thrombotic limb and a symptomatic limb, and the one recommendation this section carries applies to adults with severe, acute manifestations and to nobody else.

16 · Neurosarcoidosis, CNS vasculitis and antiphospholipid syndrome with psychiatric features

Neurosarcoidosis, primary vasculitis of the central nervous system and antiphospholipid syndrome are filed together as rare systemic causes of psychiatric presentation, and that filing is very nearly the only thing they share. Each arrives in psychiatry by a different route, each is settled by a different kind of evidence, and for one of them the psychiatric literature this text could locate is thin enough that saying so is the correct answer rather than an embarrassment. This section installs what is actually sourced for neurosarcoidosis, what is actually sourced for antiphospholipid syndrome, and a worked method for answering on primary central nervous system vasculitis without inventing a phenotype for it. The rare causes are examinable for exactly this reason: they are where a candidate's discipline about evidence becomes visible inside a single paragraph.

16.1 Three names, three different examination problems

The grouping is administrative. A syllabus has to put the uncommon systemic causes somewhere, and the shared shelf tempts a tired candidate into one undifferentiated answer about inflammation reaching the brain. That answer earns very little. The examiner assumes you accept that systemic autoimmune disease can affect the mind, and is asking something else: whether you can tell these conditions apart with a patient in front of you, and whether you know which parts of the answer are yours to give.

It helps to sort them on a single axis: where the discriminating information sits. For **neurosarcoidosis**, the sourced material puts discriminating information inside the mental state itself, because the described presenting picture assembles features that a purely functional psychosis would not usually assemble in the same person at the same time. For **antiphospholipid syndrome**, the sourced material does the opposite: the reported psychiatric syndromes are the ordinary syndromes of general adult psychiatry, so the mental state is precisely where the discrimination is not. For **primary central nervous system vasculitis**, the discriminating information sits in a specialty literature that this text could not quote, and that changes the shape of an honest answer without making the condition irrelevant.

Those are different problems and they take different answers. The first is a recognition problem: can you notice, in a mental state you are already examining, a combination that sends you outside psychiatry. The second is an attribution problem: with a systemic diagnosis on the file and an ordinary psychiatric syndrome in front of you, how much may you claim about the relation between them. The third is an evidence problem: what do you say when the psychiatric claim you would like to make is one your sources will not carry. A candidate who answers all three with one paragraph about immune-mediated brain injury has told the examiner that the differences are invisible to them. The generic immune-to-brain framework belongs elsewhere in this volume and is deliberately not rebuilt here.

16.2 Neurosarcoidosis: a picture that can arrive before the diagnosis

The sourced statement for neurosarcoidosis is a statement about presentation. Reported neuropsychiatric manifestations include **confusion, dysfunction in orientation or memory, hallucinations, delusions and catatonia**, and these are described as manifestations that can be the presenting features of the condition. Read the load-bearing word carefully. Presenting means the psychiatric picture can be the first thing anyone sees, which in turn means the referral may reach a psychiatrist while the systemic diagnosis is still unmade, unsuspected and absent from the file you are handed.

That is the examinable point, and it is about sequence rather than rarity. Were the systemic diagnosis always established first, this condition would be a footnote in liaison practice, because the medical team would carry it. Because the neuropsychiatric picture can come first, the recognition burden lands on psychiatry, during an ordinary assessment of what looks like a first episode of psychosis.

The discriminating feature is the combination rather than any single item. The sourced list runs across layers that psychiatric nosology usually keeps apart: a confusional and cognitive layer (**confusion, dysfunction in orientation or memory**), a psychotic layer (hallucinations and delusions), and a motor and behavioural layer in the form of catatonia. A patient in whom orientation and memory are disturbed, psychotic phenomena are present, and catatonic signs are observable has a picture crossing those layers at once. Crossing them is the signal, and the response is a decision to widen the question beyond the psychiatry room rather than a diagnosis made inside it.

Be equally precise about what the source does not license. The material behind that list is a report of a single presentation, and its framing describes these manifestations as rare. A statement of that kind establishes possibility. It does not establish frequency, it does not rank the features against one another, and it supplies no proportion of patients in whom any of them occurs. So there is no percentage in this section, and a candidate who supplies one has supplied it from memory. The marks here are for recognising the pattern and routing the patient, never for a prevalence figure.

RULE **A presenting feature is not a common feature.** The sourced claim about neurosarcoidosis licenses recognition: you keep the condition available when a picture crosses cognitive, psychotic and catatonic layers together. It licenses no statement about how often that happens, and none that this presentation is typical of the disease. State the possibility, then say what you would do about it.

16.3 What psychiatry contributes when neurosarcoidosis is the question

Once the question is open, the psychiatrist's contribution is a description good enough for someone else to act on. Record phenomenology in the patient's own words rather than in summary labels, because the summary label is what a later reader will doubt. Record catatonic phenomena as observed signs with the manoeuvres that elicited them, so that a neurologist reading your entry can see what you saw. Date the cognitive change against the psychiatric history: a disturbance of orientation and memory that arrived with the psychotic phenomena

reads very differently from one that has been present for years, and only the psychiatric assessment is positioned to establish that ordering.

The rest of the pathway belongs to other owners, and naming them accurately is part of a complete answer. Imaging and cerebrospinal fluid evaluation of a suspected inflammatory brain process, with immunotherapy decisions, sits with the neurology and psychiatry interface material. Assessment and management of delirium as a syndrome, including its bedside instruments, sits with the delirium and focal brain syndromes material. Corticosteroid-related psychiatric disturbance, relevant once systemic treatment begins, is owned by its own routing section and by the endocrine and metabolic material. Adjustment of psychotropic prescribing for organ impairment sits with the psychopharmacology material. Capacity, if it arises, is decision-specific and time-specific, and the statutory test belongs to the mental health legislation material.

Routing is not evasion. An answer that names the right owner for each limb of the work shows the examiner how you manage a shared patient, and in particular that you know how each team assumes the other has already done the thinking.

TRAP **Recording catatonia as a diagnosis rather than as a set of signs.** Once catatonia is entered as a conclusion, the file behaves as though the question has been answered, and the systemic question quietly stops being asked. Candidates fall into this because catatonia feels like an endpoint: it is a named syndrome, it has a management pathway, and naming it produces the relief of having decided something. Written as observed signs instead, it stays what it is, which is a finding that still needs an explanation.

16.4 Primary central nervous system vasculitis: a declared gap, and how to answer it

Here this text has to say something uncomfortable and say it precisely. What was sought was a self-contained quotation from a primary study, a guideline or a systematic review that explicitly links primary central nervous system vasculitis to a psychiatric manifestation. No such quotation was located for this fragment. That is the gap, stated with its exact boundaries, and the boundaries matter: the search was for an explicit link to a psychiatric manifestation, quotable on its own, in a source of that standing.

Note what the gap is not. It is not a finding that the condition has no psychiatric presentation, and converting it into that claim would be a worse error than the one it replaces, because a categorical negative asserts far more than a failed search can support. Absence of a located quotation is a fact about this text's sources. It is not a fact about the disease.

Say also whose claim it would be. A statement linking this condition to a specific psychiatric syndrome would rest on the authors of peer-reviewed clinical studies of the condition and on the neurology and rheumatology specialty literature, where cohorts of these patients are described. Psychiatry cannot assume it by analogy with the conditions on either side of it in this section. The specialty that follows these patients owns the phenotype, and the honest position is to defer to it by name rather than fill the space.

Clinically the gap changes little about what you do and a great deal about what you write. The condition stays available as an inflammatory cerebrovascular possibility in a patient whose

presentation has crossed into neurological territory, and its evaluation sits with neurology as the neurosarcoidosis workup does. What changes is that you refrain from attaching a named psychiatric syndrome to it in a case note, a referral letter or an examination answer, because you would be attributing on authority you lack. You describe the presentation, record why the possibility is open, and let the specialty that can settle it settle it.

This is worth practising as a sentence, because candidates who have never said it aloud tend to bluff instead. A usable form runs: the psychiatric phenotype of this condition is not something I can state from a specific source, the neurology and rheumatology literature holds it, and my contribution is the described presentation and the referral. That answer is short, it is true, and it displays the judgement that separates a safe registrar from an unsafe one. An invented list of psychiatric features, delivered fluently, is the failure mode this discipline exists to prevent.

TRAP **Filling the vasculitis gap with borrowed lupus material.** Neuropsychiatric systemic lupus erythematosus has a rich catalogue of manifestations, it is taught in its own section of this chapter, and it sits close enough in a candidate's mind that its phenotype slides across to fill an empty space. It is a borrowed answer about a different disease, and an examiner who knows the vasculitis literature is thin will hear the borrowing at once. Name the lupus material as the neighbouring topic it is, and leave its content where it belongs.

16.5 Antiphospholipid syndrome: a psychiatric range that reads like general psychiatry

The sourced position for antiphospholipid syndrome is that a range of psychiatric disorders has been reported in these patients, and the reported range includes **psychosis, mania, depression, bipolar disorders and schizophrenia**. Read that list as a whole before reading any item in it. Taken together, **psychosis, mania, depression, bipolar disorders and schizophrenia** are close to a roster of general adult psychiatry. That is the discriminating feature of this condition, and it is a negative one in a useful sense: there is no distinctive mental state signature here to look for.

The consequence follows directly. If the reported psychiatric range is indistinguishable by name from primary psychiatric illness, the mental state examination cannot be the instrument that raises the possibility, and the discrimination has to come from elsewhere in the record. The syndrome is named for an antibody, which tells you that serology and the general medical history are where the question is settled, and that the settling belongs to the rheumatology and haematology literature. This text carries no sourced criteria for the condition, so it prints none, and a candidate who prints a criteria set from memory is doing the thing this method exists to stop.

Be careful with the verb as well. The sourced claim is that these disorders have been reported in patients with the condition. Reported in is an observation of co-occurrence in a described population, rather than a causal claim about the antibody producing the syndrome, and it carries no proportion. So a psychiatrist assessing a patient who already holds this systemic diagnosis has reason to keep the association in view while treating aetiology as unsettled. The material behind the claim is a narrative review drawing on a long publication window: a reasonable authority for the statement that a range has been reported, and a poor one for anything quantitative. There is no figure in this section either.

What this leaves you with is a clinical posture rather than an algorithm. In a patient with the systemic diagnosis on file who develops a new psychiatric syndrome, each of the easy errors is a real one. Treating the psychiatric presentation as unrelated by default discards information the record already holds. Attributing it to the systemic condition by default converts a reported association into a diagnosis and ends an assessment that had barely begun. The defensible position lies between them: keep the systemic diagnosis as a live question in the formulation, put that question in writing, and manage the psychiatric presentation on its own terms while it stays open.

RULE **Reported in is not caused by.** This distinction carries more marks across the chapter than any single disease fact, because almost every systemic association in liaison psychiatry is reported at the level of co-occurrence. Say what your source says, with the verb your source uses, then say separately what you would do clinically. An answer that keeps the moves apart reads as trained. An answer that fuses them reads as memorised.

16.6 Telling them apart in one pass

The plate for this section places the disease-specific presentations beside one another, including the neighbouring topics this section deliberately leaves to their owners: neuropsychiatric lupus, and coeliac disease and inflammatory bowel disease with the gut-brain axis. Use it as a recall grid in revision, working across the row for each condition and asking what makes that condition itself rather than the one above it. The table below does something complementary: it records where each condition in this section stands in evidence, which governs how firmly you may speak.

CONDITION	WHAT THIS TEXT CAN STATE	WHERE THE REMAINDER IS HELD
Neurosarcoidosis	That **confusion, dysfunction in orientation or memory, hallucinations, delusions and catatonia** can be presenting features, so psychiatry may see the patient first	Frequency, imaging and cerebrospinal fluid evaluation, and immunotherapy: neurology and the interface material
Primary central nervous system vasculitis	A declared gap: no self-contained quotation explicitly linking it to a psychiatric manifestation was located for this fragment	The psychiatric phenotype, if described: authors of peer-reviewed clinical studies and the neurology and rheumatology literature
Antiphospholipid syndrome	That a range of psychiatric disorders has been reported, including **psychosis, mania, depression, bipolar disorders and schizophrenia**	Diagnostic criteria, serology and proportions: the rheumatology and haematology literature

16.7 Answering on the rare causes without over-claiming

A complete answer here has a recognisable shape, worth rehearsing because the content is thin enough that structure carries the marks. Five moves carry it.

- Name the three conditions separately, and refuse the single undifferentiated paragraph about inflammation reaching the brain that the shared shelf invites.
- Neurosarcoidosis: give the discriminating feature as a presenting picture crossing cognitive, psychotic and catatonic layers together, and say that the response is to widen the question rather than to make the diagnosis in the psychiatry room.
- Antiphospholipid syndrome: give the discriminating feature as a reported psychiatric range that reads like general adult psychiatry, so the discrimination lives outside the mental state, in the record and the serology.
- Primary central nervous system vasculitis: give the discriminating feature as a psychiatric literature this text could not quote, held by the specialties that follow these patients, and name it as theirs rather than filling the space.
- Mark explicitly which parts of your answer rest on a source and which rest on clinical reasoning, then route the workup to its owner.

Recognition	**Attribution**	**Evidence**	**Reported, not caused**
THE NEUROSARCOIDOSIS PROBLEM	THE ANTIPHOSPHOLIPID PROBLEM	THE VASCULITIS PROBLEM	THE VERB THAT CARRIES THE MARKS

The first three tiles are the three problems this section separates, one to each condition, and a candidate who can say which of the three a question is asking has already done most of the discriminating. The fourth is the verb discipline that runs underneath all three.

The shape survives contact with a question you were not expecting, which is its real value. The neighbouring topics in this chapter, paraneoplastic neuropsychiatric syndromes, neuropsychiatric lupus, immunosuppressant neurotoxicity after transplantation, and the gut-brain topics, are owned elsewhere in this volume and each carries its own sourced material. Name them as neighbours when a question invites the comparison, and resist teaching them inside an answer about these conditions. Examiners read borrowed content as padding, and padding is expensive in a viva.

IN INDIA

No Indian series or Indian guidance on the psychiatric presentation of these conditions was located for this fragment, and that is a limit of this text rather than a claim about Indian practice or the Indian literature. What travels regardless of setting is the referral, and it is worth writing well. Specialist assessment may be separated in time from the presentation you witnessed, so the phenomenology, the catatonic signs with their eliciting manoeuvres, and the dating of cognitive change against the psychiatric history are things only your entry preserves. Where rheumatology, neurology or immunology assessment means travel and delay for the family, the referral letter is what keeps the question alive until they are seen.

GOOD TO REMEMBER

Ask the systemic question at the moment a psychiatric picture stops behaving like one, rather than at the end when everything else has failed. For neurosarcoidosis the trigger is a presentation crossing cognitive, psychotic and catatonic layers together. For antiphospholipid syndrome it comes from the record rather than the mental state, because the reported syndromes are the ordinary ones. For primary central nervous system vasculitis it is the neurological unease that would prompt any inflammatory cerebrovascular referral, and your written answer stops short of naming a psychiatric syndrome for it. Describe fully, attribute cautiously, route to the specialty that can settle it.

Three conditions, three different problems: neurosarcoidosis is a recognition problem, antiphospholipid syndrome an attribution problem, primary central nervous system vasculitis an evidence problem, so describe fully, attribute cautiously, and route each to the specialty that can settle it.

17 · Coeliac disease, inflammatory bowel disease and the gut-brain axis in psychiatry

Two bowel diseases reach the psychiatry examination for opposite reasons: coeliac disease for what it is associated with, and inflammatory bowel disease for how it behaves over time. Both are usually taught under the single heading of the gut-brain axis, and that heading does them a disservice, because it places an epidemiological observation and a mechanistic theory in the same sentence and lends them the same confidence. This section separates them: what the comorbidity evidence in coeliac disease states and what its design will bear, what the bidirectionality evidence in inflammatory bowel disease states and what it will not bear, what each finding changes in front of a patient, and where the mechanistic account stops being evidence and becomes a story that is merely satisfying. The examinable skill here is discrimination, not enthusiasm.

Several neighbouring topics are named here and left to the sections that own them. Neuropsychiatric systemic lupus erythematosus, neurosarcoidosis, central nervous system vasculitis and antiphospholipid syndrome each have their own home in this chapter, and their manifestations are not repeated here. The general immune-brain vocabulary of inflammatory signalling and sickness behaviour belongs to the earlier notes that own it and is used here only as a name. Steroid-induced psychiatric disturbance is owned by the routing section of this chapter and appears below only as a confound to be excluded before anything else is concluded. Delirium assessment belongs to the notes on stroke, traumatic brain injury and focal brain syndromes. Organ-adjusted prescribing, including any adjustment for malabsorption, belongs to the psychopharmacology notes, so no dose, no interaction ranking and no drug choice is stated in this section.

17.1 Three different claims wearing one name

The phrase **gut-brain axis** is used to make at least three claims that differ sharply in how well they are supported, and most of the confusion in this topic comes from sliding between them

inside a single answer.

- A **co-occurrence claim**: people carrying the bowel diagnosis carry more psychiatric morbidity than some comparison group. This is a question about counts in two populations, and it is the easiest of the three to test.
- A **covariation claim**: within a single person, the state of the bowel and the state of the mind move together over time, and each may carry information about the other's future. This needs longitudinal data and is harder to establish.
- A **mechanistic claim**: a specified biological pathway carries the influence in one direction or both. This is the claim the informal literature makes most often and the one it supports least well.

The two diseases in this section illustrate the first two claims respectively. Neither of the findings taught below is a mechanistic claim, and keeping that straight is most of the work an examiner is testing.

17.2 Coeliac disease: the comorbidity signal, and the shape of it

The finding to carry into the examination comes from a systematic review that compared people with the diagnosis against healthy controls. It **identified a significant increased risk for autism spectrum disorder, attention deficit hyperactivity disorder, depression, anxiety and eating disorders in patients with coeliac disease compared with healthy controls**. That sentence rewards slow reading, because each of its parts constrains what may afterwards be done with it.

The comparator is **healthy controls**, and that one phrase governs the whole interpretation. A comparison against healthy people cannot separate what belongs to coeliac disease specifically from what accompanies having any chronic diagnosis at all: a restrictive treatment, a lifetime of appointments, a label, and the ordinary demoralisation of managing a condition that never resolves. A candidate who says the finding demonstrates a disease-specific psychiatric vulnerability has reported a comparison that was never made. The defensible formulation is that psychiatric morbidity is raised relative to people without the disease, and that the design does not tell us whether it is raised relative to people carrying a different chronic illness of similar burden.

The outcome list is broad rather than narrow. It **spans neurodevelopmental, affective, anxiety and eating-disorder categories** rather than picking out a single syndrome, and breadth of that kind fits a general elevation of psychiatric morbidity better than it fits a specific lesion producing a specific disorder. That does not make the finding uninteresting. It makes it a reason to take a full psychiatric history in this population rather than a reason to go looking for one particular condition.

The design is a synthesis of observational comparisons. It establishes that the categories co-occur more often than expected, and it establishes nothing at all about which of them came first. The

rival explanations that survive a design of that kind are set out below, and reciting them is the cheapest mark available in this topic.

TRAP **Converting an association list into a causal list.** The review reports raised risk across several diagnostic categories against healthy controls. Candidates routinely restate this as the bowel disease causing the psychiatric disorders, then reason forward to a treatment implication that no observational synthesis can support. The finding tells you whom to assess carefully. It does not tell you what to attribute, and it does not tell you what to treat first.

17.3 What the coeliac finding actually changes in the room

Two situations arise, and they call for different work. In the first, the bowel diagnosis is already made and the person is referred with a psychiatric presentation. Here the finding does one useful thing: it removes any temptation to treat the psychiatric picture as an obvious reaction to illness that needs no further characterisation. Because the associated categories **include neurodevelopmental as well as affective and anxiety conditions**, a history that stops at mood in the last few months is too short. The developmental history, the school history and the pattern of attention and social functioning across the lifespan belong in the assessment, and they are the parts most often skipped when a medical diagnosis is already on the front of the file.

The entry in that list which most changes a consultation is **eating disorders**, because the management of coeliac disease is itself dietary exclusion. A medically required restriction and a symptomatic restriction occupy the same behavioural territory, and each can conceal the other. A person whose intake has narrowed far beyond what the bowel disease requires may be presented by the family, and even by the treating team, as unusually adherent. The clinically useful question is not whether the person avoids particular foods, which the treatment already dictates, but what the avoidance is for, what happens when it is breached, whether weight and shape have entered the reasoning, and whether the restriction has spread past the medical boundary into foods it never covered. That distinction is doable in the consultation and is where the psychiatric contribution is greatest.

In the second situation the bowel diagnosis has not been made and the temptation is to reason towards it from the psychiatric presentation. Here the section declares a limit rather than manufacturing guidance. No evidence row available to it grounds a screening rule, a case-finding threshold, an at-risk group worth testing or any performance figure for a diagnostic test, so none is printed. What survives without a row is procedural, and it is enough for most examinations: gastrointestinal symptoms, unexplained weight loss and unexplained nutritional deficiency belong to the referring physician, and the right move is a clear referral question rather than a test ordered from the psychiatry clinic.

17.4 Inflammatory bowel disease: two arrows, and the modal verb that carries them

For inflammatory bowel disease the claim to hold is different in kind. A systematic review and meta-analysis concluded that **bidirectional effects of the brain-gut axis are present in inflammatory bowel disease and may influence both the natural history of the disease and psychological health**. This is a covariation claim, not a co-occurrence claim, and it is the more

clinically interesting of the two because it concerns what happens next rather than what is present now.

Unpacked, **bidirectionality** is two arrows, and the conclusion adds a qualifier. The first arrow runs from the bowel to the mind: the state of the disease bears on psychological health. The second runs from the mind to the bowel: psychological state bears on the natural history of the disease. A complete answer names both, because naming only the first reduces psychiatric symptoms to an understandable reaction and leaves the psychiatrist with nothing to offer except sympathy, while naming only the second overstates what any single service can influence.

The qualifier matters as much as the arrows. The conclusion is that these effects **may influence** disease course and psychological health. It is a statement that bidirectional effects are present and that influence is possible. It is not a magnitude, it is not a treatment claim, and it is emphatically not a finding that treating a mood disorder alters the course of the bowel disease. That last step is the one candidates take without noticing, usually in the final sentence of an otherwise careful answer, and it converts a defensible position into an unsupported one.

TRAP **Promoting a bidirectional association into a therapeutic promise.** "The brain-gut axis is bidirectional, therefore treating depression will improve the bowel disease" reads as a natural inference and is a different claim from the one the evidence makes. Treat the psychiatric disorder because it is a disorder and it causes suffering. Do not sell it to the patient or to the gastroenterology team as disease-modifying therapy.

17.5 Working both arrows at the bedside

Bidirectionality becomes concrete as soon as it is turned into a sequence of consultation moves. Because **the effects run in both directions**, the first task is to work out where in the cycle the person currently sits. The psychiatric history on its own will leave that question open.

Begin with disease activity. Before attributing fatigue, poor concentration, disturbed sleep and reduced appetite to a mood disorder, establish with the treating team whether the bowel disease is currently active, when it was last assessed, and what the plan is. The activity measures used by the gastroenterology service are theirs to interpret and are not named or scored here; what the psychiatrist needs is the team's answer, in writing, to the question of whether this is active disease. A psychiatric formulation written without that answer is a guess about which arrow is operating.

Next exclude the treatment as the explanation. Corticosteroid-induced psychiatric disturbance is taught in full by the section of this chapter that owns it, and is named here only so that it is excluded before anything is attributed to the axis. Establish what the person is taking, what was started or stopped in the weeks before the change in mental state, and whether the timing fits. A mental state change that tracks a treatment change is a treatment problem until proven otherwise, and no amount of gut-brain reasoning displaces that.

Then characterise the illness experience rather than assuming it. Urgency, incontinence, nocturnal symptoms and the resulting narrowing of where a person is willing to go are facts that patients rarely volunteer, because they are humiliating. Asking about them directly is the part of

the interview that most often turns a generic depressive episode into a specific and modifiable set of avoidances.

RULE **Establish disease activity and treatment timing before you formulate.** In a person with inflammatory bowel disease, low mood, fatigue and poor concentration have at least four candidate origins: active disease, the treatment, the restriction the illness imposes on daily life, and an independent psychiatric disorder. These are not mutually exclusive and they demand different responses. Writing to the gastroenterology team with a specific question, and waiting for the answer, is faster than treating the wrong one.

17.6 Mechanism, and where a careful answer stops

This is the part of the topic where overreach is routine, so the boundary is worth stating plainly. The mechanistic literature on gut-brain communication is large and is written with more confidence than its evidence carries. Several families of explanation are commonly offered: immune and inflammatory signalling, the gut microbial community and its products, nutritional and absorptive consequences of the bowel disease, the effects of treatment, shared genetic or developmental liability acting on both systems, and the ordinary psychological load of a chronic, unpredictable and stigmatising illness. These are named here as categories only.

No evidence row available to this section grounds any specific mechanism in either disease, and none is asserted here. That is a declared gap, not an oversight: the two findings this section owns are an association in coeliac disease and the presence of bidirectional effects in inflammatory bowel disease, and neither identifies a pathway. A candidate asked to explain the axis mechanistically should name what is proposed, mark it as proposed rather than established, and then give what the evidence does support, which is the two claims above.

Four alternatives should be recited whenever an association in this field is offered as though it were a mechanism. The association may run in the reverse direction from the one assumed. A third factor may produce both. Detection may be differential, so that people already in one care system are more thoroughly investigated in the other. And the association may be entirely real yet wholly mediated by the burden of chronic illness, which is a psychological explanation and needs no biology at all. An answer that lists these four has demonstrated the exact competence being examined.

TRAP **Reaching for the microbiome because it is available.** A fluent mechanistic paragraph is easy to produce in this topic and is the commonest way of converting a good answer into a suspect one. Fluency is not evidence. Say what is proposed, mark it as proposed, and spend the remaining time on the two findings you can actually defend and on what they change in the consultation.

17.7 Where these two sit among the systemic presentations

It is worth placing coeliac disease and inflammatory bowel disease alongside the other systemic conditions that reach psychiatry, because they belong to a different column. The autoimmune conditions taught elsewhere in this chapter present as direct neuropsychiatric manifestations, in which the illness process itself produces the psychiatric picture and the diagnostic task is to recognise it. The two bowel diseases here are not taught that way. Their psychiatric relevance is

an elevated burden of common psychiatric disorders in coeliac disease and a bidirectional relationship with disease course in inflammatory bowel disease, which is a question of comorbidity and prognosis rather than of a direct neuropsychiatric syndrome.

The systemic autoimmune comparator plate sets each systemic presentation in this chapter beside its discriminating feature, so the conditions can be told apart in the moment rather than merely listed. Read it as a routing aid: the discriminating feature is what should redirect the assessment when it is present. The two entries relevant here are the ones whose discriminating feature is not a neuropsychiatric sign at all, and noticing that asymmetry is the point of putting them on the same plate.

That plate is printed with the neuropsychiatric lupus section, where it places these two conditions beside the other four, and it is the fastest way to revise the discriminating features together.

17.8 What a complete answer contains

A structured answer on the gut-brain axis contains five things in this order: the definition with the three claims separated; the coeliac finding stated with its comparator, which is the part most answers drop; the inflammatory bowel disease finding stated with both arrows and the qualifier intact; the design limitations, meaning direction, shared liability and differential detection; and the consultation consequences, meaning a lifespan history in coeliac disease, the required-versus-symptomatic restriction question, and disease activity and treatment timing established before any psychiatric attribution in inflammatory bowel disease.

QUESTION	COELIAC DISEASE	INFLAMMATORY BOWEL DISEASE
Type of claim the evidence supports	Co-occurrence against a comparison group	Covariation over time, in both directions
What is stated	**Increased risk of autism spectrum disorder, attention deficit hyperactivity disorder, depression, anxiety and eating disorders against healthy controls**	**Bidirectional brain-gut effects are present and may influence disease course and psychological health**
What is not stated	Direction of causation, disease specificity against another chronic illness, any mechanism	Any magnitude of effect, and any claim that psychiatric treatment alters the natural history
First move in the consultation	Full lifespan psychiatric history, developmental included, and the required-versus-symptomatic restriction question	Establish current disease activity and treatment timing with the treating team, in writing
Commonest error	Reading the association list as a causal list	Promising disease modification from psychiatric treatment

IN INDIA

No Indian prevalence figure, service audit or cost estimate for either disease appears in this section's evidence rows, so none is printed; the shortfall is declared rather than filled. What can be said is organisational. The gastroenterology service is usually in a different building from psychiatry and often in a different institution, so the liaison here is a written one, and it should carry a specific question rather than a request for an opinion: is the disease active, what changed in the treatment and when, and what is the plan for the coming months. The dietary restriction that treats coeliac disease is negotiated inside a shared kitchen, with food prepared for the household at once, so an adherence discussion aimed only at the patient misses where the decisions are actually made. And where a person travels a long distance to a tertiary clinic, align the psychiatric and gastroenterology reviews to the same visit wherever the services can manage it, because a review that needs a second journey is a review that will not happen.

GOOD TO REMEMBER

In coeliac disease, widen the history: the associated categories reach into development and into eating behaviour, so a history that covers only the last few months of mood is the wrong shape. In inflammatory bowel disease, narrow the question: find out whether the disease is active and what the treatment has been doing before you attribute anything to the axis. In both, treat the psychiatric disorder because it causes suffering and is treatable, and resist the pull to sell it to the patient or to the referring team as a way of changing the bowel disease.

MNEMONIC

Widen, then narrow

Widen for coeliac disease, because the associated conditions cross the lifespan and the eating-behaviour boundary. Narrow for inflammatory bowel disease, because the useful question is which arrow is operating right now, and that is answered by disease activity and treatment timing rather than by mechanism.

Three, not one	Comorbidity, not cause	Possible, not measured	Proposed, not established
CLAIMS INSIDE THE ONE PHRASE	WHAT THE COELIAC REVIEW SUPPORTS	WHAT THE QUALIFIER LEAVES OPEN	HOW MECHANISM MUST BE MARKED

Coeliac disease gives you a comorbidity fact and no direction; inflammatory bowel disease gives you a direction in both senses and no magnitude. Everything beyond those two sentences that sounds mechanistic is a proposal, and saying so out loud is what separates a careful answer from a fluent one.

18 · Burn injury and its psychiatric course

Burn injury is the one medical event in this chapter where the psychiatric question runs forwards and backwards: what the injury has done to the patient, and what the patient may have been doing when it happened. Everything examinable here follows from holding those

apart. This section installs a timeline, because each phase of burn care asks a different question, then a question that sits outside the timeline because it is asked at presentation and concerns the moment before the flame. It also states what this text can source and what it cannot, since burns psychiatry is taught nowhere else in these notes.

18.1 The acute phase: the ward you are called to

The referral comes from a surgical or burns team, and psychiatry walks into an environment organised around fluid balance, wound and airway. The mental state you are asked about is produced by several processes at once, and the acute-phase skill is separating them rather than settling on a label. Distress belonging to the event, distress belonging to the pain, a disturbance of consciousness and the effects of what is given for the pain are all live in the same bed at the same hour, and each responds to something different.

What was sought here, and not obtained in verifiable form, was a quotable statement from a burns or trauma source able to carry it that psychological shock, **dissociation** and **acute stress disorder** may occur in the acute phase after burn injury. State the reason exactly, because it changes what the gap means: of the burns records assembled for this section, several hosts refused automated retrieval, one address no longer resolved, and one quotation could not be found in the document actually served. Each of those is a fact about retrieval, none about burn patients. The claim, if made, would rest on the burns and trauma literature, where acute-phase presentations after burn injury are described from real cohorts, and which psychiatry reads rather than owns.

Without the figure, you describe. Acute distress after a burn injury is observable at the bedside and needs no prevalence attached. Record the phenomenology in the patient's own words, date it against the injury and against each operation, and note whether it fluctuates. That record lets a later clinician settle the question you could not, and it beats a confident figure from nowhere.

One process must be named and handed straight over. Disturbed consciousness, disorientation and fluctuating attention occur in burn patients, and the acute burns bed is one place a psychiatrist meets them. This chapter states that they occur and stops there. Assessment, motoric subtypes and management belong to the Stroke, TBI, delirium and focal brain syndromes notes. The routing is deliberate: a second delirium pathway written inside a burns section is how sibling chapters come to disagree, and a disagreement about an acutely unwell patient is a safety defect rather than an editorial one.

RULE **In the acute phase, ask which process, not which label.** Acute distress, pain, disturbed consciousness and drug effect co-occur in a burns bed and respond to different things. The examinable act is separating them at the bedside and routing each to its owner: the disturbance of consciousness out of this chapter, analgesia to the treating team.

18.2 Pain, and the part of it that is yours

Pain is the largest psychiatric problem of the acute phase and the one most easily handed back whole. Burn pain is unusual in being renewed deliberately: dressing changes, debridement and physiotherapy each produce a further episode of severe pain at a time the patient can predict, so

anticipation becomes part of the pain. That anticipatory limb is psychological and sits inside your remit whether or not the analgesic chart does.

The sourced statement available here is modest and useful. In a systematic integrative review of psychotherapeutic interventions for burns patients, **relaxation breathing techniques are an intervention to be considered for pain and anxiety relief in the care of burns patients**. The pairing is the teaching: the intervention is offered for pain relief and for anxiety relief together, which matches what the ward presents, a patient whose pain and whose fear of it have become one complaint by the third dressing change.

Its practical virtues belong in an answer: it needs no equipment, costs nothing, can be delivered by nursing staff once taught, and can be rehearsed with the relative at the bedside. It is an adjunct, and an adjunct sits alongside analgesia rather than in place of it. Analgesic prescribing stays with the treating team and this chapter prints no drug and no dose. Psychotropic selection and any organ-related adjustment belong to the Psychopharmacology II: emergencies, resistance, monitoring and interactions notes, which this chapter adds no further version of.

TRAP **Handing pain back whole.** Candidates write that analgesia is the surgical team's business, which is true of the prescription and false of the problem. Anticipatory anxiety before a predictable painful procedure is a psychological mechanism with a psychological adjunct, and an answer that omits it surrenders the part of burn pain psychiatry was consulted about.

18.3 Sleep, and where its causes sit

Sleep fails early after burn injury, and it fails for reasons that come from more than one place. What was sought here, and not obtained in verifiable form for the retrieval reasons already given, was a quotable statement that poor sleep in burn patients reflects psychological factors and the hospital environment together. That claim would rest on the burns and trauma literature, and on the ward-based sleep work within it.

The gap costs little at the bedside, because the instruction underneath it survives intact. Take the sleep history so that the sources separate: on one side what the ward is doing, meaning light through the night, noise, observations, position and procedures timed for the day list; on the other what the mind is doing, meaning intrusive re-experiencing, nightmares, worry about appearance and money, and the anticipated dressing change. Ask about each explicitly, because a patient asked only whether they are sleeping badly will say yes and tell you nothing actionable. Then correct the environmental limb first, since a hypnotic prescribed into a bright and noisy bay treats a cause that was never in the patient.

18.4 The subacute phase: appearance and disfigurement

As the patient stabilises, the question changes from survival to what the body now looks like and what that will mean for the life attached to it. Keep these apart. **Disfigurement** is a visible difference in the body, assessable by anyone looking at it. **Appearance concern** is what the person makes of that difference, assessable only by asking them. An assessment that measures the burn and infers the distress has measured the wrong variable, and it will be wrong in either direction.

The sourced finding comes from a multi-centre prospective cohort of adults hospitalised after burn injuries. **Increased psychological flexibility during hospital admission after a burn injury predicts lower appearance concerns over time, although not over the effect of baseline appearance concerns.** Each part carries an examinable point. The exposure was measured during the admission, so the acute admission is a measurement window and not only a treatment window, and the outcome came later, so an acutely measured variable travels forward into a later-phase result. That is why this section is a timeline rather than a list of complications. And the qualification is what an examiner will press: the prediction does not hold over and above baseline appearance concerns, so what the person already thinks about their appearance carries the prognostic weight.

The clinical consequence is direct. Ask about appearance concern during the admission, because it carries the prognostic weight and costs one question, and record it so that later change can be read against something. Be careful, though, describing any intervention aimed at flexibility, because the study supporting the association is the study that qualifies it.

TRAP **Reading a predictor as a treatment target.** A variable that predicts an outcome, then loses its prediction once a baseline is controlled for, is telling you where to look rather than what to change. Candidates keep the first clause of that sentence and drop the second, and the dropped clause is why the finding was reported as it was.

Social re-entry also begins in the ward rather than at the front door, and the work is concrete: rehearsing the first meeting with people who have not yet seen the scars, deciding what the patient will say when a stranger asks, and preparing the family, whose answers to the same question set the tone the patient has to live with.

PHASE	THE QUESTION IT ASKS	WHERE IT IS ANSWERED
Acute	Which process is producing this mental state, and what does each need?	This section, for acute distress and for the psychological limb of pain and sleep.
Acute	Is this a disturbance of consciousness, and how is it assessed and managed?	The Stroke, TBI, delirium and focal brain syndromes notes. Named here, never taught here.
Subacute	What does this person make of the disfigurement, and how closely are they followed?	This section, with its prevalence gaps declared.
Any phase	Which psychotropic, and how is it adjusted for organ impairment?	The Psychopharmacology notes. This chapter adds no further version.
Any phase	Does this patient have capacity to refuse the dressing change?	The Mental health legislation notes. Capacity is decision-specific and time-specific; the test is taught there.
At presentation	How did this burn injury happen, and was it an act?	This section, as an antecedent question.

18.5 Depression and post-traumatic stress: what is missing, and what holds

Here the declared gaps bite hardest. What was sought first was a citable prevalence of post-traumatic stress disorder after burn injury, as a proportion of a defined cohort at stated follow-up points inside the first year. What was sought second was a pooled estimate linking burn extent, as a proportion of **total body surface area** above a stated threshold, to an increased risk of depression expressed as a risk multiple. Neither was obtained in verifiable form, so this text supplies neither the proportions, nor the threshold, nor the multiplier. Each would rest on the burns and trauma literature, on primary follow-up cohorts for the first and a quantitative synthesis of such cohorts for the second.

Now the part that carries marks. A missing figure is not a negative finding, and converting one into the other is a larger error than the one it replaces. To say that post-traumatic stress after burn injury is uncommon, or that burn extent is unrelated to later depression, asserts far more than a failed retrieval can support. The correct sentence is short: the figure exists in the burns literature, I cannot state it from a source, and it does not change what I do with the patient in front of me. It does not, because prevalence tells you how hard to look, never whether this patient has the syndrome. That is settled by assessment, applied during admission, at the transition out of acute care, and across the long rehabilitation tail when the operations have stopped and the attention has thinned.

What holds, sourced and directive, is what to do with a patient who screens badly. European practice guidance for burn care states that **patients with an abnormal psychological profile, including suicidal ideation, should be given special attention, adequately monitored and regularly followed by appropriate mental health professionals**. Notice its shape. It is written around a profile rather than a diagnosis, so it is actionable on the day of the assessment and before any diagnostic conclusion is reached. It names obligations of distinct kinds, attention and monitoring and follow-up, which are commonly collapsed into one vague plan. And it names suicidal ideation inside the profile, which is the hinge into the last part of this section.

TRAP **Turning a missing number into a negative finding.** The candidate who cannot recall a prevalence says the association is weak or the disorder is rare. That is a categorical claim built on a failure of memory, unsafe in exactly the domain where being wrong costs a life. Name the gap, name whose literature holds the figure, and state what you do anyway.

18.6 Rehabilitation, and the return to work

Rehabilitation after a major burn injury is measured in years. Scars mature slowly, contractures develop and are released, and each new operation re-opens the acute and subacute questions in a patient who had begun to leave them behind. The consequence is unglamorous and examinable: psychiatric follow-up must outlast surgical follow-up, and the plan should say so in writing, because attention thins once the wounds are closed and it is then that appearance concern and social re-entry become the whole of the patient's day.

A further gap is declared here. What was sought was a comparison of work-reintegration outcomes between burn survivors whose injury happened at work and survivors injured

elsewhere, with the direction of the difference stated. It was not obtained in verifiable form, and it would rest on the burns rehabilitation and occupational health literature, where such cohorts are followed and outcomes defined well enough to compare.

The instruction underneath it survives the gap: ask where the burn injury happened, because the answer reshapes the rehabilitation problem. When the injury occurred at work, the site of the injury and the site of return are the same place, and that single fact brings in fear of the machine or the flame, the employer's response, blame, compensation and colleagues who witnessed it. A domestic injury raises its own version, since the kitchen is also a place the patient must return to. In Indian practice much work is informal, so returning to work may mean returning to a stove, a field or a roadside stall, with no graded return to arrange and financial pressure that overrides advice framed as a preference.

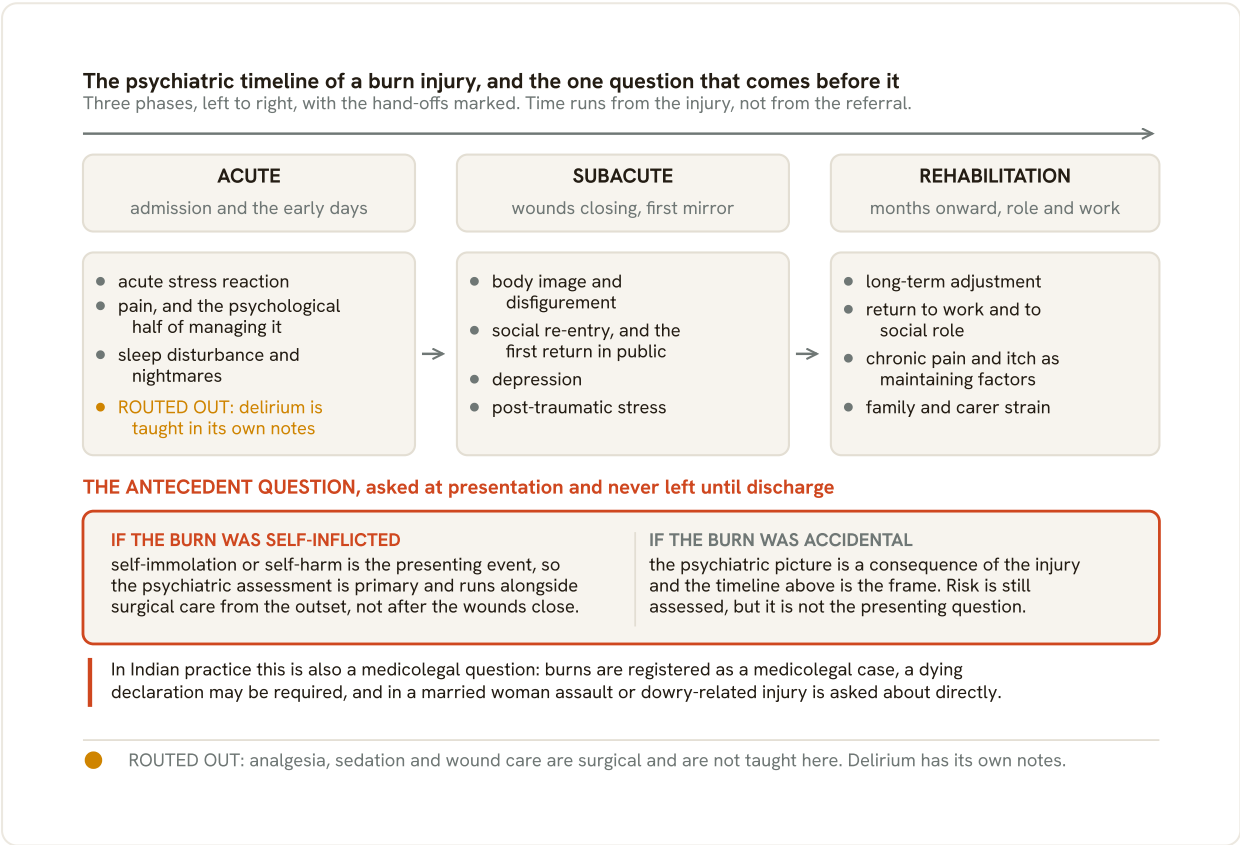

Fig 40.10 The psychiatric course of a burn injury across three phases, with the antecedent question set apart from the timeline because it is not part of it. This is the only plate in the series that teaches burns, so it is built to carry the whole construct rather than illustrate one corner of it. The phases are not merely descriptive: each has a characteristic failure, which is treating acute distress as a disorder before the wounds have closed, missing the subacute period in which body image and the first return in public do most of the psychological damage, and discharging at wound healing as though the rehabilitation phase were somebody else's problem. The block below the timeline is the discrimination that changes management, because a self-inflicted burn makes the psychiatric assessment the primary one from the outset rather than a consequence to be picked up later, and in Indian practice the same question carries a medicolegal weight that an accidental injury does not. From memory you should be able to name the phase-specific problems in order, say where the delirium pathway hands off, and state why self-harm is asked about as an antecedent at presentation rather than as an outcome at discharge.

18.7 The antecedent question, asked at presentation

Everything above treats psychiatric problems as consequences of burn injury. There is a question that runs the other way, asked at presentation rather than at discharge: was the injury itself an

act, and was psychiatric disorder present before the flame rather than after it. That discrimination changes management, and it is the one most often lost when a burns referral is treated as routine liaison work.

It changes management because consequence-work and antecedent-work run on different clocks. Consequence-work is rehabilitative and can be paced. Antecedent-work is risk-work and cannot be: if the burn injury was self-inflicted, the patient is already inside a high-risk period, the method remains available on discharge, the collateral history has to be taken while relatives are still in the hospital, and the discharge destination becomes a psychiatric question. A service that asks how the burn happened only when the wounds are healing has asked its most important question several weeks late.

The Indian relevance is direct, because self-immolation presents to burns units here rather than to psychiatric services, and because the Indian psychiatric literature has examined it. **An Indian report investigated four cases of attempted self immolation, with the psychiatric evaluation done according to DSM-III-R criteria.** Be exact about what that establishes. It is a small clinical series with formal diagnostic assessment applied to it, so it establishes the construct as one Indian psychiatry has studied with criteria rather than impression. Its denominator is small, and using it as though it described a national picture would misuse it.

The same literature shows that self-immolation is heterogeneous rather than a single clinical entity. In an Indian psychosocial study, **22 young people, mostly students, had indulged in this act to protest against the decision of the Government of India**. A public and collective act of protest is a different phenomenon from a private act of self-harm during a domestic crisis, and a formulation built for one will misdescribe the other. So the clinical question is simple to ask and easy to skip: what was the act for. The same physical burn injury can follow any of these.

- A suicidal act, after which the patient is already inside a high-risk period and the method remains available on discharge.
- A public and collective act of protest, which is the phenomenon the Indian psychosocial study above describes.
- An impulsive act inside a family quarrel, a private act during a domestic crisis rather than a public one.
- An accident, where the injury was not an act, though the question of psychiatric disorder before the flame still stands.
- An assault by another person, which this section names as an antecedent and does not follow further.

Those demand different assessments and different collateral sources, which is why the question belongs at presentation rather than in the discharge summary.

Where the assessment finds a self-inflicted injury, ongoing suicidal ideation, or a disturbed psychological picture of any kind, the sourced instruction above applies directly and is worth quoting rather than softening: **special attention, adequate monitoring and regular follow-up**

by appropriate mental health professionals. In a burns unit those obligations have concrete forms. Attention means your assessment happens at presentation, in the surgical bed, written where surgical staff will read it. Monitoring means the nursing observations reflect a psychiatric risk as well as a wound. Follow-up means a named appointment attached to a named person, since a patient who leaves with a dressing clinic date and a vague psychiatric intention will attend the first and lose the second. Where such a patient then refuses dressing changes, capacity becomes the question, and capacity is decision-specific and time-specific; the statutory test is taught in the Mental health legislation and forensic psychiatry notes.

IN INDIA

The burns unit is usually where this psychiatry happens, because the patient occupies a surgical bed and the psychiatric team travels to them. Your assessment competes with a dressing round, your notes are read by surgeons, and the family outside is your collateral source and the carer who will do the dressings at home. Distance and cost shape everything after discharge, so a plan written for a family travelling a long way looks different from one written for a city outpatient. Note what this text could and could not verify. The acute-phase, prevalence and work-reintegration claims were not obtained in verifiable form here, which is a limit of this text rather than a statement about the Indian record. What was verified is Indian, the self-immolation work, and that tells you where Indian psychiatry has looked hardest at burns.

GOOD TO REMEMBER

Ask how the burn injury happened before you ask how the patient is coping with it, because the first question sets the urgency of everything else. In the acute bed, separate the processes and route the disturbance of consciousness to the Stroke, TBI, delirium and focal brain syndromes notes rather than managing it here. Treat anticipatory pain as your work and offer the psychological adjunct alongside the analgesia the team prescribes. Ask about appearance concern during the admission, because it is the early variable that carries the later weight. Where a figure is missing, name the gap and the literature that owns it, then say what you will do anyway.

Antecedent first ASKED AT PRESENTATION, NOT AT DISCHARGE	**Which process, not which label** WHAT THE ACUTE BURNS BED ASKS OF YOU	**Asked in the admission** WHEN APPEARANCE CONCERN CARRIES THE LATER WEIGHT	**A named gap, not a negative** WHAT A PREVALENCE YOU CANNOT SOURCE BECOMES

Burns psychiatry runs in two directions: ask at presentation whether the injury was an act, then follow the timeline of what the injury has done, separating process from label in the acute bed, treating anticipatory pain as your work, asking about appearance concern during the admission, and naming a missing figure rather than converting it into a negative finding.

Consolidate and apply

19 · Worked vignettes

Every referral in this chapter asks one question in a different accent: is this the medical illness, is this the treatment for it, or is this a psychiatric disorder that would have arrived anyway. The sections before this one supply the content for that triage. What they cannot supply is the experience of running it against a referral somebody else has already answered, where all three possibilities are live at once and the answer is sometimes that the question belongs to another chapter.

Each vignette opens with the referral as it would arrive, works the reasoning in the open, and closes with what an examiner is testing. Where a vignette reaches a specific taught elsewhere, it names the owning section and stops. Two end without a treatment, because the answer is a hand-over. One ends without a fact, because the fact was sought and not established.

19.1 A decompensated heart and a request for an antidepressant

The referral. From a cardiology ward. A man in his sixties, readmitted within months for decompensated heart failure. Low mood, poor appetite, sleeping badly, no motivation, will not participate with physiotherapy. Please see and start an antidepressant.

The reasoning. The failing circulation can generate every item on that list. Fatigue, appetite change, broken sleep, slowed movement and withdrawal from effort follow equally from the haemodynamic state, from the treatment burden, or from a mood disorder, so the count will be high whichever is true and says nothing about which of the three is operating.

What separates them is **order in time**. Put the cardiac events, the regimen changes and the mood changes on one timeline. If low mood arrived with the decompensation and lifted as it settled, that is a person responding to a frightening event. If the breathlessness and the swelling improved while interest, outlook and self-reproach stayed where they were, the mood state has separated from the haemodynamic state and behaves as an illness in its own right. The second lever is pleasure in things costing no physical effort, because a man too breathless to reach the gate can still say whether a visit gives him anything.

Keep the treatment limb open: the regimen is itself a psychological load, and admission interrupts whatever he took at home. Then look again at what was asked. The note has gone straight to a prescription, and agent choice in established cardiac disease is not this chapter's to give: it belongs to the psychopharmacology notes, and prognosis with safe antidepressant choice after myocardial infarction to the merged organ-interface routing section.

TRAP **Accepting the triage the referral has already performed.** The note has decided that this is a psychiatric disorder and that the intervention is a tablet. A consultation answering only the question asked leaves the attribution unmade, and reads later as agreement.

What the examiner is testing. Whether you attribute symptoms rather than count them, and whether you can be useful to a cardiology team without writing a prescription.

19.2 Breathless at night, and already told it is anxiety

The referral. A woman in her forties with obstructive airways disease is referred to the psychiatric outpatient clinic with episodes of breathlessness, palpitations and terror that wake her from sleep. The letter states that her airflow is stable in clinic, that the attacks are anxiety, and asks for treatment of panic.

The reasoning. The whole difficulty sits in the letter's middle clause. Breathlessness and panic recruit the same body and the loop closes both ways: airflow limitation produces fear, fear produces hyperventilation, hyperventilation produces breathlessness. The features shifting the probability, the temporal shape of the episode, its relation to exertion or rest, and the content of the thought at the peak, are taught by this chapter's section on obstructive airways disease, asthma and chronic respiratory failure. This vignette is about what a clinician does when they shift the probability without settling it, which is most of the time.

Two things follow. The commonest correct answer here is both rather than either, and each condition worsens the other, so treating one and closing the case leaves the disability where it was. And the errors are not symmetrical: calling a panic attack an exacerbation costs an unnecessary course of treatment, while calling an exacerbation a panic attack can cost a life. Note too what the letter's premise establishes. Stable airflow in a daytime clinic is a statement about the clinic, not about three in the morning, and nocturnal episodes have nocturnal explanations a daytime measurement cannot exclude.

One contribution is concrete, not diagnostic. Coexistent anxiety and depressive symptoms are routinely used to filter patients out of the rehabilitation that treats the deconditioning, and the guideline position blocking that is stated, in its exact wording, by the owning section.

RULE **When the triage cannot be completed in the room, complete the safe half and record why.** Treat the airway first, interrogate the psychiatry once the episode has settled, and write down which half you answered today. A note committing to a label it cannot defend is more dangerous than one stating what it could not decide.

What the examiner is testing. Whether you accept a triage handed to you in a letter, which direction of error is dangerous, and whether you can offer both with an order of work.

19.3 Confusion after transplantation, and what the psychiatrist does not own

The referral. A call from a transplant unit, late evening. A man in his thirties, some weeks after receiving a kidney from a living relative, has become confused, does not recognise the ward and has had a witnessed seizure. He is on maintenance immunosuppression and has had pulsed treatment for suspected rejection in recent days.

The reasoning. Several sufficient causes are present at once, so the examinable skill is **sequencing rather than selection**. He may be septic while barely febrile, metabolically deranged from graft dysfunction, hyponatraemic or hypoxic, on more than one agent with recognised

neuropsychiatric toxicity, and carrying the load of a graft he believes he could lose. An opinion that names the drug and stops has chosen the interesting cause rather than the first one.

The sequence that survives scrutiny puts reversible and dangerous causes first, keeps the immunosuppressants permanently on the list rather than at the end of it, and treats the psychological formulation as concurrent rather than as a diagnosis of exclusion. One technique is worth taking away whole: the drug history here is a sequence, not a list. What was given, when relative to the event, and what was given alongside it carry the information, because the labelled neurotoxicity signals in this class attach to changes of regimen and to combinations. A chart transcribed as current medicines has discarded the structure that made them reportable.

The boundary, which is the point of this case. Three of the four things the team wants are not yours here. Detection, rating and management of the acute confusional state, including any drug used for it, are taught once, with the stroke, traumatic brain injury, delirium and focal brain syndromes notes. Which co-prescribed agent alters exposure to which immunosuppressant belongs to the psychopharmacology notes. The named neurotoxic syndromes of the individual agents belong to this chapter's transplant and immunosuppressant section. You hand over a dated mental state, a timeline against the drug chart, a statement that the immunosuppressant stays on the differential, and the pathway for each remaining question.

RULE **A referral you cannot answer completely is answered by naming who answers each part.** Routing is a clinical act, not an evasion. It moves a question to somebody who can settle it, and it beats a partial version from memory that commits the receiving team to something you never verified.

What the examiner is testing. Whether you sequence rather than select, and whether you can be useful while declining most of what was asked.

19.4 Two questions from a surgical ward, and three different owners

The referral. The surgical team telephones the day after major abdominal surgery on a man in his seventies. He is agitated at night, pulling at his lines, settled by day. They ask two things in one breath: does he have capacity to refuse the drain, and is this postoperative cognitive dysfunction.

The reasoning. Separate the questions before answering either, because one undifferentiated account of postoperative confusion loses marks in both directions at once. Capacity first. One property does most of its work here: capacity is decision-specific and time-specific. The question is never whether this man has capacity as a standing attribute, but whether he can make this decision at this hour, and the perioperative window supplies an unusual density of moments in which the answer moves: pain, disturbed sleep, premedication and the hours after anaesthesia all sit inside it. An opinion formed on the morning round does not transfer to a conversation that evening. Everything else is elsewhere: the statutory test and the procedure following a finding of incapacity are taught with the mental health legislation notes and are neither restated nor cited by provision. You describe functional ability for the named decision at the named time; the decision belongs to the treating team.

The second question turns on an observation nobody on the ward tonight can make. A cognitive change is a relation between two observations, so the first thing to establish is not what is wrong with him tonight but whether anybody measured him before the operation. If not, the trajectory question cannot be settled on this ward at all, and the honest note says so, manages the acute presentation through the chapter owning it, and arranges the review after discharge.

TRAP **Recording a global capacity verdict.** A note stating that the patient lacks capacity, with no decision named and no time attached, is unusable by the team receiving it and indefensible if later examined. Worse, it travels: written once, it is quoted for months across decisions it was never about.

What the examiner is testing. Whether you separate two questions that arrived as one, and whether your first move is to ask about the measurement taken beforehand.

19.5 A cognitive complaint after treatment ended, and a question the evidence does not answer

The referral. A woman in her fifties, referred by her family doctor some months after finishing chemotherapy. Back at work on reduced hours, she cannot hold several things in mind, loses words she knows well, and works far more slowly than before. She asks whether this will get better, and when.

The reasoning. Take the complaint seriously and decline its title. The word patients use for this names a cause and, in naming it, stops the clinician looking. Run the triage instead.

- The illness limb: fatigue, disturbed sleep, pain and the endocrine consequences of treatment all fail exactly these effortful functions.
- The treatment limb: the cytotoxic agents are one contributor inside a bundle that also holds the surgery and the anaesthesia.
- The independent-disorder limb: an untreated depressive episode degrades attention, retrieval and speed, and is the most treatable thing on the list.
- A fourth possibility peculiar to this population: something still evolving underneath, so whether the picture is static or moving is asked deliberately.

The declared gap, which is her actual question. She has asked for a trajectory. What can be said is that a change of this kind has been reported as still detectable at the end of the interval examined, and that an interval describes how far the looking reached, not where the problem ended. A statement of the course beyond that point would rest on longer follow-up in the oncology and neuropsychology literature, and none was obtained for these notes in a quotable form. The absence describes what these notes opened, is not evidence that the course is unknown to the field, and must not be converted into reassurance or into catastrophe.

What you do without it is concrete. Treat the mood, sleep and pain degrading her cognition today. Characterise the deficit well enough for her workplace to be adjusted around it, whether or not it reverses. And set a review point, because the moment the oncology course closes is the moment nobody is reviewing cognition.

RULE **A declared gap is a complete answer and it has three parts.** State what was sought, on whose authority the missing claim would rest, and what you do without it. An answer carrying only the first two is a shrug; one supplying a remembered figure is worse, because the person receiving it cannot correct it.

What the examiner is testing. Whether you convert an observation window into a prognosis, and whether you can still leave the room with a plan after saying that something is not established.

19.6 Psychosis in an established autoimmune disease, where the differential is the whole answer

The referral. A woman in her twenties with a long-standing systemic autoimmune disease is admitted under medicine with a few days of persecutory beliefs, hallucinations and badly disturbed sleep. Her disease has recently been treated more intensively. The team has sent a serology panel and asks whether this is the disease affecting her brain.

The reasoning. The referral asks for an attribution and offers a laboratory result as the route to it, and neither is available in the form requested. Serology establishes that she has the disease. It does not establish that the syndrome in front of you was produced by it, and a patient with unambiguous systemic disease can equally have an infection, a drug effect or an unrelated psychiatric illness. Attribution here is a reasoned judgement rather than a measurement, and it is the substance of the case rather than its preliminary.

So the answer is an ordered list of what must be displaced before the disease is credited. Infection first, and staying first, in a person immunosuppressed by both the illness and its treatment. Metabolic derangement and blood pressure next, particularly where there is renal involvement. Then the drug chart, read with the dates set against the onset, because the treatment of the disease and the cause of the psychiatric picture can be the same agent; that construct is owned by the routing section of this chapter and by the endocrine and metabolic notes, and is named here only as a direction of travel. Then a primary psychiatric illness with a history predating the systemic diagnosis, and the ordinary response of a young person to a relapsing, visible illness with an unpleasant treatment.

The order matters because of where the answers lead: one path escalates immunosuppression and the other reduces it, so an attribution made the wrong way round is a harm rather than an inelegance. The manifestations and the treatment recommendation with its restrictions are taught in full by this chapter's section on the condition. Give the attribution as a judgement, with the observations it rests on and what would change it. A judgement can be revised without anybody losing face; a finding is very hard to withdraw.

TRAP **Treating a positive result as the answer to the referral question.** The panel speaks to the disease, never to the syndrome, and answering with it skips the contested part of the case. It is also how a commoner and more reversible cause is missed while treatment is escalated in the wrong direction.

What the examiner is testing. Whether you recognise that the difficulty is attribution and not treatment, and whether you write a judgement rather than a verdict.

19.7 A burns unit referral, where the first question is how the injury happened

The referral. A burns unit asks for review of a man in his twenties, several weeks into an admission after a flame injury. He is described as low, withdrawn, sleeping badly and increasingly reluctant to cooperate with dressing changes. The unit would like something started.

The reasoning. The triage runs as it does everywhere in the chapter, but the order is unusual, because one question sits underneath all three limbs and gets harder to ask with every day it is left. Ask first how the injury happened. A burn may be an accident, an assault or a deliberate self-inflicted act, and the risk formulation, the conversation with the family, the safeguarding position and the discharge plan differ completely across those possibilities. Ask early, ask directly, ask the patient and whoever brought him, and record the accounts separately, because they harden with retelling and a unit that has settled on an accident will not re-open it.

Then work the ordinary causes, which this setting stacks. Pain, and specifically the anticipatory dread of a procedure repeated on a schedule, which is a distinct and treatable phenomenon rather than a general lowness. Sleep, degraded by the injury, by the analgesia and by a unit lit and busy through the night. Acute confusional states, which occur here and route out entirely to the notes owning delirium, including their bedside assessment and every drug used for them. And an independent depressive episode, which this setting makes easy to explain away, since comprehensibility is not a diagnostic exclusion.

The word uncooperative is a **referral word**, and it repays unpicking, because it names a behaviour and hides its reason. Reluctance to face dressings after a course of them is a learned response to a procedure that has reliably hurt, and it answers to analgesia, prediction, a share of control and specific procedural anxiety work, none of which is persuasion and none a tablet. Beyond the admission sit appearance, the reactions of others and return to work, all of which belong to this chapter's burns section.

TRAP **Reading a refusal of dressings as either a character problem or a depressive symptom.** Both readings skip the mechanism and both produce the wrong intervention, one punitive and one pharmacological. The behaviour is usually specific to the procedure, which is exactly what makes it treatable.

What the examiner is testing. Whether you ask how the injury happened before anything else, and whether you can decompose an unhelpful referral word into treatable parts.

The cases share a shape: the referral names a symptom and requests a treatment, while the real question is about order in time, the rest of the chart, or ownership.

THE REFERRAL AS WRITTEN	THE QUESTION UNDERNEATH IT	WHO OWNS THE ANSWER
Low mood in heart failure, start an antidepressant	Did mood move with the circulation, or separately	Attribution here; agent choice, psychopharmacology
Her attacks are anxiety, treat panic	What drives tonight's episode, and which error is dangerous	The obstructive airways and respiratory failure section
Confused and fitting after transplantation	What was given, when, and alongside what	Sequence here; the state itself, delirium; interactions, psychopharmacology
Capacity to refuse, and is this cognitive dysfunction	Which decision, at what hour, and was he measured beforehand	Decision-specific and time-specific here; the test, legislation
Will my concentration come back	What degrades it today, and over how long a window was it observed	Function here; the findings, the chemotherapy and interferon section
Is this the disease affecting her brain	What must be displaced before the disease is credited	Attribution here; manifestations, the owning section
He is not cooperating, start something	How did the injury happen, and what is he avoiding	Circumstances here; the sourced material, the burns section

Four moves sit under these cases, and not one of them is a fact to recall.

Re-opened, not accepted	**Attributed, not counted**	**Named, not attempted**	**Sought, not remembered**
THE TRIAGE THE REFERRAL ARRIVES WITH	WHAT MAKES A SYMPTOM LIST MEAN ANYTHING	THE PART OF THE REFERRAL THAT IS NOT YOURS	A FACT THESE NOTES COULD NOT ESTABLISH

The first move is the one the other three depend on, because a triage accepted at the door decides the answer before any of them is run.

IN INDIA

Several features of Indian practice change how these cases run, and none is a figure, because this section holds none. Referrals arrive late and pre-decided, so the first act is usually to re-open a triage somebody else closed. A relative is almost always present, which makes collateral history easy to obtain and the funding question answerable at the bedside. Follow-up happens by travel rather than by schedule, so a plan whose active ingredient is a review appointment fails quietly. And how a burn happened carries legal consequences from the day of admission.

GOOD TO REMEMBER

Before answering any referral in this chapter, ask three questions in order. What has changed, in what order, against what baseline. What else on the chart or in the admission could produce the same picture. And which part of this is not mine to answer here. The first settles the attribution, the second protects the patient, the third makes the note usable.

Run the triage rather than accepting the one in the referral letter, attribute by order in time rather than by symptom count, name the owner of every question that is not yours, and where a fact was not established, say what was sought, whose claim it would be, and what you do anyway.

20 · Consolidation and quick review

In the last week before the examination the useful question is not what this chapter contained but what it installed, and it installed one triage, one habit of naming owners, and one way of saying what is not established. Every section is a worked instance of those three. This consolidation restates them in the smallest form that survives questioning: the triage and the evidence deciding each limb, a grid of the features separating the conditions the chapter teaches, the boundaries stated as a competence rather than an apology, the shapes an answer should take, and what to read if there is an hour left. Read it as a set of moves rather than a summary, because content cannot be compressed without becoming the thin second copy the chapter argued against, whereas reasoning can.

20.1 The three-way question, and what each answer is made of

The chapter opens with a triage and never leaves it. A psychiatric presentation in a medically unwell patient is being produced by the illness acting on the brain, by the treatment for that illness acting on the brain, or by a psychiatric disorder that is the patient's own and has arrived in a body that happens to be unwell. Those are the three limbs, and the chapter is training in choosing between them and in saying why.

Two properties matter more than the categories. The limbs are not exclusive, and more than one is usually running at once, so the working question is never which limb is true but which is doing the most damage now and which, if missed, is dangerous. Each is settled by a different kind of evidence, which is why an answer naming a syndrome without placing it against a limb has skipped the examined step.

LIMB	WHAT SETTLES IT	WHAT IT OBLIGES	CHARACTERISTIC FAILURE
The illness acting on the brain	Disease course and physiology, read against the timing of the psychiatric change	Establish it and hand back for treatment of the disease, since no psychiatric intervention holds while the process continues	Waiting for a confirmatory test in conditions that have none
The treatment acting on the brain	Exposure and interval, from the drug chart and the procedure record rather than the mental state	Raise the exposure with the treating team, and keep the agent permanently on the differential	Pairing an agent with a syndrome and omitting the interval
A psychiatric disorder in a medically ill person	The patient's own history, baseline and account of themselves, with collateral	Treat it in full as a psychiatric disorder, since this limb is the largest and the least delegable	Accepting that anyone would reasonably feel that way, and stopping there

RULE **Name the limb, then name the evidence that placed you in it.** The follow-up at this interface is one question in different words: what else could be producing this, and what would separate them. An answer that has already given the limb and its evidence has answered it in advance, and bought time to be interesting about the rest.

20.2 The manoeuvres that decide it, in the order they are done

The triage is settled by history far more often than by investigation, and by a small set of manoeuvres that cost nothing and are frequently skipped. They are worth more than any single association here, because they transfer to every section and beyond the chapter.

- **Get a baseline from someone who is not the patient.** A medically unwell patient is observed by staff who never met the well version, so the comparison that would make a change visible does not exist on the ward until a psychiatrist goes and gets it.
- **Read the tempo, and read it for mismatch.** A presentation moving faster than the condition it resembles, continuing to move while adequate treatment is given, or following a slope rather than an episode, is telling you something.
- **Lay the psychiatric change against two timelines.** The medical course with its events dated, and the drug chart read for start dates rather than for the current list. This is the single act that separates the first limb from the second.
- **Ask what was stopped as well as what was started.** An admission removes alcohol, nicotine and whatever else was used at home, and often interrupts the patient's own long-term treatment. That syndrome, on a medical ward, is read as a complication of the illness.
- **Take consciousness and attention before anything else in the mental state.** A fluctuating disturbance of attention and awareness changes the entire answer and moves the problem into territory this chapter names and does not teach. Recognising the shape and handing it on is the examinable act.

TRAP **Settling for understandable.** The commonest error at this interface is not a wrong diagnosis but an abandoned one: the low mood is judged a reasonable response to a serious illness and the assessment stops. Comprehensibility is not a diagnostic exclusion and would not be accepted for any other comorbidity. It costs more here, because the psychiatric state is often what removes the patient from the treatment that would have changed the medical trajectory.

20.3 Telling the systemic conditions apart, which is how they are examined

The autoimmune and inflammatory band is not examined by asking what each condition is. It is examined by asking how you would tell them apart with a patient in front of you, which is why the six conditions sit together on one comparator plate. Two axes organise them: where the discriminating information sits, and what kind of claim you hold when you speak.

CONDITION	WHERE THE DISCRIMINATION SITS	THE SEPARATING SENTENCE	WHAT YOU MAY NOT SAY
Neuropsychiatric lupus	In the reasoning, not in any test	Diagnosis is **attribution**, built from timing against disease activity, absence of a competing explanation, and the character of the syndrome	That any investigation confirms it, or that one manifestation is the commonest
Neurosarcoidosis	Inside the mental state you are already examining	A picture crossing the confusional, psychotic and catatonic layers together, and it can present before the systemic diagnosis exists	That the presentation is common or typical, since a presenting feature is not a frequent one
Primary vasculitis of the central nervous system	In a specialty literature this text could not quote	A declared gap: no self-contained quotation explicitly linking it to a psychiatric manifestation was located	Any named psychiatric syndrome, and equally that it has none, since a failed search is not a negative finding
Antiphospholipid syndrome	Outside the mental state, in the record and the serology	The reported psychiatric range reads like a roster of general adult psychiatry, so the mental state is where the discrimination is not	That the antibody caused the syndrome, since the source reports co-occurrence and no proportion
Coeliac disease	Not in a neuropsychiatric sign at all	A co-occurrence claim against healthy controls, spanning neurodevelopmental, affective, anxiety and eating-disorder categories	That it is disease-specific or causal, since the comparison against another chronic illness was never made
Inflammatory bowel disease	Not in a sign either, but in the course over time	A covariation claim with two arrows and a qualifier: effects run both ways and may influence disease course and psychological health	That psychiatric treatment alters the natural history of the bowel disease

Read the third and fourth columns before the first. Two of the six carry no neuropsychiatric sign of their own, and that asymmetry is the point of putting all six on one plate, because the row not behaving like its neighbours is the row an examiner will use. One condition sits adjacent to the family without belonging to it: the paraneoplastic syndromes are immune-mediated, but their defining feature is a sequence rather than a sign or a claim type, since the syndrome arrives before the malignancy is detected, which is why suspicion, and not the workup, is the psychiatrist's part.

RULE **Name the kind of claim before you name its content.** Reported in is not caused by, an association is not a mechanism, a co-occurrence is not a covariation, and a presenting feature is not a frequent one. This distinction carries more marks than any single disease fact, because almost every systemic association here is reported at the level of co-occurrence. An answer keeping the claim and the clinical move apart reads as trained; one fusing them reads as memorised.

20.4 The treatment limb, compressed to exposure and interval

The second limb has one shape wherever it appears. The decisive evidence is in the drug chart and the procedure record, the question is what was given and when relative to the change, and the detail of what to do belongs elsewhere. What you carry is an agent family paired with a syndrome and with an **interval**, meaning the time between that exposure and the psychiatric change.

EXPOSURE	THE QUESTION IT FORCES	WHERE THE DETAIL IS TAUGHT
Calcineurin inhibitors after transplantation	What else was given, in what order, and when relative to the event, since one labelled signal is conditional on a combination	Label level in the transplant section; prescribing and interactions in the psychopharmacology notes
Corticosteroids	Which agent, over what course, and what was the earlier psychiatric history	Routed out of the chapter to the endocrine and metabolic notes, so there is exactly one place to look
Cytotoxic treatment	Did the difficulty predate the first cycle, does it fluctuate within a day, is it evolving, and has it an objective correlate	The cancer-drug section, which teaches the construct rather than the patient's word for it
Interferon-alpha	Prevention before a defined course or treatment after the syndrome declares itself, and in which population	The cancer-drug section; interferon-beta belongs to the neurology and psychiatry interface notes and is never pooled with it
Anaesthesia and surgery	Was anything measured before the operation, since a change is a relation between two observations	The perioperative section for the construct; the delirium notes for the acute confusional presentation
What the admission removed	What was stopped, at what hour, and whether the timing fits	Here, in the triage, since no other chapter owns a removal as an exposure

TRAP **Naming the agent and the syndrome and stopping.** That pairing is the least discriminating half of the topic and every candidate has it. The interval tells a clinician when to look and which explanations compete at that moment, and the population tells them whether the finding applies to this patient. A finding stated without its population is not a shorter answer, it is a weaker and different one.

20.5 The boundaries, which are a competence and not an apology

Liaison work is routing, so a chapter that practises routing is teaching the clinical skill rather than excusing an omission. A referral arrives from another team, written by someone unsure what to ask, and the first act is deciding whose question it is: the treating team's, the psychiatrist's, or a third specialty's. A resident who has practised deciding where a construct belongs has been practising the job; one who memorised a thin paragraph about each has not, and the paragraph fails the follow-up. Stating the boundary aloud also makes an answer checkable: whether a chapter mentioned a topic is a test it can pass while leaving its reader unable to answer anything, whereas whether the reader can name the owner has a right answer.

THE QUESTION IN FRONT OF YOU	WHO OWNS IT
Which agent, at what dose, adjusted how for renal, hepatic, cardiac or transplant disease	The psychopharmacology notes. This chapter teaches everything preceding a prescription and adds no second version of it.
Is this an acute confusional state, and how is it assessed and managed	The stroke, traumatic brain injury, delirium and focal brain syndromes notes, including every bedside instrument, rating scale and drug choice.
Does this patient have capacity, and what is the statutory test	The mental health legislation notes. This chapter says only that capacity is decision-specific and time-specific.
The general immune-to-brain model, and the workup of an autoimmune encephalitis	The neurology and psychiatry interface notes, which also own interferon-beta. This chapter owns the disease-specific presentation and the decision to suspect.
Depression and anxiety after infarction, critical-illness sequelae, dialysis, depression and demoralisation in cancer, palliative care psychiatry	The consultation-liaison, geriatric and transcultural psychiatry notes. These are defined by a setting and a referral pathway, not by an organ.
Psychiatric disturbance following a corticosteroid	The endocrine and metabolic psychiatry notes. This row follows a drug rather than a setting, and follows it out of the chapter.
A possible assault, and the pathway that follows it	The forensic and medicolegal material. Raised in the burns section as an antecedent question and handed on at once.

RULE **Route in the answer, out loud, before you are asked to.** A cross-reference is an address and not an abstract, so it will not survive being revised from, and the failure mode is a candidate who knows a construct matters but cannot say what it does. Say what you are handing on, who holds it, and what you keep. That costs seconds and demonstrates the judgement this interface examines.

20.6 The declared gap, and the sentence that carries it

This chapter declares more gaps than most, deliberately, and the declarations are examinable content rather than housekeeping. A candidate who can say what is not established, and on whose authority a statement would rest, answers better than one who fills the space, because a filled space is unverifiable and frequently wrong. A **declared gap** has three parts, each load-bearing.

State what was sought, with its boundaries: a quotable statement of a particular kind, linking a named condition to a psychiatric manifestation, in a source of stated standing, rather than a vague admission of uncertainty. Name whose literature would hold it, since the specialty following these patients owns the phenotype and deferring to it by name is a different act from not knowing. Then say what you do anyway, because a missing figure never changes the clinical move: prevalence tells you how hard to look, never whether this patient has the syndrome.

The declared gaps are worth revising as a set, since a stem often lands on one. The psychiatric phenotype of primary vasculitis of the central nervous system was not obtained, nor was a specialty statement binding psychiatric management to chronic respiratory failure. No prognostic figure is attached to anxiety after infarction, and no usable recommendation was opened on the desire for hastened death. The epidemiological specifics ordinarily quoted about the stress-precipitated cardiomyopathy are absent, and that section teaches what such a figure would have to measure. In lupus the antibody associations and every frequency statement are absent, because without a diagnostic gold standard each study defines its own numerator. In burns the prevalence figures and the work-reintegration comparison were not obtained, while the Indian work on self-immolation was. And no mechanism is asserted anywhere in the gut-brain material, because neither finding it holds identifies a pathway.

TRAP **Turning a missing number into a negative finding.** The candidate who cannot recall a figure says the association is weak or the condition is rare. That is a categorical claim built on a failure of memory, and it is unsafe where being wrong costs a life. Absence of a located quotation is a fact about a text's sources, never about the disease.

20.7 Answer structures, and the order the reasoning goes in

Almost every question at this interface is one of four stems, and each has an opening move that buys the rest of the answer. Learn the openings, because the first two sentences are where an examiner decides whether you are reasoning or reciting.

- **Discuss the psychiatric aspects of a named medical condition.** Open with the triage applied to that condition, naming which limb it mostly trains and why. Do not open with a list of syndromes that could have been written about any organ.
- **When would you suspect an occult or systemic cause.** Open with the readings that raise suspicion: tempo, the company the syndrome keeps, and whether a general history was taken at all. Then say what you would do, and route the investigation to its owner.
- **How would you tell these conditions apart.** Open by naming them separately and refusing the single undifferentiated paragraph about inflammation reaching the brain. Give each its discriminating feature, and mark which parts rest on a source and which on clinical reasoning.
- **A patient with a known systemic diagnosis presents with a psychiatric syndrome.** Open with the exclusions, in the order infection, metabolic disturbance, the treatment itself, and primary psychiatric illness, because those are commoner and more reversible than the manifestation being reached for.

The body then runs in the same order whichever stem opened it. Characterise the syndrome in the patient's own words. Place it against the three limbs and say what put it there. Give the discriminating feature separating it from its nearest neighbour. State each finding with its population and observation window attached. Name every construct you hand on, with its owner. Declare the gap where the chapter has one, in its three parts. Then say what you would do on the ward tomorrow, which is what most answers run out of time for and what a marker is waiting on.

Two sentences are worth rehearsing aloud, because candidates who have never said them tend to bluff. The gap sentence: that figure is held in the specialty's literature, I cannot state it from a source, and it does not change what I do with this patient. The attribution sentence: this is my judgement rather than a finding, and here is what would change it.

Both sentences are built from the same four fields, and so is every finding this page asks you to state. The comparator grid asked what kind of claim each row holds, the exposure table asked when and in whom, and the gap declaration asked what is not established and whose literature would hold it. Four words carry all of it.

MNEMONIC

Claim, Population, Window, Gap

Claim: say what grade of statement you are holding before you say what it contains, since the grades separated in the comparator rule above are not interchangeable and the weakest of them is the one most often reported here. **Population**: say in whom it was observed, because leaving that out does not shorten the answer, it changes what the answer means. **Window**: give the period over which it was seen, and on the treatment limb the gap between the exposure and the change, which is the half of that pairing most candidates omit. **Gap**: where nothing can be stated, declare it in the three parts set out above. Asked in that order the four survive a condition this chapter never taught, which is why they outlast any single row in the tables.

20.8 If there is an hour left

Revise in this order and stop when the hour is gone rather than when the chapter is finished. The order puts first what an unfamiliar stem is most likely to need.

- **The triage and its evidence table.** It is the answer structure for every question here, it works on constructs you have never met, and it is the shortest thing on this page.
- **The manoeuvres.** Baseline, tempo, the two timelines, what was stopped, and consciousness first.
- **The comparator grid for the six systemic conditions.** Work across each row, ask what makes that condition itself rather than the one above it, and check what kind of claim the row is.
- **The owner list.** Prescribing, delirium, capacity, the immune-brain model with the encephalitis workup, the setting-defined liaison constructs, and the corticosteroid.
- **The gap sentence in its three parts,** and the specific gaps this chapter declares, since a stem landing on one is a stem you answer well while others invent.

- **The exposure and interval table,** read for the second column, because agent to syndrome is the half everybody has and the interval is the half that separates.

What can safely be left is every list this chapter did not build an answer around, and every construct owned elsewhere. Reading a routed topic here in the last hour costs the material only this chapter carries: systemic and inflammatory disease presenting psychiatrically before anybody knows it is present.

If only one card leaves the hour with you, make it this one: four questions to ask of any finding before you say it aloud.

Claim	Population	Window	Gap
WHAT KIND, BEFORE WHAT CONTENT	IN WHOM IT WAS OBSERVED	OVER WHAT PERIOD, AND HOW LONG AFTER	NOT ESTABLISHED, AND WHOSE LITERATURE HOLDS IT

IN INDIA

No India-specific figure is held for this consolidation and none has been invented. The Indian point is about information rather than epidemiology. In most Indian general hospitals the psychiatrist reaches the medical bedside on request, usually late, to a referral question written by a team under pressure, and often without the previous notes, the collateral history or the drug chart's start dates. Every manoeuvre above depends on information the referring team holds and the letter rarely contains, so the skill is asking for it explicitly and early rather than working from the mental state alone. Distance and cost then decide whether the plan is followed at all.

GOOD TO REMEMBER

Three questions at the bedside, one afterwards, and one sentence held in reserve. Is the disease doing this? Is the treatment doing this? Is this a psychiatric disorder in a person who is unwell? Then, before writing: whose chapter answers the part I am handing on? And in reserve, for the moment a figure is wanted and cannot be sourced: that number is held in the specialty's literature, I cannot state it, and here is what I would do regardless.

The chapter installs a triage, a habit of naming owners, and a way of declaring what is not established. Everything else in it is worked examples, and a candidate who holds those three can answer on a condition this chapter never mentioned.

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