WEAVE SPRINT

PAPER IV · VOLUME ONE

MD and DNB psychiatry, theory revision

Neurology, Medicine and Recent Advances

Epilepsy and Clinical Neurology

The two-way traffic between seizures and the mind. Then the neurology a psychiatrist actually needs: the lesion, the headache, the sleep disorder.

Dr. Wilfred D'souza · Dr. Niharika Reddy

WEAVE

CENTRE FOR INTEGRATIVE PSYCHIATRY

Weave
Centre for Integrative Psychiatry

WEAVE SPRINT
PSYCHIATRY OUTREACH PROGRAMME

Psychiatry that listens before it prescribes.

Therapy-informed psychiatry for adults, children, couples and families. Online across India, in person in Mumbai.





Assessments

ADHD, autism and developmental. Anxiety, mood, attention and personality patterns.



Consultations and sessions

Psychiatric consultations, therapy sessions, medication review and couples sessions.



Family Library

Free plain-language guides for families, in five languages.

`weave.clinic/family`

REFER, OR SEND SOMEONE OUR WAY

Your patient contacts Weave directly and decides whether to book.

You remain the referring clinician. Any coordination between clinicians happens with the patient's permission.

CONTACT

`weave.clinic/contact`
`WhatsApp +91 7625004229`

Dr. Niharika Reddy and Dr. Wilfred D'souza, Consultant Psychiatrists. Runwal Forests Clinic, Kanjurmarg West, Mumbai, and online.

WEAVE.CLINIC

Routine referrals only. This is not an emergency route.

WEAVE SPRINT · PAPER IV

Neurology, Medicine and Recent Advances

THE WEAVE SPRINT SERIES

TITLE

Weave Sprint, Paper IV, Volume One: Epilepsy and Clinical Neurology

Part of the Weave Sprint series, MD and DNB psychiatry theory revision.

WRITTEN BY

Dr. Wilfred D'souza and Dr. Niharika Reddy

PUBLISHED BY

Weave, Centre for Integrative Psychiatry · weave.clinic

EDITION

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

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

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

ACKNOWLEDGEMENT

A word of thanks

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

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

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

Dr. Wilfred D'souza

Foreword

This first volume is the neurological interface, and it opens where psychiatry and neurology have argued longest over the same patient. Epilepsy is the model case: the seizure changes the mind, the mind changes the seizure, and the drugs for one act on the other. The neurology chapter that follows is the working minimum, learned so that the referral is made at the right time.

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

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

HOW TO USE THIS BOOK

The colour memory code

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

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

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

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

Contents

FRONT MATTER

Foreword 7

How to use this book 8

PART I **Epilepsy and Psychiatry** 11

- Epilepsy for the psychiatrist: classification, localisation and the interface 11
- The peri-ictal axis: syndromes by their timing to the seizure 60
- The psychoses of epilepsy 108
- Mood, anxiety and suicide in epilepsy 139
- Personality, behaviour and cognition in epilepsy 168
- Psychogenic non-epileptic seizures 204
- Antiepileptic drugs: the psychiatric interface 239
- Consolidate and apply 269
- References 282

PART II **Neurology Foundations for Psychiatrists** 284

- The bedside examination, what it localises, and the investigations it earns 284
- Headache and the psychiatric interface 338
- Sleep: the architecture and its disorders 365
- Consolidate and apply 392
- References 405

BACK MATTER

Subject index 407

About the authors 411

PART I

Epilepsy and Psychiatry

The two-way traffic between seizures and the mind: the peri-ictal syndromes, the psychoses of epilepsy, non-epileptic attacks, and prescribing across both illnesses at once.

01 Epilepsy for the psychiatrist: classification, localisation and the interface

02 The peri-ictal axis: syndromes by their timing to the seizure

03 The psychoses of epilepsy

04 Mood, anxiety and suicide in epilepsy

05 Personality, behaviour and cognition in epilepsy

06 Psychogenic non-epileptic seizures

07 Antiepileptic drugs: the psychiatric interface

08 Consolidate and apply

09 References

VISUAL 1.1



The three possible authors of a psychiatric symptom

- Seizure-linked
- Treatment-linked
- Independent psychiatric condition
- Timing and collateral
- Reassess

A psychiatric symptom in epilepsy may arise from the seizure disorder, its treatment, or an independent condition; timing and collateral evidence determine attribution.

Source lock: day-30.final.html#sec-t01

VISUAL 1.2



The peri-ictal axis

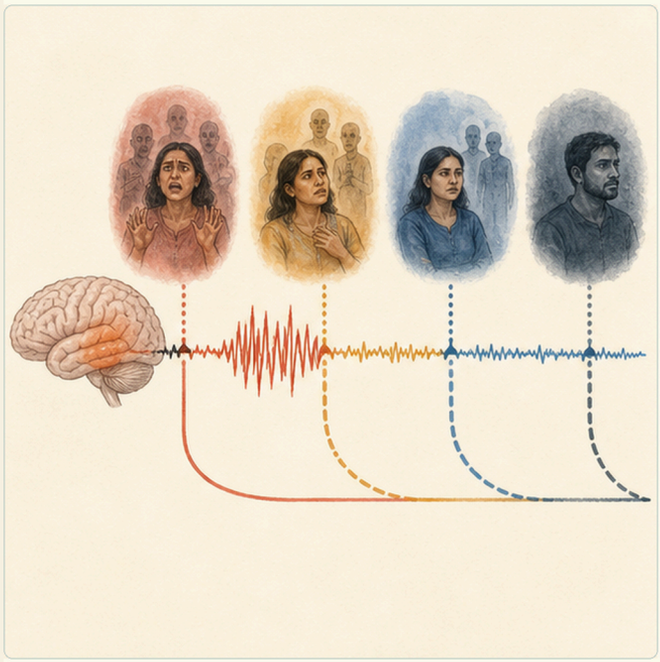
- Preictal
- Ictal
- Postictal
- Interictal
- Lucid interval

Peri-ictal psychiatric syndromes are classified by their temporal relation to the seizure; the lucid interval is especially important in postictal psychosis.

Source lock: day-30.final.html#sec-t09

VISUAL 1.3

Psychosis across the seizure timeline



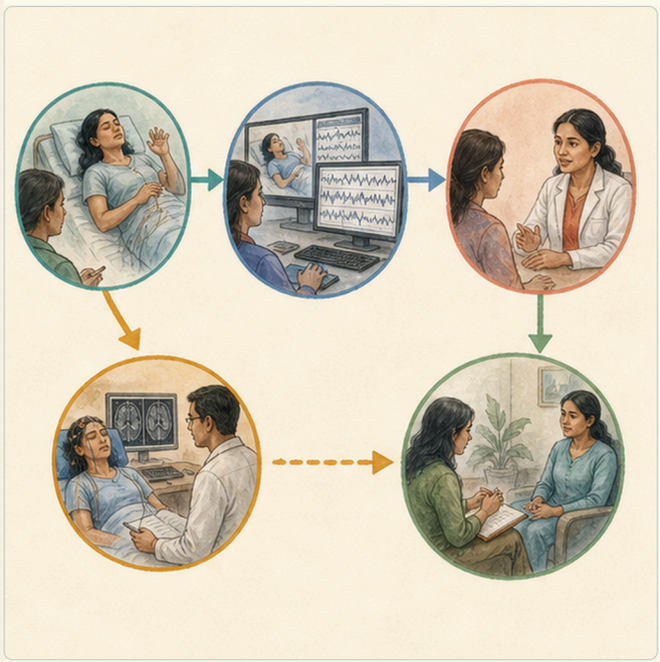
- Ictal
- Postictal
- Interictal
- Primary psychosis
- Seizure timeline

Psychosis associated with epilepsy is interpreted through timing, seizure relationship, recovery pattern and longitudinal course rather than symptom form alone.

Source lock: day-30.final.html#sec-t19

VISUAL 1.4

PNES is a positive diagnosis



- Positive semiology
- Video-EEG
- Communicate the diagnosis
- Psychological treatment
- Coexisting epilepsy

PNES is diagnosed positively, communicated clearly and treated psychologically while coexisting epilepsy remains an explicit possibility.

Source lock: day-30.final.html#sec-t37

PAPER IV

Epilepsy and psychiatry

Seven bands . one complete notes document

Epilepsy for the psychiatrist: classification, localisation and the interface

1 · Epilepsy and psychiatry: the interface, and how to read this chapter

Epilepsy is the disorder neurology and psychiatry have never managed to divide cleanly between them. A seizure is an electrical event, but the cortex it discharges through is the same organ that generates mood, thought, perception and personality, so a large part of what epilepsy does to a patient lands squarely in the psychiatrist's clinic. Candidates lose marks in this territory not because the facts are hard but because the material sits astride a border they were trained to treat as a wall. This chapter is written to dissolve that wall. It is a standalone reference: it assumes you can already recognise a seizure, and asks instead what the psychiatrist must add once epilepsy is on the table.

The examiner is rarely testing whether you can list the psychiatric complications of epilepsy, because any textbook can supply that list. The real test is whether you can take a single presenting symptom - a wave of fear, a hallucination, a spell of low mood, an aggressive outburst - and reason about it the way a clinician who understands epilepsy would: in time, in space, and by cause. Those three questions organise everything that follows, and this opening section exists to install them before the detail arrives. A reader who reaches the later sections without them will be forced to memorise each syndrome as an isolated fact; a reader who carries them will find the same syndromes deducible, which is both faster and more durable under examination pressure.

1.1 Why this material sits in the psychiatry syllabus

For most of recorded medical history epilepsy was construed as a disorder of the soul long before it was understood as a disorder of the cortex, and the **interface** between seizures and psychiatric phenomena is therefore not a modern administrative overlap but one of the oldest observations in clinical medicine. The reason it endures is structural rather than historical. The circuits that discharge in the commonest human epilepsies are the same limbic and paralimbic systems that regulate emotion, memory and drive, so disturbance in those circuits announces itself in the currency of psychiatry: fear, elation, depersonalisation, psychosis, altered sexuality and changed personality. A psychiatric symptom in a patient with epilepsy is frequently not a coincidental second illness but a direct expression of the seizure disorder or of the brain that hosts it.

This matters for the examination because it reframes the whole subject. Psychiatric phenomena in epilepsy are best approached not as a catalogue of comorbidities to be memorised but as the predictable behavioural output of a known lesion in a known circuit. Once that stance is adopted, the individual syndromes stop being arbitrary and start being derivable. The chapter is organised to reward exactly that reasoning, and the sections ahead repeatedly ask you to move from the

seizure to the symptom and back, rather than treating the two as separate examinable objects that happen to share a patient. It is also why the psychiatrist cannot delegate this material to the neurologist and vice versa: the seizure and the symptom are the same event described in two vocabularies, and the patient is served only when someone reads both.

1.2 A relationship that runs in both directions

The single most important conceptual correction this chapter makes is that the link between epilepsy and psychiatric disorder is not a one-way causal arrow but a genuinely **bidirectional relationship**. The obvious direction is familiar: living with a seizure disorder raises the risk of depression, anxiety, psychosis and suicide, through mechanisms that are partly neurobiological and partly the burden of a chronic, stigmatised, unpredictable illness. The direction candidates neglect is the reverse one. A history of a primary psychiatric disorder, depression in particular, precedes and predicts the later onset of epilepsy, which cannot be explained by the psychological reaction to seizures because the mood disorder comes first. The two conditions appear to share vulnerability in the same circuits, so each raises the probability of the other.

Holding both arrows in mind changes clinical practice. It means a psychiatric history is part of the epilepsy assessment and a seizure history is part of the psychiatric assessment, and it means the two disorders are best thought of as neighbours on a shared substrate rather than as cause and consequence. The epidemiological weight of this two-way traffic - how much commoner these disorders actually are in epilepsy, and how the risk is distributed - is quantified where the chapter turns to prevalence, incidence and the bidirectional data; this section installs the frame, and that section supplies its magnitude.

IN INDIA

The bidirectional interface is not an abstraction in Indian practice. Epilepsy still carries heavy social stigma that bears on marriage, employment and schooling, and much of that burden is psychological and therefore the psychiatrist's to manage. Care is frequently split across a neurologist or general physician and a psychiatrist with little formal liaison, and the psychiatrist is often the first clinician a patient reaches when the presentation is behavioural rather than convulsive. Reading the relationship as two-way, rather than assuming the psychiatric picture is merely a reaction to fits, is what keeps that first contact from missing a treatable seizure disorder.

1.3 The first question - when, relative to the seizure?

The organising spine of the entire subject is temporal. Almost every psychiatric phenomenon in epilepsy is defined less by its cross-sectional appearance than by its timing against the seizure, and the chapter therefore introduces a **peri-ictal temporal axis** as its master framework. The axis sorts any symptom into one of four windows according to when it occurs relative to the fit: before it, during it, immediately after it, or in the seizure-free intervals between attacks. A fear that is **ictal** is the seizure itself; the same fear arising in the settled periods between seizures is an **interictal** anxiety phenomenon with a different mechanism, course and treatment. The words are similar; the disorders are not.

Pre	Ictal	Post	Inter
BEFORE THE SEIZURE - PRODROME AND PREICTAL CHANGE	DURING THE SEIZURE - THE DISCHARGE ITSELF	AFTER THE SEIZURE - THE RECOVERY WINDOW	BETWEEN SEIZURES - THE SETTLED INTERVAL

This axis is the framework that the peri-ictal band of the chapter unfolds in full, placing each syndrome - the preictal prodrome, the ictal affective and psychotic phenomena, postictal confusion and psychosis, and the interictal disorders - into its correct window. For orientation, only the discipline of the question matters: get into the habit of establishing the temporal relationship of a symptom to the last seizure before you reach for a diagnostic label, because the label often changes entirely once the timing is known.

RULE **Timing comes before naming.** A psychiatric symptom in a patient with epilepsy is characterised first by where it sits on the peri-ictal axis and only then by its content. The same phenomenology means different things in different windows, so the temporal relationship to the seizure is the first datum to establish, not an afterthought once the diagnosis has been guessed.

1.4 The second question - where is the focus?

Once timing is settled, location does much of the remaining explanatory work. Epilepsy is not a single disease but a family of disorders, and the psychiatric colour of a given case depends heavily on where in the brain the seizures begin. The chapter opens with classification precisely because the distinction between focal onset and generalised onset, and within focal epilepsy the identity of the lobe, is what predicts the psychiatric phenotype. **Localisation** is therefore not a neurologist's technicality that psychiatrists can skip; it is the variable that tells you which behavioural syndrome to expect.

Two regions carry most of the psychiatric salience, and each has its own section. Temporal lobe epilepsy, arising from limbic and mesial temporal structures, is the substrate for the richest affective, psychotic and personality phenomena, while frontal lobe epilepsy produces its own striking behavioural and motor presentations that are easily mistaken for functional or primary psychiatric disorder. The chapter also devotes a mechanism section to the limbic system, kindling and the neurobiology that links repetitive seizure activity to durable behavioural change; the full structural anatomy of that system belongs to the Functional Neuroanatomy and Neurophysiology notes and is cross-referenced there rather than rebuilt here. Reading the **semiology** of a seizure as a map to its origin is the skill these sections train.

The practical lesson of localisation is that the psychiatrist should never receive the word "epilepsy" as a sufficient diagnosis. Which epilepsy, from where, matters as much as the fact of seizures, because a mesial temporal focus and a frontal focus generate different behavioural risks and warrant different anticipatory questions. When the referring information gives only the bare label, the localising detail - the aura, the ictal behaviour, the imaging and the electroencephalographic focus - is worth actively seeking, because it is frequently the single fact that makes the psychiatric presentation intelligible.

1.5 The third question - disease, drug or neither?

The final master question is the one that separates safe clinicians from dangerous ones, and it recurs so often that the chapter is deliberately built around it. Any psychiatric symptom in a patient with epilepsy has three broad possible authors, and **attribution** to the correct one dictates management. The symptom may be produced by the epilepsy itself, whether as a direct peri-ictal event or as an interictal consequence of the seizure disorder. It may be produced by the treatment, since the antiepileptic drugs carry their own well-described psychiatric and cognitive adverse effects. Or the paroxysmal event may not be epileptic at all, but a functional, non-epileptic attack that mimics a seizure.

This attribution problem is why several parts of the chapter are built as explicit, disjoint pairs: cognitive change from the disease set against cognitive change from the drugs; mood and suicidality intrinsic to epilepsy set against mood and suicidality attributable to antiepileptic medication. In each case the examiner is testing whether you can assign the same clinical picture to disease or to drug, because the two demand opposite responses. The point of naming the three authors at the outset is to make you ask, of every symptom, which one you are looking at before you act on it.

TRAP **Blaming the disease for what the drug is doing, or the reverse.** Candidates confidently ascribe apathy, slowed cognition or low mood to "the epilepsy" when the antiepileptic drug is the cause, or reassure that a symptom is a harmless drug effect when it is the seizure disorder declaring itself. The cross-sectional picture cannot settle it; only a careful account of onset, dose changes and timing can.

PITFALL

Adjusting psychotropic medication in a patient with epilepsy without speaking to the treating neurologist. A change made in isolation can destabilise seizure control, compound an antiepileptic drug's adverse effect, or collide with an enzyme interaction that the psychiatrist never saw coming. The interface is bidirectional in management too: neither clinician should prescribe across it blind to the other.

1.6 A map of the chapter

With the three questions in place, the architecture of what follows becomes legible. The chapter moves in bands, each answering the master questions for a defined slice of the material, and the accompanying map sets out how they fit together. The early bands establish the substrate - how seizures are classified and localised and how the two conditions relate epidemiologically. The middle bands work through the psychiatric syndromes in the order the peri-ictal axis dictates, then the psychoses, then mood, anxiety and suicide, then the personality, behavioural and cognitive changes. The later bands address the non-epileptic attacks that mimic all of the above, and finally the antiepileptic drugs viewed as objects of psychiatric interest in their own right.

BAND OF THE CHAPTER	WHAT IT COVERS	THE DISCRIMINATION IT DRILLS
Classification and localisation	how seizures are classified and where in the brain they arise	focal versus generalised onset; which lobe is involved
The peri-ictal axis	psychiatric syndromes sorted by their timing to the seizure	ictal versus postictal versus interictal
The psychoses of epilepsy	ictal, postictal and chronic interictal psychotic states	epileptic psychosis versus primary schizophrenia
Mood, anxiety and suicide	depression, anxiety disorders and suicide risk in epilepsy	intrinsic to the disease versus attributable to the drug
Personality, behaviour and cognition	interictal personality change, aggression and cognitive effects	the epilepsy versus its treatment versus surgery
Non-epileptic seizures	functional, psychogenic attacks and their management	epileptic versus non-epileptic events
Antiepileptic drugs at the interface	the drugs as mood agents, as offenders and as interacting molecules	therapeutic effect versus adverse psychiatric effect

The chapter closes with worked vignettes and an active-recall consolidation section, so the reading load is front-weighted and the application is deliberately saved for last. Nothing in the map needs memorising as a list; it is here so that when a later section names a syndrome you already know which question it is answering and which neighbour it borders.

1.7 What the examination actually rewards

The subject has a small number of recurring intellectual moves that earn most of the marks, and recognising them in advance is worth more than any single fact. Across every band, the questions that discriminate strong candidates from weak ones ask essentially the same things in different clothing, so it is worth naming them plainly before the detail begins.

- Placing a symptom on the peri-ictal axis before diagnosing it, so that an ictal, postictal and interictal version of the same complaint are never confused.
- Reading localisation and semiology together to predict the psychiatric phenotype, rather than treating the lobe as a neurological detail.
- Attributing a symptom to the disease, the drug or a functional cause, since the same picture demands opposite management under each.
- Discriminating the epileptic from the primary psychiatric disorder that it mimics, using course and relationship to seizures rather than cross-sectional appearance.
- Coordinating psychiatric treatment with seizure control, so that neither is optimised at the other's expense.

A candidate who carries these five habits into the rest of the chapter will find that the individual syndromes fall into place as instances of a general method, and that the awkward look-alikes the examiner favours become tractable rather than treacherous. The detail still has to be learned, but it is learned faster when it hangs on a frame.

GOOD TO REMEMBER

Take a seizure history from every patient who presents with episodic or paroxysmal psychiatric symptoms, and in every patient with known epilepsy establish the temporal relationship of any new psychiatric symptom to the last seizure before you name it. Those two habits, applied on Monday morning, catch most of the errors this chapter is written to prevent.

1.8 Holding it together

Everything in this chapter can be reduced to a single disciplined sequence applied to whatever symptom the patient brings. First locate the symptom in time against the seizure, then locate the seizure in space within the brain, then decide whether the disease, the drug or a functional process is its author. Only once those three are settled does the diagnostic label become safe to attach, because each of them can change the label entirely. The syndromes that fill the rest of the chapter are simply what this sequence produces when it is run on the real presentations epilepsy generates. None of the sequence is exotic; its power lies in being applied consistently, to the fear and the hallucination and the outburst alike, so that no presentation is ever named on its surface appearance before its timing, its origin and its cause have been established.

MNEMONIC

Localise, Time, Attribute

The three master questions of the whole chapter, in the order the clinic uses them. **Localise**: where do the seizures arise, and what phenotype does that predict? **Time**: where on the peri-ictal axis does this symptom sit? **Attribute**: is its author the disease, the drug, or a non-epileptic process? Run all three before you name anything, and the look-alikes that trap candidates come apart in your hands.

Before you name a psychiatric symptom in a patient with epilepsy, place it in time against the seizure and in space against the focus, and decide whether disease, drug or a functional process produced it.

2 · ILAE seizure classification (focal, generalised, unknown onset) and clinical relevance to psychiatry

Classification is where the clinical thinking begins, not where it ends. When the International League Against Epilepsy last recast how seizures are named, in 2017, it fixed the discipline's opening question as something deceptively plain: where does the seizure start? Every seizure is now sorted by its **point of onset** into one of three families, focal onset, generalized onset, or unknown onset, and that single decision organises almost everything downstream of it. It shapes the syndrome the patient is likely to have, the causes worth pursuing, the investigations that earn their keep, and the treatment that is rational rather than reflexive. For the psychiatrist this is not neurology's private bookkeeping. A seizure and a psychiatric episode can present as near-identical events across the same consulting-room desk, and the only disciplined way to tell them apart is to know what a seizure is, how it is classified, and what each class is capable of imitating.

What this topic owns is the top of the scheme: the division of every seizure by where it begins, the reason that division is the first question asked, the closed list of causes that sits beneath it, and the single idea that earns all of this a place in a psychiatry chapter, that seizures and psychiatric episodes can impersonate one another. The finer subdivision of focal seizures by awareness, the anatomy and semiology of temporal and frontal lobe epilepsy, the reading of the electroencephalogram, the neurobiology that links limbic circuitry to behaviour, and the epidemiology of the epilepsy-psychiatry association are each developed in their own right elsewhere in this chapter; here they are named, not reopened.

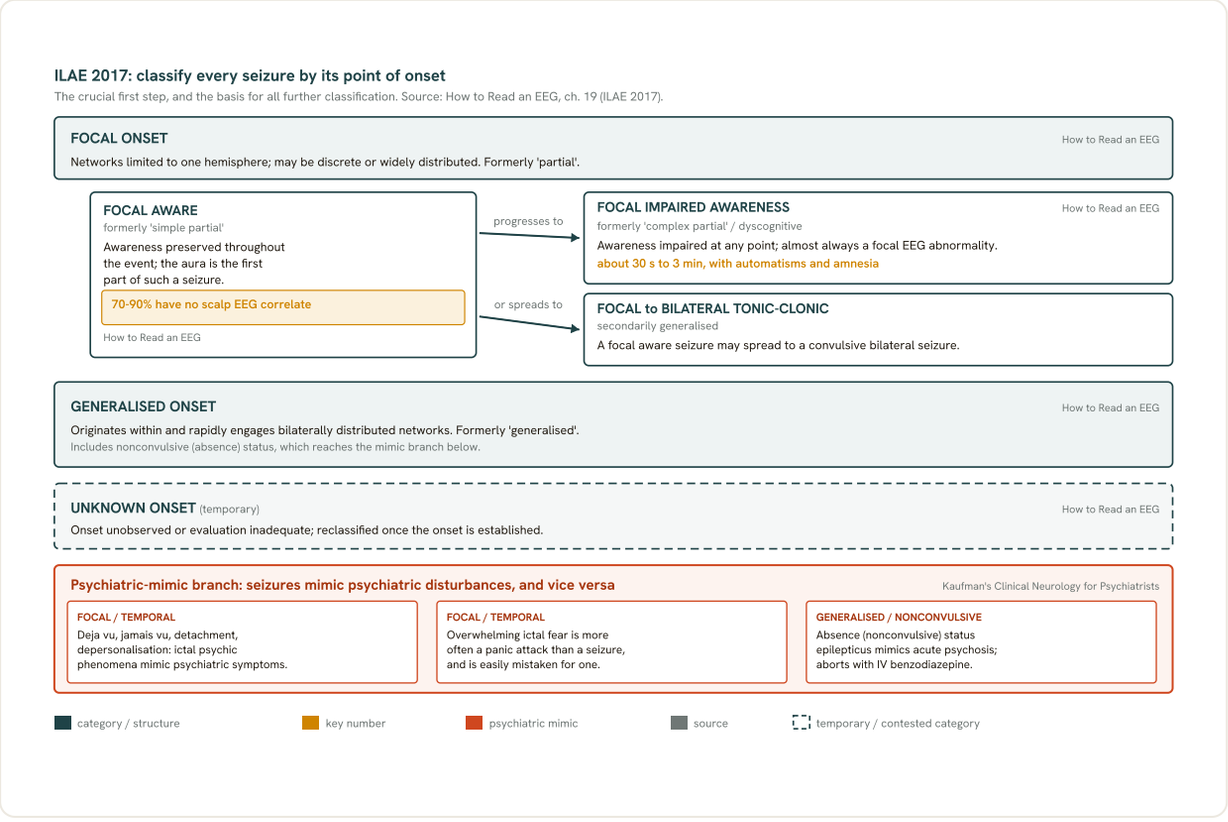


Fig 2.2 **ILAE 2017 seizure classification.** Every seizure is first sorted by point of onset into focal, generalised or unknown; focal onset is further split by whether awareness is preserved, with a focal aware seizure able to progress to impaired awareness or spread to a bilateral tonic-clonic seizure. The coral band collects the presentations that mislead the psychiatrist, since ictal phenomena mimic psychiatric disturbances and the reverse also holds.

2.1 The framework: seizures named by where they begin

A stepwise instrument, not a single label. The scheme works in tiers. The first tier concerns onset alone; only afterward does it ask what kind of epilepsy the person has, whether the picture fits a recognised epilepsy syndrome, and what is driving it. Naming the onset is therefore the doorway to a longer chain of reasoning, and it is not read off any one source. It is assembled from three streams held together: the semiology of the attack as it is witnessed or recounted, the electroencephalographic signature, and neuroimaging. None of the three is sovereign. A vivid eyewitness account with no electrical correlate, or a florid tracing with no history, leaves the onset a matter of inference rather than fact, and the classification is candid that its categories are provisional and sharpen as evidence accumulates. This is a genuine advance over older habits, which reached for a syndrome label first and let the description follow; anchoring the whole

edifice to the plain question of onset forces the clinician to earn each later refinement. The reading of the discharges themselves, ictal against interictal, is a craft of its own and is developed separately, as are the individual localisations that give focal epilepsies their names. What matters at this level is the shape of the system: a small set of onset families sitting above a short, closed list of causes, with the individual patient slotted in as the data allow. Holding that shape in mind stops the clinician leaping to a syndrome or a cause before the humbler question, where did it begin, has been honestly answered.

2.2 Focal onset: seizures that begin on one side

Confinement at the start is the defining feature. **Focal onset** seizures, the events once grouped together as partial seizures, begin within networks confined at their outset to a single hemisphere. That confinement is what defines them, but it should not be read as "small". The originating network may be tightly discrete, a single sharply localised generator, or it may be widely distributed across that hemisphere while still remaining on one side. Two seizures can both be focal in onset and yet look utterly different at the bedside, one a fleeting subjective symptom, the other a florid motor or behavioural event, because the size and situation of that one-hemisphere network vary enormously. What unites them is provenance, not appearance. This is also why focal onset is the family that carries most of the localising information a clinician can act on: because the seizure starts somewhere in particular, its earliest features often betray where, and it is those early, still-focal features, rather than any later spread, that point to a lobe and, with it, to a characteristic set of behavioural and experiential accompaniments. The subdivision of focal seizures by whether awareness is retained or impaired, and the specific psychiatric mimics that go with each, belongs to its own discussion, as do the individual localisations, the temporal and frontal presentations that dominate the psychiatric literature. Here the point is simply that a focal seizure is defined by its one-sided beginning and by nothing about how dramatic it looks.

2.3 Generalized onset: seizures that begin on both sides at once

Bilateral from inception, not a focal seizure that has spread. **Generalized onset** seizures are the mirror image: from the very beginning they arise within and rapidly recruit networks distributed across both hemispheres, so there is no single side of origin to find. The event is bilateral at its inception. This distinction is easy to state and genuinely hard to make at the bedside, because a focal seizure that propagates widely and a seizure that was generalized from the outset can converge on the same dramatic convulsion, and the observer who arrives late sees only the shared endpoint. The generalized family spans a wide clinical range, from brief lapses of awareness, through myoclonic jerks, to the major convulsion, and that range matters to psychiatry for one reason above all others: some generalized seizures are nonconvulsive. They produce no jerking and no fall, only an altered state, and a nonconvulsive generalized seizure that persists can present as a change in behaviour and awareness with nothing motor to declare it a seizure at all. That single possibility, a seizure with no convulsion, is where the classification stops being an academic exercise for the psychiatrist and becomes an urgent differential diagnosis, as the closing section of this discussion sets out in full.

2.4 Unknown onset: an honest placeholder

The third family is an admission, not a third kind of seizure. **Unknown onset** is used when the beginning of the event was not observed, or when the available evaluation is simply inadequate to place it, and it is explicitly a temporary designation, meant to be revised the moment better information arrives and the onset can be established. It exists because the honest alternative, forcing every event into focal or generalized on insufficient evidence, is worse, manufacturing a confidence the data do not support. A seizure that occurs in sleep with no witness, or a first event described only at third hand, may be genuinely unclassifiable at first contact, and the scheme would rather say so than guess. Importantly, an event can still be described by some of its features while its beginning stays unsettled, so "unknown onset" is not the same as "unknown event"; it is a statement about one axis only. The category is thus a discipline as much as a label, committing the clinician to returning to the question rather than closing it. It should never harden into a permanent verdict, because the whole point of naming a seizure is lost if the name is a shrug that no one revisits.

ONSET FAMILY	NETWORK AT THE START	STATUS OF THE LABEL	BEARING ON THE PSYCHIATRIC DIFFERENTIAL
Focal onset	One hemisphere	Definitive once established	Predicts a focal, experiential repertoire that can imitate panic or dissociation
Generalized onset	Both hemispheres at once	Definitive once established	Includes nonconvulsive states that can imitate acute psychosis
Unknown onset	Not established	Provisional, to be revised	Withholds the differential until the onset is known

The three onset families set against the axes that matter to a psychiatrist; the label's provisional or definitive status is itself part of the diagnosis.

2.5 The first fork: focal against generalized

One split does the heaviest clinical work. Of the three onset families, the division that carries the load is the split between focal and generalized, and it is worth pausing on why. The two describe genuinely different machinery. A seizure confined at its start to networks in one hemisphere behaves, spreads, responds and recurs unlike one that engages both hemispheres from the outset, and almost every subsequent decision keys off which of the two is in front of you. That is why the classification treats it as the pivot rather than a detail. Getting it right narrows the field of possible syndromes, points the causal work-up in a direction, and steers treatment toward what suits that seizure type; getting it wrong sends the entire work-up down the wrong branch, where every later step only lends false confidence to the original error. The fork determines, in turn:

- which epilepsy syndromes are even in contention;
- which causes justify a costly or invasive work-up;

- how the electrical and imaging investigation is targeted;
- which broad treatment pathway is rational for the seizure type.

RULE **Settle focal versus generalized before anything else.** The **focal-versus-generalized distinction** is the first and most consequential division in the whole scheme, because syndrome, cause and the entire work-up descend from it. Every later refinement is a branch of this first choice, which is why an unclassified seizure is only ever a half-diagnosed one.

TRAP **A witnessed bilateral convulsion is not proof of generalized onset.** A focal seizure can spread until both hemispheres are engaged and the patient convulses; what an observer sees at the peak says little about where the event began. Candidates equate the dramatic generalized motor phase with generalized onset and miss the focal beginning, which is exactly the beginning that carries the localising and psychiatric information.

Name the onset first: focal, generalized or unknown is the fork from which the syndrome, the cause and the psychiatric differential all descend.

2.6 The aetiological axis: cause as a separate question

A closed list of causes, considered after the type is settled. Once the seizure has an onset and the epilepsy has a type, the classification turns to cause, and here too the answer is drawn from a defined list rather than an open field. The **aetiological axis** sorts the epilepsies into **six** categories, structural, genetic, infectious, immune, metabolic and unknown, and cause is examined only after the seizure and epilepsy type are established, not before. The categories are not mutually exclusive in spirit, since a single patient may carry a structural lesion that is itself genetic in origin, but the axis forces the clinician to ask the question deliberately and to hold a place for each kind of answer. The sobering counterweight to all this machinery is how often it returns nothing: in surveyed populations the cause is never identified in about **two-thirds** of people with epilepsy, a fraction that holds broadly across age groups. That figure is not a failure of the scheme so much as an honest measure of the field, and it is why "unknown" is a named category rather than an embarrassed silence. For the psychiatrist the axis carries a specific warning: the structural, infectious, immune and metabolic causes are precisely the ones that also produce psychiatric symptoms directly, so a seizure filed into one of those causal boxes is a seizure whose psychiatry may be organic rather than reactive, and worth chasing to its root.

MNEMONIC

Onset first, cause second

Read every seizure in that order: fix where it begins before arguing about why it happens. The onset families sit above the closed list of causes, and jumping to aetiology before onset is settled is how a focal, structurally driven seizure gets mislabelled as a generalized epilepsy with no lesion to find.

PITFALL

Do not let the two "unknowns" blur into one. Unknown onset is a statement about the seizure, that we could not see where it began. Unknown cause is a statement about the epilepsy, that we could not find why it happens. They are independent: a seizure can have a confidently classified onset and still, like the majority of people with epilepsy, no identifiable cause at all. Writing "unknown" once and quietly meaning both is a classification error that buries real information.

2.7 Why the psychiatrist needs the classification: the logic of mimicry

The interface is a two-way street. The reason a scheme built by neurologists earns space in a psychiatry chapter is a single, unnerving fact: **psychiatric mimicry** runs in both directions. Seizures can counterfeit psychiatric disturbance, and psychiatric events can counterfeit seizures, so a clinician who cannot classify the first cannot safely diagnose the second. Focal seizures in particular can deliver brief experiential symptoms, fear, an altered sense of familiarity, a wave of unreality, that resemble a panic attack or a dissociative episode, while paroxysmal behavioural events with no electrical basis can arrive dressed as convulsions. The finer subdivision of focal seizures by preserved or impaired awareness, and the specific mimics that follow, is taken up separately, as are the ictal fear and panic phenomena that most closely imitate a psychiatric attack. Getting the onset family right is simply the first filter, because each family predicts a different repertoire of subjective events and therefore a different set of look-alikes. This is a different question from how often epilepsy and psychiatric disorder occur together and raise the risk of one another; that bidirectional comorbidity, and its epidemiology, is developed in its own right elsewhere in this chapter. The concern here is phenomenological rather than statistical: not how commonly the two coexist, but how convincingly one can pass for the other in a single patient on one occasion, and why the remedy is disciplined classification rather than pattern-matching.

IN INDIA

In much of Indian practice the psychiatrist is the first physician a family consults about a "behavioural spell", and the event itself is often unwitnessed by anyone who can describe it, with routine electroencephalography and especially video-electroencephalography unevenly available outside larger centres. More events therefore arrive already provisional, their onset genuinely undetermined rather than merely unrecorded, and more of them are carried for a long time under a psychiatric or a faith-healing label before a seizure is even considered. The discipline of classifying by onset, and of holding the category open until better evidence comes, matters more, not less, exactly where that evidence is hardest to obtain.

2.8 Absence status epilepticus: the seizure that impersonates psychosis

One generalized state is a near-perfect forgery of a psychiatric emergency. The clearest single reason a psychiatrist must own this material sits at the far end of the generalized family. **Absence status epilepticus**, the generalized nonconvulsive form, produces no dramatic convulsion at all. Instead the patient is altered: apathetic, confused, slowed, sometimes mute or oddly disinhibited, a state that can persist for **several hours** and reads convincingly as acute psychosis or an acute

confusional presentation. There is no aura to report and no fall to witness, so the history offers nothing to flag a seizure, and the referral arrives already labelled psychiatric. The distinguishing move is electrographic: the diagnosis is confirmed on the electroencephalogram, and the episode characteristically resolves once an intravenous benzodiazepine is given under that electrographic confirmation. That responsiveness is itself informative, because a primary psychosis does not lift within minutes of a benzodiazepine. The lesson generalises well beyond this one syndrome. A generalized nonconvulsive state can wear the face of psychiatry precisely because it strips away the motor signature that would otherwise announce a seizure, and it is only classification, knowing that a generalized onset can be nonconvulsive, that keeps the possibility alive in the clinician's mind. The peri-ictal psychiatric syndromes proper, sorted by their timing to the seizure, are taken up in their own right; here the point is narrower and prior, that the very existence of a nonconvulsive generalized seizure is a standing reason to classify before you diagnose.

GOOD TO REMEMBER

When a patient is sent to you for "acute psychosis" but the mental state is fluctuating, the affect blunted and the sensorium clouded, put nonconvulsive status on the list before you reach for an antipsychotic, and ask for an electroencephalogram. A single benzodiazepine given under electrographic cover can be both the test and the treatment, and getting there first changes the whole trajectory of the presentation.

2017	**six**	**two-thirds**
ILAE ONSET-BASED RECLASSIFICATION	AETIOLOGICAL CATEGORIES OF THE EPILEPSIES	EPILEPSY WITH NO IDENTIFIED CAUSE

3 · Focal aware versus focal impaired-awareness seizures and their psychiatric mimics

This is where the psychiatrist meets epilepsy first, and often without recognising it. A patient describes a recurring wave of unreality, a flood of familiarity in a strange room, a rising dread that peaks and passes in a minute, or brief lapses during which relatives say the person fumbled with their clothes and remembers nothing. Each of these is a psychiatric complaint on its face, and each can be the whole of a focal seizure. The examinable heart of this topic is the single distinction that organises focal seizures for the modern clinician, whether awareness is kept or lost, and the corollary that matters to us specifically: the symptoms produced can be indistinguishable at the bedside from panic, depersonalisation, dissociation and fugue. Reading the awareness axis correctly, and holding the psychiatric mimics in mind, is what separates a resident who commits a patient to years of the wrong treatment from one who asks the right next question.

Focal seizures are those that begin in one part of one hemisphere, and their overarching place within the broader division into focal, generalised and unknown-onset seizures is set out in its own topic and not reopened here. What this topic owns is the layer beneath that division: how a focal seizure is subclassified by awareness, what each subtype looks like and how long it runs, why the surface electroencephalogram behaves so differently across the two, how one grows into a

bilateral convulsion, and, throughout, the psychiatric conditions these events counterfeit. The anatomy and semiology of temporal lobe epilepsy, the substrate of most of these auras, are developed in their own right elsewhere in this chapter, as are frontal lobe epilepsy and its behavioural presentations; here they are named, not re-taught.

3.1 The single dividing line: awareness

One question splits the whole category. A focal seizure is classified by whether the patient is aware of themselves and their surroundings while it happens. A **focal aware seizure** is one in which awareness is preserved throughout the entire event, so the patient can later give an account of it from the inside; a **focal impaired-awareness seizure** is one in which awareness is disturbed at any point during the event, even briefly, even if it is regained before the seizure ends. Awareness here is used in a precise operational sense: the patient's connectedness to the event and their capacity to recall and respond during it, not merely whether they stay upright or their eyes stay open. The reason a single binary carries so much weight is that it maps onto everything the clinician then wants to know. The aware patient is a reliable narrator of their own aura and can warn of what is coming; the patient whose awareness is breached cannot, so the history has to be built from a witness, and the event carries a different risk to driving, to work and to safety. These two labels describe a state during a seizure, not two separate diseases: the same person can have both, and the same seizure can begin in one state and pass into the other.

RULE **Awareness, not motor drama, is the classifier.** A focal seizure is aware if consciousness of the self and surroundings is preserved from start to finish, and impaired-awareness if it is disturbed at any moment during the event. The presence or absence of convulsive movement, falling or eye-opening does not decide the class; the integrity of awareness does.

3.2 The names that were retired, and why the change matters

Old vocabulary, familiar to examiners, now superseded. The current terms replaced an older pair that still surfaces in wards, in question banks and in the memories of senior colleagues, so both must be recognised. What is now called a focal aware seizure was formerly the **simple partial** seizure, and what is now the focal impaired-awareness seizure was the **complex partial** seizure, a category some sources also labelled **dyscognitive**. The relabelling is not cosmetic. The words "simple" and "complex" described the seizure's apparent elaborateness, which is exactly the wrong axis, because a focal aware seizure can be a rich, frightening, deeply strange experience while a focal impaired-awareness seizure can look outwardly quiet. Anchoring the name to awareness rather than to how complicated the event seems forces the clinician to ask the one clinically decisive question and stops the reflexive assumption that a dramatic-looking spell must be the more serious category. For the psychiatrist the shift is doubly useful: the experiential auras that most concern us occur with awareness fully intact and sat awkwardly under a label like "simple".

TRAP **Reading "impaired awareness" as "unconscious".** Candidates equate the impaired-awareness seizure with loss of consciousness, coma or collapse. Impaired awareness need not mean unconscious: the patient may remain standing, eyes open, carrying out automatisms, and simply be disconnected from and later amnesic for the event. Describing it as a faint or a blackout misses the point and loses the mark.

3.3 Duration, automatisms and amnesia of the impaired-awareness seizure

A stereotyped shape: a short spell, purposeless activity, a hole in memory. The focal impaired-awareness seizure has a characteristic tempo and content that make it recognisable once the pattern is known. It typically lasts **about 30 seconds to 3 minutes**, a duration that is one of its most useful diagnostic anchors because it sits between the momentary and the prolonged. During the event the patient commonly shows purposeless automatisms with dense amnesia for what happened, and, depending on the region involved, transient disturbances such as aphasia may colour the picture. Automatisms are the involuntary, often repetitive fragments of behaviour that surface when higher control is offline: lip-smacking, chewing or swallowing, fumbling or picking at clothing, aimless walking, or repeating a snatch of speech. They look semi-purposeful precisely because they are assembled from over-learned motor programmes running without the awareness that would normally direct them, which is why the patient can appear to be doing something while being genuinely absent from it. The amnesia is not incidental; it is the behavioural signature of the awareness breach that defines the class. A witness who watched the whole spell is therefore worth more than the patient's own account, since by definition the patient did not fully register it. The regional flavour of the automatisms and language change is where the semiology of specific lobes begins, and the fuller anatomy belongs to the topics that own it; what matters here is the generic template of a brief spell, automatism and amnesia that flags the impaired-awareness class.

3.4 The electroencephalographic divergence

The scalp trace lies far more often for the aware seizure. One of the highest-yield and most counter-intuitive contrasts between the two subtypes is how they appear, or fail to appear, on the surface electroencephalogram. In the great majority of focal aware seizures there is no accompanying surface change at all: no scalp electroencephalographic correlate is found in **70 to 90 per cent** of them, so a completely normal routine trace recorded during a genuine focal aware seizure is the rule rather than the exception. The focal impaired-awareness seizure behaves in the opposite way: it almost always shows a focal ictal abnormality, a discharge or rhythmic ictal activity that can then spread to involve both hemispheres. The mechanism behind the difference is instructive and worth carrying. A focal aware seizure can arise from a discharge confined to a small volume of cortex, too limited in extent, or too deep, to project a detectable signal through the skull to scalp electrodes, whereas the loss of awareness that defines the other subtype requires recruitment of a larger, often bilateral network, and that broader synchronised activity is what the surface electrodes can finally see. The general principles of the electroencephalogram and of ictal versus interictal discharges are set out in their own topic; what this contrast establishes is a specific and clinically loaded fact about these two seizure types.

PITFALL

Concluding that a patient does not have epilepsy because a routine electroencephalogram recorded during, or between, their spells was normal. For focal aware seizures a normal scalp trace is expected, not reassuring, and even an ictal recording may show nothing. A normal electroencephalogram never excludes a focal seizure; the diagnosis rests on the clinical account, and the investigation is chosen to confirm, not to rule out.

3.5 Evolution: the aura and the march to a bilateral convulsion

The aware seizure is often only the first chapter. A focal aware seizure is not always a self-contained event. It may progress to impaired awareness, or spread further to a **focal-to-bilateral tonic-clonic seizure**, the event formerly called a secondarily generalised convulsion. This is the single most important reframing in the topic for a psychiatrist, because it dissolves the apparent gap between a strange subjective symptom and a full convulsion. The warning that patients and clinicians call an **aura** is not a herald that precedes the seizure; it is the first portion of the seizure itself, the focal aware phase experienced before awareness is lost or the discharge generalises. Understanding this changes how the history is heard. When a patient reports a consistent rising sensation, an odd smell or a wave of familiarity that on some occasions is followed by a convulsion and on others resolves on its own, they are not describing two problems but the same seizure captured at two lengths. It also explains why the aware phase is so precious diagnostically: it is the only part of the event the patient can narrate from within, and its content often points to where the seizure begins. The sequence is best held as a spectrum of extent rather than as separate diagnoses.

- The focal aware phase, the aura, in which the discharge is contained and awareness is intact.
- Progression to impaired awareness as the network recruited by the seizure widens.
- Spread to a focal-to-bilateral tonic-clonic seizure when the discharge generalises across both hemispheres.

3.6 Psychic and experiential auras as psychiatric mimics

Now the reason these seizures land in a psychiatry chapter at all. Because the focal aware phase is experienced and remembered, its content can be purely mental, and when it is, it counterfeits psychiatric illness with uncanny fidelity. Kaufman's Clinical Neurology for Psychiatrists describes the **experiential or psychic aura** as exactly this class of phenomenon: a subjective, often emotional or perceptual experience generated by the seizure. The mimics that recur are a small, memorable set. There is a sudden, unearned sense of familiarity, deja vu, and its mirror image, an alienating unfamiliarity within well-known surroundings, jamais vu. There is a feeling of unreality and self-estrangement that is clinically inseparable from **depersonalisation** and derealisation, and a sense of detachment from the self and the world. Presented in a clinic without the surrounding history, any of these reads as a dissociative symptom, a depersonalisation-derealisation complaint, or an anxiety phenomenon. Two features rescue the diagnosis. The first is form over content: an ictal experience tends to be brief, stereotyped and recurrent, and it may shade into a lapse or a convulsion in a way a primary psychiatric symptom

does not. The second is a discipline of suspicion, particularly where the same experience arrives in identical form again and again. One caution belongs with the enthusiasm, though: these experiential phenomena are not reliable for localisation, so their presence flags a possible seizure without pinning down where it starts.

MNEMONIC

BASE

What tilts a paroxysmal "mental" symptom away from primary psychiatry and toward a focal seizure. **B**rief, running seconds to a few minutes rather than a sustained state. **A**mnesia for part of the event once awareness is breached. **S**tereotyped, reproducing nearly the same experience each time. **E**pisodic and recurrent, arriving out of the blue. Each letter is a feature this topic states; none is decisive alone, and the base rates still matter.

3.7 Ictal fear and the panic-attack differential

The commonest and most consequential mimic of all. Of the experiential auras, the one that most often reaches a psychiatrist is a paroxysm of sudden, overwhelming fear, and it is the trickiest because it collides with one of our commonest diagnoses. A focal seizure, typically of temporal origin, whose entire content is a surge of **overwhelming fear** can be almost impossible to tell from a panic attack on phenomenology alone: both bring abrupt terror, autonomic arousal and a sense of impending doom, both peak fast and subside, and both leave the person shaken. The honest teaching point, and the one Kaufman's Clinical Neurology for Psychiatrists makes, is a probabilistic one: an isolated attack of overwhelming fear is more likely to represent a **panic attack** than a seizure, simply because panic is so much commoner, even as the two are genuinely easy to mistake for each other in both directions. The reflex should not be to relabel every panic patient as epileptic, but to keep ictal fear on the differential when the attacks are very brief, stereotyped, accompanied by any clouding of awareness or automatism, or followed on occasion by a convulsion. The ictal fear phenomenon itself, its amygdalar origin and the full range of ictal affective experience, is developed in its own topic and is not taught here; what this topic owns is the clinical differential with panic disorder, the psychiatric-mimic angle. Anxiety disorders in epilepsy, and the separation of an interictal anxiety disorder from an ictal one, are likewise handled in their own right elsewhere in this chapter.

3.8 The psychiatrist's discipline: reading a paroxysmal mental symptom

Put the axis and the mimics together at the bedside. The practical synthesis is to hold two questions at once for any patient with paroxysmal, unexplained mental phenomena. First, place the event on the awareness axis: was the person connected to and able to recall the episode throughout, which points to a focal aware event, or was there a breach of awareness with automatisms and amnesia, which points to the impaired-awareness class and mandates a witness account. Second, ask whether a symptom that presents as psychiatric, the depersonalisation, the deja vu, the wave of terror, might be the experiential content of a focal aware seizure rather than a primary disorder, and let the form of the episode guide the suspicion. Neither question is answered by a single investigation; the electroencephalogram in particular is expected to be silent

in the aware seizures that most often masquerade as psychiatric illness. The comparison that follows lays the two subtypes side by side on the axes that separate them, so the whole contrast can be weighed at once rather than feature by feature.

AXIS	FOCAL AWARE SEIZURE	FOCAL IMPAIRED-AWARENESS SEIZURE
Awareness	Preserved throughout the event	Impaired at some point during the event
Former term	"simple partial"	"complex partial", also dyscognitive
Consciousness	Fully retained	Impaired, though not necessarily lost
Automatisms and amnesia	Absent; the patient recalls the event	Purposeless automatisms with amnesia
Scalp EEG during the event	Usually no surface correlate	Almost always a focal ictal change
May evolve to	Impaired awareness or a bilateral convulsion	A focal-to-bilateral tonic-clonic seizure
Psychiatric mimic	Depersonalisation, dissociation, panic	Dissociative or fugue-like states

The two focal subtypes compared on the axes that separate them; awareness is the classifier, and the electroencephalographic and psychiatric-mimic rows are the ones that most often mislead.

30 s to 3 min	**70 to 90%**	**almost always**
FOCAL IMPAIRED-AWARENESS SEIZURE DURATION	FOCAL AWARE SEIZURES WITH NO SCALP EEG CORRELATE	FOCAL ICTAL CHANGE IN IMPAIRED-AWARENESS SEIZURES

IN INDIA

Two realities sharpen this topic in Indian practice. Access to routine and especially prolonged video-electroencephalography is concentrated in tertiary centres, so the aware seizures that carry no surface correlate are even more likely to be dismissed when the one available trace comes back normal; the diagnosis has to be carried on the clinical account. And where neurological services are distant, a patient whose only seizure is an experiential aura of deja vu, unreality or paroxysmal fear frequently enters care through a psychiatric or a faith-healing route and is labelled with an anxiety or dissociative disorder for a long time before a focal seizure is considered. A patient reporting stereotyped, brief, recurring mental symptoms deserves that question asked explicitly, exactly where confirmatory testing is scarce.

GOOD TO REMEMBER

When someone describes a recurring, identical, out-of-the-blue mental experience that lasts seconds to a couple of minutes, whether it is deja vu, a sense of unreality, or a surge of fear, add one question to the psychiatric history: does it ever end in a lapse of awareness, an automatism, or a convulsion, and does a witness describe absence or fumbling. A normal electroencephalogram does not close the matter. The awareness axis and the form of the episode, not the trace, carry the diagnosis.

Focal seizures are classified by awareness alone: preserved throughout in the focal aware seizure, breached at any point in the focal impaired-awareness seizure; the aware phase is the experienceable aura that mimics depersonalisation, deja vu and panic and that usually leaves the scalp electroencephalogram normal, so a brief, stereotyped, recurrent mental symptom is a possible seizure until the awareness axis and a witness say otherwise.

4 · Temporal lobe epilepsy: anatomy, semiology and psychiatric salience

No other epilepsy sits so squarely on the psychiatrist's desk. The temporal lobe is where seizure and mental phenomenon become the same event: an attack here can be a wave of fear, a flood of the uncanny, a phantom smell, a rising sickness in the gut, a minute of vacant fumbling the patient does not remember. Because these experiences are subjective and strange rather than convulsive, the person who has them is far more likely to reach a psychiatric clinic than a neurology ward, and far more likely to be told the trouble is anxiety, dissociation or a personality problem than to be recognised as having focal epilepsy. The marks here are for holding the anatomy, semiology and psychiatric consequences of **temporal lobe epilepsy** as one connected picture, so that an experiential aura is read as a possible seizure, a discrete and often operable lesion is looked for behind it, and the disproportionate psychiatric burden the temporal lobe carries is anticipated rather than discovered late.

The account moves from where these seizures come from, through what they look and feel like and how they mark the electroencephalogram, to why the temporal lobe is the seizure focus that psychiatry cannot ignore. The broader ILAE framework that names a seizure focal, generalised or of unknown onset, and the distinction between focal aware and focal impaired-awareness seizures, are developed in their own right elsewhere in this chapter. The interictal personality change this same epilepsy can produce, and the named Gastaut-Geschwind cluster, are constructs of their own and taught in their own right elsewhere in this chapter; this topic supplies the substrate on which they rest but does not teach them.

4.1 The temporal lobe as the home of impaired-awareness seizures

Where consciousness clouds, look first at the temporal lobe. The single anatomical fact from which everything else follows is that **focal impaired-awareness seizures** - the events an older nomenclature called complex partial seizures - arise most often from the temporal lobe, according to Kaufman's Clinical Neurology for Psychiatrists. The qualifier matters as much as the rule: the frontal lobe can also harbour the focus, and frontal lobe epilepsy with its own

behavioural and psychiatric presentations is a distinct topic taught in its own right elsewhere in this chapter. Within the temporal lobe the origin is not evenly spread. Kaufman's puts it precisely: **about 80%** of complex partial seizures originate from the medial, or mesial, surface of the temporal lobe rather than the outer convexity. That concentration is the reason electrodes placed close to the skull base, the nasopharyngeal and sphenoidal electrodes, were devised, since a discharge buried on the mesial surface may be too far from the standard scalp array to register clearly. The take-home is directional: a patient whose seizures involve altered awareness, automatism and amnesia has, until proven otherwise, a temporal, and probably mesial temporal, focus, and the diagnostic effort should be pointed there.

4.2 Mesial temporal sclerosis: the commonest substrate

The lesion behind the syndrome. If most temporal seizures begin on the mesial surface, the next question is what is wrong with that tissue, and here temporal lobe epilepsy has an answer unusually specific for a seizure disorder. Kaufman's Clinical Neurology for Psychiatrists names the commonest cause as **mesial temporal sclerosis**, a combination of sclerosis of the hippocampus with atrophy of the temporal lobe. This is a discrete, identifiable and often surgically remediable pathology, which is why establishing it is worth the trouble: unlike a diffuse encephalopathy, a sclerotic hippocampus can in selected patients be removed, and the seizures with it. The origin of the sclerosis carries an examinable subtlety that is easy to garble. Kaufman's is explicit that it is prolonged febrile seizures, not the brief occasional ones, that are implicated in mesial temporal sclerosis; the association is strong but the direction of causation is still debated, and the ordinary short febrile convulsion of childhood does not, as a rule, scar the hippocampus. The same source implicates perinatal anoxia and herpes simplex encephalitis among the other insults that leave this signature. A long, complicated febrile seizure in early childhood is thus a meaningful pointer toward later mesial temporal lobe epilepsy, whereas a run-of-the-mill febrile fit is not, and conflating the two is a classic error.

TRAP **Attributing mesial temporal sclerosis to any febrile seizure history.** The examiner offers a patient with temporal lobe epilepsy and a childhood of febrile convulsions and invites an indiscriminate link. The link is real but conditional: the prolonged, complicated febrile seizure is implicated in mesial temporal sclerosis, while brief, occasional febrile seizures are not. Counting every febrile fit as a cause loses the distinction the question tests; denying the association altogether loses the other half of it.

4.3 The mesial aura: the experiential front door

The seizure that arrives as a feeling. Mesial temporal seizures are mistaken for psychiatric events because their onset is subjective and non-motor. How to Read an EEG describes a mesial temporal seizure as beginning with a nonmotor onset and behavioural arrest, the patient falling still and inaccessible, preceded or accompanied by an aura drawn from a small and recognisable family. There is the **rising epigastric sensation** and its autonomic accompaniments, an ascending visceral feeling that patients struggle to name. There is the **olfactory or gustatory aura**, a hallucinated smell or taste, usually unpleasant. There is the cognitive or experiential aura, the intrusions of **déjà vu and jamais vu**, the false familiarity of the new or the false strangeness

of the known. And there is the emotional aura, a sudden, motiveless fear welling up without a frightening thought behind it. Each is routinely misread as a primary psychiatric symptom: the epigastric rush as a panic attack, the fear as an anxiety disorder, the déjà vu and depersonalised strangeness as dissociation, the phantom smell as a psychotic hallucination. The dedicated treatment of ictal fear and ictal panic as amygdalar phenomena, and of the psychiatric mimics of focal seizures, belongs to topics of their own elsewhere in this chapter; the fact to hold is that these auras are the semiology of the mesial temporal lobe, and their psychiatric costume is what makes them dangerous.

MNEMONIC

FROG

The mesial temporal aura families in four beats, each one a feature How to Read an EEG actually names. **F**ear, rising without a reason. **R**ising epigastric sensation with its autonomic tail. **O**lfactory or gustatory hallucination, a smell or taste that is not there. **G**nostic disturbance, the déjà vu and jamais vu of a mind briefly unsure what it knows.

4.4 Automatisms and the vacant minute

The motor signature is quiet, repetitive and unremembered. Once awareness is impaired, the temporal seizure fills the gap with automatism rather than the violent movement of a frontal attack. Kaufman's Clinical Neurology for Psychiatrists fixes the frequency: **automatisms** are present in **about 80%** of seizures originating in the temporal lobe, making them the rule rather than the exception. They fall into two families the source names directly. The oral automatisms are the lip-smacking, chewing and swallowing movements of the mouth; the manual automatisms are the fumbling, picking and fidgeting of the hands. To a bystander this looks like a person who has drifted off and is aimlessly busy, not like a seizure, and afterwards the patient has no memory of the interval. The mimicry deepens when a minority of patients utter brief, stereotyped phrases, so that a fragment of speech emerges from someone who is not, in any usual sense, present. The yield is that a witnessed spell of altered contact filled with oro-alimentary and hand automatisms, followed by amnesia, is temporal lobe epilepsy until proven otherwise, however psychological the surface behaviour appears.

4.5 The electroencephalographic signature of the mesial focus

The record points to the anterior temporal region. The EEG in mesial temporal lobe epilepsy has a recognisable grammar that converts a suggestive history into a localised diagnosis. How to Read an EEG describes the interictal recording as showing anterior temporal discharges maximal at the F7 and F8 electrodes, and, in a proportion of patients, **temporal intermittent rhythmic delta activity**, or TIRDA, a run of rhythmic slow waves over the temporal region that is a useful lateralising sign. When a seizure is captured, the same source describes an ictal pattern of rhythmic alpha-theta activity or spikes over the involved temporal region, and a postictal record that commonly shows slowing on the same side as the focus, a further lateralising clue in the aftermath. The general principles of the EEG, and the difference between ictal and interictal discharges as they inform psychiatric assessment, are set out in their own right elsewhere in this

chapter; what belongs here is the fingerprint of the mesial temporal focus. That fingerprint is intermittent, which is the source of a common and costly misreading.

PITFALL

A single normal routine interictal EEG is taken to exclude temporal lobe epilepsy. The anterior temporal discharges are intermittent, and temporal intermittent rhythmic delta activity may only declare itself in drowsiness or sleep, so a brief waking recording can fall between the discharges and read as clean. Treating that clean trace as a rule-out abandons a patient with genuine, and potentially operable, mesial temporal epilepsy. The discipline is to repeat the study, capture drowsiness and sleep, and weigh the record against the semiology rather than let one normal trace close the question.

4.6 Lateral neocortical temporal epilepsy: the other half of the lobe

Not all temporal seizures are mesial. The examinable counterpoint is the lateral, or neocortical, temporal focus, which differs in aura, electrode localisation and behaviour, and holding the two apart is often the whole point of a question. How to Read an EEG characterises **lateral neocortical temporal lobe epilepsy** by an aura that is auditory or vertiginous rather than visceral or experiential: a hallucinated sound or a sense of spinning, reflecting the auditory cortex and its neighbours on the lateral surface. The interictal discharges shift with the anatomy, appearing over the mid and posterior temporal electrodes, T7 and T8, rather than the anterior temporal maxima of the mesial form. And the seizures spread differently: the same source notes a tendency to more frequent and more rapid secondary generalisation, so a lateral temporal seizure is more apt to march quickly into a bilateral convulsion than its mesial counterpart, which stays local longer. An auditory or vertiginous aura should therefore move the clinician's mental map from the mesial structures to the lateral neocortex and raise the expectation of quicker generalisation, changing both what is looked for on the EEG and what is warned about clinically.

FEATURE	MESIAL (MEDIAL) TEMPORAL	LATERAL (NEOCORTICAL) TEMPORAL
Characteristic aura	Rising epigastric, olfactory or gustatory, déjà vu or jamais vu, fear	Auditory or vertiginous
Onset character	Nonmotor onset with behavioural arrest	Sensory aura referable to the lateral surface
Interictal discharge maximum	Anterior temporal, F7 and F8; TIRDA	Mid and posterior temporal, T7 and T8
Spread to a bilateral convulsion	Tends to remain local longer	More frequent and more rapid secondary generalisation

The two temporal syndromes on their shared axes: the mesial focus announces itself through visceral and experiential auras and anterior temporal discharges, the lateral neocortical focus through auditory or vertiginous auras, more posterior discharges, and a readier march to generalisation.

4.7 Why the temporal lobe is psychiatry's seizure focus

The disproportionate mental burden. The temporal lobe earns its central place because, more than any other seizure origin, it is bound up with psychiatric illness, and the tightest of those bonds is with psychosis. The schizophrenia-like psychosis of epilepsy - the chronic interictal psychosis that can, over years, come to resemble schizophrenia - is especially strongly associated with temporal lobe epilepsy, a link brought into focus by the work of Slater and reported in Lewis Child and Adolescent Psychiatry. The entity itself, its full clinical features, its relation to the epilepsy and its management, is a construct owned by its own topic and taught in its own right elsewhere in this chapter; what this topic asserts is the salience, that of all the localisations it is the temporal lobe whose seizures most consistently keep company with this psychosis. That salience ties the earlier anatomy to a psychiatric outcome: the same mesial structures whose sclerosis generates the seizures sit within the limbic system that governs affect, memory and meaning, so their chronic irritation may express itself, in some patients, as disordered belief and perception. The functional link between the limbic system, kindling and behaviour is the mechanism step of this chapter and is taught in its own right elsewhere in it; the point to carry is that a temporal seizure focus is a psychiatric risk factor, and a patient with longstanding temporal lobe epilepsy who develops a psychosis is showing a recognised association, not a coincidence.

The same lobe is also the substrate of the interictal personality change and the eponymous behavioural cluster that longstanding temporal epilepsy can bring, both taught as constructs of their own elsewhere in this chapter and not reopened here. Together they make the temporal lobe the one seizure focus a psychiatrist must know in anatomical detail: its seizures wear psychiatric clothes at onset and its chronic course carries psychiatric consequences at the far end.

GOOD TO REMEMBER

When a patient presents with unexplained episodic fear, a rising sensation in the stomach, recurrent déjà vu or a phantom smell, do not close on a primary anxiety, panic or dissociative label without asking whether these are auras; a witnessed spell of altered awareness with lip-smacking or fumbling, followed by amnesia, points the same way. The move that changes the course is to think temporal lobe epilepsy, take a history for prolonged childhood febrile seizures, and arrange neurological assessment with an EEG that captures drowsiness.

RULE **An experiential or visceral aura is a temporal lobe seizure until proven otherwise, and its commonest substrate is a discrete lesion.** Most focal impaired-awareness seizures are temporal in origin, most temporal seizures begin on the mesial surface, and the commonest thing wrong with that surface is mesial temporal sclerosis. The chain matters because its end point is remediable: a subjective, easily psychologised aura can trace back to a sclerotic hippocampus that surgery may cure, which is why the aura must be taken seriously as neurology and not filed as psychiatry.

IN INDIA

Mesial temporal sclerosis is among the most surgically remediable of the epilepsies, yet the investigations that identify it, dedicated epilepsy-protocol MRI and prolonged or video-EEG, are concentrated in tertiary and academic centres, while most patients are first seen where only a routine interictal recording, or none, is available. A patient whose experiential auras and automatisms are the face of an operable lesion is thus often carried for years as treatment-resistant anxiety, dissociation or psychosis. The corrective is a low threshold to elicit the aura and the childhood febrile-seizure history, to name temporal lobe epilepsy, and to refer toward the imaging and monitoring that can turn a chronic psychiatric label into a treatable neurological diagnosis.

4.8 Recognising temporal lobe epilepsy: a practical synthesis

The diagnosis is assembled from convergent clues, not clinched by one test. No single feature is decisive, and the surface EEG can miss the mesial focus between attacks; what secures the recognition is the way the strands run together and against the psychological first impression they create. Each of the following should move temporal lobe epilepsy up the differential rather than let it slide off.

- An onset that is a feeling: a rising epigastric sensation, a phantom smell or taste, déjà vu or jamais vu, or motiveless fear, rather than a movement.
- A vacant interval of impaired awareness filled with oral and manual automatisms and followed by amnesia for the episode.
- A childhood history of a prolonged, complicated febrile seizure, which points toward mesial temporal sclerosis where a brief febrile fit would not.
- An interictal EEG showing anterior temporal discharges or temporal intermittent rhythmic delta activity, remembering that a single clean waking trace does not exclude the diagnosis.
- An auditory or vertiginous aura with more posterior temporal discharges when the focus is lateral rather than mesial.
- A longstanding course in which a psychosis strongly associated with temporal lobe epilepsy may emerge, marking the same lobe as the psychiatric one.

Read together, these turn a presentation that reads as anxiety, dissociation or a personality problem into one that reads as focal epilepsy with a nameable, often operable substrate. The clinician who has internalised the pattern treats an aura as neurology to be investigated rather than psychology to be soothed, a normal routine EEG as a reason to record again in sleep rather than to close the case, and a temporal seizure focus as a standing psychiatric risk to be watched, not a finished neurological fact to be filed away.

~80% OF COMPLEX PARTIAL SEIZURES ARE MESIAL TEMPORAL IN ORIGIN	**~80%** OF TEMPORAL-LOBE SEIZURES SHOW AUTOMATISMS	**mesial temporal sclerosis** COMMONEST SUBSTRATE OF TLE	**F7 / F8** ANTERIOR TEMPORAL INTERICTAL MAXIMUM

Temporal lobe epilepsy is the commonest source of focal impaired-awareness seizures, most of them mesial in origin and most often built on mesial temporal sclerosis; it announces itself through experiential and visceral auras and oro-alimentary automatisms rather than convulsion, and it carries the strongest link of any seizure focus to the schizophrenia-like psychosis of epilepsy, so its psychologically disguised onset must be read as neurology.

5 · Frontal lobe epilepsy and its behavioural and psychiatric presentations

This is the great impostor among the epilepsies. More often than any other seizure disorder, **frontal lobe epilepsy** reaches the psychiatrist before it reaches the neurologist, because its attacks look like almost anything except epilepsy. They erupt at night, throw the body into violent thrashing or rigid posturing, last only seconds, leave the patient clear-headed almost at once, and are accompanied by an electroencephalogram that is frequently, maddeningly, normal. To an eye trained on the slow, aura-led, confusion-trailed temporal lobe seizure, none of this reads as a fit. It reads as a nightmare, a tantrum, a functional display, or an act. The marks in this topic are awarded for resisting exactly that first impression, because the price of yielding to it is a patient with real, treatable epilepsy who is instead labelled hysterical, psychogenic or a fabricator and left unprotected.

The reason the mistake is so easy is that every feature which should signal a seizure is either absent or actively misleading. The behaviour is bizarre rather than orderly; the duration is too short to resemble a classical convulsion; awareness is often preserved, which the untrained observer takes as proof the patient is acting deliberately; and the surface EEG, the one investigation that ought to settle the matter, commonly shows nothing or is buried under the noise of the movements themselves. This account sets out the semiology, the timing, the electrographic pitfalls and the behavioural presentations of frontal lobe seizures so that the pattern, once seen whole, becomes recognisable rather than deceptive. The framework that separates focal aware from focal impaired-awareness seizures, and the fundamentals of the EEG, are developed in their own right elsewhere in this chapter; here the concern is what the frontal lobe in particular does, and why it fools people.

5.1 The motor semiology: hyperkinetic thrashing and tonic posturing

Dramatic movement is the signature. Frontal lobe seizures announce themselves through their motor content, and it is the character of that movement, not any accompanying inner experience, that defines them. How to Read an EEG describes two dominant patterns. In the first, the patient is thrown into **hyperkinetic** activity: agitated, complex and often forceful movements of the limbs and trunk, such as thrashing, kicking, bicycling, pelvic thrusting and rocking, which can look purposeful and are readily mistaken for a rage outburst or an emotional scene. In the second, one or more limbs are locked into **asymmetric tonic posturing**, a sustained and often bizarre stiffening in which the body is held in an unnatural attitude. Neither pattern resembles the slowly evolving oro-alimentary automatisms of the temporal lobe, and both look far stranger than a clinician expects a seizure to look. Just as important, awareness during these attacks may be preserved or lost, and it is the combination of odd, vigorous, apparently coordinated movement

with a patient who can sometimes still respond that most powerfully suggests the behaviour is willed. The formal distinction between seizures with and without preserved awareness belongs to the focal-seizure classification set out elsewhere in this chapter; what matters here is that retained awareness does not argue against a frontal seizure, and must never be pressed into service as an argument for a psychogenic one.

5.2 The timing: nocturnal, out of non-REM sleep, and clustering

They are creatures of the night. A second defining feature is when the seizures happen. How to Read an EEG emphasises that frontal lobe seizures are characteristically **nocturnal**, arising specifically out of non-REM sleep rather than from wakefulness, and that they tend to occur in clusters rather than as isolated events. This fact does enormous diagnostic damage in the wrong direction. A patient who, night after night, sits up, thrashes, vocalises, postures and then settles, only to do it again, presents to the world as someone with a sleep disorder or a night-time behavioural disturbance, not as someone with epilepsy. The nocturnal, sleep-bound pattern is precisely what aligns frontal seizures with the parasomnias in the mind of an observer, and the clustering deepens the impression of a recurring nocturnal event rather than a scattered seizure tendency. For the psychiatrist the practical yield is high: any history of repeated, brief, stereotyped nocturnal episodes emerging directly from sleep should raise frontal lobe epilepsy explicitly, because the sleep-relatedness that seems to argue against a seizure is in fact one of its strongest fingerprints.

5.3 The temporal profile: brief, aura-free, and quick to clear

On and off, with nothing before and little after. Kaufman's Clinical Neurology for Psychiatrists lays out the course that separates a frontal seizure from a temporal one at the bedside. Frontal lobe seizures are brief, typically lasting **under one minute**; they are not heralded by a premonitory aura; and they are followed by little or no postictal confusion, so the patient returns to normal almost as soon as the movement stops. Each of these is the mirror image of the temporal lobe seizure, whose anatomy, semiology and psychiatric salience are taught in their own right elsewhere in this chapter: the temporal seizure classically opens with an experiential or visceral aura, unfolds more slowly, and trails a period of postictal clouding. The abruptness at both ends is one of the features most often misread: an event that starts without warning and stops as suddenly, leaving a lucid patient, does not fit the naive template of a seizure and is easily taken for a behavioural outburst the patient could have contained. Kaufman's also fixes the epidemiological frame that completes the picture: frontal lobe seizures tend to begin in adult life and to recur relatively often, on the order of **several times a month**. An adult with frequent, brief, aura-free nocturnal spells is the prototypical presentation, and it walks into psychiatric and sleep clinics far more readily than into a neurology ward.

5.4 The electroencephalographic problem: a seizure the EEG hides

The one test you trust is the one that fails you. The feature that turns frontal lobe epilepsy from a difficult diagnosis into a treacherous one is its behaviour on the EEG. Kaufman's Clinical Neurology for Psychiatrists is explicit that frontal seizures generate paroxysmal discharges which are hard to capture: the interictal recording is often entirely normal, and even the ictal recording,

taken while the seizure is actually in progress, is frequently **obscured by movement artifact**, since the very thrashing that characterises the attack drowns the electrical signal in muscle and motion noise. The anatomy compounds the problem: much of the frontal cortex lies on mesial and basal surfaces, folded away from scalp electrodes, so discharges there may never reach the surface at readable amplitude. A patient whose seizure is genuine but whose EEG is silent is the single most dangerous configuration here, because a normal EEG is precisely what an observer reaches for to declare the episodes non-epileptic. Kaufman's does offer a way through: repeated recordings may eventually reveal bifrontal or midline discharges, so persistence with the EEG, and recourse to prolonged or sleep-capturing techniques, is often what finally secures the diagnosis. The lesson is not that the EEG is useless but that a single normal study proves nothing, and the burden of proof must not be laid on an investigation this particular epilepsy is built to evade.

RULE **A normal interictal EEG does not exclude frontal lobe epilepsy.** Of all the localisation-related epilepsies, this is the one in which the routine scalp recording is most likely to be clean between attacks and swamped by artifact during them. A negative EEG is therefore worthless as a rule-out here, and the diagnosis must rest on the clinical pattern, with prolonged, sleep-capturing or video recording pursued rather than the single normal trace accepted.

5.5 False localisation and false generalisation

Even when the EEG speaks, it may lie about where. When a frontal discharge does break through to the surface, a further trap opens, and it is one of the most examinable points in the topic. How to Read an EEG warns that a frontal ictal pattern may appear to arise from the temporal lobe on the same side as the true focus, a false localisation to the ipsilateral temporal region; or it may spread so quickly across the midline that the record looks generalised from the outset, a phenomenon of **secondary bilateral synchronisation**. Both errors point the interpreter away from the frontal lobe where the seizure actually begins. The clinical stakes are considerable, because localisation drives the choice of imaging, the interpretation of the surgical work-up, and ultimately the decision about which region, if any, might be resected; a patient whose frontal seizures are read as temporal or as primarily generalised is investigated and counselled along an entirely wrong axis. The defence is the same discipline the whole topic demands: the electrographic impression must be weighed against the semiology and the timing, never taken at face value, because in the frontal lobe the surface record is prone to misdirect both about whether a seizure is present and about where it is coming from.

PITFALL

A scalp EEG that captures a frontal seizure but appears to localise it to the ipsilateral temporal lobe, or to be generalised from onset, can steer the entire work-up wrong. The interpreter, trusting the trace, sites the focus in the wrong place, and the imaging, the presurgical evaluation and the counselling follow the error. When the semiology is unmistakably frontal, a temporal or generalised-looking ictal pattern should prompt suspicion of false localisation or secondary bilateral synchronisation rather than a revised anatomical diagnosis.

5.6 Bizarre behaviour and the question of aggression

The most behaviourally florid of the focal epilepsies. The frontal lobe earns its place in a psychiatry chapter because of what its seizures do to behaviour. Kaufman's Clinical Neurology for Psychiatrists makes the sharp point that frontal lobe seizures, even set beside temporal lobe seizures, are the most apt to produce genuinely **bizarre behaviour** and the rare instances of aggressive violence seen in the ictal state. This is not a licence to read violence into every frontal seizure; ictal aggression remains uncommon, and when it occurs it is crude, brief and undirected rather than organised. The general subject of aggression, irritability and episodic dyscontrol in epilepsy, and the vexed forensic question of criminal responsibility, is a construct in its own right and is taught elsewhere in this chapter. The point specific to the frontal lobe is directional and comparative: of all the seizure localisations, this is the one from which strange, disinhibited and occasionally aggressive ictal behaviour is most likely to spring, more so even than the temporal lobe with which it is usually contrasted. That comparative primacy is why frontal seizures so often land in psychiatric hands: the behaviour is loud, the movement is odd, and the whole presentation begs to be read psychologically, when the discipline of the topic is to remember that a seizure can produce all of it.

MNEMONIC

FRONTAL

The signature of the frontal lobe seizure in seven beats, each one a feature the topic actually states. **F**requent, several times a month. **R**apid and brief, over in under a minute. **O**ut of non-REM sleep, so characteristically nocturnal. **N**o aura to warn of onset. **T**onic asymmetric posturing, or hyperkinetic thrashing, as the motor content. **A**wareness that may be kept or lost. **L**ittle or no postictal confusion, clearing at once.

5.7 The great mimic: psychogenic seizures and sleep disorders

Where all the threads converge. Everything set out so far, the bizarre movement, the preserved awareness, the abrupt on and off, the nocturnal timing and the silent EEG, gathers into a single clinical hazard. Kaufman's Clinical Neurology for Psychiatrists states it directly: because their behavioural manifestations are so bizarre and their EEG changes usually undetectable, frontal lobe seizures are the epileptic events most readily mistaken for **psychogenic non-epileptic seizures**, and, when they arise at night, for a sleep disorder. This is the psychiatric heart of the topic. Psychogenic non-epileptic seizures, their semiology, their investigation with video-EEG and their management, are defined and taught across their own dedicated topics elsewhere in this chapter; the fact that belongs here is the reverse direction, that frontal lobe epilepsy is the organic diagnosis most often lost to a functional label. The asymmetry of harm is what should be held: to call a psychogenic attack epileptic risks needless drugs, whereas to call a frontal seizure psychogenic risks abandoning treatable epilepsy and, with it, the seizure control on which so much else depends. A confident functional or sleep-disorder label placed on a bizarre nocturnal episode, on the strength of a normal routine EEG, is the archetypal error.

TRAP **Calling a frontal seizure psychogenic because it looks bizarre and the EEG is clean.** The examiner's favourite version of this topic offers a young adult with brief, dramatic, hyperkinetic or posturing nocturnal spells, preserved awareness, rapid recovery and a normal routine EEG, and invites the candidate to diagnose a non-epileptic or functional attack. Every one of those features is a positive fingerprint of frontal lobe epilepsy. The bizarre look and the normal EEG are not evidence against a seizure; in the frontal lobe they are evidence for one.

FRONTAL SEIZURE FEATURE	WHAT IS SEEN	WHY IT MISLEADS
Onset and frequency	Adult onset, recurring several times a month	An established, repetitive pattern reads as a behavioural or sleep habit
Duration and course	Under a minute, no aura, clears at once	Abrupt on and off looks non-physiological and suggests a willed act
Timing	Nocturnal, out of non-REM sleep, clustering	Sleep-bound recurrence mimics a parasomnia
Semiology	Hyperkinetic thrashing or asymmetric posturing	Odd, forceful movement is read as functional or emotional
Awareness	May be preserved through the attack	Retained awareness with strange movement suggests volition
Interictal EEG	Frequently normal	A clean record is wrongly taken to exclude epilepsy
Ictal EEG	May falsely localise or appear generalised	Even the ictal trace can point to the wrong lobe

The diagnostic signature of the frontal lobe seizure: each feature that ought to confirm a seizure is instead the feature that pushes the unwary observer toward a psychogenic or sleep-disorder diagnosis.

IN INDIA

The equipment that resolves this diagnosis is unevenly distributed. Prolonged and video-EEG monitoring is concentrated in tertiary and academic centres, while most patients are first seen where only a routine interictal recording is available. A single normal trace from a district setting cannot exclude frontal lobe epilepsy, yet it is often read as if it can, and the bizarre nocturnal spells that follow are relabelled as a psychogenic disturbance, a sleep problem or a primary psychiatric complaint. Sleep-related and behaviourally florid nocturnal seizures should be actively considered rather than assumed functional, and referral for prolonged or video-EEG, rather than reassurance drawn from a clean routine study, is what protects a patient with genuine, treatable epilepsy from being written off.

5.8 Recognising the pattern: a practical synthesis

The diagnosis is made by pattern, not by any single test. No single feature is decisive on its own, and the surface EEG, the investigation a clinician instinctively leans on, is the least reliable arbiter in this setting. What secures the diagnosis is the convergence of the features, read together and against the grain of the first impression they create. Each of the following should move frontal lobe epilepsy up the differential rather than down it.

- Repeated, brief, stereotyped nocturnal episodes that emerge directly out of sleep and tend to cluster.
- Hyperkinetic thrashing or fixed asymmetric posturing as the motor content, however bizarre it appears.
- Attacks that begin without an aura and clear at once, without the trailing confusion of a temporal seizure.
- Preserved awareness during obviously abnormal movement, which argues neither way and must not be read as proof of volition.
- A normal or unrevealing routine EEG that has been offered, wrongly, as reassurance.

Held together, these turn a presentation that reads as psychiatric or functional into one that reads as neurological, by refusing to let any single misleading feature carry the decision. The clinician who has internalised the pattern treats a normal EEG as a reason to record for longer rather than to close the case, treats preserved awareness as neutral rather than as proof of pretence, and treats the very strangeness of the behaviour as a pointer toward the frontal lobe rather than away from epilepsy. That reorientation is what keeps a treatable epilepsy from being mislaid among the disorders it so faithfully imitates.

GOOD TO REMEMBER

When a patient gives a history of repeated, brief, bizarre nocturnal spells and arrives with a normal routine EEG, do not let the clean trace close the door. Keep frontal lobe epilepsy on the differential, ask specifically about sleep-bound clustering, preserved awareness and rapid recovery, and arrange prolonged, sleep-capturing or video-EEG through neurology rather than settling for a functional or sleep-disorder label. The normal EEG is the trap, not the answer.

under 1 min	NREM sleep	several / month	often normal
TYPICAL SEIZURE DURATION	WHEN THEY ARISE	USUAL FREQUENCY IN ADULTS	INTERICTAL SCALP EEG

Frontal lobe seizures are brief, aura-free, nocturnal attacks of hyperkinetic thrashing or asymmetric posturing that clear at once and sit on a frequently normal interictal EEG; that pairing of bizarre behaviour with an unrevealing record is why they are the epileptic events most often misread as psychogenic or as a sleep disorder, and why a normal EEG must never be allowed to exclude them.

6 · Epidemiology and bidirectional relationship of epilepsy and psychiatric disorders

The interface begins with counting. Before the peri-ictal syndromes, the psychoses and the prescribing dilemmas of the topics that follow, the psychiatrist needs a working sense of how common epilepsy is, how it behaves across a life, and how tightly it is bound to psychiatric illness. Two ideas carry most of the marks in this area, and both reward being held clearly. The first is that epilepsy is common, chronic and, for the majority, remitting, so that its frequency at a single

moment and its frequency across a lifetime are genuinely different quantities answering different questions. The second, and the one candidates most often underrate, is that the link between epilepsy and psychiatric disorder runs in both directions: seizures raise the risk of psychiatric illness, and psychiatric illness raises the risk of seizures. Together these turn a short list of prevalence figures into a way of thinking about the shared ground where neurology and psychiatry meet.

The examinable content divides into the numbers a candidate is expected to quote and place in the right conceptual slot, and the two-way relationship whose logic reorganises how a clinician reads the patient in front of them. Drawing on Kaufman's Clinical Neurology for Psychiatrists, Lewis's Child and Adolescent Psychiatry and the Maudsley Prescribing Guidelines, it is the chapter's hinge: the peri-ictal, psychotic, mood and drug topics that follow all assume the frame set out here.

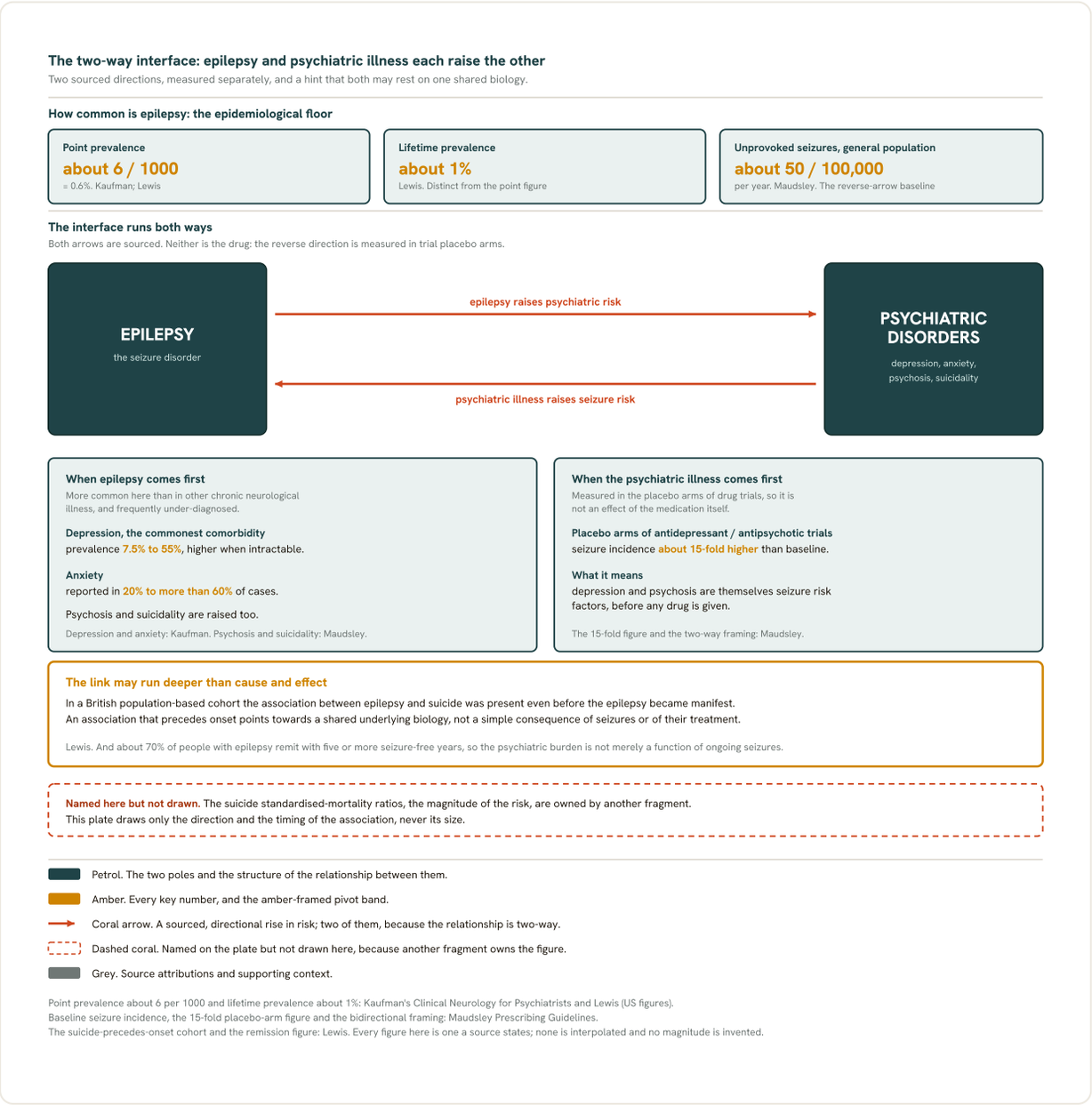


Fig 6.1 **The two-way relationship.** Epilepsy and psychiatric illness set as two poles with a sourced arrow running each way: epilepsy raises the risk of depression (about 7.5% to 55%), anxiety (about 20% to more than 60%), psychosis and suicidality, while psychiatric illness raises seizure risk, shown by a seizure incidence about 15-fold above the general-population baseline of roughly 50 per 100,000 per year in the placebo arms of antidepressant and antipsychotic trials. Because the reverse direction is read from placebo arms, it is a property of the illness rather than of the drug. The amber band carries the deeper point: in a British cohort the epilepsy-suicide association preceded epilepsy onset, which points to a shared biology rather than simple cause and effect. The suicide risk-ratio figures themselves are owned by another fragment and are named but not drawn here.

6.1 How common is epilepsy: prevalence and incidence

Three quantities answer three different questions, and examiners exploit the confusion between them. **Point prevalence** is the proportion of a population who have active epilepsy at a single instant, the figure that tells you how many people in a clinic or on a ward today actually carry the disorder; it sits at about **six people in every 1000**. **Lifetime prevalence** is the proportion who will satisfy the criteria for epilepsy at some time across their lives, and it is necessarily the larger number, of the order of **1%**, because it accumulates everyone who has ever had the condition, including the many now in remission. **Incidence** is different again: it counts new cases arising each year, and for epilepsy it is conventionally expressed as the annual rate of

unprovoked seizures, running at roughly 50 per 100,000 persons in the general population. An unprovoked seizure is one occurring without an immediate provoking cause such as acute metabolic derangement, intoxication or recent head injury, and it is the unprovoked event, rather than any convulsion whatever, that signals the underlying tendency to seize.

Two clarifications keep these numbers honest. A single seizure is not epilepsy; the disorder is defined by an enduring predisposition to recurrent unprovoked seizures, and the formal seizure and epilepsy classification that underpins that definition is the business of the seizure-classification account in this chapter, not of the epidemiology. And acute symptomatic, or provoked, seizures, those driven by a transient insult, are counted separately, because they carry a different prognosis and do not by themselves establish epilepsy. Keeping provoked events out of the count is what makes incidence a measure of the underlying tendency, not of every fit that reaches an emergency department. For the psychiatrist the practical message is twofold: epilepsy is common enough that it will recur throughout a career, in general and liaison settings and in the assessment of paroxysmal behavioural disturbance, and the measures are not interchangeable. Quoting a lifetime figure when asked about point prevalence, or the reverse, signals that a candidate has not grasped what each one measures.

MEASURE	THE QUESTION IT ANSWERS	WHY IT MATTERS TO THE PSYCHIATRIST
Point prevalence	How many people have active epilepsy right now?	Sets the expected caseload burden at any single moment
Lifetime prevalence	How many will carry the diagnosis at some time?	Captures patients now in remission who still bring psychiatric risk and history
Incidence of unprovoked seizures	How many new cases arise each year?	The baseline rate against which seizure risk in psychiatric populations is judged

The three epidemiological quantities distinguished by the question each answers; a lifetime figure and a point figure are not interchangeable, and the incidence of unprovoked seizures is the general-population baseline used later to gauge the reverse direction of the relationship.

6.2 A remitting disorder: why lifetime and point prevalence diverge

The gap between the lifetime figure and the point figure is not a rounding artefact; it is the signature of a condition that most people eventually leave behind. **Remission**, here meaning sustained freedom from seizures over a span of several years whether or not the person stays on medication, is the usual long-term outcome: about **70%** of people with epilepsy eventually reach it. Because the large majority pass into remission, the pool of people with active disease at any instant is smaller than the pool who have ever had it, and that single fact reconciles the two prevalence numbers. It also carries a clinical implication that is easy to miss: a great many patients who carry a past diagnosis of epilepsy are no longer having seizures at all, yet they may still bring the psychiatric and social sequelae of the years in which they were, the interrupted schooling, the surrendered driving licence, the accumulated sense of being marked by the illness.

A minority run a very different course. **Drug-resistant epilepsy**, epilepsy that continues to seize despite adequate trials of appropriate medication, concentrates much of the psychiatric burden, because refractory seizures, repeated hospital contact and the steady erosion of independence are precisely the conditions under which depression and anxiety take hold. The prognostic split therefore matters to the psychiatrist as much as to the neurologist. The person in durable remission and the person with refractory disease sit at opposite ends of a psychiatric risk gradient, and knowing where a given patient falls on it often does more to predict their psychiatric vulnerability than the seizure type alone.

6.3 The bidirectional relationship, in one idea

The organising idea of this whole area is that epilepsy and psychiatric illness are linked both ways. The relationship is **bidirectional**: people with epilepsy carry a raised risk of psychiatric disorder, and people with psychiatric disorder carry a raised risk of going on to develop epilepsy. This is not the older, intuitive picture in which psychiatric symptoms are simply the emotional fallout of living with seizures. That forward pathway is real but it is only one arm of the relationship. The reverse pathway, in which psychiatric illness precedes and predicts seizures, is now equally well established, and it is what lifts the topic from a footnote in a neurology text to a genuine two-way interface deserving a psychiatrist's attention. As the accompanying figure makes clear, the traffic moves in both directions rather than one, and the same psychiatric domains recur on each arm.

MNEMONIC

DAPS

The psychiatric domains for which the two-way link is best demonstrated: **D**epression, **A**nxiety, **P**sychosis and **S**uicidality. Each is more common in people with epilepsy than in the general population, and each, in turn, is associated with a raised risk of seizures.

RULE **The epilepsy-psychiatry relationship is genuinely two-way, not a one-way consequence of seizures.** Any account that explains psychiatric comorbidity solely as a reaction to having epilepsy has described one arm and missed the other. The reverse arm, psychiatric illness as a risk factor for seizures, is where the marks and the clinical surprises both lie.

6.4 The forward arrow: psychiatric disorder is over-represented in epilepsy

That people with epilepsy carry more psychiatric illness than the general population is the older and better-recognised half of the relationship, and the epidemiology is genuinely striking. Depression is the single most common psychiatric **comorbidity** of epilepsy. Reported prevalence spans a wide band, from roughly 7.5% to 55%, with the higher figures clustering among people whose seizures are intractable. The range is broad because studies differ in setting, in how they define a case and in the instruments they use, but even its lower bound sits above the rate seen in most other chronic neurological conditions, and depression here is characteristically both under-recognised and under-treated. The detailed phenomenology and screening of depression in epilepsy belong to the dedicated account of that disorder later in this chapter; what

matters for the epidemiology is its sheer frequency and its claim to first place among the comorbidities.

Anxiety is comorbid almost as often. It is reported in anywhere between about 20% to more than 60% of people with epilepsy, once again with a wide spread that reflects how variably it is defined and measured, and its full clinical treatment is developed in the anxiety-in-epilepsy account of this chapter rather than here. Beyond mood and anxiety, the psychoses of epilepsy and a raised risk of suicide complete the forward picture; each is taken up in full in its own account, and neither should be collapsed into a single number in this topic. The epidemiological point holds across every one of these domains: whatever the precise figure, psychiatric morbidity in epilepsy is not an occasional complication but an expected one.

GOOD TO REMEMBER

Because psychiatric comorbidity in epilepsy is the rule rather than the exception, active enquiry for depression and anxiety belongs in every epilepsy review, not only when the patient volunteers distress. Waiting for a spontaneous complaint is precisely how a frequent, treatable and under-recognised comorbidity is allowed to run unchecked.

PITFALL

Attributing low mood in a person with epilepsy to an understandable reaction to a chronic illness, and therefore watching rather than treating it. The reaction may be genuinely understandable and the depression still be a discrete, biologically driven disorder that warrants the same treatment it would receive in anyone else. Explaining a symptom is not the same as excusing yourself from treating it.

6.5 The reverse arrow: psychiatric illness as a risk factor for seizures

The counterintuitive half of the relationship, and the part examiners most enjoy probing, is that psychiatric illness precedes and predicts epilepsy rather than merely trailing after it. The cleanest quantitative evidence comes from an unlikely place: the placebo arms of randomised trials of antidepressants and antipsychotics. In those arms, where patients receive no active drug at all, the incidence of unprovoked seizures runs about **15-fold higher** than the general-population baseline set out earlier. Since the placebo patients are taking nothing that could lower the seizure threshold, the excess cannot be a drug effect and must instead be attributed to the psychiatric illness itself. Depression and psychosis, on this evidence, behave as seizure risk factors in their own right, and the comparison holds only because there is a stable general-population incidence to measure the trial arms against.

The suicide literature makes the same argument from a different angle. The association between suicide and epilepsy is present even before epilepsy has declared itself: in population-based data the raised risk is already detectable in the years preceding the first seizure. Because that excess cannot be a consequence of seizures that have not yet happened, nor of drugs not yet prescribed, it points instead to a **shared neurobiological diathesis** underlying both conditions. The magnitude of the suicide risk itself, and the specific factors that drive it, are the province of

the suicide-in-epilepsy account and are not quantified here; the lesson at this point is narrower and cleaner, that the timing of the association argues for common ground rather than simple one-way causation.

TRAP **Assuming that seizures in a psychiatric patient must be drug-induced.** Antidepressants and antipsychotics can lower the seizure threshold, but the placebo-arm data show that the underlying illness raises seizure risk on its own, before any drug is given. A candidate who credits every seizure in a depressed or psychotic patient to the medication has quietly denied the reverse arm and lost the point examiners are testing for.

6.6 Reading the two arrows: shared biology, confounding and causation

A two-way association immediately raises the question of what actually connects the two conditions, and here a little epidemiological caution is worth more than a confident answer. Three explanations coexist, and they are not mutually exclusive:

- **Two-way reverse causation** - seizures and the disruption they bring can precipitate mood and anxiety disorders, while depression and psychosis, and the physiological changes that accompany them, can lower the threshold for seizures.
- **Confounding by shared antecedents** - early adversity, head injury, substance use and social disadvantage each raise the risk of both epilepsy and psychiatric illness, and can manufacture an association without either condition causing the other.
- **A common biological substrate** - a shared vulnerability expressed as both a tendency to seize and a tendency to psychiatric symptoms.

What that substrate might actually be belongs elsewhere in this chapter. Its detailed account is the business of the chapter's topic on kindling and the limbic basis of behaviour, and the full anatomy of the structures involved is set out in the Functional Neuroanatomy and Neurophysiology notes; both are named here rather than re-taught. The discipline for the reader is to resist collapsing the three explanations into one. The two-way finding does not, on its own, prove shared biology, since it is equally compatible with reciprocal causation and with **confounding**. What tips the balance towards a common substrate is the observation, from the suicide data, that risk is already raised before epilepsy begins, a pattern that seizures and their treatment simply cannot explain. It is this temporal ordering, more than the bare fact of association, that carries the argument. Applied with that care rather than as a slogan, it is **reverse causation** reasoning at its most defensible.

6.7 Why the epidemiology changes practice

Numbers earn their place only when they change behaviour. The prevalence and the two-way findings converge on a small set of habits that separate careful from careless practice across the neurology-psychiatry border:

- Screen actively for depression and anxiety in everyone with epilepsy, given how frequently they occur and how quietly they are missed when the clinician waits for a spontaneous complaint.

- Take a seizure and a spell history in psychiatric patients, remembering that the illness itself raises seizure risk and that a first seizure can surface on that background rather than only as a drug effect.
- Treat comorbid psychiatric disorder on its own merits rather than writing it off as an inevitable reaction, while staying alert to seizure-threshold effects when selecting an agent.

The last point sits at the centre of the therapeutic tension that runs through the rest of this chapter. Psychiatric illness in epilepsy must be treated, yet the drugs that treat it can influence seizure control, and the drugs that control seizures can in turn influence mood, cognition and behaviour. The epidemiology is exactly what makes that tension unavoidable: if comorbidity were rare, the prescribing dilemma would seldom arise, but because it is the norm, every clinician working at this interface will meet it, and meet it often. The specifics of choosing drugs against the seizure threshold, and of the mood and cognitive effects of the antiepileptics themselves, belong to later topics; the point to carry from here is that the frequency of comorbidity is what forces those choices into the open.

IN INDIA

The population burden of epilepsy is amplified by a wide treatment gap, with a large share of people who have active seizures receiving no consistent antiepileptic care, particularly in rural and under-served districts. In the same high-volume clinics, where a consultation is understandably consumed by seizures and drug supply, the psychiatric comorbidity this topic insists on is even easier to overlook, which is exactly where deliberate, structured enquiry earns its place.

6.8 Pulling the picture together

Common, chronic, mostly remitting, and bound to psychiatric illness in both directions. The topic resolves into a short chain. Epilepsy is common, with a point prevalence lower than its lifetime prevalence because most people eventually remit, and the gap between those two figures is the natural history of the disease made visible rather than a contradiction to be explained away. The relationship with psychiatric disorder is genuinely two-way: the forward arm, in which depression leads a frequent and under-recognised set of comorbidities, and the reverse arm, in which psychiatric illness raises seizure risk in its own right, shown most cleanly by the excess seizures in trial placebo arms and by a suicide association that predates the epilepsy itself. That last point nudges the explanation towards a shared substrate without proving it. Everything the rest of the chapter does, from the peri-ictal syndromes to the prescribing dilemmas, stands on this frame.

~6 / 1000	~1%	~15-fold higher	~70%
POINT PREVALENCE	LIFETIME PREVALENCE	SEIZURES, PLACEBO ARMS VS POPULATION	EVENTUALLY REMIT

Epilepsy is common and mostly remitting, and its relationship with psychiatric illness is bidirectional: seizures raise the risk of psychiatric disorder, and psychiatric disorder raises the risk of seizures, a two-way link best demonstrated for depression, anxiety, psychosis and suicidality.

7 · EEG fundamentals and ictal versus interictal discharges in psychiatric assessment

The electroencephalogram is the one bedside investigation that speaks directly to the border between epilepsy and psychiatry. It is also the investigation psychiatrists most often misread, trusting a normal tracing too far or over-reading an incidental sharp transient in a patient whose real trouble is functional rather than epileptic. Electroencephalography was born inside psychiatry, and the discipline has kept a fascination with the electrical brain ever since. A psychiatrist rarely reports an EEG but is constantly asked to act on one: to decide whether an odd spell was a seizure, whether an atypical psychosis sits on an epileptic substrate, or whether a normal study can be allowed to close a question. This section builds the vocabulary needed to read a report critically rather than take its conclusion on trust. It covers the normal background rhythms and their frequency bands, the morphology of the epileptiform discharge, the sharp limits on what the scalp can and cannot see, and above all the distinction between activity recorded between seizures and activity recorded during one. That last contrast, interictal against ictal, is the electrographic spine of the whole framework that later classifies psychiatric syndromes by their timing to the seizure.



Interictal EEG: Javidan M, Epilepsy Res Treat 2012;2012:637430, Fig 2, CC BY 3.0



Ictal EEG: Javidan M, Epilepsy Res Treat 2012;2012:637430, Fig 3, CC BY 3.0

Fig 7.1 Interictal versus ictal EEG in temporal lobe epilepsy. Above, the record between seizures: intermittent sharp waves over the anterior and sphenoidal temporal channels, the electrical signature that points to the epileptogenic focus without any clinical event. Below, the record during a seizure: a rhythmic discharge builds and evolves over the temporal channels, the hallmark of a focal seizure in progress. The distinction is the whole point of the recording. Interictal sharpening localises the focus; the evolving ictal rhythm confirms a seizure, and the two must never be read as the same thing.

7.1 Where the EEG came from and what the scalp records

A psychiatrist's instrument from the start. The recording of rhythmic electrical activity from the intact human scalp was first achieved by **Hans Berger**, a German psychiatrist, who published the human electroencephalogram in 1929 and in doing so founded electroencephalography as a discipline. The rhythm he described over the back of the head in relaxed wakefulness became the reference point for everything that followed. What the surface electrode actually measures is worth stating plainly, because most misreadings begin with a wrong mental model of the signal.

The EEG does not record action potentials or the firing of single neurons. It records the summed excitatory and inhibitory postsynaptic potentials of large populations of cortical pyramidal cells, whose parallel radial orientation lets their minute extracellular currents add together into a field large enough to reach the scalp. For that summation to become visible, thousands of neurons must be active in near synchrony; sparse or asynchronous activity cancels out and disappears. The signal is then further attenuated and smeared by the meninges, skull and scalp that lie between cortex and electrode, so that what reaches the recording is a blurred, low-amplitude average of a great deal of cortical work. This is why the EEG is exquisitely sensitive to changes in cortical state, the very thing that shifts in delirium, encephalopathy and sedation, yet spatially coarse and blind to much of what the brain is doing. Holding that model steady, a synchronised population average heavily filtered by the head, makes sense of both the power and the limits that the rest of this topic explores.

7.2 The frequency bands of the normal background

Four bands, slowest to fastest. The continuous background of the EEG is described by carving its frequencies into named bands, and four of them carry the clinical weight a psychiatrist needs. Following the conventions of the standard primer How to Read an EEG, they are set out in the accompanying table. The **posterior dominant rhythm** of the healthy, relaxed adult with eyes closed sits in the alpha range and attenuates when the eyes open or attention engages; faster beta activity accompanies alertness and is a familiar signature of sedative-hypnotic drugs; the slower theta and delta frequencies are physiological in drowsiness, sleep and childhood but become pathological when they intrude on the waking adult record. That intrusion is the single most common reason a psychiatrist meets an abnormal EEG. Diffuse slowing of the background into the theta and delta range is the electrographic hallmark of a diffuse encephalopathy, the state that underlies delirium, whereas slowing confined to one region points to a focal structural lesion beneath it. One caution belongs with the numbers. The exact cut-offs between adjacent bands, and the theta boundary in particular, are drawn slightly differently by different authorities, so a boundary figure should be held as a working convention rather than a law of nature.

BAND	FREQUENCY	WHERE IT BELONGS
Delta	0.5 to 4 Hz	Deep sleep in health; pathological as diffuse or focal slowing in the waking adult
Theta	4 to 7 Hz	Drowsiness and light sleep; normal and prominent in childhood
Alpha	8 to 13 Hz	The posterior dominant rhythm of relaxed, eyes-closed wakefulness
Beta	14 to 30 Hz	Active concentration; accentuated by sedative-hypnotic drugs

The four clinically weighted frequency bands of the background, from slowest to fastest.

7.3 What the scalp can and cannot see

Most of the cortex is effectively silent to the scalp. The most consequential limitation of the routine study is spatial. Only about **one-third** of the cortical surface, the crowns of the superficial gyri lying just beneath the electrodes, generates a signal the scalp can read well. The remainder is electrographically quiet from the surface: the cortex buried in the depths of the sulci, the basal surfaces, the mesial walls between the hemispheres and the insula folded deep within the Sylvian fissure all sit too far from any electrode, or are oriented so that their fields point away from it, to register reliably. This single fact of geometry drives much of the clinical trouble a psychiatrist inherits, because the regions that are hardest to see are precisely the ones of greatest psychiatric interest.

- The mesial temporal structures, the seat of temporal lobe epilepsy, lie deep and generate weak, often absent, scalp signals.
- The mesial and orbital frontal surfaces, the substrate of frontal lobe epilepsy, are similarly buried and poorly represented.
- Deep and basal sources in general are attenuated by distance and by the intervening tissue before they ever reach the recording.

The clinical reading of this is uncomfortable but essential: a scalp EEG can be repeatedly and genuinely normal in a patient who is having unmistakable seizures from a deep or mesial focus. The tracing has not failed; it is reporting faithfully on the third of the cortex it can reach and staying silent about the rest. For the psychiatrist weighing an atypical presentation that might be epileptic, this means a normal surface study can never be treated as a clean bill of health for the deep structures where the epilepsy-behaviour interface actually lives.

TRAP **Reading a normal interictal EEG as proof the patient does not have epilepsy.** The scalp sees only the convexity, and a single routine tracing captures a minority of established cases; a mesial temporal or deep frontal focus can discharge freely while the surface record stays stubbornly normal. A normal EEG lowers the probability of epilepsy, it never closes the question.

7.4 Epileptiform discharges: the spike and the sharp wave

Two pointed transients that differ only in duration. When the EEG is abnormal in a way that suggests epilepsy specifically, it is usually because of an **epileptiform discharge**: a brief, pointed waveform that stands out abruptly from the surrounding background, typically followed by a slower after-going wave. Two forms of this transient are distinguished, and the difference between them is purely one of duration. A **spike** is the briefer of the two, lasting **longer than 20 but under 70 milliseconds**, while a **sharp wave** is the broader form, lasting **between 70 and 200 milliseconds**. Both are pointed, both arise from the synchronised, paroxysmal depolarisation of a population of neurons, and both belong to the family of interictal epileptiform abnormalities; morphology does not separate them, only the clock does. It matters to hold this firmly, because not every sharp-looking transient in an EEG is epileptiform. The background is full of benign sharp elements and artefacts that a careful electroencephalographer distinguishes from a true discharge by their field, their after-going slow wave, their disruption of

the surrounding rhythm and their location, and the untrained eye that calls every pointed waveform a spike will generate false alarms. The genuine epileptiform discharge is a specific object with defined temporal limits, not merely anything that happens to look sharp.

MNEMONIC

Spike is Speedy, Sharp is Slower

The two interictal transients are separated on one axis only, their duration: the **sp**ike is the briefer, faster peak and the **sh**arp wave is the broader, longer one. Both are pointed epileptiform discharges, so it is the clock, and not the shape, that tells them apart.

7.5 Reading the discharge: probability, not proof

An epileptiform discharge is a strong pointer, not a verdict. The single most important interpretive principle is that a spike or a sharp wave is not pathognomonic of epilepsy. It is not, on its own, proof that the person has the disorder; what it establishes is a strong predisposition to seizures rather than the disorder itself, and a small proportion of entirely healthy people carry such discharges without ever having a seizure. The way to make this quantitative is to separate two properties that candidates routinely confuse. The epileptiform EEG is highly specific but poorly sensitive for epilepsy, and that asymmetry is the whole point. Its high specificity is what gives a discharge its weight: when one is genuinely present, epilepsy becomes a strong bet. Its low sensitivity is the other blade: most people with epilepsy will not show a discharge on any given routine recording, so the absence of one proves very little. The prevalence of epileptiform discharges in the healthy population anchors the specificity from the other side, low enough that an incidental discharge is uncommon yet not so low that it can be dismissed out of hand. Reading a report therefore means holding the result against the clinical pre-test probability. In a patient with a convincing seizure history, a discharge confirms what was already likely; in a patient with none, the same discharge should prompt caution rather than a diagnosis, because the base rate of epilepsy in that person is low and even a highly specific test throws the occasional false positive.

~95%	~30%	<1%	<4%
SPECIFIC FOR EPILEPSY	SENSITIVE FOR EPILEPSY	OF HEALTHY ADULTS	OF HEALTHY CHILDREN

RULE **The epileptiform EEG confirms far more reliably than it excludes.** A discharge is a strong pointer because the finding is highly specific, but its poor sensitivity means a single recording misses most people who have epilepsy. Read every result against the clinical pre-test probability: the EEG moves the odds, it does not settle the diagnosis by itself.

PITFALL

The mirror error to the examination trap is over-reading. A psychiatrist who orders an EEG as a routine screen for organicity in every atypical presentation will sooner or later be handed a report noting a sharp transient, and will be tempted to rediagnose on the strength of it. Because such discharges occur in a small minority of entirely healthy people, an incidental finding in someone with no clinical seizures rarely changes the picture and should not, by itself, convert a functional or affective presentation into epilepsy. Order the study to answer a defined question, not as a fishing expedition.

7.6 Why one tracing is rarely enough

The single routine EEG is the least sensitive form of the test. The low sensitivity of the interictal recording has a direct practical consequence: **fewer than half** of people with established epilepsy show any epileptiform abnormality on a single routine scalp EEG. A normal first study is therefore the expected result in a large share of genuine cases, and it must never be allowed to overturn a good clinical history. The corollary is that yield can be raised, and the ways of raising it are the practical heart of ordering the test well. Timing is the most powerful lever, since the chance of capturing a discharge is highest soon after a seizure and greatest within roughly the first 24 hours of an event, so an EEG requested promptly after a first seizure earns far more than one deferred for weeks. Beyond timing, the sensitivity of the study itself can be increased.

- Repeating the recording, since serial studies accumulate the chance of catching an intermittent discharge that any one tracing misses.
- Recording during drowsiness and sleep, and using sleep deprivation, which brings out discharges that a fully alert cortex suppresses.
- Prolonging the recording, since a longer or ambulatory study simply samples more time and more brain states.

GOOD TO REMEMBER

Two habits improve the yield without any new equipment. First, record early, because an EEG requested at once after a first event is worth far more than one booked for a month later. Second, read a single normal routine study for exactly what it is, the least sensitive form of the test, rather than as evidence that epilepsy has been excluded. Between them, these two habits prevent the commonest ways a psychiatrist is misled by a scalp recording.

IN INDIA

A routine scalp EEG is inexpensive and widely available across the country, and that cuts both ways. Because it is so easy to obtain, it is frequently over-requested in psychiatric and functional-seizure workups and its incidental findings over-interpreted, while the studies that actually raise the yield, prolonged, sleep-deprived and video-EEG telemetry, remain concentrated in tertiary and academic centres. The practical stance for a psychiatrist in a district setting is to lean on a careful clinical history, to treat a single normal routine EEG as non-exclusory rather than reassuring, and to refer onward when the question genuinely needs activity captured during an event, including the separation of functional from epileptic seizures.

7.7 Interictal against ictal: the distinction that organises everything

The contrast the whole peri-ictal framework turns on. Everything discussed so far, the spike and the sharp wave, has been an **interictal discharge**: an abnormality recorded between seizures, when the patient is clinically well. It testifies to a brain that is capable of seizing, but it is not itself a seizure. An **ictal pattern**, by contrast, is the electrographic seizure itself: not a single transient but a rhythmic, evolving run of activity that builds, changes in frequency and amplitude, spreads across the head and then subsides. The two were mapped onto epilepsy early in the field's history. **Gibbs and Jasper** first reported the interictal discharge of focal epilepsy in 1936, and the most famous ictal and interictal signature of all, the generalised **3 Hz** spike-and-wave discharge of idiopathic generalised epilepsy with absences, was demonstrated by **Gibbs, Davis and Lennox** in 1935. The distinction is not a piece of electrophysiological trivia; it is the difference between a finding that suggests a diathesis and a finding that catches the disorder in the act.

FEATURE	INTERICTAL DISCHARGE	ICTAL PATTERN
When it occurs	Between seizures, with the patient clinically well	During the seizure itself
Form	A brief, stereotyped transient, the spike or sharp wave	A rhythmic run that evolves in frequency, amplitude and distribution
What it establishes	A predisposition; a brain able to seize	That a seizure is happening at that moment
Bearing on behaviour	Supports an epileptic tendency but cannot time a given spell	If locked to an observed spell, proves the spell is that seizure

The interictal and ictal patterns compared, and what each does and does not prove.

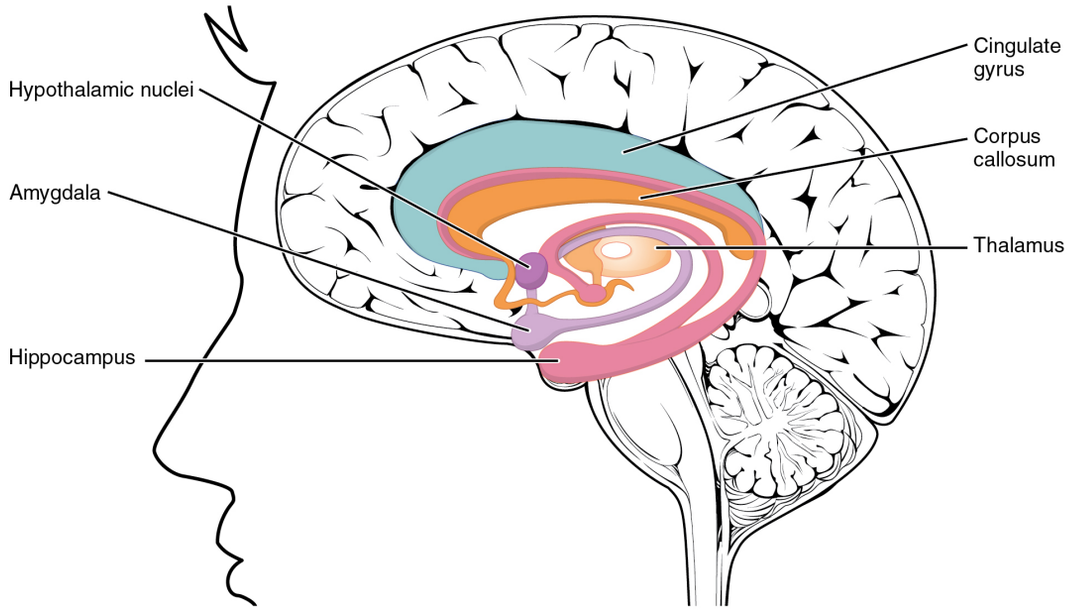
The clinical payoff of the distinction is precise. An interictal discharge supports the presence of an epileptic tendency but cannot, on its own, tell you that any particular episode was a seizure. An ictal pattern does exactly that: when a rhythmic electrographic seizure is recorded in lockstep with an observed behavioural change, a spell of fear, a period of altered awareness, an automatism, it proves that the behaviour in front of you is that seizure and not something else. This is why the capture of an event on a recording is the aim of the more elaborate studies, and why the interictal-ictal distinction is the electrographic foundation of the framework that

classifies psychiatric syndromes by their timing to the seizure. To place a preictal, ictal, postictal or interictal label on a behavioural disturbance is, at bottom, to make a statement about its relationship to an electrographic seizure, and that statement rests on the difference this section has drawn.

> Interictal discharges tell you a brain is capable of seizing; an ictal pattern tells you it is seizing at that moment, and only the second can prove that the behaviour in front of you is itself a seizure.

8 · Limbic system, kindling and the neurobiology linking epilepsy to behaviour

Epilepsy is at root a disorder of abnormal electrical discharge, yet the reason it earns a place in a psychiatry examination is that it so reliably changes behaviour, mood and thought. The question a candidate is really being asked, whenever this material appears, is a mechanistic one: by what route does a paroxysm of electrical activity in the brain translate into a durable psychiatric change that can outlast any single seizure by years. This section is the chapter's mechanism step. It does not catalogue the peri-ictal syndromes, the personality change of long-standing epilepsy or the several psychoses of epilepsy, each of which is described in its own right elsewhere in this account; it supplies the neurobiology that makes all of them intelligible. Two ideas carry the weight. The first is anatomical: the seizures that matter most for behaviour arise in or spread to the limbic system, the emotional and mnemonic core of the brain. The second is physiological and is the heart of the topic: the phenomenon of kindling, a laboratory model in which repeated limbic seizure activity leaves a lasting change in how the brain works. Hold those two together, a vulnerable anatomical stage and a mechanism that makes seizure activity self-perpetuating and behaviourally consequential, and the whole clinical picture of epilepsy-related psychiatric disorder acquires a spine.



OpenStax College, Anatomy and Physiology, "The Limbic Lobe", CC BY 3.0, via Wikimedia Commons

Fig 8.1 The limbic circuit and the mesial temporal structures that matter in temporal lobe epilepsy. The amygdala and the hippocampus sit at the medial edge of the temporal lobe and are the commonest origin of focal seizures that carry psychiatric colour; through the limbic ring they connect to the cingulate gyrus, the hypothalamus and the thalamus. This is the anatomy behind the affective and experiential aura, behind the memory disturbance of mesial temporal sclerosis, and behind the kindling model in which repeated limbic discharge lowers the threshold for the next.

8.1 The limbic system as the anatomical stage

Why behaviour and seizures meet in the same territory. The limbic structures are where the epilepsy-behaviour interface physically lives, and this is no coincidence. The same medial temporal and basal forebrain regions that generate and propagate the seizures of greatest psychiatric interest are the regions that subserve emotion, motivation, fear and the laying down of memory. When a discharge arises in or recruits this circuitry, it is not activating neutral tissue; it is driving the very apparatus of affect and salience, which is why limbic seizures so often carry an emotional or experiential colour and why chronic limbic epilepsy has behavioural sequelae that a purely motor epilepsy does not. The limbic system also has a low threshold for the propagation and self-reinforcement of abnormal activity, a property that becomes central once kindling is introduced. The full anatomy of this system, its component structures, their interconnecting circuitry and its wider projections, is a neuroscience topic in its own right and is set out in the Functional Neuroanatomy and Neurophysiology notes; it is named here and not re-taught. The one name to carry across is the classical loop itself, the **Papez circuit**, which runs from the hippocampus through the fornix to the mammillary bodies, on to the anterior thalamus and the cingulate gyrus and back by way of the entorhinal cortex, the anatomical ring through which emotion and memory are bound together; the amygdala sits within the limbic system but outside that canonical loop. What matters for the present purpose is only the functional consequence: the limbic system is simultaneously the brain's principal seizure-prone gateway and its emotional engine, so activity that disturbs it disturbs behaviour almost by definition.

Everything that follows is an account of how repeated disturbance of this stage produces changes that persist.

8.2 Kindling: the experimental paradigm

A small, repeated stimulus that grows its own seizure. The single most important laboratory model linking seizure activity to lasting change is **kindling**. In the classic preparation, a brief, low-intensity electrical stimulus is delivered periodically to the amygdala or another limbic structure. The first stimulation is deliberately weak: it evokes only a brief electrographic seizure, an **afterdischarge** visible on the recording, with no outward change in the animal's behaviour at all. Left there, nothing would come of it. The defining observation is what happens on repetition. When the same modest stimulus is applied again and again over successive sessions, the evoked response does not stay constant; it escalates. The afterdischarges lengthen and spread, and behavioural accompaniments appear and intensify in a stereotyped progression, until a stimulus that once produced only a silent electrographic flicker now reliably provokes a full, generalised behavioural seizure. As the standard pharmacology account in Goodman and Gilman's therapeutics text describes the model, it takes only **about 10 to 20** such stimulations for this progressive intensification to unfold. The power of the paradigm is precisely that the stimulus never changes. Nothing is made stronger from the outside; the brain itself becomes progressively more responsive to an unchanging input, manufacturing an ever-larger seizure from an ever-constant spark. That is the observation the rest of the topic has to explain, and it is the reason kindling, rather than any single acute seizure, is treated as the model of how epilepsy leaves a mark.

- An initial weak stimulus evokes only a brief afterdischarge, with no behavioural change.
- Repetition of the identical stimulus lengthens and spreads the electrographic seizure.
- Behavioural seizure components appear and escalate in a stereotyped sequence.
- The endpoint is a full generalised seizure elicited by the same stimulus that once did nothing.

8.3 Why the amygdala kindles most readily

The emotional structure is also the most excitable one. Kindling can be established at several limbic sites, but they are not equivalent, and the difference is itself instructive. The **amygdala** is the ideal site for kindling because seizure activity can be induced there most rapidly, a point emphasised in the account of the phenomenon in Panksepp's Affective Neuroscience. This is not an incidental piece of laboratory trivia. The amygdala is the brain's principal mediator of fear, threat appraisal and the emotional tagging of experience, so the fact that it is also the tissue most readily driven into a self-amplifying seizure is exactly the anatomical coincidence that makes limbic epilepsy behaviourally loud. A structure that kindles quickly is a structure whose repeated activation most efficiently installs the lasting change that kindling produces, and when that structure is the seat of emotion, the lasting change is liable to be emotional and behavioural in kind. The rapidity of amygdalar kindling therefore does double duty in the argument: it identifies the most vulnerable node in the limbic circuit, and it explains why the psychiatric consequences of chronic limbic seizures so often take the form of altered affect, fearfulness and reactivity

rather than, say, a pure motor or sensory residue. The anatomy of the amygdala as a structure belongs to the neuroanatomy account; what is owned here is the physiological claim that it is the fastest-kindling site and the behavioural inference that follows from it.

8.4 The nature of the change: functional, not structural

A lasting reorganisation you cannot see on a scan. The most consequential feature of the kindled state is what kind of change it is. Kindling induces a permanent change in the functional organisation of the brain with no change evident at the structural level. Once an animal is kindled it stays kindled: the heightened seizure susceptibility does not fade with the passage of time, yet nothing corresponding can be found when the tissue is examined for gross structural damage. The change is in how the circuit works, in the strength and pattern of its synaptic connections and the ease with which activity propagates through it, not in any visible lesion. Conceptually this belongs with the broad family of use-dependent plasticity, the same class of lasting, activity-driven strengthening of synaptic transmission that underlies ordinary learning and memory; kindling can be read as a pathological cousin of that machinery, in which the thing being learned is the seizure itself. This is the property that makes kindling such a satisfying bridge between an electrical event and an enduring behavioural one. A single seizure comes and goes; a functional reorganisation that persists indefinitely is exactly the kind of durable substrate a durable behavioural change requires. It also disciplines how the clinician thinks about a normal scan in a patient whose behaviour has shifted after years of seizures, because the model predicts precisely the situation of a real change in brain function with an unremarkable image.

MNEMONIC

Small spark, lasting fire

Kindling's lesson compressed: a stimulus too **small** to change behaviour at first, once repeated, leaves a **lasting** reorganisation of function that does not fade and does not show on structural imaging. The name is the memory aid - a fire kindled from small sparks that, once lit, stays lit.

PITFALL

A normal MRI is taken to mean the brain is structurally intact and therefore that a patient's behavioural or affective change cannot be seizure-related. Kindling predicts the opposite situation directly: a genuine, permanent reorganisation of function can coexist with a wholly unremarkable structural scan. Reassurance drawn from clean imaging should not be allowed to dismiss a real, biologically grounded change in a patient with long-standing limbic epilepsy.

8.5 One phenomenon reached by several routes

Not an artefact of the electrode. A reasonable first suspicion is that kindling is a quirk of pushing current through an electrode, an experimental artefact with no bearing on natural disease. The convergence of routes is what answers that objection. Kindling can also be induced by periodically administered seizure-inducing drugs, so-called **chemical kindling**, and by repeated loud auditory stimulation in susceptible animals, or **auditory kindling**. That the same progressive, self-amplifying, permanent phenomenon can be produced electrically,

pharmacologically and by a purely sensory stimulus tells us it is a property of the nervous tissue and not of any one method of provoking it. Whatever repeatedly drives the limbic circuitry into brief seizures, whether an implanted electrode, a convulsant compound or an intense recurring sound, sets the same machinery in motion. This generality is the reason kindling can be offered as a model of human disease at all. Human beings are not stimulated through amygdalar electrodes, but they do sustain recurrent limbic seizures from natural causes over years, and the multiplicity of experimental routes makes it plausible that the natural process engages the identical mechanism. The routes differ; the outcome, a lasting escalation of seizure susceptibility, does not.

- Electrical: brief, low-intensity periodic stimulation of a limbic site, the original paradigm.
- Chemical: periodically administered seizure-inducing drugs producing the same escalation.
- Auditory: repeated loud auditory stimulation in susceptible animals.

8.6 From kindled seizure to altered behaviour

This is the step the whole topic exists to make. A model of escalating seizures is only half of what a psychiatrist needs; the other half is the link from seizure activity to behaviour. Kindling supplies it. Repeated kindling produces a durable change in behaviour in animals, and it is this observation, not any single acute seizure, that provides the neurobiological model linking limbic seizure activity to altered behaviour. The animal does not merely become more seizure-prone; it becomes, in lasting ways, a behaviourally different animal. That is the bridge in its cleanest experimental form. From it, two lines of clinical thought are drawn, and the discipline of this section lies in naming them without annexing them. The enduring behavioural residue of chronic human limbic epilepsy, the interictal personality change of long-standing temporal lobe epilepsy, is the human counterpart of this animal observation, but it is a topic in its own right and is not taught here. Separately, the concept of kindling is invoked as an etiological model to help explain the emergence of psychotic phenomena in epilepsy, offering a mechanism by which a psychiatric syndrome can arise gradually over years of accumulating limbic seizure activity rather than appearing fully formed. The psychoses of epilepsy themselves, ictal, postictal and chronic interictal, are described elsewhere; kindling is only the candidate mechanism that makes their slow emergence intelligible. What this fragment owns is the bridge; what the bridge leads to is owned by others.

RULE **Repeated limbic seizure activity leaves a permanent functional change that durably alters behaviour.** This single proposition is the neurobiological link between epilepsy and its psychiatric consequences. It is why a chronic epilepsy can reshape affect and conduct over years, and why the relevant lesion is one of function rather than structure. Every peri-ictal syndrome, personality change and epileptic psychosis discussed elsewhere ultimately draws on this mechanism.

GOOD TO REMEMBER

When a patient with years of limbic epilepsy shows a settled change in mood, reactivity or thought, the kindling model gives you a reason to treat that change as biologically grounded rather than as a purely psychological reaction to having a chronic illness. It does not license a deterministic story, but it does argue against dismissing a durable behavioural shift as merely reactive in someone whose limbic circuitry has been seizing for a long time.

8.7 Epileptogenesis: how an epileptic focus is acquired

From a single insult to a chronic disorder. Kindling models the progressive facilitation of seizures; a related but distinct body of work models how a brain that has never been epileptic acquires a chronic epilepsy, the process of **epileptogenesis**. Here the experimental insult is not a train of small stimulations but a single severe one. In the status-epilepticus models, a chemoconvulsant, either **kainic acid** or **pilocarpine**, is used to provoke prolonged seizure activity, and the animal is then followed. After a latent interval of weeks, during which the animal appears well, spontaneous recurrent seizures emerge and the hippocampus is found to be sclerosed. The sequence is the point: a single acute insult, a silent maturation period, and then a self-sustaining epilepsy with a characteristic hippocampal lesion. This maps with striking directness onto a well-recognised human pathway, in which complicated febrile seizures in early childhood precede, after a long silent interval, the mesial temporal sclerosis that underlies temporal lobe epilepsy, the substrate detailed in the dedicated account of that condition. The animal work makes that clinical association mechanistically credible: it shows, in a controlled setting, that an early episode of intense limbic seizure activity can causally produce the sclerotic focus years before the chronic epilepsy declares itself. The accompanying figure sets kindling and this chemoconvulsant model side by side as the two complementary halves of the neurobiological story, one explaining escalation, the other explaining acquisition.

FEATURE	KINDLING	CHEMOCONVULSANT STATUS MODEL
Provoking insult	Brief, low-intensity stimulation, repeated periodically	A single episode of chemically induced prolonged seizure activity
What it models	Progressive facilitation of seizures and durable behavioural change	Acquisition of a chronic epilepsy from an initial insult
Time course	Stepwise intensification across successive stimulations	A silent latent interval, then spontaneous seizures
End state	Permanent functional reorganisation, no structural lesion	Spontaneous recurrent seizures with hippocampal sclerosis
Human parallel	Limbic seizures linked to lasting altered behaviour	Complicated febrile seizures preceding mesial temporal sclerosis

The two complementary limbic models: kindling explains escalation and behavioural change, the chemoconvulsant status model explains how a focus is acquired.

8.8 What the model establishes, and what it does not

A powerful bridge, held at the right altitude. The value of kindling to the psychiatrist is that it converts a plausible clinical intuition, that years of limbic seizures can change a person, into a demonstrated biological mechanism with a defined form: escalation from an unchanging stimulus, a permanent functional reorganisation, and a durable behavioural residue, reached by several independent routes. That is a great deal, and it is what the marks reward. What the model does not do is prove, in any individual human patient, that a given psychiatric syndrome was caused by kindling. It is a model and a heuristic, established in animals and invoked to make human phenomena intelligible, not a diagnostic test that can be applied at the bedside. The distance between a controlled animal preparation and a particular person's psychosis or personality change is real and must be respected. Read at the right altitude, kindling earns its central place: it is the reason the chapter can speak of an epilepsy-behaviour interface at all, the mechanism that underwrites the peri-ictal syndromes, the personality change and the epileptic psychoses without itself being any of them.

Amygdala	Functional	Durable	A model
THE FASTEST-KINDLING SITE	PERMANENT REORGANISATION, NOT STRUCTURAL	BEHAVIOURAL CHANGE IN ANIMALS	FOR EMERGENT PSYCHOSIS, NOT A PROOF

TRAP **Treating kindling as proven causation in the individual patient rather than as an animal model and heuristic.** Candidates who have grasped the mechanism are tempted to state that a patient's epilepsy caused their psychosis or personality change by kindling. The evidence supports kindling as a model that explains how such changes can emerge; it does not license a causal verdict in a single case. State the mechanism as a model, and the direction of the claim survives.

Kindling shows that repeated limbic seizure activity leaves a permanent, purely functional reorganisation of the brain that durably alters behaviour, and that single fact is the neurobiological bridge from an electrical disorder to its psychiatric consequences.

The peri-ictal axis: syndromes by their timing to the seizure

9 · Classification of peri-ictal psychiatric syndromes by temporal relationship to seizure

The single most useful question at the bedside is not what the symptom is, but when it sits. A patient with epilepsy who turns frightened, confused, elated, paranoid or profoundly low presents the psychiatrist with a phenomenology that, taken in cross-section, can be indistinguishable from a primary psychiatric disorder. What tells them apart, and what organises the otherwise sprawling behavioural landscape of epilepsy into something teachable, is the relationship each disturbance holds to the seizure itself. That temporal relationship is the classifying axis of this topic, and mastering it earns disproportionate marks because it is the scaffold on which every other peri-ictal syndrome hangs. Get the timing right and a wide

differential collapses to a short candidate list; get it wrong and a self-limiting, seizure-driven state is mistaken for a lifelong illness.

The anchor of the whole scheme is the seizure event itself, against which every psychiatric phenomenon is dated. Working outward from that anchor, a disturbance may coincide with the discharge, may cluster in the hours around it, may occupy the long stretch between attacks, or may float free of any dependable temporal tie. Each position carries its own diagnostic weight, its own list of candidate syndromes and, decisively, its own management logic. This account builds the axis from its vocabulary upward, places the categories of psychiatric disturbance onto it, and then draws out the one management principle the timing dictates. The syndromes that occupy each position are developed in their own right elsewhere in this chapter; here they are only located, never re-taught.

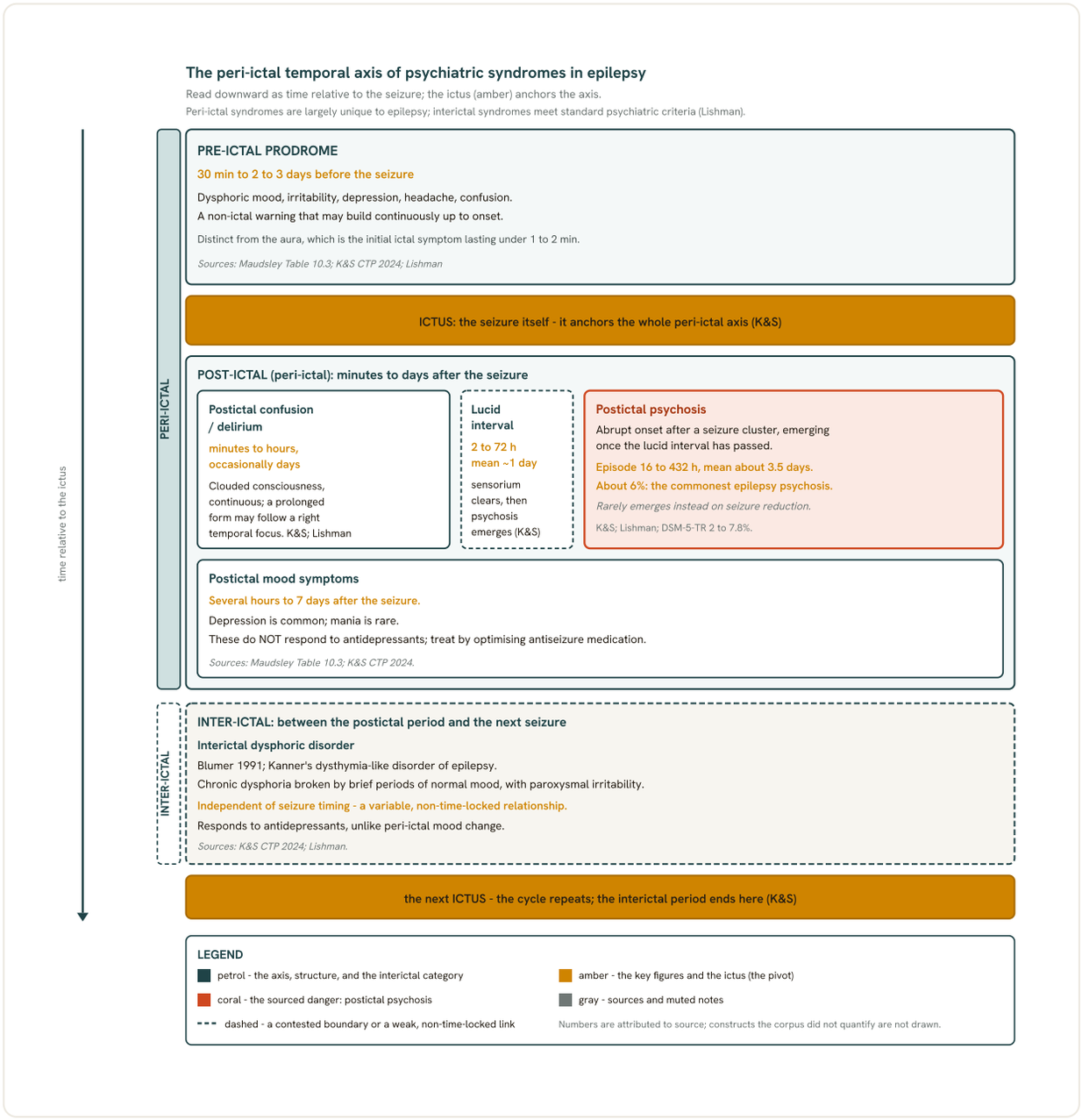


Fig 9.1 **The peri-ictal axis.** Psychiatric syndromes of epilepsy arranged by their timing relative to the seizure. Peri-ictal syndromes (the pre-ictal prodrome, ictal states, and postictal states, including postictal psychosis after a lucid interval) cluster around the ictus and are largely unique to epilepsy, whereas interictal syndromes such as the interictal dysphoric disorder are not time-locked and meet standard psychiatric criteria.

9.1 The vocabulary of timing: ictus, peri-ictal, interictal

Fix the reference points before anything is placed against them. The classification cannot be used until its temporal terms are pinned down, because each names a window in time rather than a symptom. The **ictus** is the seizure event itself; it is the zero point of the timeline, and Kaplan and Sadock's Comprehensive Textbook of Psychiatry treats it as the anchor from which the entire temporal axis is drawn. The **interictal period** is the stretch that lies between the settling of one seizure's aftermath and the onset of the next, which is to say the ordinary baseline state in which a person with epilepsy spends the great majority of life. The **peri-ictal period** is the narrow band just before or just after the ictus, and it carries a deliberate looseness in its very definition: it is the term reached for precisely when there is not enough information to say exactly where the seizure begins or ends.

That looseness is not sloppiness but clinical honesty. The electrical event has a beginning and an end that are frequently invisible without simultaneous recording, so a behavioural change that seems to precede a convulsion may in fact be the earliest, purely subjective fragment of the discharge, and an apparent aftermath may be the tail of ongoing seizure activity. Naming such a phenomenon peri-ictal, rather than committing it to ictal or postictal, is an open admission that without electrographic confirmation the exact boundary is unknown. This is the real work the peri-ictal label does: it holds together the phenomena that hug the seizure without forcing a precision the clinic rarely possesses. The interictal state, by contrast, is defined by its distance from the fit. It is what remains once the postictal disturbances have cleared and before the next attack announces itself, and it is against that quiet baseline that any enduring, seizure-independent psychiatric disorder must be judged.

9.2 The categories, by relationship to the seizure

The spine of the whole topic. With the vocabulary fixed, the psychiatric manifestations of epilepsy sort into **four** categories defined solely by their relationship to the ictus. Kaplan and Sadock's Comprehensive Textbook of Psychiatry sets them out as the **ictal** phenomena, which are the psychiatric expression of the discharge itself and of which the psychic auras are the paradigm; the peri-ictal phenomena clustered around the fit, of which postictal confusion is the everyday example; the interictal disorders arising in the baseline state, of which an interictal psychosis is representative; and a fourth, variably related group whose binding to the discharge is loose, and into which most mood disorders fall. The categories are exhaustive and they are mutually exclusive at the level of a single episode, which is exactly what makes the scheme usable at speed. The ictal category is reserved for a phenomenon that is unambiguously the discharge itself; where the boundary between the discharge and its aftermath cannot be drawn, the phenomenon is placed in the peri-ictal category rather than the ictal one, and it is that rule that keeps the two from overlapping.

CATEGORY	TIMING RELATIVE TO THE ICTUS	REPRESENTATIVE DISTURBANCE	DIAGNOSTIC FOOTING
Ictal	during the discharge	psychic auras	part of the seizure itself
Peri-ictal	the hours just around the fit	postictal confusion	largely unique to epilepsy
Interictal	the baseline between attacks	interictal psychosis	meets standard psychiatric criteria
Variably related	loose or uncertain tie to the fit	most mood disorders	relationship to the discharge not established

The categories of psychiatric disturbance in epilepsy, ordered by their temporal relationship to the seizure; each representative disturbance is developed as its own topic elsewhere in this chapter and is named here only to locate it.

The ordering is not arbitrary. Read from the discharge outward, each category is one step further removed from the electrical event, and that distance tracks a real shift in what the phenomenon means and what to do about it. An ictal disturbance is the seizure wearing a psychiatric face; a peri-ictal one is the brain's disturbed state in the immediate orbit of the fit; an interictal one is a

disorder living in the quiet between attacks; and the variably related group is the honest acknowledgement that some disturbances refuse to keep a schedule. Holding these in mind as a single ordered axis, rather than as unrelated headings, is what lets a clinician move from a raw presentation to a working category in a single step.

RULE **Classify by timing before you classify by symptom.** In epilepsy the same cross-sectional picture can arise in any of the categories, so the phenomenology alone will not place it. The relationship to the seizure is the primary sort key, and it must be established first, because it, and not the symptom list, decides both the diagnosis and the first treatment move.

MNEMONIC

AXIS

The positions on the axis, held as one word. **A**round the fit is the peri-ictal band. **X** marks the discharge itself, the ictal category. **I**n between the attacks is the interictal baseline. **S**lackly bound, keeping no reliable schedule, is the variably related group where most mood disorders sit. Each letter asserts only a timing this topic states.

9.3 What the peri-ictal category gathers together

One category, several kinds of phenomena. The peri-ictal bin is the busiest of the categories and the one most often misread, because it is not a single syndrome but a gathering of **three** temporally distinct groups. Kaplan and Sadock's Comprehensive Textbook of Psychiatry, in its table of behavioural disorders in epilepsy, collects within the peri-ictal category the prodromal disturbances that precede the fit, the postictal disturbances that follow it, and the boundary-blurred phenomena in which it cannot be said cleanly whether a disturbance is still the discharge or already its aftermath.

- The **prodromal** phenomena, such as the dysphoria and irritability that can build over hours to a few days before a seizure, taken up as the preictal prodrome in its own dedicated discussion.
- The postictal phenomena that emerge as the discharge subsides, spanning the confusion and delirium of the immediate aftermath through to the postictal psychoses that declare themselves only after a symptom-free interval.
- The boundary-blurred phenomena, including the peri-ictal psychoses, where the very lack of information about when the seizure starts and stops is what forces the peri-ictal label onto the picture.

Grouping these under one heading is not a filing convenience; it reflects a shared biology. Each is a direct product of a brain destabilised by the seizure or by the state immediately surrounding it, which is why they arrive stereotyped, run a limited course, and settle as that state resolves. It also explains why the peri-ictal category behaves so differently from the interictal one at the level of workup and treatment, a difference taken up next. Keeping the prodromal, postictal and mixed subgroups mentally separate, while recognising that they share the peri-ictal home, prevents the

common error of treating everything that happens near a seizure as a single undifferentiated event.

9.4 Peri-ictal versus interictal: unique to epilepsy, or not

The split that carries most of the diagnostic weight. The sharpest and most examinable line on the axis runs between the peri-ictal and the interictal categories, and Lishman's Organic Psychiatry frames it in a way worth memorising. The peri-ictal presentations, the prodrome and the postictal psychosis among them, are largely unique to epilepsy: they have no clean counterpart in general psychiatry, so their very form points back toward the seizure and is close to diagnostic of an epilepsy-related state. The interictal disorders, by contrast, meet the standard psychiatric criteria used in patients who have no epilepsy at all, and they look like the disorders seen everywhere else. That contrast has a direct clinical consequence. A peri-ictal presentation is essentially telling you its own origin, whereas an interictal presentation must be worked up as you would work up any psychiatric disorder, with the epilepsy standing as context and vulnerability rather than as the proximate cause. The two categories demand different reasoning even when their surface phenomenology briefly resembles, and collapsing them loses the point of the axis.

TRAP **Treating every psychiatric disorder in epilepsy as epilepsy-specific.** Candidates lose the mark by assuming that because a disturbance occurs in a patient with epilepsy it must be a peri-ictal, seizure-bound phenomenon. The interictal disorders meet ordinary psychiatric criteria and are diagnosed exactly as they would be in anyone else. Reserve the epilepsy-unique framing for the peri-ictal band, and keep the interictal disorders on standard footing.

9.5 The fourth category: disturbances only loosely tied to the fit

Not everything dates cleanly to the seizure, and the scheme says so. The fourth category is easy to forget precisely because it is defined by the absence of a firm temporal tie, yet naming it is itself examinable. Kaplan and Sadock's Comprehensive Textbook of Psychiatry places into this **variably related** group the disturbances whose relationship to the discharge is weak or not established, and it notes that most mood disorders belong here. A depressive or anxious presentation in epilepsy sometimes clusters before or after seizures, sometimes runs steadily in the interictal baseline, and often shows no dependable relationship to the fits at all, which is exactly why the classification declines to force it into a single slot. The honesty of this fourth bin is its strength. It resists the reflex, common in candidates and clinicians alike, to assume that every disturbance in epilepsy must lock onto the seizure, and it leaves room for the mood syndromes that genuinely do keep a schedule, such as the postictal mood changes and the interictal dysphoric disorder of epilepsy, to be recognised and taught in their own right elsewhere in this chapter. What sits in the variable category is the broad mass of mood disturbance that will not be pinned to the discharge, and treating that mass as though it were peri-ictal is a category error the axis is designed to prevent.

9.6 Peri-ictal psychosis and the paradox of seizure frequency

A disturbance that can worsen when seizures get either worse or better. The peri-ictal psychoses illustrate a counter-intuitive feature that the axis makes sense of. As a rule, peri-ictal disturbance tracks seizure burden, so that a run of fits is the usual precipitant and better seizure

control brings better behaviour. But the **peri-ictal psychosis** can move in either direction: it may worsen as seizure activity increases, or, less commonly, it may emerge precisely as seizures come under control and the record quietens. That second, decreased-frequency form is the bridge to forced normalisation, the phenomenon in which behavioural disturbance appears as seizure activity is suppressed, which is developed in its own dedicated discussion elsewhere in this chapter. The clinical lesson is that a psychosis appearing on the axis cannot be read off seizure frequency alone; the direction of change matters as much as the fact of it, and improving the electroencephalogram is not always the same as improving the patient.

PITFALL

When a patient's seizures have just been brought under good control and psychosis then appears, the reflex is to read it as an unrelated new illness and to press on with the successful anticonvulsant. In the decreased-frequency form the improving seizure control may itself be driving the disturbance, so intensifying suppression can make matters worse. A psychosis that arrives as the fits recede should prompt a rethink of the regimen, not an automatic escalation of it.

9.7 The management principle the timing dictates

The axis is not only diagnostic; it sets the first move in treatment. Once a psychiatric disturbance in epilepsy has been placed in the peri-ictal band, its management follows a principle that the timing itself dictates, and the guidelines that govern it are, for the seizures, the International League Against Epilepsy and the National Institute for Health and Care Excellence epilepsy guidance, and, for the psychiatric layer, the epilepsy-specific psychiatric-management literature together with the Maudsley Prescribing Guidelines. The organising claim is blunt and high-yield: every peri-ictal psychiatric symptom is first treated by optimising the **antiseizure medication**, whether the symptom is preictal, postictal or of blurred boundary, because there is no evidence that psychotropic drugs prevent the peri-ictal symptoms themselves.

The **LOCUS** of that care is usually the outpatient clinic, where the antiepileptic regimen is reviewed and tightened; it escalates to the inpatient ward or the emergency department only when a peri-ictal state such as a florid postictal psychosis or severe agitation threatens safety. The **focus** shifts by phase: the acute target is the disturbed behaviour of the current episode and the risk it carries, the short-to-mid-term target is the seizures that generated it, and the long-term aim is durable seizure control so that the peri-ictal disturbances do not recur. The **modus** is led by the pharmacological mode, but pointed at the seizures rather than at the symptom, optimising the anticonvulsant before reaching for any psychotropic; the psychological, social and procedural modes support this work, through education, safety planning and attention to the precipitants of seizure clusters, but none of them substitutes for getting the seizure treatment right. The whole plan is an application of the axis: because the disturbance is peri-ictal, the lever that moves it is the one that moves the seizures.

IN INDIA

The rule that peri-ictal symptoms are managed first by optimising seizure control lands with particular force in Indian practice, where the epilepsy treatment gap is wide and many patients reach the psychiatrist with poorly controlled or unmonitored seizures. Inexpensive agents such as phenobarbital remain in first-line use in resource-limited settings, and video-electroencephalography to confirm the exact timing of an episode is available only in tertiary centres, so the peri-ictal placement is usually a clinical judgement made from a careful history. In this setting, getting the anticonvulsant regimen right is frequently the single most effective psychiatric intervention available, and it is reachable without the specialist infrastructure that the psychotropic-first reflex would otherwise wait for.

9.8 Putting the axis to work at the bedside

One question, asked first, reorganises the whole assessment. In practice the axis is used as a first filter. Faced with any psychiatric presentation in a patient with epilepsy, the clinician asks where it sits in relation to the last seizure and the next, and the answer routes the entire workup. A phenomenon locked to the discharge points toward the ictal syndromes, ictal fear and the ictal affective phenomena, ictal psychosis and the ictal depressive phenomena. A phenomenon hugging the fit points into the peri-ictal band, the preictal prodrome of dysphoria and irritability, postictal confusion and delirium, postictal psychosis with its lucid interval, and postictal depression, mania and anxiety. A phenomenon settled in the quiet baseline points to the interictal disorders, the interictal dysphoric disorder of epilepsy and the schizophrenia-like psychosis of epilepsy. And a mood disturbance that will not date cleanly sits, honestly, in the variable category. The accompanying figure lays the same axis out visually for revision.

Held this way, the axis earns its marks by doing several things in one move:

- It narrows a wide differential to a short list of candidate syndromes, because each position on the axis carries only a handful.
- It separates the epilepsy-unique peri-ictal states from the interictal disorders that meet ordinary psychiatric criteria, which is the split that most often decides the diagnosis.
- It fixes the first treatment move, since a peri-ictal placement points straight at optimising seizure control rather than at a psychotropic.

GOOD TO REMEMBER

Before reaching for an antidepressant, antipsychotic or anxiolytic in a patient with epilepsy, timestamp the symptom against the seizure. Where it sits, coincident with the discharge, hugging the fit, in the quiet baseline or floating free, changes the diagnosis you reach and the very first prescription you write. The timing is not background detail; it is the sort key for the whole assessment.

Ictal	Peri-ictal	Interictal	Variable
THE DISCHARGE ITSELF	AROUND THE FIT: PRODROME, POSTICTAL	THE BASELINE BETWEEN ATTACKS	MOST MOOD DISORDERS, NO FIXED TIE

Every psychiatric disturbance in epilepsy is classified first by its timing to the seizure, ictal, peri-ictal, interictal or only variably related, and that timing, not the symptom in cross-section, is what sets both the diagnosis and the first move in management.

10 · Ictal fear, ictal panic and ictal affective phenomena

The fear that opens a seizure is an examiner's favourite, and with reason. Among the peri-ictal psychiatric phenomena, the syndromes sorted by their timing relative to the discharge, ictal affect is the material that arises during the electrical event itself, and of all ictal affects the abrupt surge of dread is both the commonest and the one most readily mistaken for a primary psychiatric disorder. A candidate is expected to recognise it on sight, localise it, and lift it out from the interictal anxiety disorders that also afflict people with epilepsy. This topic takes the ictal slot on the temporal classification of peri-ictal syndromes and fills it with the affective phenomenology that lives there; it names that classifying axis but does not re-derive it, because the framework is developed as its own topic. What follows is the phenomenology of ictal fear and panic, the wider palette of ictal affect around them, and the single differential that carries most of the marks.

10.1 Ictal fear as a psychic aura

Ictal fear is fear generated by the seizure discharge itself, experienced as a **psychic aura** rather than as a reaction to anything in the patient's surroundings. Its intensity is not fixed. The same underlying mechanism yields a faint, barely nameable unease in one patient and overwhelming terror in another, so the phenomenon is best held in mind as a spectrum that runs from vague apprehension at one pole to abject fright at the other, as it is set out in Kaplan and Sadock's Comprehensive Textbook of Psychiatry. The clinically decisive fact is that this fear can constitute the whole seizure. It may occur with no motor sign, no automatism and no march into anything more obviously epileptic, which is precisely why it so often reaches a psychiatrist as an unexplained anxiety symptom long before epilepsy is entertained. The subjective report is frequently hard to put into words, and a patient may reach for figurative language, a sense of impending doom, of something terrible about to happen, of the ground giving way, none of which maps onto a real event; it is this ineffable, causeless quality that both defines the experience and marks it as arising from within rather than from the world.

Fear of this ictal type is also the commonest **affective aura** of temporal lobe epilepsy, and its defining temporal signature, described in Lishman's Organic Psychiatry, is that it wells up suddenly and without provocation. A patient who is calm and unstressed is abruptly gripped by dread that has no object and no trigger, and then it recedes of its own accord. That combination, maximal at onset, unprovoked, and self-limiting, is the phenomenological fingerprint that will later separate an ictal panic from a psychiatric one, and it repays being fixed firmly at the outset. Temporal lobe epilepsy supplies the substrate for this and for much of the affective phenomenology of seizures, but its anatomy and semiology are developed as their own topic; here it is enough to know that the fear and the temporal lobe travel together, and that a psychic aura of pure dread is a temporal-lobe event until something else is shown.

10.2 The ictal amygdala phenomenon and the fear network

The reason ictal fear binds so tightly to the temporal lobe is anatomical. The affect is manufactured by discharge in the medial temporal structures, and above all in the amygdala, the limbic node whose physiological task is the tagging of salience and threat; when a seizure recruits it, the output is fear with no frightening world to justify it. Lishman's Organic Psychiatry names this the **ictal amygdala phenomenon**, and it is the mechanistic heart of the topic. The wider structural account of the limbic system, its nuclei and its circuits, belongs to the Functional Neuroanatomy and Neurophysiology notes and is not rehearsed here; the point that matters clinically is that the fear is produced by the brain, not perceived by it, so there is no proportionate threat to be found and none should be sought. This also accounts for the sheer conviction the affect carries. Because it is generated by the same machinery that ordinarily signals real danger, the patient does not experience it as an idea about fear but as fear itself, fully embodied, which is part of why it is so distressing to endure and so persuasive to the clinician who has not yet placed it.

RULE **Sudden unprovoked fear that is the entire seizure points to the medial temporal lobe.** The commonest affective aura of temporal lobe epilepsy arises from amygdala discharge, so an isolated psychic aura of dread is, until shown otherwise, a mesial temporal event and not a mood, a nerve or a reaction to circumstance. Localisation follows from the phenomenology before any recording is made.

Intense fear, however, is not purely amygdalar. As the seizure grows, the affect maps onto a **distributed limbic network** that draws in the medial temporal lobe together with orbitofrontal cortex and the anterior cingulate, and the clinical escalation from a mild change in mood to frank terror tracks the spread of the discharge on the electroencephalogram towards the frontal regions. This yields a useful teaching image: the strength of the fear is, in effect, a readout of how much cortex the seizure has captured. It also explains why the most severe ictal fear tends to accompany the seizures that recruit the widest network rather than the briefest, most contained auras, and why a patient's worst episodes may look and feel categorically different from the mild apprehension that heralds a milder attack, even though both are the same phenomenon at different amplitudes.

10.3 Behavioural accompaniments and the paradox of preserved awareness

What the observer sees is as diagnostic as what the patient feels. When ictal fear reaches high intensity it drives a small, recognisable set of behaviours that Lishman's Organic Psychiatry sets out, and knowing them lets a clinician read the episode from the outside even when the patient cannot report it in the moment. The accompaniments cluster into three forms:

- a sudden call for help, the patient turning to or calling out to whoever is present;
- marked agitation, restless and driven rather than purposeful;
- **frightened immobility**, the patient frozen and mute, held still by terror.

The feature that catches candidates out is that through all of this awareness is usually preserved. The patient is frightened, may cry out, looks about, and can afterwards describe the experience in

detail, all while the seizure is in progress. This is the expected state of a focal event that has not yet impaired consciousness, and it is the source of a recurring clinical error, because the retained, reportable quality of the episode is read as evidence against a seizure rather than as a normal property of one.

PITFALL

Because the patient can call out, scan the room and later narrate the whole episode, clinicians conclude that it "cannot be a seizure" and reach instead for a functional or a purely anxious explanation. Preserved awareness is the expected accompaniment of this kind of focal event, not an argument against it, and treating retained consciousness as exclusionary is a well-worn route to a missed diagnosis.

10.4 Beyond fear: the wider menu of ictal affect

Fear is the loudest ictal affect but it is not the only one, and a complete account of the topic has to name the rest of the palette. At the opposite emotional pole are the pleasurable auras, transports of joy, elation and even ecstasy; these are the classical **ecstatic auras** associated with Dostoyevsky, described in Kaplan and Sadock's Comprehensive Textbook of Psychiatry, and while they are genuine seizure phenomena they are distinctly less frequent than fear. Their importance out of proportion to their rarity lies in their capacity to mislead: a patient who describes a recurring rapture, a sense of revelation or of perfect wellbeing, may never think to mention it as a symptom at all, and the clinician who does not ask will not hear of it. In the older literature such states drew attention out of proportion to their frequency because of their link to religious and mystical experience, and the practical residue of that history is a standing reminder to take an account of transcendent episodes seriously as a possible seizure rather than dismissing it as fancy or as devotion.

The unpleasant end of the palette also extends past fear. Lishman's Organic Psychiatry records that a seizure can manufacture depression and **ictal guilt**, and, rarely, anger, so the affective aura is capable of counterfeiting a fair range of primary mood states in miniature.

- pleasurable auras of joy, elation or ecstasy, less common than the fearful ones;
- ictal depression and guilt, momentary and tied to the discharge;
- ictal anger, which is rare.

Two boundaries need holding here. The depressive end of this menu, the ictal depressive phenomena proper, is developed in full alongside the ictal psychoses as its own topic and is only named here, not taught. And these ictal affects are all momentary, locked to the electrical event, and must not be confused with the sustained interictal dysphoric disorder of epilepsy, a separate, enduring mood syndrome that keeps its own company elsewhere in this chapter. The unifying feature of everything in this section is transience: an ictal affect, whether dread, ecstasy or guilt, is the seizure wearing an emotion, and it lifts as the discharge passes.

10.5 Where ictal affect sits among ictal psychiatric symptoms

The ranking is worth memorising because it predicts what the clinic actually delivers. Standard tabulations of ictal psychiatric symptomatology, such as the one in the Maudsley, arrange the phenomena by frequency, and the order is stable and high-yield. Ictal fear and **ictal panic** head the list as the most common presentation, ictal depressive symptoms come next, and ictal psychosis is rare. Read as a single hierarchy, this tells the psychiatrist that when a seizure declares itself through a psychiatric symptom, the odds overwhelmingly favour fear or panic, and it is the presentation to expect and to be ready to recognise.

Commonest	Next	Rare
ICTAL AFFECT: FEAR OR PANIC	ICTAL DEPRESSIVE SYMPTOMS	ICTAL PSYCHOSIS

Both the depressive and the psychotic ends of that ranking belong to the neighbouring topic on ictal psychosis and ictal depressive phenomena, and neither is developed here. What matters for the present topic is that fear and panic sit unambiguously at the top of the list, so that recognising an ictal affect is, most of the time, the work of recognising ictal fear. The hierarchy also carries a quiet warning against the reverse inference: the rarity of ictal psychosis does not make it impossible, and a strange, brief, stereotyped psychotic fragment in a person with epilepsy deserves the same timing question that fear does. For the trainee the corollary is a habit rather than a fact: when epilepsy and a psychiatric symptom coincide, expect fear first, and let that expectation shape the very first questions asked about the timing of the episode, its stereotypy and the company it keeps.

10.6 Ictal panic versus a primary panic disorder

This is where the marks are won and lost. The single most examined point in this topic is the separation of an ictal panic from a primary, interictal panic or anxiety disorder, because the two can feel identical to the patient and yet demand completely different management. The interictal anxiety disorders that people with epilepsy also develop are a genuine and separate problem, taken up among the anxiety disorders in epilepsy, and an ictal panic must be lifted out from among them before either is treated. The features that betray an ictal origin are its brevity, its stereotypy and its **epileptiform semiology**: an ictal panic is short and self-limiting, replays in almost the same form each time, and carries the hallmarks of a seizure, so that it may cloud consciousness or evolve into recognisable seizure activity, whereas a psychiatric panic attack runs longer, varies from episode to episode, and leaves consciousness entirely clear. The stakes of getting this wrong run in both directions. A missed ictal panic leaves a focal epilepsy untreated and exposes the patient to the hazards that unrecognised seizures carry, while an ictal panic wrongly assumed in someone who in fact has an ordinary anxiety disorder invites an unnecessary anticonvulsant and delays the psychological treatment that would actually have worked.

FEATURE	POINTS TO AN ICTAL PANIC	POINTS TO A PRIMARY ANXIETY DISORDER
Onset	abrupt, unprovoked, maximal at the start	may build, often situational or cued
Course	brief and self-limiting	typically longer and more variable
Stereotypy	highly stereotyped, recurs near-identically	content and setting vary
Consciousness	may be impaired; automatisms may follow	clear throughout
Correlate	epileptiform semiology; other seizure signs may appear	no epileptiform correlate

Features that separate an ictal panic from a primary interictal anxiety disorder; the interictal disorders themselves are developed as their own topic and are named here only for the contrast.

TRAP **Labelling a brief, stereotyped ictal panic as panic disorder and treating it psychiatrically.** Candidates anchor on the shared subjective experience and prescribe an antidepressant or refer for cognitive therapy, leaving the underlying focal seizures untreated. The stereotypy and the epileptiform features, not the feeling, decide the diagnosis, and the feeling is the one thing the two conditions genuinely share.

MNEMONIC

Four S's: Sudden, Short, Stereotyped, Semiology

When a surge of fear is **S**udden and unprovoked, **S**hort and self-limiting, **S**tereotyped from one episode to the next, and carries epileptiform **S**emiology, it is a seizure until proven otherwise. Each letter asserts only a property this topic states of ictal fear and panic.

GOOD TO REMEMBER

Any patient whose "panic attacks" are brief, arrive out of nowhere, replay in nearly the same form every time, and are followed by a patch of amnesia deserves an electroencephalogram and a neurology opinion before the label of panic disorder is allowed to settle. The cost of asking is small; the cost of missing a focal epilepsy dressed as anxiety is not.

A brief, stereotyped, unprovoked surge of fear that recurs identically, and may cloud awareness, is a seizure until proven otherwise.

10.7 Recognition, and what follows from it

Because ictal fear and panic are seizures, their management is the management of the epilepsy, and the psychiatrist's decisive contribution is diagnostic: to recognise the phenomenon, name it correctly, and route the patient appropriately rather than to reach for a psychotropic. The guideline anchors for that work are the International League Against Epilepsy and the National Institute for Health and Care Excellence epilepsy guidance, supported by the epilepsy-specific

psychiatric-management literature; none of them treats ictal fear as a mood or an anxiety disorder in its own right, and all of them place the target on the seizure.

The **LOCUS** of care is overwhelmingly the outpatient clinic, shared between psychiatry and neurology or a dedicated epilepsy service; the emergency department enters only when an aura evolves into a prolonged or repetitive seizure, or when the terror drives behaviour that threatens safety. The **focus** shifts by phase: the first target is the diagnosis itself, and thereafter the symptom that must be controlled is the seizure, so that the affect settles as seizure control improves, with immediate safety the aim in the acute event, seizure reduction over the short and mid term, and durable prevention of recurrence over the long term. The **modus** runs across every available mode. The drug mode is antiseizure medication, whose selection belongs with the antiepileptic drugs used in epilepsy and is not rehearsed here; the psychological mode is psychoeducation and reassurance that reframes the experience as a seizure rather than as madness or a heart attack, which is often profoundly relieving in itself; the procedural mode, in drug-resistant focal epilepsy, is evaluation for epilepsy surgery; and the social mode covers the driving, employment and family counselling that any focal epilepsy demands. The organising idea is that the lever which moves the fear is the one that moves the seizures.

IN INDIA

A fearful or ecstatic psychic aura is readily read through a spiritual or possession framework, and families frequently consult faith healers or temples before a physician, so the seizure origin surfaces late. Limited and unevenly distributed access to electroencephalography compounds the delay, and stigma around both epilepsy and mental illness discourages disclosure of experiences that a patient may already fear will be taken as madness. The practical lesson is to ask directly, in anyone presenting with sudden unexplained terror or with recurring rapturous experiences, whether the episodes are brief and stereotyped, and to lower the threshold for referral rather than assume a primary psychiatric or spiritual cause.

11 · Ictal psychosis and ictal depressive phenomena

When the disturbance is the seizure, the treatment is to stop the seizure. Two of the loudest psychiatric presentations in epilepsy, a florid psychosis and a profound depression, can each occur as the direct expression of an ongoing electrical discharge. When they do, they sit in the ictal slot of the peri-ictal axis, reserved for phenomena that are not merely provoked by a seizure but are the seizure, worn as a psychiatric face. This is what makes **ictal psychosis** and **ictal depression** distinct from every syndrome that follows a fit across a gap. The marks are here because the ictal versions are the most dangerous to misread: a psychosis that is actually a status is worsened by the antipsychotic reflex, and a depression that is actually a discharge can drive a patient to self-harm while the clinician waits for a mood disorder to declare itself.

These presentations belong to the temporal classification of peri-ictal psychiatric syndromes, developed as its own framework elsewhere in this chapter, and their defining feature is the one the axis makes visible: there is no interval between the discharge and the disturbance because they are the same event, which dictates a management logic closer to neurology than to

psychiatry. The account below teaches what an ictal psychosis and an ictal depression actually are, why each is missed, and the load-bearing distinction that keeps them apart from their postictal mimics. The ictal affective phenomena proper, ictal fear and ictal panic, are taken up in their own topic and are only named here.

11.1 The raw material: ictal psychic phenomena

A focal discharge can generate experience, not just movement. A focal seizure arising in or spreading through limbic and temporal cortex need not announce itself as a convulsion; it can present entirely as altered inner experience. Kaplan and Sadock's Comprehensive Textbook of Psychiatry gathers these under the heading of **ictal psychic phenomena**, the substrate from which both an ictal psychosis and an ictal depression are built.

- Mood changes, in which the affect itself is generated by the discharge rather than reflecting the patient's circumstances.
- Derealization and depersonalization, the sense that the surroundings or the self have become unreal or detached.
- **Forced thinking**, the intrusion of a stereotyped, unbidden thought the patient cannot resist and does not recognise as their own initiative.

What unites these is their origin. Each is a fragment of experience manufactured by abnormal cortical activity, which is why they arrive suddenly, feel alien, and are stereotyped from one seizure to the next. When the discharge is brief, these phenomena are the psychic auras a focal seizure can carry; when it is sustained, the same machinery produces something that looks less like a fleeting aura than like a psychiatric illness in its own right. That transition, from a momentary psychic symptom to a sustained state driven by continuous seizure activity, is exactly what an ictal psychosis is.

11.2 Ictal psychosis is nonconvulsive status epilepticus

The identity that unlocks the whole topic. The single most important thing to grasp is that ictal psychosis is not a psychosis that happens to accompany a seizure; it is a seizure that happens to look like a psychosis. Kaufman's Clinical Neurology for Psychiatrists makes the point directly: the behaviour of a **nonconvulsive status epilepticus** can be so bizarre, disorganised and psychiatric in flavour that it earns the label of ictal psychosis. There is no separate disease to diagnose. The patient is in continuous or near-continuous seizure activity that never crosses into visible convulsion, and the disturbance the clinician observes is the outward face of that ongoing discharge.

Framing it this way reorganises everything downstream. If the psychosis is the discharge, then it is concurrent with the discharge, persists for as long as the discharge persists, and resolves the moment the discharge is aborted, none of which is true of a psychiatric illness that merely coexists with epilepsy. It also explains the strangeness of the presentation: depending on where the activity sits and how it spreads, nonconvulsive status can produce anything from a fluctuating confusional state to a densely psychotic picture with disorganised behaviour. Holding ictal psychosis as an electrographic event rather than a mental illness is the conceptual move that

makes diagnosis, timing and treatment fall into place, and the one candidates most often fail to make.

11.3 How long it lasts and how it looks

Sustained, fluctuating, and cognitively disorganised. Because the disturbance is a continuing discharge rather than a passing aura, it is not measured in seconds. Kaufman's Clinical Neurology for Psychiatrists describes the aberrations of nonconvulsive status running across **hours to days**, a duration that marks the state out from an ordinary focal seizure and gives it the sustained quality that makes it resemble a primary psychiatric disorder. The picture is dominated by disorganisation. The same source characterises the state by thought disorder, impairment of language, and an **altered sensorium**. The patient is therefore muddled, disconnected and hard to reach rather than cleanly psychotic in the way a person with schizophrenia is, with awareness that waxes and wanes.

That combination is diagnostically useful. A florid but clear-sensorium psychosis in a fully alert patient sits uneasily with ictal psychosis; a fluctuating, disorganised state in which language and coherence come and go, in a person with known epilepsy, sits comfortably with it. The waxing and waning itself is a clue, because the mental state tracks the shifting intensity of the discharge. That texture is quite different from the loud, affectively coloured, clear-headed psychosis that arrives after a lucid interval in the postictal form.

MNEMONIC

BEND

The identity of ictal psychosis in four beats. **B**enzodiazepine given intravenously is the first move, aborting the status and lifting the mental state. **E**EG makes the diagnosis, because the psychosis is a discharge and nothing else. **N**o lucid interval separates the seizure from the disturbance; they are one event. **D**ischarge itself is the disorder, a nonconvulsive status wearing a psychiatric face. Each letter asserts only what this topic states.

11.4 Why it is missed: the mimics

Everything it resembles is a psychiatric or neurological emergency of its own. The danger of ictal psychosis lies less in the disorder itself, which is eminently treatable once recognised, than in the company it keeps. Kaufman's Clinical Neurology for Psychiatrists notes that it readily masquerades as several other conditions, each of which pulls the clinician toward a different and often wrong response.

- It looks like delirium, a clouded, fluctuating, disorganised state, and the reflex is to hunt for a metabolic or infective cause while the discharge goes unrecorded. Delirium in its own right, its assessment and subtypes, belongs to the Stroke, TBI, delirium and focal brain syndromes notes.
- It looks like the aftermath of traumatic brain injury, especially where a seizure has caused a fall or head injury and epilepsy coexist, so the disturbance is attributed to structural damage rather than a live electrical event.

- It looks like an acute psychotic episode, and here the error is most costly, because the instinct is to reach for an antipsychotic and admit the patient as a first psychiatric break.

The common thread is that every mimic invites a plan built for a different disease, and none includes the one investigation that settles the matter, so a delirium workup, a brain-injury attribution or a first-episode-psychosis admission can proceed for hours while the patient remains in status. The lesson is not that ictal psychosis is hard to treat but that it is easy to overlook, and the cost of overlooking it is prolonged, unrecognised seizure activity. Where a psychosis in a person with epilepsy is disorganised, fluctuating and clouded, the possibility that it is a discharge should be actively excluded.

TRAP **Treating the status as a functional psychosis.** The classic error is to see a disorganised, agitated, psychotic patient with epilepsy and start an antipsychotic, without an electroencephalogram, as though this were an acute psychiatric break. That misses the diagnosis, delays the only effective treatment, and reaches for a drug class chosen for the wrong disease. The corrective is a reflex: disorganised psychosis plus epilepsy means record the brain before you sedate the mind.

11.5 The load-bearing distinction: no lucid interval

The one line that separates ictal from postictal psychosis. If a single fact from this topic is worth carrying into an examination, it is the temporal relationship each psychosis holds to the seizure. Kaufman's Clinical Neurology for Psychiatrists and Kaplan and Sadock's Comprehensive Textbook of Psychiatry converge on the discriminator: the ictal psychosis is concurrent with the ongoing discharge and has no lucid interval at all, whereas the postictal form declares itself only after a symptom-free gap of apparent recovery. The **lucid interval** is the pivot on which the two diagnoses turn. In the ictal form the patient is disturbed continuously from within the seizure, with no window of return to clarity, because the process has never stopped. In the **postictal psychosis**, developed in full as its own topic, the seizure ends, the patient appears to recover, and only then does the psychosis erupt.

FEATURE	ICTAL PSYCHOSIS	POSTICTAL PSYCHOSIS
Relation to discharge	concurrent with ongoing discharge	after the seizure has ended
Lucid interval	**none**	present, a symptom-free gap
Underlying process	nonconvulsive status epilepticus	a seizure-driven aftermath state
Diagnosis	electroencephalogram shows the discharge	reconstructed from the timing
First-line treatment	intravenous benzodiazepine to abort the status	self-limiting; antipsychotic or benzodiazepine as needed

The ictal versus postictal psychosis distinction, the load-bearing separation in this part of the chapter; the postictal entity, its lucid interval and its numbers are taught in full in its own topic and are named here only to draw the contrast.

The distinction matters because it changes the first thing the clinician does. Read the psychosis as ictal and the priority is to record the brain and abort a status; read it as postictal and the priority

is safety and containment of a self-limiting episode. Getting the timing wrong sends the patient down the wrong pathway from the outset, which is why the discriminator should be stated before any description of symptoms.

RULE **Ictal psychosis is the discharge; postictal psychosis follows it.** The ictal form coincides with ongoing seizure activity and has no lucid interval, so it is diagnosed on the electroencephalogram and terminated by aborting the status; the postictal form arrives after a gap of apparent recovery and is a separate syndrome. Every decision in this topic descends from that one structural difference.

11.6 Ictal depression and its danger

A discharge can be low, not just frightened or elated. The affective side of the ictal slot is easy to underplay, because a brief ictal mood change can seem trivial next to a psychosis. It is not, for two reasons Kaplan and Sadock's Comprehensive Textbook of Psychiatry sets out. First, unlike most ictal phenomena that end when the seizure ends, an ictal depression can extend for **days or longer** after the seizure has passed, so the low mood outlives the discharge that generated it and comes to look like an ordinary depressive episode. Second, and this carries the weight, though it is rare an ictal depression can outlast the ictus and, at its most dangerous, drive the patient to suicide. Such a state is a genuine risk, because its severity can be out of proportion to any situational trigger and it can persist long enough to act on.

The significance is that an ictal depression breaks the comfortable assumption that ictal phenomena are always brief and self-cancelling. Most are; this one need not be. A patient whose depression is the tail of a seizure discharge may present days later with a mood state that is severe, endogenous in quality and dangerous, and the epileptic origin is easy to overlook once the seizure is out of the immediate history. Depression in epilepsy and its screening, and the general question of suicide risk in epilepsy, are each developed elsewhere in this chapter; what is owned here is that a depressive state can be an ictal event in itself and carry lethal risk while it lasts.

PITFALL

A severe low mood in the days after a seizure is easily filed as an understandable reaction to having fits, or a mild postictal dip, and its danger discounted on the grounds that seizure-related mood changes are brief. An ictal depression can persist well beyond the fit and carry a real risk of suicide. Do not let the epileptic context soften the risk assessment; a persistent, severe depressive state after a seizure deserves the same safety scrutiny as any other, and arguably more.

11.7 Management

Here the psychiatrist thinks like a neurologist. The management of ictal psychosis is unusual because the target is not the mental state but the seizure that produces it, and the governing guidance is drawn accordingly from the International League Against Epilepsy and the National Institute for Health and Care Excellence epilepsy guidelines for the seizure, together with the epilepsy-specific psychiatric-management literature for the behavioural layer. The organising claim from Kaufman's Clinical Neurology for Psychiatrists is that nonconvulsive status is diagnosed on the electroencephalogram and treated first with an intravenous benzodiazepine, a

single move that both aborts the status and awakens the patient, so diagnosis and treatment are demonstrated in the same act.

The **LOCUS** of care is not the outpatient clinic. Because ictal psychosis is a nonconvulsive status, it is a neurological emergency requiring the emergency department or an inpatient setting with electroencephalography, intravenous access and monitoring; a disorganised, clouded patient cannot be safely held in the community while a status runs unrecorded. The **focus** shifts by phase: the acute target is to confirm and abort the discharge, the short-to-mid-term target is to find what precipitated the status and prevent recurrence by optimising the antiseizure medication, and the long-term aim is durable seizure control. The **modus** is led by the pharmacological mode but aimed at the seizure: the intravenous benzodiazepine first, then optimisation of the antiepileptic regimen, whose seizure-type-led selection is a topic in its own right and is not reopened here; the procedural mode, the electroencephalogram, is both the diagnostic instrument and the measure of response. This is the sharpest contrast with the postictal psychosis, which is self-limiting and contained with an antipsychotic or benzodiazepine: in the ictal form an antipsychotic is not the answer, and the question of which antipsychotic is safe against the seizure threshold belongs to its own topic. For an ictal depression the same principle applies once the discharge is settled, but the psychological and social modes come forward, because the acute priority is protection from the suicide risk the state can carry.

IN INDIA

The diagnosis rests on capturing the discharge, yet access to electroencephalography, let alone urgent or continuous recording, is concentrated in tertiary centres. In much of Indian practice such a patient reaches a clinician who cannot immediately record the brain, and the risk is that a nonconvulsive status is labelled an acute psychosis and started on an antipsychotic. The practical safeguard is a high index of suspicion and a low threshold for a therapeutic trial of an intravenous benzodiazepine, which is widely available and inexpensive, when the psychotic state is clouded rather than clear; a rapid awakening is both treatment and diagnosis. Where the treatment gap leaves seizures poorly controlled, the sustained discharges underlying ictal states are correspondingly more common.

11.8 Placing both at the bedside

One question routes both syndromes. A clouded psychosis and a persistent depression look nothing alike, yet they are managed by the same first question: is this the seizure, or something the seizure left behind. For the psychosis, concurrent-with-the-discharge means a status to abort, and the absence of any lucid interval confirms it. For the depression, an ictal origin means a mood state that can outlast the fit and turn lethal, deserving active safety measures rather than reassurance. Both answers come from timing the disturbance against the discharge, the discipline the temporal classification of peri-ictal syndromes teaches for the whole family. The differential is short: postictal confusion and the postictal psychosis, which follow the seizure across a gap; and, for the mood side, the interictal dysphoric disorder of epilepsy and the general depression of epilepsy, which are not ictal at all. The accompanying figure locates both ictal syndromes on the peri-ictal axis for revision.

Hours to days	None	Days or longer	IV benzodiazepine
DURATION OF ICTAL PSYCHOSIS	LUCID INTERVAL IN THE ICTAL FORM	AN ICTAL DEPRESSION CAN PERSIST	FIRST-LINE, ABORTS THE STATUS

GOOD TO REMEMBER

Any prolonged, fluctuating, clouded psychotic state in a person with epilepsy should prompt an electroencephalogram to exclude a nonconvulsive status before a psychiatric diagnosis is committed to, because the treatment is to abort a seizure, not to start an antipsychotic. And a severe depression that appears or persists in the days after a fit should be taken as seriously as any other, because it can be ictal in origin and carry a real suicide risk.

Ictal psychosis is a nonconvulsive status epilepticus, concurrent with the discharge and without a lucid interval, diagnosed on the electroencephalogram and aborted with an intravenous benzodiazepine; ictal depression is the seizure's own low mood, which can outlast the fit for days or longer and, rarely, prove fatal.

12 · Preictal prodrome: dysphoria and irritability before seizures

One clean discrimination and one clinical restraint carry most of the marks here. The **preictal prodrome** is the change of mood and behaviour that gathers in the run-up to a seizure and heralds it, and its examinable identity rests on a single idea: it is defined by where it sits in time, not by any symptom peculiar to it. It occupies the leading edge of the peri-ictal band, before the ictus, and belongs to the scheme that sorts the psychiatric disturbances of epilepsy by their timing to the seizure, the framework that organises the whole peri-ictal group. Two things earn easy marks. The first is that the prodrome is told apart cleanly from the aura, with which candidates perennially confuse it. The second is that a recurrent pre-seizure dysphoria is not misread as an independent psychiatric illness and treated as one.

Both errors are common and both are avoidable. The prodrome is one of the quieter members of the peri-ictal family, easy to overlook because its symptoms are ordinary, yet it rewards attention: a mood change that keeps appearing just before the fits is a datum about the epilepsy, not a separate disorder.

12.1 What the preictal prodrome is, and where it sits

A warning that comes from the seizure, before the seizure. The prodrome is the phase of altered mood and behaviour that precedes a seizure, arising from the same destabilised brain that will shortly discharge but preceding the discharge itself. Its core, as the Maudsley Prescribing Guidelines set out in their account of pre-ictal symptoms, is a **dysphoric mood** that gathers before the attack: a darkening of affect, often with tension and a shortened fuse, that the patient or those around them come to read as a sign a fit is on the way. It sits at the pre-ictal end of the axis that classifies the psychiatric disturbances of epilepsy by their timing to the seizure, the organising framework of the whole peri-ictal band, and it is that position, rather than the flavour of the mood change, that gives it its meaning. Unlike an interictal mood disorder, which lives in

the quiet baseline between attacks, the prodrome is tethered to the seizure ahead of it, and unlike the aura, it is not part of the discharge at all.

Because it is a herald rather than a disorder in its own right, the prodrome is defined less by a checklist of symptoms than by its relationship to the event it precedes. The same low, irritable state, encountered without reference to the seizures, would be worked up as a primary mood disturbance; encountered as a recurring overture to a patient's attacks, it is a prodrome, and it lifts as the seizure it announces arrives. This is why the topic is taught, above all, as a lesson in timing.

RULE **The prodrome is defined by its timing, not its symptoms.** The same dysphoria and irritability could belong to a primary mood disorder; what makes them a prodrome is that they reliably precede the patient's seizures and lift once the fit occurs. Establish the temporal relationship first, because it, and not the symptom picture, settles both the diagnosis and the first treatment move.

12.2 The clinical picture: a dysphoric, irritable build-up

Low mood and a short temper, with a scatter of somatic complaints. The prodromal state is dominated by affective and behavioural change. Kaplan and Sadock's Comprehensive Textbook of Psychiatry lists its cardinal features as **irritability** together with depression, headache and confusion, so that the picture is not purely one of low mood but of a tense, easily provoked, mildly clouded state with a somatic edge. Lishman's Organic Psychiatry describes much the same thing as **an ill-defined prodrome** of irritability, disturbed sleep, anxiety, nausea and headache appearing in the run-up to the attack, and the word ill-defined is doing real work: the prodrome rarely presents as a clean syndrome, more as a vague, uncomfortable shift that the patient struggles to name and that a spouse or parent often notices first.

- Irritability and a lowered threshold for anger, frequently the change a family reports before the patient does.
- A depressed, dysphoric mood, sometimes coloured by anxiety and inner restlessness.
- Disturbed sleep in the nights before the attack.
- Somatic complaints, with headache and nausea prominent among them.
- A degree of confusion or mental dulling, a sense of not being quite right.

The value of holding the whole cluster in mind, rather than the low mood alone, is that it keeps the clinician from mistaking the prodrome for a straightforward depressive episode. A build-up that couples irritability with disturbed sleep, headache and a vague clouding, recurring before the seizures, has a texture of its own, and recognising it is often what first raises the possibility that the mood change is seizure-related.

MNEMONIC

Long fuse, short spark

The prodrome is the **long fuse**: a non-ictal build-up of mood and behaviour that can smoulder for a variable stretch before the fit. The aura is the **short spark**: the seizure's own brief opening symptom. Holding the pair as fuse and spark fixes the two features that separate them, their length and their nature, and asserts only what this topic states.

12.3 Beyond mood: the motor prodrome

Not every warning is affective. Although dysphoria and irritability dominate the account, the prodrome is not exclusively a mood phenomenon, and one motor form is worth knowing because it is concrete and observable where the affective prodrome is vague. Lishman's Organic Psychiatry notes that in patients prone to myoclonus a **motor prodrome** may appear, the myoclonic jerks increasing in frequency over the hours leading up to a tonic-clonic seizure, so that the escalation of jerking is itself the herald of the impending convulsion. This matters clinically for two reasons. First, it is a warning the patient can often feel and a bystander can sometimes see, more reliable than the diffuse affective shift, and it therefore lends itself to precautionary action. Second, it locates the prodrome firmly within the epilepsy rather than in a psychiatric overlay: a crescendo of myoclonus is unambiguously seizure-related, and its build-up before a generalised convulsion is a reminder that the prodromal phase reflects mounting instability in a brain moving toward discharge. The affective and the motor prodrome are two faces of the same pre-ictal process, one experienced as mood, the other expressed in movement, and both are absorbed into the seizure they precede.

12.4 The temporal signature: how long before, how long lasting

The interval is wide, and its width is the point. What most sharply characterises the prodrome is not its content but its timing, and here the sources give a usable range. The Maudsley Prescribing Guidelines place the prodromal window anywhere from **thirty minutes to two or three days** before the seizure, wide enough to cover both a brief foreboding an hour before a fit and a mood that darkens across a day or two. Kaplan and Sadock's Comprehensive Textbook of Psychiatry frames the same phenomenon operationally: the prodromal symptoms begin **at least thirty minutes before** the seizure and may run on for ten minutes to three days as a continuous disturbance rather than a momentary flicker. The two accounts agree on the essentials: the prodrome opens no sooner than about half an hour before the attack and can persist for up to a few days, which is exactly the feature that separates it from the aura at one end and from an enduring interictal mood disorder at the other. A clinician who fixes this window in mind can place a reported mood change against it: a shift falling in the half-hour to a few-days range, recurring before the fits, fits the prodrome, whereas a change lasting weeks and unrelated to the attacks does not.

30 min to 2-3 days	10 min to 3 days	under 1-2 min
PRODROME PRECEDES THE SEIZURE	HOW LONG THE SYMPTOMS LAST	THE AURA, BY CONTRAST

12.5 Prodrome versus aura: the discrimination that carries the marks

Two warnings, but only one of them is the seizure. The single most examined point in this topic is the line between the prodrome and the aura, because both precede the visible convulsion and both are experienced as a warning, yet they are fundamentally different events. Lishman's Organic Psychiatry draws the distinction crisply: the prodrome is a non-ictal warning lasting minutes to days, whereas the **aura** is the initial ictal symptom, the very first fragment of the seizure itself, and is gone within **under a minute or two**. The consequences of that difference run in every direction. The aura, being the opening of the discharge, is part of the seizure and carries localising value about where the seizure begins, a matter developed with the focal seizures elsewhere in this chapter; the prodrome, being outside the discharge, localises nothing and predicts only that a seizure is coming. The accompanying figure lays out the same contrast for revision.

FEATURE	PREICTAL PRODROME	THE AURA
Position in time	a non-ictal warning before the fit	the seizure's own first symptom, at ictal onset
Duration	minutes to days	under a minute or two
Relation to the discharge	outside the electrographic seizure	the earliest part of the electrographic seizure
Typical content	a change of mood and behaviour	the seizure's opening subjective symptom
Clinical use	a warning that permits precautions	points to the seizure focus, taught with the focal seizures

The prodrome and the aura compared on the axes that separate them: the prodrome is a non-ictal warning outside the discharge, the aura the discharge's own brief first symptom, and duration together with relation to the seizure is what tells them apart.

TRAP **Calling the pre-seizure mood change an aura.** Because both precede the convulsion, candidates collapse the two, yet the aura is itself the opening of the seizure and lasts under a minute or two, while the prodrome is non-ictal and can span minutes to days. The giveaway is duration and nature: a warning measured in hours or days that lies outside the discharge is a prodrome, never an aura.

The prodrome is recognised by its timing, not its content: a non-ictal change of mood and behaviour that precedes the seizure and settles with it, unlike the aura, which is the seizure's own brief opening symptom.

12.6 Telling the prodrome from its neighbours

Several seizure-related mood states share its cross-section. The prodrome has to be separated not only from the aura but from the other affective disturbances that cluster around the seizure, and the discriminator, once again, is timing. A dysphoria that follows the fit rather than preceding it belongs to the postictal mood disturbances, the postictal depression, mania and anxiety taught in their own right elsewhere in this chapter, while a fear or affective experience that is itself the

discharge belongs to the ictal affective phenomena. A chronic, interictal irritability and dysphoria that runs in the baseline between attacks, waxing and waning without a fixed tie to any single fit, points instead to the interictal dysphoric disorder of epilepsy, a distinct entity developed separately. The prodrome is distinguished from all of these by its position: it comes before the seizure, is relieved by it, and recurs as a herald rather than persisting as a baseline state.

Hardest of all is the separation from a primary mood disorder, because the phenomenology can be identical. What settles it is the pattern over time. A depressive or irritable state that reliably appears in the day or two before seizures and remits once they occur is prodromal, however depressive it looks in cross-section; a mood disturbance that runs independently of the fits, present in the long interictal stretches and unrelieved by seizures, is a disorder in its own right and is treated as one. The two can coexist, which is why the history must map the mood onto the seizure calendar rather than assessing it in isolation.

PITFALL

A patient reports feeling low, tense and irritable for a day or two before most of their seizures, and the reflex is to diagnose a depressive disorder and start an antidepressant. When the disturbance is time-locked to the fits and lifts with them, it is the prodrome, not an independent illness, and the lever that helps is better seizure control rather than a mood drug. Treating the prodrome as a standing mood disorder both medicates the wrong target and exposes the patient to an agent whose seizure-threshold effects are weighed in their own right elsewhere in this chapter.

12.7 Why recognising the prodrome matters

Beyond the examination, the prodrome earns its keep as a clinical tool. A reliably recognised prodrome is one of the few advance warnings epilepsy offers, and it has genuine practical value. A patient who knows that a day of irritability and broken sleep tends to precede a fit can take sensible precautions, keeping away from heights, water and driving, arranging not to be alone, and ensuring any rescue medication is to hand. Families who learn the pattern can prepare rather than be caught unawares. The prodrome is also a datum for the clinician: a change in its frequency or character can signal worsening seizure control, and its consistent appearance strengthens the case that a given mood disturbance is seizure-related rather than free-standing.

The recognition cuts the other way too, protecting the patient from over-treatment. A pre-seizure dysphoria correctly identified as prodromal is not a new depressive illness and does not call for an antidepressant; naming it for what it is spares the patient an unnecessary diagnosis and an unnecessary drug. This is why the history should ask explicitly, of both patient and family, about mood in the hours to days before the attacks, and why the answer belongs in the seizure record.

GOOD TO REMEMBER

Ask every patient with epilepsy, and a family informant, specifically about a change of mood or behaviour in the hours to days before their seizures, and record it in the seizure diary. A prodrome that the patient and family learn to recognise becomes a usable warning that allows sensible precautions, and a documented pre-seizure dysphoria that clears with the fit spares the patient a mistaken diagnosis of a primary mood disorder.

IN INDIA

In much of Indian practice the family's presenting complaint is precisely the prodrome: he becomes irritable and withdrawn a day or two before the fit. Where continuous monitoring and specialist follow-up are scarce, this family-observed change is often the only warning available, and it is clinically valuable. The risk is that the pre-seizure mood change is read as a separate psychiatric illness, or attributed to non-medical causes and taken first to a faith healer, delaying the seizure-focused care that actually helps. Naming it as part of the epilepsy, and folding it into the seizure history rather than a standalone mood assessment, keeps the treatment pointed at the fits.

12.8 Management: treat the seizures the prodrome announces

The prodrome is a peri-ictal symptom, so it follows the peri-ictal rule. Because the disturbance belongs to the orbit of the seizure, its management is not a mood-disorder algorithm but the shared peri-ictal principle: it is answered first by **optimising the antiseizure medication** rather than by a psychotropic aimed at the mood. The guidelines that anchor this are, for the epilepsy itself, those of the International League Against Epilepsy (ILAE) and of NICE, with the epilepsy-specific psychiatric-management literature covering the behavioural layer; all of them frame a recurring pre-ictal disturbance as a marker that seizure control is imperfect and to be corrected there.

The **LOCUS** of care is almost always the outpatient neurology-psychiatry interface, where the regimen is reviewed and the prodrome is logged against the seizure record; it escalates to the ward or the emergency department only in the uncommon case where the prodromal state itself carries a risk of harm or heralds a severe cluster. The **FOCUS** shifts by phase: the acute target is safety through the warning window, the short-to-mid-term target is tighter seizure control, and the long-term aim is to reduce the seizures so that their heralds recede with them, while reassessing whether any mood disturbance that persists in the quiet baseline is truly interictal and deserves its own treatment. The **MODUS** spans every mode. The drug mode leads, but is pointed at the seizures: optimising the anticonvulsant is the single most useful move, and a prodrome that shortens or disappears as control improves confirms its peri-ictal nature. The psychological mode carries much of the rest, through psychoeducation, a seizure diary and teaching the patient and family to recognise the prodrome and act on it. The procedural mode enters only within the wider epilepsy workup, for the minority whose seizures resist medication. The social mode is activity and safety planning across the warning window and family involvement in spotting it. A psychotropic is reserved for a genuinely independent, interictal mood disorder, never for the prodrome itself.

13 · Postictal confusion and postictal delirium

The commonest psychiatric sequel of a seizure is also the one most often misjudged. Almost every patient who has a convulsion is briefly muddled afterwards, and for the great majority this settles without comment. **Postictal confusion** earns its place in an examination not because it is exotic but because its ordinary, self-limiting version has to be told apart from two states that share its cross-section and are anything but benign: a seizure that is still quietly running, and a psychosis that is about to erupt. It occupies the postictal slot of the axis that sorts the psychiatric disturbances of epilepsy by their timing to the seizure, the framework that organises the whole peri-ictal band, and its diagnosis is almost entirely a matter of reading that timing correctly.

The reason the marks cluster here is that the safe-looking answer is the trap. A confused, disoriented, sometimes agitated patient in the wake of a fit invites the label "postictal" and an instruction to wait, and most of the time waiting is correct. But the same picture, when it is prolonged or atypical, can be the surface expression of continuing seizure activity, and a clouded aftermath that clears and then yields to a florid disturbance is a separate syndrome altogether. The topic is taught as much by its boundaries as by its core: what postictal confusion and postictal delirium are, how the normal aftermath unfolds and clears, how seizure type and localisation reshape it, and how to distinguish it from its look-alikes. The general syndrome of delirium, its formal assessment, its subtypes and its prevention are developed in the Stroke, TBI, delirium and focal brain syndromes notes; the concern here is strictly the peri-ictal form and where it sits beside the seizure.

13.1 What postictal confusion is

A confusional state that follows the discharge and clears on its own. Postictal confusion is the clouding of consciousness that follows a seizure while the brain re-establishes its ordinary working state, the exhausted aftermath of the electrical storm rather than a disorder in its own right. Kaplan and Sadock's Comprehensive Textbook of Psychiatry frames it as a **confusional state** lasting **minutes to hours, and occasionally days**, and that single range carries most of the clinical meaning. At the short end it is the trivial, expected grogginess that needs nothing more than time and a safe place to recover; at the long end, when the state stretches towards its outer limit, it stops being reassuring and becomes a reason to look harder for something that is prolonging it. The phenomenology is that of any acute disturbance of consciousness, here generated by the seizure itself.

- Disorientation to time and place, often with a slowed, effortful grasp of what is being asked.
- Impaired attention and a reduced level of arousal, so that the patient is drowsy, distractible or difficult to rouse fully.
- Blunted, delayed responses, with fragmentary or absent recall for the period afterwards.
- Restlessness, agitation, or aimless semi-purposeful wandering in the more disturbed cases.
- A gradual, fluctuating clearing rather than an abrupt return to normal, the hallmark of a state that is resolving.

Because the disturbance is driven by a discrete, self-terminating event, its natural course is towards recovery, and that is the single most important thing to hold. It is the everyday example of the peri-ictal category, the disturbance clustered in the immediate orbit of the fit, and it is precisely its ubiquity and usually benign course that set the diagnostic trap: the clinician who has seen it resolve a hundred times assumes it always will.

13.2 When confusion becomes delirium

The same aftermath, read at greater severity. Where the postictal state is more than transient grogginess, with a clouded sensorium, prominent disorientation, perceptual disturbance and agitation, it is spoken of as **postictal delirium**. The distinction is one of degree rather than kind: both are disturbances of consciousness generated by the seizure, on a continuum from mild, brief clouding to a fuller delirious picture. What matters for the examination is not a sharp line between the two labels but that the peri-ictal form is recognised as a variant of delirium tied by its timing to a fit, and therefore expected to clear as the postictal state resolves. The formal apparatus for delirium in general, its standardised assessment, subtypes, aetiology and prevention, belongs to the Stroke, TBI, delirium and focal brain syndromes notes and is not reopened here; this topic owns only its peri-ictal expression. Naming it delirium matters because it carries delirium's cardinal feature, a globally clouded and continuously impaired consciousness, which turns out to be the key to separating it from the syndrome with which it is most dangerously confused.

13.3 The normal arc of recovery

You cannot call an aftermath prolonged without knowing the usual one. The whole clinical judgement in this area rests on an internal template of how long recovery ordinarily takes, because "prolonged" and "atypical" are defined against that baseline. Lishman's Organic Psychiatry gives the reference figure for the commonest scenario: after a tonic-clonic seizure the patient is usually alert again within **about 15 minutes**, even though a fuller sense of being back to normal, with restored energy, concentration and mood, may not return for a day or two. The gap between those two milestones is instructive. Returning to alertness is not the same as returning to baseline: a patient oriented within a quarter of an hour may still be slowed, tired and cognitively below par for a considerably longer stretch. Holding both figures keeps the clinician from two opposite errors: expecting complete restoration too soon, and, more importantly, failing to register when even the first milestone is missed. A patient who is not clearly alert well past the expected window has departed from the normal arc, and that departure is the signal to stop waiting and start investigating.

13.4 How seizure type and localisation shape the aftermath

The aftermath is patterned, not random. The duration and quality of postictal recovery track the type of seizure and the region it arose from, and these regularities are examinable because they run against intuition: one might expect a dramatic convulsion to have the shortest tail and a subtle focal event the longest, but the opposite is closer to the truth. Lishman's Organic Psychiatry records that recovery is more protracted after a generalised tonic-clonic seizure and, strikingly, surprisingly abrupt after frontal seizures, so that a patient may switch off a frontal

seizure and be back almost at once while another remains muddled far longer after a generalised fit. Localisation within the temporal lobe matters too, with prolonged postictal confusion particularly following **right temporal focal seizures**.

SEIZURE TYPE OR FOCUS	CHARACTERISTIC POSTICTAL COURSE	SOURCE
Generalised tonic-clonic	recovery more protracted than after a focal frontal event	Lishman's Organic Psychiatry
Frontal (focal)	surprisingly abrupt recovery, often almost immediate	Lishman's Organic Psychiatry
Right temporal (focal)	the substrate for a prolonged postictal confusion	Kaplan & Sadock CTP
Temporal lobe epilepsy	slow, gradual recovery with delirium unfolding over **several minutes**; postictal headache common	Lishman's Organic Psychiatry

Postictal recovery patterned by seizure type and localisation; each row is a signature the source states, and the counter-intuitive contrast between the protracted generalised aftermath and the abrupt frontal one is the examinable point.

The value of these regularities is that they let a clinician calibrate expectation to the specific seizure rather than to a single generic template. A slow, gradual clearing with a postictal headache in someone with temporal lobe epilepsy, whose anatomy and semiology are set out in their own right elsewhere in this chapter, is an unalarming pattern for that patient; the identical duration after a frontal seizure would be out of keeping and would demand explanation. The temporal and limbic structures that generate the slow, delirious, headache-laden recovery are the same ones that bind seizure activity to mood, memory and consciousness, which is why a temporal-lobe aftermath is richer and longer than the swift reset following a frontal discharge.

MNEMONIC

Frontal fast, temporal slow

The counter-intuitive recovery gradient in one line. Recovery is **surprisingly abrupt after frontal seizures**; it is **more protracted after a generalised tonic-clonic seizure**; and in **temporal lobe epilepsy it is slow and gradual over several minutes, with a postictal headache**, prolonged confusion particularly following right temporal focal seizures. Each beat asserts only a course this topic states.

13.5 Twilight states: when the boundary blurs

Neither cleanly ictal nor cleanly postictal. Not every peri-ictal disturbance resolves into a tidy discharge followed by an aftermath. A **twilight state** is the prolonged, clouded episode that arises when ictal and postictal processes run together, so that the disturbance is a protracted period of intermixed ictal and postictal changes rather than a discharge with a clean end and a recovery beginning after it. Kaplan and Sadock's Comprehensive Textbook of Psychiatry describes exactly this blurring, and it matters for two reasons. First, it explains why the neat vocabulary of the temporal axis sometimes fails at the bedside: the electrical event does not

always have a boundary that behaviour can respect, and a twilight state is the honest name for that failure of separation. Second, a state in which seizure activity and its aftermath are intermixed and prolonged is precisely when a clinician must ask whether seizure activity is still going on, which leads directly to the most important differential in this topic.

13.6 The look-alike that must not be missed: non-convulsive status

A prolonged postictal delirium and an ongoing seizure can look identical. The highest-stakes discrimination in this topic is between a genuinely prolonged postictal delirium and continuing seizure activity that never actually stopped. Lishman's Organic Psychiatry states the difficulty plainly: a prolonged postictal delirium may be very hard to distinguish from **non-convulsive status epilepticus**, because both present as a clouded, impaired consciousness in a person with epilepsy and neither announces itself with an obvious convulsion. The clinical lever the source offers is to watch for signs that the brain is still seizing: impaired consciousness accompanied by intermittent, often subtle motor signs should push the diagnosis towards ongoing status rather than a resolving aftermath. Subtle facial or eyelid twitching, rhythmic jerks, automatisms or fluctuating responsiveness in a patient who is not clearing are the features that raise the alarm. The distinction cannot always be settled clinically, and the investigation that resolves it, an electroencephalogram capturing continuing epileptiform activity, is the arbiter; the specifics of that recording belong to the EEG discussion elsewhere in this chapter. The stakes are one-directional: a resolving delirium needs only time and protection, whereas continuing status worsens the longer it runs.

TRAP **Writing off a prolonged clouded state as "just postictal".** The classic error is to attribute a persistent, fluctuating impairment of consciousness after a seizure to a slow postictal recovery and to keep waiting, thereby missing non-convulsive status epilepticus. Clinicians make it because the ordinary postictal state is so common and so reliably benign that a prolonged one is assumed to be the same thing writ large. The corrective is a rule of thumb: when a postictal state outlasts the expected arc, especially with any intermittent motor signs, treat it as possible ongoing status until an EEG says otherwise.

IN INDIA

The postictal-delirium-versus-status distinction is exactly the point at which resource constraints bite. Urgent electroencephalography, and video-EEG in particular, is concentrated in tertiary centres and is often unavailable when the decision has to be made, so the judgement frequently rests on clinical observation of consciousness and intermittent motor signs rather than on a confirmatory recording. A large epilepsy treatment gap means poorly controlled seizures, and therefore prolonged and atypical postictal states, are seen commonly. The pragmatic posture is a low threshold for suspecting continuing seizure activity in a patient who is not clearing, arranging transfer or an EEG where feasible, and not defaulting to reassurance because the confirmatory recording is out of reach.

13.7 Continuous clouding versus the lucid interval

The second boundary is drawn by the sensorium. The other syndrome that shares this territory is postictal psychosis, and here the discriminator is not severity but the continuity of

consciousness. Postictal confusion and postictal delirium cloud consciousness continuously: the patient never returns to a clear sensorium between the fit and the disturbed state, because the disturbed state simply is the unbroken aftermath of the seizure. Postictal psychosis, by contrast, declares itself only after a **lucid interval** of clear sensorium, a window of apparent full recovery before the psychosis erupts; its lucid interval, clinical features, course and management are developed in its own dedicated topic and are not repeated here. Kaufman's Clinical Neurology and Lishman's Organic Psychiatry both hang the distinction on this single hinge. The practical test is therefore about the timeline rather than the symptoms: was there a genuine return to clarity in between? If consciousness was clouded throughout, the syndrome is the confusional or delirious aftermath; if it cleared and then broke down again, it is the postictal psychosis, while the postictal affective disturbances, postictal depression, mania and anxiety, are named and taught separately. The examinable elegance is that one property, the presence or absence of an intervening clear sensorium, sorts three neighbouring postictal syndromes at a stroke.

RULE **Clouding that never clears is the signature.** Postictal confusion and delirium impair consciousness continuously from the seizure onward, with no intervening return to a clear sensorium. A state that follows a genuine lucid interval of clarity is, by definition, no longer this syndrome, and reclassifying it correctly changes the entire management plan.

13.8 Approach and management

The management is mostly good observation with a few decisive exceptions. Care of the postictal confusional and delirious state is anchored in optimising seizure control and in excluding the dangerous mimics, for which the ILAE and NICE epilepsy guidelines set the standard of care, with the epilepsy-psychiatry literature, Lishman's Organic Psychiatry among it, guiding the disturbed state itself. The governing principle is that a self-limiting aftermath should be supported and protected rather than aggressively treated, while the two look-alikes that are not self-limiting are actively sought.

The **LOCUS** of care follows severity and duration. Most postictal confusion is managed wherever the seizure was, at home, on the ward or in the emergency department, and needs only a safe, low-stimulation environment while it clears; a prolonged, atypical or non-clearing state warrants inpatient observation with access to EEG so that non-convulsive status can be excluded, and marked agitation with a risk of harm may itself justify admission. The **focus** shifts by phase: the acute target is safety together with the active exclusion of continuing seizure activity; the short-term target is supportive containment, reorientation and prevention of injury while the state resolves; and the mid-to-long-term target is improving seizure control so that the fits, and therefore their disturbed aftermaths, become less frequent. The **modus** spans every mode. The nursing and psychological mode carries most of the work: calm reassurance, a quiet setting, protection of a wandering or resistive patient, and orientation as awareness returns. The procedural mode is the EEG that settles the status question. The pharmacological mode is used sparingly: sedation is avoided in an ordinary resolving aftermath because it deepens the very clouding one is trying to read, and is reserved for confirmed continuing seizure activity, where treatment is that of the underlying status; any antipsychotic given for postictal agitation must be

weighed against its effect on the seizure threshold, a decision handled in its own dedicated topic. The social mode is family education about what a postictal state is, how long it should last and when to seek help, which also improves the collateral history on which the whole assessment depends.

PITFALL

The acutely confused postictal patient who is restless, resistive or wandering is easily met with physical restraint and heavy sedation, both of which tend to escalate injury and agitation and cloud the picture further. The confusion is transient and the patient is not wilfully uncooperative; the correct response is a calm, low-stimulation environment, gentle redirection and protection from harm while the state clears, reserving sedation for the specific situation of confirmed ongoing seizure activity.

GOOD TO REMEMBER

When a patient does not become clearly alert within the expected window after a tonic-clonic seizure, do not simply keep waiting. A postictal state that overstays its normal arc, particularly with any intermittent motor signs, should prompt an EEG to exclude non-convulsive status, and a state that follows a genuine return to clarity should redirect you towards postictal psychosis rather than a persisting delirium.

about 15 min	a day or two	minutes to hours	several minutes
ALERT AFTER A TONIC-CLONIC FIT	UNTIL FULLY RESTORED	TYPICAL CONFUSION, OCCASIONALLY DAYS	SLOW TEMPORAL-LOBE RECOVERY

Postictal confusion and delirium are a self-limiting, continuously clouded disturbance of consciousness in the immediate aftermath of a seizure; the two states that must never be mistaken for it are non-convulsive status epilepticus, betrayed by a non-clearing state with intermittent motor signs, and postictal psychosis, betrayed by an intervening lucid interval of clear sensorium.

14 · Postictal psychosis: lucid interval, clinical features, course and management

The mark is in the timing, not the symptom. Almost every psychotic feature of **postictal psychosis** can be produced by a dozen other conditions; what fixes the diagnosis is where the episode sits in relation to the seizure. It is a brief, **self-limiting** psychosis of abrupt onset that follows a seizure, characteristically a cluster of generalised tonic-clonic fits, and it declares itself only after a deceptive symptom-free gap. The reason it earns marks is that it is common, eminently treatable, and repeatedly misread: as a primary psychotic illness that then attracts lifelong medication, as unresolved postictal clouding, or as a psychosis arising from the seizure itself. Getting the timing right changes the management plan, the drug decision and the prognosis in a single stroke.

These peri-ictal presentations are sorted along an axis defined by their temporal relationship to the seizure, from preictal through ictal to postictal and interictal, and postictal psychosis occupies the postictal slot: it arrives after the fit but only across a delay. That single fact separates it from its two nearest mimics. It is not **ictal psychosis**, in which the psychotic phenomena are the electrographic event and there is no interval at all, and it is not the persisting **postictal confusion** of a clouded, delirious aftermath, in which the patient never returns to clarity before turning psychotic. The whole diagnosis turns on the interval, and so must the answer.

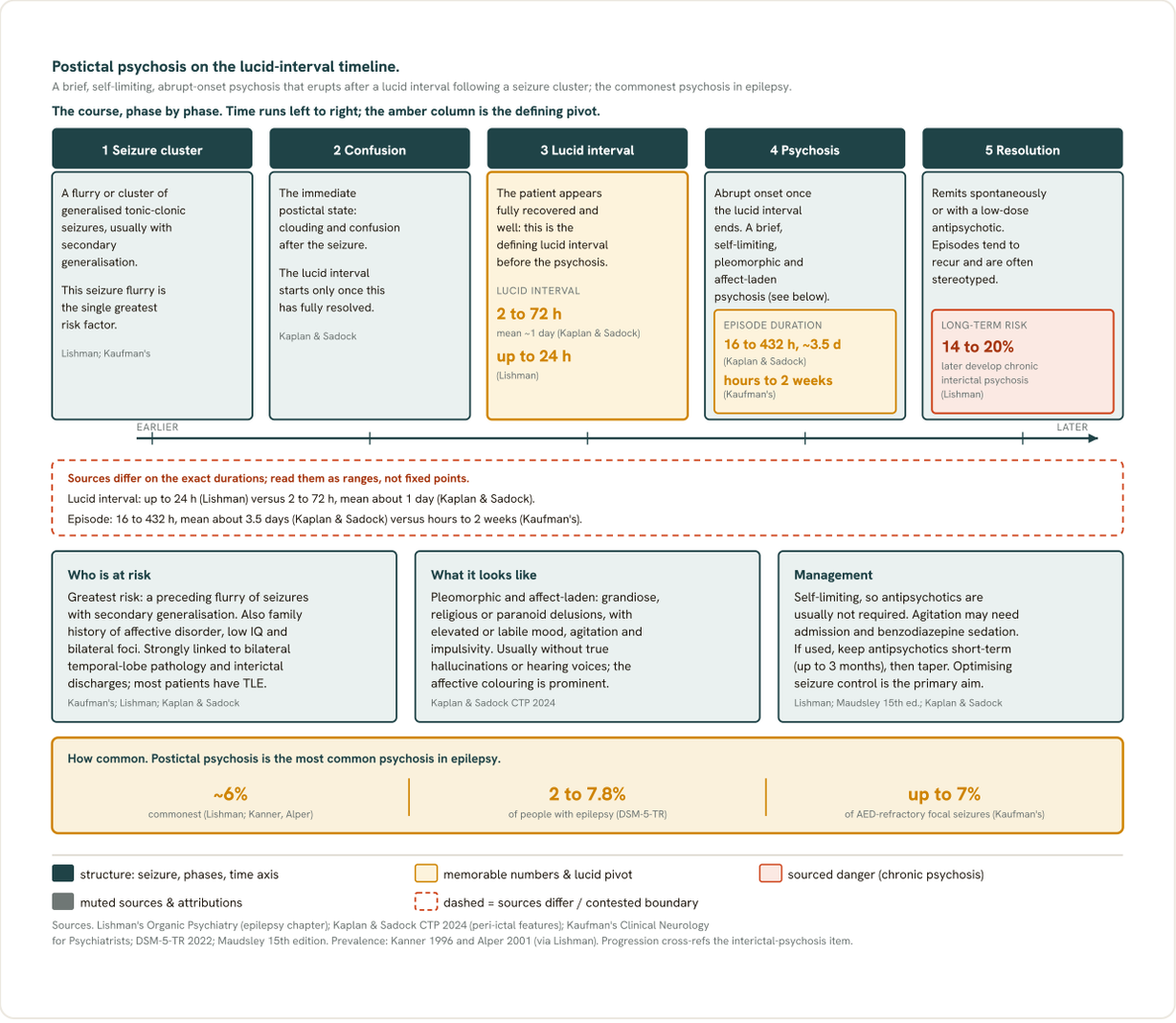


Fig 14.1 **Postictal psychosis on the lucid-interval timeline.** After a cluster of generalised tonic-clonic seizures and a brief spell of postictal confusion, the patient appears fully recovered for a defining lucid interval (2 to 72 hours, mean about 1 day in Kaplan & Sadock; up to 24 hours in Lishman) before an abrupt, brief, self-limiting and affect-laden psychosis erupts (16 to 432 hours, mean about 3.5 days; hours to 2 weeks in Kaufman's). It is the commonest psychosis in epilepsy (about 6 per cent; 2 to 7.8 per cent in DSM-5-TR), usually needs no antipsychotic, but 14 to 20 per cent later develop chronic interictal psychosis.

14.1 What postictal psychosis is

A secondary psychosis, phenotypically loud but biologically brief. Postictal psychosis is best held as a self-terminating disturbance driven by the brain state that a heavy seizure burden leaves behind, rather than as an independent psychotic disorder that merely happens to co-occur with epilepsy. Kaplan and Sadock's Comprehensive Textbook of Psychiatry and Lishman's Organic Psychiatry both describe the core picture the same way: an abrupt onset, a short course, and full

recovery, set off by seizures and usually by a run of them rather than an isolated fit. Because it is provoked by a medical event and remits when that event settles, the current classifications file it as a secondary or organic psychosis; **psychotic disorder due to another medical condition** is the DSM-5-TR home for it, and ICD-11 likewise treats the epilepsy psychoses as secondary presentations rather than as schizophrenia. Framing it this way is not pedantry. It is what justifies a short, targeted intervention and an expectation of recovery, in place of the open-ended commitment a primary psychotic diagnosis would imply. The corollary is a mechanistic one, and it is worth carrying because it explains almost every other feature of the syndrome. If the psychosis is the product of a transient, seizure-driven brain state rather than of a fixed disease process, then it should begin abruptly, run a limited course and clear as that state resolves, which is exactly what is observed. The same logic accounts for the requirement of a seizure burden, usually a cluster rather than an isolated fit, to push the brain past the threshold at which the disturbance emerges. Holding postictal psychosis as a provoked, time-limited event rather than as latent schizophrenia unmasked by epilepsy keeps the clinician from over-reading a single florid presentation.

14.2 The lucid interval: the defining feature

Recovery, then relapse. The sequence is stereotyped and is the examinable heart of the topic. The seizures end; a period of ordinary postictal confusion follows and then clears, so that the patient appears to have recovered fully; and only after that apparent recovery does the psychosis erupt. The window of apparent normality between the settling of the confusion and the onset of psychosis is the **lucid interval**. Kaplan and Sadock give it as **2 to 72 hours**, with a mean of about one day, while Lishman's Organic Psychiatry frames the same window as lasting up to 24 hours, during which the patient looks genuinely well. The interval is what makes the diagnosis feel counter-intuitive at the bedside, because the team relaxes precisely when the psychosis is about to begin. Two practical consequences follow. First, the delay means the diagnosis is frequently made in reverse, once the psychosis is under way, so the history has to be reconstructed backward to the seizure cluster and the intervening period of apparent recovery, often with a collateral informant who watched the patient look well before turning disturbed. Second, the interval is a window of opportunity as much as a diagnostic sign: a patient known to be prone to postictal psychosis, who has just had a run of fits and is currently lucid, is a patient in whom the episode can be anticipated and safety arranged before it declares itself. The gap is not empty time; it is the period in which the provoking process is still evolving toward its psychotic expression.

RULE **No lucid interval, not postictal psychosis.** The entity is defined by a symptom-free gap of hours to a few days between the seizure or cluster and the onset of psychosis. A psychosis that is continuous with the fit, or that emerges out of unbroken clouding, is a different syndrome and is managed differently.

TRAP **Collapsing the interval.** Candidates lose the mark by describing a psychosis that begins the instant the seizure stops. That erases the very feature that names the disorder and pushes the answer toward ictal psychosis or an unresolved postictal delirium. Always place the delay, and the return to lucidity, at the centre of the description.

14.3 How common it is, and the denominator problem

The commonest of the epilepsy psychoses, but the figure depends on who is counted. Postictal psychosis is the most common psychotic disorder in epilepsy, yet the reported frequency swings widely with the population sampled and the way cases are ascertained, so a single number recited without its denominator is a weak answer. The sources set out below are best read as three different questions rather than three attempts at one constant.

SOURCE	REPORTED FREQUENCY	POPULATION COUNTED
Lishman's Organic Psychiatry	about 6%	epilepsy patients in seizure-monitoring (telemetry) series
DSM-5-TR	2% to 7.8%	individuals with epilepsy
Kaufman's Clinical Neurology for Psychiatrists	**up to 7%**	drug-refractory focal seizures with impaired awareness

Reported frequency of postictal psychosis by source and denominator; the figure rises in the refractory and monitored populations, which is why the denominator must always travel with the number.

The lesson to carry into an answer is methodological. Frequencies gathered from video-telemetry units or from drug-refractory clinics are enriched for exactly the patients most prone to postictal psychosis, so they run higher than a figure drawn from an unselected epilepsy population. DSM-5-TR adds a demographic note worth holding: among older individuals the disorder is more prevalent in women. State the population, and the apparently conflicting numbers stop conflicting.

14.4 The seizure substrate and who is at risk

A bilateral, limbic disorder with a proximal trigger. Postictal psychosis is strongly associated with **bilateral temporal lobe pathology** and with bilateral, independent interictal epileptiform discharges, and most affected patients have temporal lobe epilepsy, whose anatomy and semiology are set out in their own right elsewhere in this chapter. Lishman notes that the discharges often involve the left amygdala, and, clinically important, that bilateral independent foci should be looked for and excluded before a temporal lobectomy is contemplated, because they mark a brain unlikely to be cured by removing one side; the psychiatric outcomes of epilepsy surgery are a separate topic. On this bilateral, limbic background the episode is set off by a proximal trigger, and recognising the risk profile lets you anticipate an episode before it declares itself.

- A preceding flurry or cluster of seizures with secondary generalisation, which is the **greatest single risk factor** and the usual immediate precipitant.
- A family history of affective disorder.
- Low intelligence.

- Bilateral independent seizure foci.

The cluster is doing double duty here: it is both the defining antecedent in the very definition of the syndrome and the strongest predictor that a given patient is about to develop it. A run of secondarily generalised fits in a person with bilateral temporal disease should raise the alarm before the lucid interval has even begun. The bilaterality is the thread that ties the risk profile together. A brain in which epileptiform activity is established independently on both sides, and in which the limbic structures that link seizure activity to mood and belief are implicated, is a brain more likely to generate a florid affective psychosis once a heavy seizure load is imposed on it, and less likely to be rendered seizure-free by removing a single focus. That is why the search for independent bilateral discharges is not an academic exercise but a decision that bears directly on whether surgery can help and on how closely the patient should be watched.

14.5 Clinical features: a pleomorphic, affectively coloured psychosis

Loud affect, changeable content, an affect-congruent psychosis. The picture is **pleomorphic** and carries a prominent affective colouring that is one of its most useful signatures. Kaplan and Sadock describe grandiose, religious and paranoid delusions, an elevated or labile mood, agitation and impulsivity. Hallucinations may occur, and can be prominent, but when present they tend to be affect-congruent, in keeping with the elevated or grandiose mood, rather than the sustained running commentary that marks a chronic schizophrenic illness. The mood is often high or unstable rather than flat, and the content shifts, so the cross-sectional impression can resemble mania as readily as psychosis. This affective, changeable quality, arriving abruptly after a lucid interval in a person known to have epilepsy, is what allows the syndrome to be told apart from a primary psychotic disorder; the systematic discrimination of the epilepsy psychoses from primary schizophrenia is set out in its own topic, and the phenomenology of schizophrenia itself belongs to the Schizophrenia: aetiology, phenomenology, classification and course notes. The affective loading is also why the episode is not benign in the moment. Grandiosity and religious preoccupation can carry conviction and drive action, and the mixture of elevated or unstable mood with impulsivity is the substrate for the disturbed and occasionally violent behaviour that brings these patients to attention. The content is worth taking seriously while it lasts even though the episode as a whole will pass, because the risk it generates is real during the days it runs. Reading the picture as loud but time-limited, rather than as either trivial or permanent, is the balanced clinical stance.

MNEMONIC

CLASP

The spine of postictal psychosis in five beats. **C**luster of seizures precedes it, usually secondarily generalised. **L**ucid interval before the psychosis begins. **A**ffectively coloured and pleomorphic, with grandiose, religious or paranoid delusions and any hallucinations affect-congruent. **S**elf-limiting and brief. **P**rogression, in a minority, onward to a chronic interictal psychosis. Each letter is a feature the topic actually states.

PITFALL

An abrupt, grandiose, religiously tinged psychosis with an elevated mood is easy to read as a manic episode or a first break of schizophrenia, and the reflex is to start a standard antipsychotic and plan for the long term. The affective loudness is exactly what misleads. In a patient with epilepsy who was lucid a day or two after a seizure cluster, resist the primary-psychosis label until the timing has been reconstructed.

14.6 Course and duration: self-limiting, recurrent, and the road to chronicity

Short by nature, but neither harmless nor always a single event. Left to itself the episode resolves. Kaplan and Sadock time the psychosis at **16 to 432 hours**, a mean of about 3.5 days, while Kaufman's Clinical Neurology for Psychiatrists describes episodes of hallucinations, delusions, agitation and occasional violence running from hours to two weeks. Two features of the course matter as much as the duration. First, the episodes tend to recur and are often stereotyped, so a patient who has had one is liable to have another with a similar shape, which is what makes a written relapse plan worthwhile. Second, and prognostically the sharpest point in the topic, a substantial minority do not simply recur but transform: **14% to 20%** of patients with postictal psychosis eventually develop a chronic interictal psychosis, the schizophrenia-like psychosis of epilepsy that is taught as its own entity elsewhere in this chapter. A recurring postictal psychosis is therefore not only a treatable acute problem but a marker to watch over years. The stereotypy is clinically useful in its own right: because a given patient tends to reproduce the same shape of episode, a previous presentation is a reasonable guide to the next, which lets the team recognise the pattern early and act before the disturbance peaks. The drift toward chronicity, by contrast, is the feature to fear, and it reframes what looks at first like a self-limiting nuisance into a longitudinal risk. It means that a patient with repeated postictal episodes deserves review over time rather than discharge after each one has settled, and that the goal of good seizure control is prophylactic against more than the next fit.

14.7 Management

What to actually do with the patient in front of you. Management is anchored first in optimising seizure control, for which the ILAE and NICE epilepsy guidelines set the standard of care, while the handling of the psychotic episode itself rests on the epilepsy-psychiatry literature, Lishman's Organic Psychiatry and the Maudsley Prescribing Guidelines. The organising principle is that a self-limiting condition should be treated proportionately and briefly.

The **LOCUS** of care follows the risk. The agitation, impulsivity and occasional violence of a florid episode, together with the safety concerns they carry, frequently justify inpatient admission; milder presentations can be held as outpatients with close and early follow-up, and the acutely disturbed patient may first reach an emergency setting. The **focus** shifts by phase: the acute target is agitation and behavioural risk, the short-to-mid-term reality is that the psychosis is self-limiting and needs containment rather than eradication, and the long-term aim is relapse prevention through seizure control together with vigilance for the slide into chronic psychosis. The **modus** spans every mode of treatment. Because episodes are self-limiting, an antipsychotic is

usually not required; agitation is managed with admission and benzodiazepine sedation, and where an antipsychotic is used it is given short-term, up to three months, and then tapered once the episode has resolved. Kaplan and Sadock and the Maudsley put the same point positively: the disorder remits spontaneously or responds to low-dose antipsychotics, with optimisation of seizure control as the primary aim. The choice of a particular antipsychotic against its effect on the seizure threshold is a decision handled in its own dedicated topic and is not reopened here. Alongside the drugs sit the social and procedural modes: securing the patient, educating the family, writing a relapse plan that exploits the stereotyped recurrence, and, at the level of the epilepsy itself, tightening antiepileptic treatment as the durable lever.

IN INDIA

Two realities shape this in Indian practice. The epilepsy treatment gap is large, so poorly controlled seizures and the clusters that trigger postictal psychosis are common, and the lever of seizure-control optimisation is often the single most useful thing to improve. Video-EEG telemetry is not widely available outside tertiary centres, so the diagnosis usually has to be made clinically, from a careful history of a seizure cluster followed by a lucid interval and then psychosis, rather than confirmed on a monitoring unit. The pragmatic acute plan, admission for safety with benzodiazepine sedation of agitation and only brief, cautious antipsychotic use, fits resource-limited settings well and avoids committing a patient with a self-limiting disorder to indefinite treatment.

GOOD TO REMEMBER

When a person with epilepsy becomes psychotic a day or two after a cluster of seizures, having clearly been lucid in between, think postictal psychosis first. Admit if there is agitation or risk, sedate with a benzodiazepine, optimise seizure control, use any antipsychotic at low dose and for a short time, and do not reflexively begin a lifelong antipsychotic for what is, by nature, a self-limiting episode.

14.8 Telling it apart from its neighbours

The same axis that classifies it also distinguishes it. Because postictal psychosis is defined by its position after a lucid interval, the cleanest way to separate it from the conditions it is confused with is to walk the timeline. It is not the psychosis that coincides with the electrographic seizure and has no interval, which is the ictal form. It is not the clouded, disoriented aftermath that never clears into lucidity, which is postictal confusion and delirium, held as its own peri-ictal syndrome. And it is not the persistent, hallucination-rich psychosis that runs on between seizures independent of them, which is the chronic interictal or schizophrenia-like psychosis, the very endpoint that a minority of postictal cases eventually reach. The formal separation of any of these from a primary schizophrenic illness, resting on preserved affect, intact premorbid function and the tight relation to seizures, is developed in the dedicated discrimination topic, and the features of primary schizophrenia sit in the Schizophrenia: aetiology, phenomenology, classification and course notes. Keep the axis in mind and the four pictures stop blurring into one another.

2 to 72 h	16 to 432 h	about 6%	14 to 20%
LUCID INTERVAL BEFORE ONSET	EPISODE DURATION	COMMONEST EPILEPSY PSYCHOSIS	PROGRESS TO CHRONIC PSYCHOSIS

Postictal psychosis is a brief, self-limiting, affectively coloured psychosis that erupts after a lucid interval of hours to a few days following a cluster of seizures; it is usually managed with containment and short-term treatment rather than long-term antipsychotics, but a minority go on to a chronic interictal psychosis.

15 · Postictal depression, postictal mania and postictal anxiety

The trap is to treat the mood when the task is to treat the seizure. When a person with epilepsy turns low, anxious or, far less often, elated in the days after a fit, the cross-sectional picture can be indistinguishable from a primary mood or anxiety disorder, and the reflex to reach for an antidepressant is strong. What separates these postictal affective disturbances from the free-standing illnesses they imitate is not their phenomenology but their position in time: they arrive tethered to the seizure, and the depressive ones in particular behave quite differently under treatment. The marks are earned by holding two facts together - that mood and anxiety symptoms clustered after a seizure are common and real, and that the treatment logic for them is inverted relative to what the symptoms alone would suggest.

These presentations are located on the axis that sorts every peri-ictal disturbance by its relationship to the seizure, from preictal through ictal to postictal and interictal. That classifying framework is developed as its own topic and is borrowed here only to place the syndromes at hand; all occupy the postictal slot, the disturbed brain state that follows the discharge. This account works through the three named members in turn - the common depressive picture, the sparser anxiety one and the rare manic one - then turns to the lateralisation question, the postictal flare of a settled interictal disorder, and the management principle that gives the topic its examination edge.

15.1 The postictal window: when these symptoms appear

Fix the timing first, because the timing is the diagnosis. The defining fact about this group is that the symptoms sit in a band after the seizure rather than floating free of it. The Maudsley Prescribing Guidelines place the postictal psychiatric symptoms - depression, anxiety, suicidal ideation and psychosis together - in a window of **several hours to 7 days** after the fit. That window does two things. It tells you where to look, so that a new low mood or a surge of anxiety in the days after a seizure should prompt the timing question before a prescription; and it tells you what company these symptoms keep, since they travel as a recognisable postictal cluster rather than as isolated events. Two members of that cluster are taught in full elsewhere and are only named here: the suicidal ideation that belongs to the account of suicide risk in epilepsy, and the postictal psychosis that declares itself after a lucid interval. The window also warns against symmetry: a disturbance that begins during the discharge is ictal, and one that runs on independently in the baseline is interictal, so a mood change dated to the days after a fit is what earns the postictal label.

15.2 Postictal depression: the common one

The everyday member of the group, and the one most often misattributed. Of the three affective pictures, **postictal depression** is the one the psychiatrist will actually meet. Kaplan and Sadock's Comprehensive Textbook of Psychiatry describes it as common and makes the clinically important point that a prolonged depressive state may follow focal impaired-awareness seizures even when the seizure itself carried no depressive content. That is the subtle observation worth carrying: it is tempting to expect a depressive aftermath only from a seizure with an unpleasant affective aura, but the low state can settle in after a fit that was, in its own experience, affectively neutral. The depression is a property of the postictal brain state rather than a carry-over of the ictal mood, and can outlast the ordinary confusion of the immediate aftermath. The label must be handled with discipline, because two other depressions in epilepsy are easily confused with it and owned by their own topics: the ictal depressive phenomena that are the psychiatric face of the discharge, and the interictal depression of epilepsy, whose prevalence, phenomenology and screening are set out separately and which behaves very differently under treatment. Naming the postictal form precisely changes the expectation of course and response: it is a self-limiting state locked to a recent seizure, not a free-standing depressive episode.

15.3 Postictal anxiety: real but thinly described

Named in the cluster, but the evidence is sparse. **Postictal anxiety** is reported as part of the postictal symptom cluster, alongside the depressive, suicidal and psychotic elements that share the same window. Beyond that listing the standard corpus says little: its phenomenology, frequency and natural history are not worked out to anything like the depth of the depressive picture, and an honest answer acknowledges that thinness rather than papering over it. What can be said with confidence is where postictal anxiety sits relative to its neighbours. It is not the brief, stereotyped ictal fear or ictal panic that is the discharge itself, taken up among the ictal affective phenomena, and it is not one of the free-standing anxiety disorders of epilepsy that run in the interictal baseline and are treated in their own right. It is a postictal phenomenon: an anxious colouring to the aftermath of a seizure, dated to the same hours-to-days band as the rest of the cluster. The consequence is modest. Recognise postictal anxiety as a legitimate member of the group, keep it separate from ictal panic and from an interictal anxiety disorder on grounds of timing, and resist importing a management or prognosis the literature has not established.

15.4 Postictal and epilepsy-related mania: the rare one

The exception, and examinable precisely because it is rare. If depression is the rule, **postictal mania** is the exception. Kaplan and Sadock's Comprehensive Textbook of Psychiatry records that manic or mixed features are much rarer than depressive features in the **mood disorder due to epilepsy** picture, so an elevated or expansive postictal mood is a genuine but uncommon event. Its rarity is the very thing that makes it a favourite discriminator, because a manic presentation in a person with epilepsy pulls the differential in several directions at once. The timing of its emergence is itself informative and slightly paradoxical: the same source notes that manic symptoms may rarely emerge either with an increase in seizure frequency or, conversely, after seizures come under control. The second route is the counter-intuitive one and deserves care,

because a mood disturbance surfacing as the fits are suppressed echoes the pattern named as forced normalisation (the Landolt phenomenon) and alternative psychosis, which is developed as its own topic and only pointed to here. The lesson is that neither a worsening nor an improvement in seizure control excludes an elevated mood; the manic picture, though rare, sits at both ends of the seizure-control spectrum rather than one predictable point.

MNEMONIC

Common down, rare up, date it to the fit

The postictal affective triad in one line. **Down** is common: postictal depression is the member you will actually see. **Up** is rare: manic or mixed features are far less frequent than depressive ones. **Anxiety** travels in the same cluster. And every one of them is **dated to the fit**, appearing in the window of hours to days that gives the group its name. Each beat asserts only what this topic states.

15.5 The lateralisation question in postictal mania

A tentative anatomy, offered with its uncertainty intact. When mania does arise in epilepsy, the natural question is whether it maps onto a particular seizure focus, and the honest answer is qualified. Kaplan and Sadock's Comprehensive Textbook of Psychiatry names a **right temporal focus** as a possible source of mania in epilepsy while being explicit that the association is not well established. That qualification is not a footnote to be dropped; it is the substance of the point. The lateralisation fits a broader intuition that right-sided limbic disturbance tends toward elevated or disinhibited states and left-sided toward the depressive, but the epilepsy data supporting it are thin. The temporal lobe epilepsy that provides the substrate has its anatomy and semiology set out in their own topic and is not re-taught. What this fragment owns is the narrow, hedged proposition itself: a right temporal focus is a candidate source of epilepsy-related mania, no more firmly than that. Carrying the uncertainty forward is what separates a good answer from an overconfident one.

TRAP **Stating the right-temporal link as established fact.** Candidates read that mania in epilepsy has been associated with a right temporal focus and then present it as a firm localising rule. The source flags the association as not well established, and the mark is in reproducing that hedge. Assert the lateralisation as possible and unconfirmed, not as a law.

15.6 Postictal breakthrough of a settled interictal disorder

A separate mechanism hiding inside the same time window. Not every postictal disturbance is a fresh, self-contained syndrome. The Maudsley Prescribing Guidelines note that patients who carry an interictal psychiatric disorder may experience a postictal worsening of previously-remitted symptoms, best held as **breakthrough symptoms** rather than as a new illness. The distinction is mechanistically real and clinically consequential. In one patient the postictal state generates a de novo depressive or anxious picture where there was no standing disorder; in another, the same state briefly reactivates a mood or anxiety disorder that had been in remission, so that what looks like a relapse is really a transient, seizure-triggered flare on the back of a recent fit. Reading the second as the first is where clinicians go wrong, and the error has a direction: it

tends toward over-treatment, because a brief reactivation of a settled disorder is easily mistaken for a durable relapse that then attracts a permanent escalation of maintenance medication. The corrective is to date the worsening to the seizure and watch it settle as the postictal state resolves, rather than to rewrite the long-term plan on the strength of a few post-seizure days. Breakthrough symptoms are the postictal window acting on an already-vulnerable brain, and belong in the differential of any apparent relapse that follows a fit.

PITFALL

A patient whose depression or anxiety had settled becomes symptomatic for a few days after a seizure, and the team reads it as a relapse of the underlying disorder and steps up the maintenance antidepressant or adds an agent for the long term. If the worsening is dated to the fit and clears as the postictal state resolves, it is a breakthrough phenomenon, not a true relapse, and a permanent change of regimen is the wrong response to a transient, seizure-driven flare.

POSTICTAL AFFECTIVE SYNDROME	FREQUENCY	CHARACTERISTIC RELATION TO THE SEIZURE	THE POINT THAT EARNS THE MARK
Postictal depression	common	may be prolonged, and can follow focal impaired-awareness seizures even when the ictus was not depressive	does not respond to antidepressants; the lever is seizure control
Postictal anxiety	reported in the cluster	shares the postictal window with depression, suicidal ideation and psychosis	corpus detail is sparse; separate it from ictal panic and from an interictal anxiety disorder
Postictal / epilepsy-related mania	rare	emerges rarely with rising seizure frequency, or after seizure control	a right temporal focus is a possible but unconfirmed source

The three postictal affective syndromes compared on frequency, relation to the seizure and examinable point; the suicidal and psychotic members of the same cluster are developed in their own topics and named here only to locate them.

15.7 Management: the antidepressant paradox

Here the topic pays off, because the obvious treatment is the wrong one. The governing principle of this group is a negative one, and it is the single most examinable fact in the fragment. The Maudsley Prescribing Guidelines state that peri-ictal depressive symptoms do not appear to respond to antidepressants and are managed instead by optimising the antiseizure medication. This inverts the instinct that a depressive picture calls for an antidepressant, and it draws the line between this topic and the interictal depression of epilepsy, set out in its own account, which does respond to antidepressant treatment. The same contrast holds against the interictal dysphoric disorder of epilepsy, the chronic baseline mood picture taught as its own entity, which likewise answers to an antidepressant. A **peri-ictal depression** tied to recent seizures is the

exception, and treating it as an ordinary depression wastes weeks on a drug that will not work while the real lever goes untouched.

The structure of care follows from that principle. The **LOCUS** of management is overwhelmingly the outpatient epilepsy or neuropsychiatry clinic, where seizure control is reviewed and adjusted; inpatient care is reserved for the few in whom postictal risk, particularly the suicidal ideation that shares the cluster, is acute, and the emergency department enters only when safety cannot be held otherwise. The **focus** shifts by phase: the acute target is safety and containment across the few postictal days, the short-to-mid-term aim is to let a self-limiting state resolve without committing the patient to a mood-specific drug the evidence says will not help, and the long-term aim is to reduce the seizure burden that keeps generating the disturbances. The **modus** is weighted away from psychotropic prescribing and toward the epilepsy itself: the primary mode is optimisation of the antiseizure regimen, supported by educating patient and family that these symptoms are seizure-driven and time-limited, with psychological support and monitoring across the postictal window rather than a reflexive antidepressant. The guideline anchor is the epilepsy standard of care set by the ILAE and NICE, within which seizure optimisation is defined, while the handling of the psychiatric symptoms rests on the epilepsy-specific psychiatric-management literature rather than the ordinary mood-disorder algorithms.

RULE **Peri-ictal depressive symptoms are treated by optimising seizure control, not by antidepressants.** They do not appear to respond to antidepressant drugs and are managed instead by optimising the antiseizure medication. This is the exact reverse of the interictal depression of epilepsy, and the reversal is the whole clinical point.

IN INDIA

The treatment gap in Indian epilepsy care means that poorly controlled seizures, and the postictal mood and anxiety disturbances they generate, are common in ordinary practice. That reality aligns with the management principle rather than fighting it: because the response to peri-ictal depression is to optimise seizure control rather than add an antidepressant, the most useful and affordable intervention is often better antiseizure treatment with the medicines already in use, not a new psychotropic. Where telemetry is unavailable the timing must be reconstructed from a careful history - a fit, then low or anxious mood over the following days - so a seizure-driven picture is not mislabelled as a primary depression and started on a drug that will not touch it.

GOOD TO REMEMBER

When a person with epilepsy becomes depressed or anxious in the days after a seizure, date the symptoms to the fit before you prescribe. If they sit in the postictal window, the first move is to review and optimise seizure control, not to start an antidepressant, and to watch a self-limiting state settle. Keep the suicidal-ideation risk of the postictal cluster in mind while you wait.

15.8 Holding the group apart from its neighbours

The same timing axis that groups these syndromes also separates them from their mimics. Because the postictal affective disturbances are defined by their band after the seizure, the

cleanest separation is to walk the timeline. They are not the ictal affective phenomena, the fear, panic or low mood that is the discharge itself and clears when the seizure ends. They are not the interictal mood and anxiety disorders of epilepsy, which run in the baseline between attacks and, crucially, do respond to the usual treatments; the interictal depression of epilepsy and the interictal dysphoric disorder both sit on that side of the line. And within the postictal band, the mood and anxiety members must be kept apart from postictal confusion and delirium, a disturbance of arousal and orientation rather than mood, and from the postictal psychosis that emerges after a lucid interval, each a peri-ictal syndrome in its own right. The discipline is the same: establish where the disturbance sits relative to the seizure, and let that placement, not the surface symptom, decide both the label and the first treatment move.

- Depressive, anxious and, rarely, manic in colour, all appearing in the postictal window rather than during the discharge or in the baseline.
- Set apart from the ictal affective phenomena by the interval after the seizure, and from the interictal mood and anxiety disorders by their tight tie to a recent fit.
- Set apart from postictal confusion and delirium, which disturb arousal and orientation, and from postictal psychosis, which follows a lucid interval.
- Managed, in the depressive case, by optimising seizure control rather than antidepressants, the reverse of the interictal picture.

Common	Rare	Up to 7 days	No AD response
POSTICTAL DEPRESSION	POSTICTAL / EPILEPSY MANIA	POSTICTAL SYMPTOM WINDOW	PERI-ICTAL DEPRESSION

Postictal depression, anxiety and the rare postictal mania appear in a window of hours to a week after a seizure; the depressive picture is common and, unlike interictal depression, does not respond to antidepressants but to optimisation of seizure control, while manic or mixed features are rare and only tentatively linked to a right temporal focus.

16 · Interictal dysphoric disorder of epilepsy

The commonest way mood disorder shows itself in epilepsy is not the one candidates are trained to look for. Faced with a person who has epilepsy and is clearly unwell in mood, the reflex is to reach for the template of a major depressive episode and test the history against it. That template usually fails, because the disturbance most such patients carry is a chronic, shifting, irritable low mood that never resolves into a clean depressive episode and never fully lifts either. Blumer gave this picture a name and a place of its own, and thought it central enough to call it **the major psychiatric disorder of epilepsy**. It earns marks precisely because it is easy to miss: it hides behind a search for classic depression, borrows features from the anxiety and even the psychotic disorders, and responds well to treatment once it is recognised for what it is.

Everything about the syndrome follows from where it sits in time. The psychiatric disturbances of epilepsy are sorted along an axis running from before the seizure, through the discharge, to its

aftermath and finally to the quiet baseline between attacks; that classification is developed in its own right elsewhere in this chapter and only borrowed here. Interictal dysphoric disorder belongs to the last position. It is an interictal disorder that runs independently of the timing of seizures, which separates it from the peri-ictal mood changes, the preictal prodrome of dysphoria and irritability and the postictal depression, mania and anxiety, that keep time with the fit and are taught as their own topics. The mood change here neither dates to the last seizure nor predicts the next; it occupies the background of the illness, not its edges.

16.1 The disorder and its two names

One entity under two labels, and a deliberate claim about its weight. The syndrome entered psychiatry through Blumer's account, published in **1991**, of a mood disturbance specific to epilepsy that did not fit the depressive categories then in use; he named it the **interictal dysphoric disorder**. More than a decade later, in **2003**, **Kanner** described the same picture as the **dysthymia-like disorder of epilepsy**, a label that foregrounds its resemblance to a chronic low-grade depression rather than to an acute episode. Both names point at one referent and both are worth carrying, because an examiner may use either. Blumer went further than merely naming it. Reviewing the psychiatric burden of epilepsy in Kaplan and Sadock's Comprehensive Textbook of Psychiatry, he characterised this condition as the major psychiatric disorder of the illness, a phrase that stakes a claim: this chronic, pleomorphic dysphoria is the one the epilepsy clinician meets most often and should know best. It is a clinically described syndrome rather than a category with its own operational criteria in the standard manuals, which is part of why it slips past a diagnostic process built around them. Naming it correctly is therefore the first clinical act, because a disorder with no line in the checklist is a disorder that goes untreated.

16.2 Interictal by definition: why the timing decides everything

The name locates the disorder before it describes it. The word interictal is doing diagnostic work. A peri-ictal mood change announces its own origin by its timing, so a dysphoria clustering in the hours around a fit is close to self-explanatory. This disorder does the opposite. Both Lishman's Organic Psychiatry and Kaplan and Sadock's Comprehensive Textbook of Psychiatry place it in the interictal baseline, arising independently of seizure timing rather than as a peri-ictal phenomenon, so it neither dates to the last seizure nor forecasts the next. That has two consequences the axis is designed to make obvious.

- It must be assessed as a mood disorder in its own right, with a full mood and risk history, rather than explained away as a transient reaction to the last seizure.
- Its treatment is aimed at the mood disorder itself and does not follow automatically from better seizure control, which is the primary lever for the peri-ictal syndromes but not for this one.

Placed rightly, the disorder is held as both truths at once: a genuine, treatable mood disorder that nonetheless belongs to the epilepsy, so that it is neither dismissed as a passing aftermath of the fits nor cut loose from its specific tie to the illness.

RULE **The disorder keeps no appointment with the seizure.** Interictal dysphoric disorder runs in the baseline state and does not track the fit, so it is diagnosed and treated as a mood disorder in its own right, with the epilepsy as vulnerability and context rather than as the proximate trigger. A dysphoria that clearly dates to the hours around a seizure is a peri-ictal syndrome and belongs to a different topic.

16.3 The core picture: a chronic dysthymia broken by brief normal mood

Chronic, but never steady. Lishman's Organic Psychiatry gives the central shape of the syndrome plainly: a **chronic dysthymia** interrupted at frequent intervals by brief periods of normal mood. Two words carry the diagnosis. It is chronic, so the low mood is the background state of the illness, present far more often than not and measured over months and years, not as an episode with a beginning and an end. And it is interrupted, so the depression is not sustained: short intervals of feeling well break it up, and those windows make the course intermittent and labile rather than a single depressive episode. The clinical lesson is that the disorder is defined as much by its rhythm as by its content. A patient seen in a good-mood window can look euthymic, and an assessment that lands in a normal-mood window will underestimate or miss the disorder. The waxing and waning is intrinsic and must not be read as a partial treatment response or a stable recovery. This fluctuating, low-grade, chronic quality is precisely why it does not answer to the description of a classic major depressive episode, whose prevalence, phenomenology and screening in epilepsy are set out as their own topic elsewhere in this chapter.

16.4 Paroxysmal irritability and agitation

Not sadness alone: the irritable, agitated edge. Kaplan and Sadock's Comprehensive Textbook of Psychiatry emphasises that the disorder is closely associated with **paroxysmal irritability** and agitation, and this is one of the features that pulls it furthest from ordinary depression. The irritability is not a steady, low-level bad temper running through the illness; it comes in paroxysms, in bursts that are sudden, disproportionate to their trigger and short-lived, with agitation alongside. The irritable component is often what the family reports, rather than the low mood the patient may never volunteer, so the complaint can be friction at home rather than sadness. It also feeds the diagnostic confusion the disorder is notorious for: the irritability can be read as a personality problem or as behavioural disturbance, each reading missing the mood disorder underneath. The irritability belongs to the dysphoric disorder as one facet of a single condition, not a separate diagnosis bolted on. Sustained interictal irritability, aggression and episodic dyscontrol in epilepsy form a behavioural syndrome with its own dedicated topic; here the irritability is one colour of the mood disturbance, arriving in paroxysms against the chronic dysthymic background rather than as a fixed trait.

16.5 The reach toward psychosis: paranoia and hallucinations

The upper border of the syndrome touches psychosis. At its more severe pole the disorder does not stay purely affective. Kaplan and Sadock's Comprehensive Textbook of Psychiatry notes that it may carry accompanying **paranoia and hallucinations**, which places it on a continuum with the psychotic disorders rather than in a sealed mood compartment. Mood and psychosis in epilepsy are not cleanly separable, and the same patient's dysphoria can shade into paranoid

ideas and perceptual disturbance without becoming a different illness. The continuum explains why a dysphoric epilepsy patient can present with a paranoid, quasi-psychotic colouring that would baffle anyone holding the disorder to be a simple depression. It should not, however, be over-read. Some paranoia or hallucination at the severe end of the dysphoric spectrum does not convert the picture into a primary psychotic illness, and the chronic interictal psychosis of epilepsy, the schizophrenia-like psychosis, is a separate entity taught in its own right. The task is to recognise the psychotic tinge as the far edge of the mood disorder while keeping the frank, persistent psychoses of epilepsy conceptually distinct.

PITFALL

When the dysphoria arrives with paranoid ideas or a hallucination, the reflex is to read it as a primary psychosis and start an antipsychotic as the main treatment. That inverts the therapeutic logic. The condition is fundamentally a mood disorder that responds to antidepressant treatment, with a low-dose neuroleptic reserved as an addition where the psychotic colouring is prominent, not as the first or the sole agent. Reaching for antipsychotic monotherapy leaves the mood core untreated.

MNEMONIC

DIP

The three domains of the disorder held in one word. **D**ysthymia that is chronic and broken only by brief windows of normal mood. **I**rritability, paroxysmal, with agitation. **P**aranoia and hallucinations at the severe pole, marking the continuum with psychosis. Each letter is a feature the disorder actually shows.

16.6 Reading the domains together: a pleomorphic disorder

Several kinds of symptom, one condition. The reason interictal dysphoric disorder resists the single-episode template is that it spreads across more than one symptom domain at once. It is depressive, in its chronic, fluctuating low mood; irritable and agitated, in the paroxysms that punctuate that background; and psychotic at its edge, where paranoia and hallucinations can appear. This pleomorphism is not vagueness but the defining texture of the syndrome, and it is exactly what a checklist built for a discrete depressive episode cannot capture. The accompanying figure lays the domains out together; the table below pairs each with its character and the misreading it invites.

SYMPTOM DOMAIN	CHARACTER IN THE DISORDER	HOW IT IS MISREAD
Depressive	chronic dysthymia broken by brief windows of normal mood	a good-mood window is mistaken for recovery or a stable baseline
Irritable	paroxysmal irritability with agitation	read as a personality trait or isolated behavioural disturbance
Psychotic-adjacent	possible paranoia and hallucinations	mistaken for a primary psychotic illness

The domains of interictal dysphoric disorder, each with the form it takes in the syndrome and the diagnostic error it invites; the disorder is a single, pleomorphic condition, not three separate diagnoses.

Filed separately, the low mood becomes a depression, the irritability a personality issue and the paranoia an unrelated psychosis, and no one treats the syndrome that unites them.

16.7 The commonest face of mood disorder in epilepsy

This, not the classic episode, is what you will meet most. The single fact that gives the topic its clinical weight is one of base rates. Kaplan and Sadock's Comprehensive Textbook of Psychiatry states that of patients who have both epilepsy and a mood disorder, most carry this dysthymia-like, interictal picture rather than a classic major depressive disorder. In the general population the prototype of a mood disorder is the depressive episode, but in the epilepsy clinic the prototype is this chronic, fluctuating, irritable dysphoria, and the clean depressive episode is the less typical presentation. The consequence is unforgiving: screening only with major-depressive-episode criteria systematically under-detects the very disorder most likely to be present. The prevalence, phenomenology and screening of depression in epilepsy are developed as their own topic; the point here is the epidemiological weighting, that when mood disorder appears in epilepsy it wears this face more often than the textbook one, so the assessment must catch a pattern rather than confirm an episode.

TRAP **Screening only for the classic depressive episode.** Candidates lose the mark, and clinicians miss the diagnosis, by testing the epilepsy patient against major depressive disorder, finding no clean episode, and concluding the mood is normal. The commonest mood presentation in epilepsy is this dysthymia-like disorder, which will not satisfy those criteria. Look for a chronic, intermittent, irritable dysphoria, not a discrete episode.

16.8 The seizure substrate and localisation

An association with frequent focal seizures, tentatively lateralised. The disorder is not evenly distributed across everyone with epilepsy. Kaplan and Sadock's Comprehensive Textbook of Psychiatry links interictal dysphoria with **frequent focal impaired-awareness seizures**, and, more tentatively, with a possible preponderance of left temporal foci, while being explicit that the lateralisation is not well established. Both halves of that claim matter, and the second must keep its caution. The association fits the wider impression that a heavier, poorly controlled seizure burden of this type carries a greater psychiatric load; the distinction between focal aware and focal impaired-awareness seizures, and the anatomy and semiology of temporal lobe epilepsy behind it, are set out in their own topics and only named here. The suggestion of a left temporal preponderance is offered as a possibility rather than a fact, and should be stated that way in an answer; asserting a firm lateralisation overstates what the source supports. The neurobiology linking the limbic structures of the temporal lobe to mood and behaviour is the chapter's mechanism topic, and it supplies the plausible substrate: an interictal disorder commoner where focal impaired-awareness seizures are frequent fits a mood disturbance generated by chronically disordered limbic function, even where the side of the focus stays uncertain.

16.9 Management

The recognition is most of the battle, but the treatment is genuinely rewarding. The management question is anchored, for the epilepsy itself, in the International League Against Epilepsy and the National Institute for Health and Care Excellence epilepsy guidelines, and, for the mood disorder, in the epilepsy-specific psychiatric-management literature described by Kaplan and Sadock's Comprehensive Textbook of Psychiatry and Lishman's Organic Psychiatry. Encouragingly, this is a treatable disorder that, once named, usually responds well.

The **LOCUS** of care is, for the great majority, the outpatient clinic, where a chronic and fluctuating mood disorder is best followed over time; it escalates to inpatient care only where severity, prominent paranoia or hallucinations, or risk make it necessary, and an acutely agitated or psychotic presentation may reach an emergency setting. The **focus** shifts by phase: the acute target is the dysphoria, the paroxysmal irritability and any distressing psychotic colouring; the mid-term target, given the chronic relapsing course, is sustained stabilisation of mood rather than the resolution of a single episode; and the long-term aim is maintenance and vigilance, because a disorder that waxes and wanes over years is one to hold in view rather than discharge after a good spell. The **modus** spans the available modes of treatment.

- The pharmacological mode is led by an **antidepressant**, to which the disorder shows a good response, with a low-dose neuroleptic added where the picture reaches toward paranoia or hallucinations; the choice of a particular antidepressant against its effect on the seizure threshold is handled in its own dedicated topic and not reopened here.
- The psychological and social modes carry real weight: explaining to patient and family that the chronic, fluctuating, irritable low mood is part of the epilepsy and is treatable reframes what has often been read as difficult behaviour, and support is needed across a long course.
- Attention to the epilepsy itself stays in the background: the disorder is commoner where focal impaired-awareness seizures are frequent, so good seizure control remains part of good care, though, this being interictal, it is not the primary lever it is for the peri-ictal syndromes.

IN INDIA

Two realities shape this in Indian practice. First, the epilepsy consultation is dominated by seizure control, often against a wide treatment gap and little time per patient, so a chronic, fluctuating, irritable dysphoria the patient never volunteers is easily overlooked; actively asking about mood and irritability, and asking the family, is what surfaces it. Second, a widespread and understandable hesitancy about prescribing antidepressants in epilepsy, for fear of provoking seizures, can leave an eminently treatable mood disorder untreated. Since the disorder responds well to antidepressants, the task is to treat it while attending to seizure-threshold safety through the dedicated topic on antidepressant choice, not to withhold treatment by default.

GOOD TO REMEMBER

When you assess a person with epilepsy, screen for a chronic, intermittent, irritable low mood, not only a classic depressive episode, and ask the family about paroxysmal irritability the patient may not report. If you find it, treat it: the disorder responds well to an antidepressant, with a low-dose neuroleptic added only where paranoia or hallucinations are prominent. Naming the pattern is the step that unlocks a good outcome.

Interictal	Dysthymia-like	Commonest	Antidepressant
RUNS FREE OF SEIZURE TIMING	CHRONIC, FLUCTUATING, IRRITABLE MOOD	MOOD PRESENTATION IN EPILEPSY	GOOD TREATMENT RESPONSE

Interictal dysphoric disorder is a chronic, fluctuating, irritable dysphoria that runs in the baseline between seizures, spreads across depressive, irritable and mildly psychotic domains, is the commonest mood presentation in epilepsy rather than a classic depressive episode, and responds well to an antidepressant, with a low-dose neuroleptic added only where paranoia or hallucinations appear.

The psychoses of epilepsy

17 · Schizophrenia-like psychosis of epilepsy (chronic interictal psychosis): features and management

This is the epilepsy psychosis that most faithfully counterfeits schizophrenia, and recognising the counterfeit for what it is carries the marks. The **chronic interictal psychosis** of epilepsy, loosely called the **schizophrenia-like psychosis of epilepsy**, is a persistent psychotic disorder that arises between seizures, in **clear consciousness**, in a person with long-standing epilepsy, and that reproduces the surface of schizophrenia closely enough to be mistaken for it on a single mental state examination. The reason it is examined so reliably is that it forces the candidate to hold two truths at once: it looks like schizophrenia, and yet it is a secondary psychosis with its own age of onset, its own temporal relationship to the epilepsy, its own risk profile and its own course. A trainee who describes only the resemblance has answered half the question; the marks are in the resemblance *and* the signature that betrays it.

The psychoses of epilepsy are sorted along an axis defined by their timing to the seizure, running from preictal through ictal to postictal and finally to the interictal, seizure-independent forms. This entity occupies the interictal, chronic slot: it runs on independently of individual seizures and of seizure control rather than being locked to a fit. That single fact separates it from its seizure-bound neighbours, each of which is taught in its own right in this chapter. It is not the ictal psychosis that is itself the electrographic discharge, it is not the brief postictal psychosis that erupts after a lucid interval following a cluster of fits, and it is not the alternative psychosis of forced normalisation, in which the disturbance appears as the electroencephalogram paradoxically normalises. The systematic discrimination of this psychosis from primary

schizophrenia is developed in its own dedicated topic, and the phenomenology of the primary illness itself belongs to the Schizophrenia: aetiology, phenomenology, classification and course notes; here the task is the epileptic entity itself, its features and its management.

17.1 What the entity is

A chronic, interictal, schizophrenia-like psychosis in a clear sensorium. The defining combination, as set out in Kaufman's Clinical Neurology for Psychiatrists, is a chronic psychotic illness that develops interictally, in clear consciousness, and that phenomenologically resembles schizophrenia. Each of those three qualifiers is doing work. *Chronic* distinguishes it from the acute, self-terminating epilepsy psychoses and signals that this is an illness measured in months and years rather than hours and days. *Interictal* places it off the seizure timeline: the psychosis is not the aura, not the fit and not the immediate aftermath, but a state that persists in the gaps between seizures and does not track them. *Clear consciousness* is the anchor that keeps it apart from every clouded, confused or deliriant peri-ictal picture, because the patient is alert, oriented and coherent in sensorium even while floridly deluded. The resemblance to schizophrenia is genuine and not merely superficial, which is exactly why the discrimination from the primary disorder had to become a discipline of its own. Framing it as a secondary psychosis, real and schizophrenia-like but arising on an epileptic brain, is what justifies taking a seizure history in every apparently primary psychosis and explains the temporal signature that no primary schizophrenia carries. Current classifications place the epilepsy psychoses among the secondary presentations rather than treating them as schizophrenia proper, which keeps the clinician's attention on the underlying epilepsy as well as on the psychotic state in front of them.

17.2 How common it is, and the epilepsy-schizophrenia association

Uncommon in absolute terms, but far above the population rate. Psychosis is not the commonest psychiatric complication of epilepsy, sitting well below the mood and anxiety disorders in frequency, yet it is disproportionately important because it is severe, chronic and closely tied to the epilepsy itself. The Maudsley Prescribing Guidelines give the point prevalence of psychosis among people with epilepsy as roughly 5%, an order of magnitude above the general population, with depression and anxiety more common still. The substrate matters as much as the headline figure. Temporal lobe epilepsy, whose anatomy and semiology are covered in their own right in this chapter, is the principal setting, and Kaplan and Sadock's Comprehensive Textbook of Psychiatry reports that up to 9% of adults with temporal lobe epilepsy develop an associated interictal psychosis. There is a laterality signal as well: the psychosis is described as more common with a left, dominant-hemisphere temporal focus than with a right-sided one, a finding that has fed the long-running argument that dominant limbic pathology is particularly psychotogenic. Stepping back from epilepsy to the population level, Lishman's Organic Psychiatry cites the Danish register work of Qin and colleagues, in which a history of epilepsy carried a relative risk of schizophrenia of 2.48 (95% CI 2.20 to 2.80), a two- to three-fold elevation. That register finding is one strand of the broader bidirectional relationship between epilepsy and psychiatric disorder, which is set out in full in its own place in this chapter; what it

establishes for the present topic is that the association is real and directional, not an artefact of shared ascertainment.

17.3 Age of onset and the temporal signature

Epilepsy in childhood, psychosis in the fourth decade, a long gap between. The most examinable feature of this illness is not a symptom at all but a chronology. Kaufman's Clinical Neurology for Psychiatrists places the typical onset of the interictal psychosis at **30 to 40 years**, in patients whose epilepsy began in childhood, characteristically in the first decade of life, so that the psychosis emerges decades after the seizures first appeared. That gap is not incidental. Lishman's Organic Psychiatry frames it as an interval of roughly 10 to 15 years separating the onset of epilepsy from the onset of the schizophrenia-like psychosis, a latency confirmed by report after report since the original description. The classic series is that of Slater, Beard and Glithero (1963), whose sixty-nine patients showed a mean age at onset of the psychosis of 30 years after a mean epilepsy duration of 14 years. The practical force of the chronology is that a first psychosis in a young or middle-aged adult who has carried epilepsy since childhood should raise this diagnosis before a first break of primary schizophrenia is assumed. The accompanying table sets the temporal anchors side by side.

TEMPORAL ANCHOR	REPORTED FIGURE	SOURCE
Age at onset of the psychosis	typically 30 to 40 years, epilepsy from childhood	Kaufman's Clinical Neurology
Latency, epilepsy onset to psychosis onset	about 10 to 15 years	Lishman's Organic Psychiatry
Classic series (Slater, Beard and Glithero)	mean onset 30 years, after 14 years of epilepsy	Lishman's Organic Psychiatry

The temporal signature from three sources: childhood-onset epilepsy, a latency of roughly a decade and a half, and a psychosis declaring itself around the fourth decade. The chronology is the discriminator that a single mental state examination cannot supply.

17.4 Clinical picture: a predominantly positive psychosis

Loud positive symptoms in a clear head. Kaufman's Clinical Neurology for Psychiatrists describes a picture that is predominantly made of **positive symptoms**: **persistent hallucinations**, **paranoid and persecutory delusions**, and a tendency to **social isolation**, all occurring in otherwise clear consciousness. The hallucinations are sustained rather than fleeting, the delusional content is characteristically persecutory, and the withdrawal from social contact can be marked, so that the cross-sectional impression genuinely reads as schizophrenia. This is the crux of the topic and the source of most of its difficulty: nothing in the roster of positive symptoms reliably points away from a primary psychosis, and a cardinal schizophrenic symptom in such a patient does not exclude epilepsy. What differs lies in the longitudinal frame rather than the cross-section, which is precisely why the systematic discrimination of this entity from primary schizophrenia is developed in its own dedicated topic and is not reopened here, and why the phenomenology of primary schizophrenia belongs to the Schizophrenia: aetiology, phenomenology, classification and course notes. For the purposes of this entity, the message is that the interictal psychosis presents as a persistent, hallucinated, paranoid state in a patient who

is fully alert, and that the clarity of the sensorium, far from arguing against psychosis, is one of its defining features.

MNEMONIC

Three P's in a clear head

The interictal-psychosis picture in four beats. **P**ersistent hallucinations. **P**aranoid, persecutory delusions. **P**oor social contact, with withdrawal into isolation. All of it in a **clear** sensorium, interictally and chronically. Each element is a feature the source actually states; the clear head is the beat that keeps it off the peri-ictal timeline.

TRAP **Expecting a clouded sensorium.** Candidates lose the mark by describing this psychosis as arising in confusion or drowsiness, importing the clouding of the ictal and postictal states into an illness defined by its absence. By definition the interictal psychosis occurs in clear consciousness; a clouded patient is having a different syndrome. State the clarity of the sensorium explicitly, because it is examined as a discriminator, not as an afterthought.

17.5 Who is at risk: severity of epilepsy and brain damage

The risk markers read as an index of epilepsy severity. The associations that predict interictal psychosis are, with striking consistency, markers of how severe and how damaging the underlying epilepsy has been, which is the single most useful way to remember them. Kaufman's Clinical Neurology for Psychiatrists groups them as follows.

- Childhood onset of the epilepsy, the same early start that opens the long latency to psychosis.
- A demonstrable neurological abnormality or evidence of brain damage.
- Low intelligence.
- Frequent seizures and multiple seizure types.
- Treatment with multiple antiepileptic drugs, itself a marker of refractory disease rather than a cause.
- A prior postictal psychosis.

Reading these as a single construct rather than a checklist is what earns the mark: they describe a brain seized early, often, in more than one way, and with structural or cognitive compromise, and it is that cumulative burden that raises the psychotic risk. The point about multiple antiepileptic drugs deserves emphasis because it is easily misread. Polytherapy here is a proxy for hard-to-control epilepsy, not evidence that the drugs are psychotogenic; the drug-attributable and forced-normalisation routes to psychosis are separate constructs taught elsewhere. The last item on the list draws an explicit bridge between the acute and chronic psychoses of epilepsy. Lishman's Organic Psychiatry, citing the work of Tarulli and colleagues, records that about **15%** of patients with a postictal psychosis, the brief seizure-locked form taught as its own entity, eventually go on to develop a chronic interictal psychosis. That progression argues that seizure-related mechanisms drive the chronic illness, and clinically it makes a recurrent postictal psychosis a longitudinal warning, not only an acute problem.

17.6 Course and prognosis

More often chronic than not, with onset speed the best guide. The name of the entity already tells the story of its course, but the figures put a shape on it. Lishman's Organic Psychiatry records that nearly half of Slater's original series ran a chronic course, and that in a longer follow-up Onuma found **64% remaining chronic over ten years**. Once established, this psychosis tends to persist rather than resolve, which is a large part of what separates it prognostically from the brief peri-ictal psychoses that recover fully. The most useful single prognostic pointer is the tempo of onset: an **acute onset carries a better prognosis** than an insidious one, so the manner in which the illness begins is worth documenting from the first contact. The chronicity has direct consequences for how the patient is followed. An illness that persists for years in a person who already carries chronic epilepsy is a long-term management problem in two domains at once, the seizures and the psychosis, and neither can be allowed to drift while the other is attended to. It also reframes the goal of treatment: the realistic aim in the majority is sustained control and preserved function over years, not a brief course of medication with an expected cure. The minority whose illness is acute and self-limiting are the exception, and identifying them early avoids over-committing them to lifelong treatment.

17.7 Management

Two illnesses in one patient, treated in parallel. Management rests on treating the psychosis while never taking the eye off the epilepsy, and it is guided by the ILAE and NICE epilepsy guidelines for seizure control, by the Maudsley Prescribing Guidelines for the antipsychotic itself, and by the epilepsy-specific psychiatric-management literature that bridges the two. The **LOCUS** of care is, for a chronic illness, predominantly the outpatient clinic, with inpatient admission reserved for acute exacerbation, behavioural risk or the acutely disturbed presentation that reaches an emergency setting. The **focus** shifts by phase. The acute target is the florid positive symptoms and any risk they generate; the short-to-mid-term target is a durable antipsychotic response together with optimisation of seizure control; and the long-term target, given the chronic course, is maintenance of remission, relapse prevention and the rehabilitation of a patient whose social contact may have narrowed. The **modus** spans every mode of treatment. Pharmacologically, Kaplan and Sadock's Comprehensive Textbook of Psychiatry notes that the majority of patients require antipsychotic treatment, although up to 15% remit without any, and that there is no clear efficacy difference between first- and second-generation agents, so the choice turns on tolerability and, crucially, on effect on the seizure threshold. That specific decision, which antipsychotic to choose against its propensity to lower the seizure threshold, is handled in its own dedicated topics and is not reopened here. Beyond the drug sit the psychological, social and procedural modes: engagement and psychoeducation, attention to the social isolation that is part of the illness, family involvement, and above all the tightening of antiepileptic treatment as a lever that acts on the shared seizure substrate.

PITFALL

Because a slice of these patients recover without medication, it is tempting to watch and wait in an alert, non-dangerous patient and defer the antipsychotic. The numbers do not support it: only a small minority remit untreated, and the great majority need an antipsychotic to get better. Waiting turns a treatable chronic psychosis into a longer one and lets the social withdrawal entrench. Treat the psychosis; do not gamble on the exception.

GOOD TO REMEMBER

When a person with long-standing, childhood-onset epilepsy develops a persistent paranoid-hallucinatory psychosis in a clear sensorium, do not reflexively file it as a first episode of primary schizophrenia. Start an antipsychotic, chosen with the seizure threshold in mind, and at the same time review and optimise the antiepileptic regimen. You are managing the psychosis and the epilepsy together, and the seizure control is part of the psychiatric treatment, not a separate errand.

IN INDIA

Two features of Indian practice sharpen this problem. The epilepsy treatment gap is wide, so childhood-onset epilepsy is often poorly controlled for years, which is precisely the severity profile that breeds the chronic interictal psychosis; optimising long-neglected seizure control is frequently the single most valuable intervention. Second, longitudinal records and prior electroencephalography are often unavailable, so a chronic schizophrenia-like picture is easily mislabelled as primary schizophrenia when no reliable seizure history is to hand. The practical safeguard is to take an active seizure history in every chronic psychosis rather than waiting for one to be volunteered, and to weigh the double stigma of epilepsy and mental illness when planning long-term, community-based follow-up.

17.8 Where it sits among the psychoses of epilepsy

The chronic, seizure-independent member of the family. Keeping this entity distinct from its neighbours is best done, once again, on the timing axis that organises the whole group. It is not the ictal psychosis, which is itself the electrographic seizure and has no interval at all. It is not the postictal psychosis, which follows a cluster of fits across a lucid interval and then remits, though a minority of those cases progress into this one. It is not the alternative psychosis of forced normalisation, a distinct construct in which the disturbance appears as the electroencephalogram normalises and seizures cease. And where an interictal presentation is dysphoric and affective rather than psychotic, the interictal dysphoric disorder of epilepsy is the neighbour to consider. The formal business of separating any of these from a primary schizophrenic illness is developed in its own dedicated topic, and the primary illness itself belongs to the Schizophrenia: aetiology, phenomenology, classification and course notes. Hold the entity by its signature and it stops blurring into the family around it.

RULE **Chronic psychosis, clear sensorium, running on between seizures in long-standing childhood epilepsy, is the schizophrenia-like psychosis of epilepsy.** Every word of that line is load-bearing: chronicity separates it from the acute peri-ictal psychoses, the clear sensorium separates it from the clouded ones, the interictal, seizure-independent course separates it from the ictal and postictal forms, and the long latency from childhood-onset epilepsy is the chronology no primary schizophrenia supplies.

up to 9%	about 5%	10 to 15 yr	2.48
INTERICTAL PSYCHOSIS IN TEMPORAL LOBE EPILEPSY	POINT PREVALENCE OF PSYCHOSIS IN EPILEPSY	EPILEPSY-TO-PSYCHOSIS LATENCY	RELATIVE RISK OF SCHIZOPHRENIA GIVEN EPILEPSY

The schizophrenia-like (chronic interictal) psychosis of epilepsy is a chronic, positive-symptom psychosis in clear consciousness, arising a decade or more after childhood-onset epilepsy, most often in temporal lobe epilepsy; most patients need an antipsychotic, the course is usually chronic, and the epilepsy must be treated alongside the psychosis.

18 · Forced normalisation (Landolt phenomenon) and alternative psychosis

This is the one epilepsy psychosis that punishes success. In almost every other peri-ictal and interictal syndrome the psychiatric disturbance tracks the seizures, so that controlling the fits tends to calm the behaviour. **Forced normalisation** inverts that comfortable rule. It names the emergence of psychotic, or less often severe affective, symptoms precisely as the seizures come under control and the electroencephalogram quietens, so that the very sign the neurologist has been working towards, a clean record, can arrive hand in hand with a new psychiatric illness. The eponymous label, the **Landolt phenomenon**, and its clinical twin, **alternative psychosis**, describe the same counter-intuitive reciprocity from two different vantage points. The marks are here because the idea is conceptually clean, historically rich, and a favourite of examiners probing whether a candidate can hold that seizures and psychosis might, in a subset of patients, behave as antagonists rather than allies.

The concept is old and its status contested, but its practical importance is out of proportion to how rarely it is convincingly documented. If it is real, the pursuit of complete seizure freedom is not an unqualified good in every patient, and the instruction that falls out of it is safe whatever one makes of the mechanism: watch the patient whose seizures have just stopped.

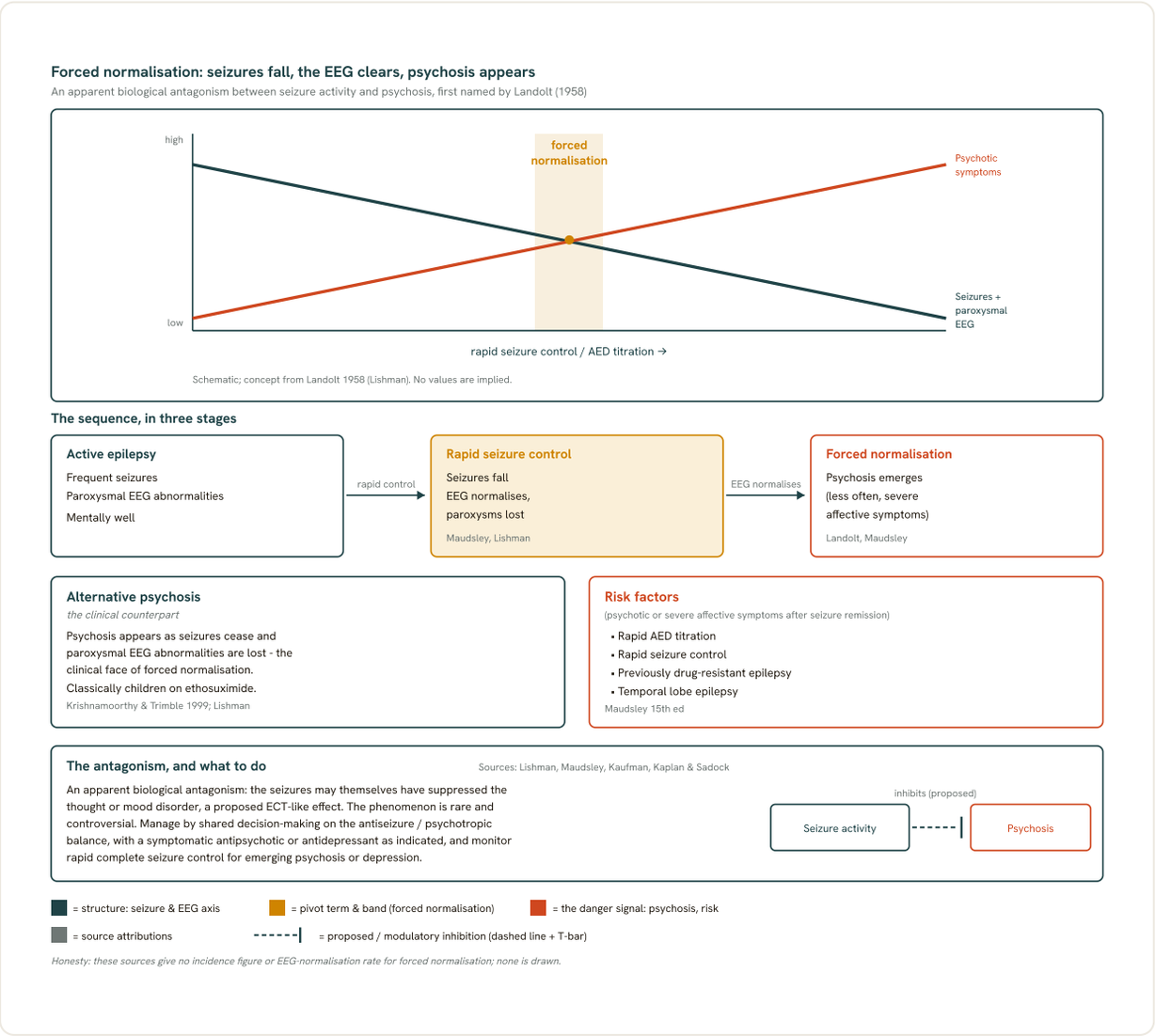


Fig 18.4 **Forced normalisation and alternative psychosis.** The EEG normalises and paroxysmal abnormalities are lost just as psychotic (or, less often, severe affective) symptoms appear, an apparent biological antagonism between seizures and psychosis first named by Landolt in 1958; alternative psychosis is its clinical counterpart, classically in children whose seizures cease on ethosuximide.

18.1 What forced normalisation is

Psychiatric symptoms that rise as seizures fall. The core definition is a temporal inversion. Forced normalisation is the appearance of psychosis, or a severe affective disturbance, at the moment when seizure frequency drops and the EEG loses its epileptiform activity, so that the improvement in the electrical disorder and the arrival of the mental disorder are yoked together in time. The Maudsley Prescribing Guidelines place it among the para-ictal episodes for exactly this reason: it is defined not by any particular content of the psychosis but by its relationship to a falling seizure burden. The essential clause, and the one that separates it from every look-alike, is that the seizure remission comes first and the psychiatric episode follows it. A psychosis that runs alongside continuing frequent seizures is not this entity; a psychosis that appears only once the fits have been suppressed is its candidate. Reading the definition this way keeps the attention on sequence rather than on symptom. The picture may be paranoid, grandiose, affectively coloured or frankly depressive, and the phenomenology alone will not name the syndrome for you; what names it is that the disturbance emerged out of a quietening brain rather than a convulsing one.

It is a syndrome of timing and reciprocity, not of a fixed clinical face, and its examinable heart is the paradox that the patient got psychiatrically worse as they got neurologically better.

18.2 Landolt and the origin of the term

An observation born at the EEG machine. The term is a piece of electroencephalographic history, and its provenance is examinable in its own right. It was **Landolt** who coined it, in **1958**, from serial EEG recordings taken through the psychotic episodes of patients with epilepsy, as documented in Lishman's Organic Psychiatry. The critical word is normalisation, and it is meant literally: Landolt was describing the record, not the patient. What he saw was that in certain individuals the EEG became normal, its paroxysmal features disappearing, at the very time the psychosis declared itself, and returned to its abnormal epileptiform pattern as the psychosis lifted. The adjective forced captures the sense that this normalisation was not the ordinary quiet of a well person but a suppression that seemed to buy the clean trace at the cost of the mind. Holding the origin in mind protects against a common misreading: forced normalisation is not a clinical description that happens to mention an EEG but a fundamentally EEG-defined phenomenon, an observation about what serial records do during psychotic episodes in epileptic patients. That distinction matters because the entity was later re-approached from the bedside, where the electroencephalogram is not always the tool in hand, and the two descriptions have to be kept aligned.

18.3 Alternative psychosis: the clinical counterpart

The same reciprocity, seen from the bedside. Because the neurophysiologist's EEG-defined term does not travel easily into everyday clinical language, the phenomenon acquired a clinical counterpart. Alternative psychosis is the name for the same reciprocal event described in behavioural terms: the psychosis appears as overt seizures cease, so that seizures and psychosis seem to alternate, one giving way to the other. Where a record happens to be taken the paroxysmal EEG abnormalities may normalise in step, which is the forced-normalisation counterpart, but the clinical term is defined by the alternation of seizures and psychosis and does not require documented EEG normalisation, which is exactly what lets it be used at the bedside where no serial EEG is in hand. Lishman's Organic Psychiatry attributes the modern framing to Krishnamoorthy and Trimble in **1999**, who set the clinical concept alongside Landolt's electrographic one, and its classic illustration is a childhood one: children whose absence seizures are rendered fully controlled on **ethosuximide** and who then develop a psychiatric disturbance as the fits vanish. The word alternative is doing careful work: it signals substitution, a swap of one state for another rather than mere coincidence, and it is the behavioural expression of what Landolt read off the trace. Keeping the pair straight is a small examination skill in itself, and the accompanying figure sets the two descriptions side by side.

AXIS OF DESCRIPTION	FORCED NORMALISATION (LANDOLT)	ALTERNATIVE PSYCHOSIS
Anchored in	the electroencephalogram	the clinical seizure state
The change that defines it	paroxysmal EEG abnormality disappears	overt seizures cease
Vantage point	the neurophysiologist reading serial records	the clinician watching the patient
Classic illustration	chronic, often drug-resistant epilepsy in adults	a child rendered seizure-free
What it asserts	one reciprocal phenomenon, seizures and psychosis alternating, described two ways	

Forced normalisation and alternative psychosis are the electrographic and the clinical descriptions of a single reciprocity; the term you reach for depends on whether the EEG or the seizure state is the thing in front of you.

18.4 The proposed mechanism: a biological antagonism

Seizures and psychosis as opposing states. The organising hypothesis is that, in some individuals, seizure activity and psychiatric disturbance stand in a relationship of **biological antagonism**, so that active epileptiform activity somehow holds the thought or mood disorder in check and its removal releases it. Lishman's Organic Psychiatry frames this as the working mechanism behind both the electrographic and the clinical descriptions: the seizures may have been suppressing an underlying psychiatric disorder, and abolishing them lifts that suppression. The most cited version of the idea invokes a parallel with electroconvulsive therapy. A seizure is, at the level of the brain, a massive synchronous discharge with recognised antidepressant and antipsychotic effects when delivered therapeutically, and the antagonism hypothesis proposes that a patient's own spontaneous seizures might exert something of the same stabilising influence, so that a brain deprived of them loses an endogenous treatment. This is a mechanistic proposal rather than an established pathway, but it has real explanatory reach. It accounts for the timing, since the disturbance follows the loss of seizures; it accounts for the affective as well as the psychotic presentations, since the postulated protective effect is not specific to one symptom class; and it fits the observation that the phenomenon is seen when control is achieved rapidly, the putative endogenous stabiliser withdrawn abruptly rather than tapered. Its value is that it converts a list of disconnected facts into a single story, and it is the point examiners probe when they ask why controlling seizures might, paradoxically, be the thing that made the patient ill.

RULE **Better seizure control can buy worse mental health.** In a susceptible minority, psychosis or a severe affective disturbance appears as seizures remit and the EEG clears, which is the reverse of the usual expectation that controlling fits calms behaviour. The corollary is practical rather than nihilistic: rapid, complete seizure control is a setting to watch, not a result to celebrate uncritically.

18.5 Who is at risk

A recognisable clinical setting, not a random event. Although uncommon, the phenomenon does not strike at random, and the reported cases share a describable profile that lets a clinician

anticipate it. The Maudsley Prescribing Guidelines set out the features that recur when psychotic or severe affective symptoms follow seizure remission, and they cohere into a picture of a hard-to-control epilepsy just brought sharply to heel. Four features travel together as the risk profile for a forced-normalisation reaction:

- Rapid up-titration of the antiseizure drug, so that the seizure burden falls steeply rather than gradually.
- Rapid and complete control of seizures, the abrupt achievement of freedom from fits.
- An epilepsy that had previously been medication-resistant, so that the sudden control is itself unusual.
- Temporal lobe epilepsy, whose anatomy, semiology and psychiatric salience are taught in their own right elsewhere in this chapter.

Each element makes sense through the antagonism lens. If spontaneous seizures were supplying an endogenous stabilising effect, the danger is greatest where that effect is removed quickly and wholly, which is what rapid titration and rapid complete control describe. The prior drug resistance marks the patients in whom sudden, thorough control is both possible and startling to the system, and the temporal lobe association ties the phenomenon to the limbic structures through which seizure activity most readily reaches mood and belief. The risk factors are therefore less a list to recite than a situation to recognise: a previously refractory patient in whom a briskly escalated regimen has just abolished the fits.

MNEMONIC

Three R's and a T

The setting in which a forced-normalisation reaction tends to be reported. **R**apid antiseizure-drug titration, **R**apid and complete seizure control, a **R**efractory (previously drug-resistant) epilepsy, and **T**emporal lobe epilepsy. Each letter is a risk feature the topic actually names, not an invented extra.

18.6 A rare and genuinely controversial entity

Real enough to teach, contested enough to qualify. The status of the phenomenon is itself examinable, since the safest answer neither dismisses it nor overstates it. Kaplan and Sadock's Comprehensive Textbook of Psychiatry describes forced normalisation as a rare and controversial phenomenon, in which psychosis coincides with improved seizure control achieved by any route: medication, vagus nerve stimulation, or epilepsy surgery. That framing carries two useful teaching points. The first is the breadth of trigger: it is not a drug effect in the narrow sense, since the psychosis can follow control secured by a stimulator or by resection just as readily as by tablets, which is what one would expect if the operative variable is the seizure suppression rather than the means used to achieve it. The psychiatric outcomes of epilepsy surgery are a topic in their own right elsewhere in this chapter; this is only their point of contact with forced normalisation. The second point is the controversy, and it deserves to be understood rather than merely stated. The phenomenon is hard to establish because its classic definition depends on serial EEG capture across an episode that is itself uncommon; ascertainment is biased toward the

cases that happened to be recorded, causality is difficult to separate from coincidence in a population already prone to psychosis, and the literature leans on case reports. None of this makes it unreal, and its corollary of vigilance stands regardless, but it does mean forced normalisation should be presented as an established concept of uncertain frequency and contested mechanism rather than as a settled quantity.

1958	1999	rare
LANDOLT NAMES THE PHENOMENON	ALTERNATIVE PSYCHOSIS REFRAMED CLINICALLY	AND GENUINELY CONTROVERSIAL IN STATUS

18.7 Recognition and the differential

Made on sequence, defended against its mimics. Because there is rarely a serial EEG to hand, recognition in practice is a clinical act that rests on reconstructing the sequence: an epilepsy, often refractory, that was brought under control, and a psychiatric episode that appeared as the control was achieved. Kaufman's Clinical Neurology for Psychiatrists turns this into an actionable caveat, advising that clinicians monitor for emerging psychosis or depression the patient who rapidly achieves complete seizure control after a change of antiseizure regimen that suppresses the seizures and the EEG. The differential is where the marks and the errors lie. The disturbance must be separated from a direct psychiatric side effect of the antiseizure drug that was escalated, the psychiatric side effects of individual antiepileptic drugs being a distinct topic in their own right; the two can look identical at the bedside and are told apart by whether the operative change was the drug itself or the remission it produced. It must be separated from the other psychoses of epilepsy that arrive on a different schedule: the postictal psychosis that erupts after a lucid interval following a cluster of fits, and the schizophrenia-like psychosis of epilepsy, the chronic interictal picture that runs on independently of seizure control and is taught as its own entity elsewhere in this chapter. Where the presentation is affective and interictal, the interictal dysphoric disorder of epilepsy is the neighbour to consider. The systematic business of distinguishing any epileptic psychosis from primary schizophrenia, on preserved affect, premorbid function and the relation to seizures, is developed in its own dedicated discussion, and the phenomenology of the primary illness itself belongs to the Schizophrenia: aetiology, phenomenology, classification and course notes.

TRAP **Blaming the drug instead of the remission.** When a patient turns psychotic soon after an antiseizure drug is pushed up and the fits stop, the reflex is to record a psychiatric side effect of that drug and switch it. Forced normalisation attributes the disturbance not to a direct pharmacological action but to the seizure suppression itself, and the distinction changes whether one would ever deliberately accept less complete control. That is the discriminating point examiners are listening for.

PITFALL

The opposite error is over-diagnosis. Not every person with epilepsy who becomes psychotic after starting treatment has forced normalisation; the phenomenon is uncommon, and reaching for it whenever seizures improve and a mental disturbance appears risks missing a coincidental primary psychosis, a direct antiepileptic side effect, or a postictal psychosis with a quite different time course. Reserve the label for the genuine temporal coupling of remission and a quietening EEG with the new symptoms.

GOOD TO REMEMBER

Whenever a change of regimen abolishes seizures quickly, especially in someone whose epilepsy had been hard to control, keep the mental state under review over the following weeks and ask the family to report emerging suspiciousness, elation or low mood. It is the low-cost instruction the topic exists to teach, and it catches the problem early without committing anyone to a change in the epilepsy plan before it is needed.

IN INDIA

Read the lesson forwards, not backwards. With a large epilepsy treatment gap, the dominant problem across most of Indian practice is under-treatment, so forced normalisation is never a reason to aim for anything less than good seizure control. Where it earns attention is the tertiary-centre patient whose refractory epilepsy is suddenly brought to heel by an optimised regimen, vagus nerve stimulation or surgery; because serial EEG follow-up is often not feasible, recognition then rests on clinical vigilance and a well-briefed family rather than on the record.

18.8 Management

The management question is a balance, not a protocol. What makes this topic unusual is that the treatment and the possible cause are the same lever, a genuine trade-off rather than an algorithm. The organising framework rests on seizure-control standards from the ILAE and the NICE epilepsy guidelines for the epilepsy itself, and on the Maudsley Prescribing Guidelines together with the wider epilepsy-psychiatry literature for the psychiatric episode. The guiding principle those sources converge on is shared decision-making with the patient and their carers about how to proceed with the antiseizure and psychotropic drugs, precisely because the values at stake, freedom from seizures against freedom from psychosis, belong to the patient to weigh.

The LOCUS of care follows severity. Most patients can be held and monitored as outpatients; a florid or high-risk psychotic or affective episode may warrant inpatient admission, and acute behavioural disturbance may present first to an emergency setting. The focus shifts by phase. The acute task is to contain the psychiatric symptoms and secure safety; the short-to-mid-term task is to settle the antiseizure-versus-psychotropic balance deliberately rather than reflexively; and the long-term aim is a sustainable equilibrium in which seizures and mental state are both tolerable. The modus spans every mode of treatment. On the drug side, symptomatic treatment with an antipsychotic or an antidepressant is used as the presentation indicates, while the separate question of which antipsychotic to choose against its effect on the seizure threshold, and the

mechanism of that threshold-lowering, are handled in their own dedicated topics and are not reopened here. The antiseizure decision itself is the pivot, and it is where the shared decision belongs: whether to hold the regimen and treat the psychiatric episode symptomatically, or, uncommonly and only by agreement, to accept marginally less complete seizure control, is a judgement made with the patient and family rather than imposed. Around the drugs sit the psychological and social modes, chiefly education of the patient and carers about the paradox they are living through, so that neither the improvement in seizures nor the emergence of symptoms is misread.

> Forced normalisation, the Landolt phenomenon, is the emergence of psychosis or severe mood disturbance as seizures are brought under control and the EEG normalises; its bedside counterpart is alternative psychosis, the working idea is a reciprocal antagonism between seizures and psychiatric illness, and the safe clinical rule is to watch the mental state of any patient whose fits have just been abolished.

19 · Differentiating epileptic psychosis from primary schizophrenia

Why the marks are here. The examiner who asks you to separate the psychosis of epilepsy from primary schizophrenia is not testing whether you can recognise psychosis, because both patients are psychotic; the test is whether you understand that a psychotic cross-section is a starting point and not a diagnosis. The problem is genuine. The chronic psychosis that arises between seizures in long-standing epilepsy, taught in full as the schizophrenia-like psychosis of epilepsy, can reproduce the surface of schizophrenia so faithfully that a single mental state examination will not tell the two apart. What separates them is almost everything the mental state examination does not capture in one sitting: the patient's affect and personality over time, the trajectory of the illness, the family background, and above all the relationship of the psychosis to a history of seizures. This topic owns that discrimination. It does not re-teach the primary illness, whose phenomenology, aetiology and course belong to the Schizophrenia: aetiology, phenomenology, classification and course notes, and it does not re-describe the epileptic entity itself, which is set out where the schizophrenia-like psychosis of epilepsy is taught.

Two boundaries keep the topic clean. The discrimination here concerns the chronic interictal presentation, the psychosis that sits between seizures and persists. The brief, seizure-locked forms are a different problem: the ictal psychosis that is itself the electrographic seizure, and the postictal psychosis that follows a cluster of fits after a lucid interval, are separated from primary schizophrenia chiefly by their timing rather than by the features discussed below, and each is dealt with as its own entity, as is the alternative psychosis of forced normalisation. So when this chapter speaks of differentiating epileptic psychosis from schizophrenia, it means the chronic, interictal, schizophrenia-like picture, because that is the one that genuinely imitates the primary disorder and forces the question.

19.1 The resemblance is real: symptoms alone will not separate them

The two overlap almost completely at the level of individual symptoms. The canonical description comes from **Slater** and colleagues, whose series of **69 patients** with epilepsy who had developed a schizophrenia-like illness established that these patients showed all the cardinal symptoms of schizophrenia, delusions, hallucinations and passivity phenomena among them, and yet in atypical combinations. In other words, nothing in the roster of positive symptoms was missing, and a cardinal schizophrenic symptom in such a patient does not point away from epilepsy. What was atypical was the pattern: the way the symptoms hung together and the relative sparing of the features that dominate severe primary schizophrenia. This is the single most important fact to carry into the examination, because the instinct of the trainee is to hunt for a symptom that rules schizophrenia in, and there is no such symptom to be found.

Understanding why the resemblance is so close also tells you where to look for the difference. Both illnesses generate psychosis, but they arrive at it by different routes. Primary schizophrenia is understood as a pervasive disorder of the developing brain that erodes the personality and the capacity for affective and social engagement, often before the first frank psychotic symptom appears. The psychosis of epilepsy is a symptomatic psychosis: a productive disturbance grafted onto a brain and a personality that were, until the illness declared itself, developing normally, and driven by a focal epileptic process rather than by the diffuse liability that underlies the primary disorder. Because the substrate differs, the surface can be identical while the depths are not, and it is in those depths, in affect, personality, course, heredity and the temporal link to seizures, that the discrimination is actually made.

TRAP **Believing that a fully schizophrenic cross-section excludes an epileptic cause.** Candidates reason that florid, well-systematised delusions or vivid auditory hallucinations must indicate the primary disorder. Slater's patients had exactly these features. The presence of cardinal schizophrenic symptoms is entirely compatible with the psychosis of epilepsy; it is their atypical combination and the surrounding history, not their mere presence, that carry the diagnostic weight.

19.2 Preserved affect and easy rapport at the bedside

Warm affect and interpersonal openness are the most useful bedside signs. The texture of affect and relatedness is where the difference first shows itself in a single interview. In the psychosis of epilepsy the affect remains, in relative terms, warm and responsive. The **preserved affect** of these patients is one of the oldest and most reliable points of separation, because the loss of affective response that characterises primary schizophrenia is here neither as early nor as marked. The patient continues to show emotional colouring appropriate to the content of thought and keeps the capacity to make affective contact with the examiner, rather than presenting the pervasive blunting and incongruity that dissolve rapport in the primary illness.

This warmth extends into the interpersonal domain. Clinically these patients tend to be approachable and willing rather than guarded, more cooperative and notably **less suspicious** than patients with primary schizophrenia. Where the schizophrenic patient is often withdrawn and hard to engage, the patient with an epileptic psychosis is frequently friendly and

forthcoming even while holding florid delusional beliefs. This is not a soft or impressionistic sign. It reflects the underlying difference between the two disorders, namely that the epileptic psychosis leaves the affective and relational core of the personality comparatively intact while superimposing a productive psychosis on top of it. The practical corollary is direct: a psychotic patient who remains emotionally engaged and cooperative, whose delusions do not come wrapped in the deep suspiciousness and affective distance of the primary illness, is a patient in whom you should be asking about seizures rather than closing the diagnosis.

19.3 A better premorbid baseline and a course that does not deteriorate

Two temporal features, one looking back and one looking forward. The impression the affect gives is reinforced from both directions in time. Looking backward, the **premorbid personality** is relatively well preserved: premorbid function is generally better than in primary schizophrenia, and a prominent personality abnormality in the years before the psychosis declared itself is uncommon. The insidious premorbid slide so often seen before a first episode of the primary illness, the quiet withdrawal and the erosion of drive and social function, is characteristically absent, and by history the patient was functioning reasonably well as a person before the epilepsy-related psychosis emerged.

Looking forward, the course diverges even more clearly. Patients with the epileptic psychosis do not show the **progressive deterioration** that is characteristic of schizophrenia. Over years the primary illness tends towards an accumulating defect state, with deepening negative symptoms and functional decline; the epileptic psychosis carries no such autonomous downhill trajectory. Whatever decline the patient shows tends to track the epilepsy and its consequences rather than an independent psychotic process. This is why the longitudinal view is so powerful. Give the two conditions time and the primary disorder tends to declare itself through deterioration, while the epileptic psychosis holds a comparatively steady line.

> **GOOD TO REMEMBER**
>
> The preserved personality and the non-deteriorating course are not merely diagnostic curiosities; they carry a better functional prognosis than primary schizophrenia and they change what you tell the family. Counselling that assumes the relentless trajectory of the primary illness misrepresents this patient's likely course, and a pessimism imported wholesale from schizophrenia is neither accurate nor kind.

19.4 The family history does not carry a schizophrenic loading

The pedigree speaks to aetiology, not presentation. Heredity gives one of the cleaner points of separation precisely because it bypasses the symptom picture altogether. Patients with the interictal psychosis of epilepsy do not show the increased **familial incidence of schizophrenia** seen in the primary disorder. Primary schizophrenia carries a well-recognised familial loading, with an increased occurrence of the illness among relatives; the epileptic psychosis does not sit on that genetic background. A family history of psychosis, where it is present, behaves at most as a modest risk factor for developing psychosis in the setting of epilepsy, not as the schizophrenia-level loading that the primary illness implies.

The reasoning follows once the aetiologies are separated. If the psychosis is being driven by an acquired, structural, epileptic process, then the inherited liability that runs in the families of people with primary schizophrenia should not be enriched in the families of these patients, and it is not. For the clinician this has two uses. It is a discriminator, in that a strong family history of schizophrenia shifts the balance towards the primary diagnosis; and it is a counselling point, because the heritable risk you would quote to the relatives of a person with primary schizophrenia does not transfer to the relatives of a patient whose psychosis is a complication of epilepsy.

19.5 The psychosis is grafted onto years of established epilepsy

Epilepsy first, by years, and then the psychosis. The most powerful contextual discriminator, and the one an examiner will reward most, is the temporal relationship between the psychosis and the seizure disorder. The interictal psychosis of epilepsy characteristically emerges only after the epilepsy has been established for a long time, on the order of a mean interval of about **14 years** of active seizures before the psychosis appears. The epilepsy comes first, by years, and the psychosis is grafted onto it. Primary schizophrenia has no such antecedent; there is neither a requirement for, nor usually any history of, a preceding seizure disorder of long standing.

This long latency matters both diagnostically and conceptually. Diagnostically, it means the single most discriminating question you can ask is about the sequence: was there a well-established epilepsy, active over many years, before the psychosis began? An affirmative answer, in a patient whose affect and personality are relatively preserved and whose course is not deteriorating, points firmly towards the epileptic psychosis. Conceptually, the long interval suggests a slow, secondary process, the gradual consequence of years of recurrent seizure activity, rather than the developmental disorder that underlies the primary illness. The mechanism linking chronic seizure activity to behavioural disturbance belongs to the chapter's account of the neurobiology of epilepsy and behaviour and is only named here, not re-derived.

> **PITFALL**
>
> The commonest way this diagnosis is missed is a seizure history that was never taken. A patient presenting with a first psychotic illness is placed on a primary-psychosis pathway, and an established epilepsy, sometimes nocturnal, sometimes long-standing focal seizures the patient no longer thinks to mention, is never elicited. Ask every psychotic patient directly about seizures, blackouts and their treatment. The temporal discriminator is worthless if the epilepsy is never uncovered in the first place.

19.6 Phenomenological texture: thought disorder and catatonia

Positive symptoms dominate, without the disorganisation and the motor phenomena. Although symptomatology cannot separate the two disorders on the presence or absence of cardinal symptoms, the emphasis within the symptom picture does differ, and two negatives are worth knowing. First, the interictal psychosis often shows a relative absence of **formal thought disorder**. The productive positive symptoms, the delusions and the hallucinations, tend to dominate, while the disorganisation of thought and language that can be so prominent in

primary schizophrenia is comparatively muted, so the patient may be floridly deluded and yet speak in a connected, followable way. Second, **catatonic phenomena** are rare in the epileptic psychosis, whereas catatonic features can arise in the course of primary schizophrenia.

These are, once again, tendencies rather than absolutes, and they have to be read alongside everything else rather than used as isolated rules. Their value is corroborative. A psychosis dominated by well-preserved, communicable positive symptoms, without significant formal thought disorder and without catatonia, in a patient who is affectively warm and cooperative, fits the epileptic picture. But because they are relative they can only tilt the balance; a patient with the epileptic psychosis may on occasion show some thought disorder, and their absence early in a primary schizophrenic illness proves nothing. The lesson that runs through every one of these discriminators is that each is probabilistic, and the diagnosis is a gestalt assembled from all of them.

Preserved	**Non-progressive**	**Absent**	**Rare**
AFFECT AND PERSONALITY	LONG-TERM COURSE	FORMAL THOUGHT DISORDER	CATATONIA

19.7 Putting the discriminators together

The whole is more than any part. No single feature listed above is pathognomonic, and any one of them can fail in an individual patient. The discrimination is made by assembling the features into a coherent picture and weighing that picture against the alternative. The comparison below sets the discriminating axes side by side; read down the epileptic column and a consistent gestalt emerges, a productive psychosis in a person whose affect, personality and course are comparatively intact, arising against a long history of seizures and without a schizophrenic family background.

DISCRIMINATING AXIS	INTERICTAL PSYCHOSIS OF EPILEPSY	PRIMARY SCHIZOPHRENIA
Affect	Relatively preserved, warm and responsive	Flattening early and marked
Interpersonal manner	Friendlier, cooperative, approachable	Guarded, suspicious, withdrawn
Premorbid personality	Relatively well preserved	Often abnormal or declining before onset
Long-term course	Non-deteriorating	Tendency to progressive deterioration
Family history	No excess familial schizophrenia	Increased familial incidence
Relation to seizures	Follows years of established epilepsy	No such antecedent
Formal thought disorder	Often absent	May be prominent
Catatonia	Rare	May occur

The discriminating axes read as a single gestalt; no row is pathognomonic on its own, and the diagnosis is made by the consistency of the column rather than by any one cell.

MNEMONIC

SPARE - the person is spared

The features that keep the diagnosis on the epileptic side spell **SPARE**. **S**eizures precede the psychosis by years. **P**remorbid personality and function preserved. **A**ffect warm and responsive. **R**elationships cooperative rather than suspicious. And no progressive d**E**terioration of course. To these the phenomenology adds positive symptoms without formal thought disorder or catatonia, and the pedigree adds no increased familial incidence of schizophrenia.

RULE **The psychosis of epilepsy is distinguished from schizophrenia longitudinally and contextually, not cross-sectionally.** Because the cardinal symptoms overlap, a single mental state examination cannot settle the question; the discriminating information lives in the history and in the course. The sequence of events and the trajectory over time outweigh any cross-sectional symptom.

19.8 Why the distinction earns its keep

The label changes prognosis, counselling and treatment. Making this differentiation is not an academic exercise, because the diagnosis alters three things at once. Prognostically, the preserved personality and the non-deteriorating course mean the outlook is generally better than in primary schizophrenia, and the patient should not be burdened with the expectations that attach to the primary illness. For counselling, the absence of a schizophrenic familial loading changes what relatives are told about their own risk. For treatment, recognising that the psychosis is a complication of epilepsy directs attention both to optimising seizure control and to choosing an antipsychotic with the seizure threshold in mind. The specific question of antipsychotic selection in epilepsy, and the mechanism by which antipsychotics lower the seizure threshold, are handled in their own right elsewhere in this chapter and are only flagged here.

In practice, a small number of questions settle most cases:

- Is there a well-established epilepsy of long standing that preceded the psychosis?
- Are the affect, personality and interpersonal manner relatively preserved?
- Is the long-term course free of the progressive deterioration of the primary illness?
- Is the family history free of a schizophrenic loading?

A final caution frames the whole topic. Every discriminator here is relative and probabilistic, and none is a rule that holds in every case; the diagnosis is the weight of evidence, not a single decisive sign. The differentiation is also specific to the chronic interictal presentation. The ictal psychosis that is a manifestation of the seizure itself, and the postictal psychosis that follows a cluster of seizures after a lucid interval, are separated from primary schizophrenia mainly by their timing and are dealt with as distinct entities. Hold the interictal discriminators for the illness they were built to describe: the chronic, schizophrenia-like psychosis that arises between seizures in

long-standing epilepsy and imitates the primary disorder closely enough to make the question worth asking in the first place.

DO NOT MISS

One organic mimic must never be missed at the other end of the timeline. A new psychosis with seizures, especially one that is acute or subacute in a young person, can be **anti-NMDA receptor encephalitis**: it classically presents in a young woman with a viral-like prodrome, a psychiatric-first picture, seizures, orofacial and limb dyskinesias, autonomic instability and a falling level of consciousness; it carries an association with an ovarian teratoma; and it is confirmed by paired serum and cerebrospinal fluid testing, with cerebrospinal fluid anti-GluN1 (NR1) IgG the decisive test because serum alone gives both false positives and false negatives. Treatment is immunotherapy together with a tumour search and resection of a teratoma when one is found, since many patients have no tumour. Its acute, dyskinetic, autonomically unstable course is the temporal opposite of the chronic interictal psychosis this topic discriminates, which is exactly why a fresh psychosis with seizures should trigger the autoimmune-antibody workup before it is filed as an epilepsy psychosis.

IN INDIA

The discrimination leans heavily on history and phenomenology, and that is exactly what makes it valuable in Indian practice. Where the epilepsy treatment gap is wide and many patients reach services only after years of poorly controlled seizures, the schizophrenia-like psychosis is a real and under-recognised presentation, and it is easily mislabelled as primary schizophrenia when longitudinal records, prior electroencephalography or even a reliable seizure history are unavailable. The clinician who cannot fall back on investigations must lean on the bedside discriminators, the preserved affect and personality, the non-deteriorating course, and above all the sequence of epilepsy first and psychosis later, and must take an active seizure history rather than waiting for one to be offered.

Preserved affect and personality, a course that does not deteriorate, a family history free of schizophrenia, and a psychosis that appears only after years of established epilepsy: these place a schizophrenia-like presentation on the epileptic side, even when the cross-sectional symptoms are indistinguishable.

20 · Antipsychotic choice and seizure-threshold considerations in epileptic psychosis

The antipsychotic is chosen against the seizure, not against the psychosis. Antipsychotic choice in epilepsy is a seizure-threshold consideration before it is a matter of psychiatric efficacy. In a patient whose psychotic episode arises on a background of epilepsy, the drugs that treat the psychosis are the same drugs that can destabilise the very seizures the neurologist has worked to control, and the whole skill of the topic lies in resolving that tension. The positive symptoms of an epileptic psychosis, whether it is a postictal psychosis, the schizophrenia-like psychosis of epilepsy or a psychosis emerging with forced normalisation, respond to antipsychotics much as psychosis does anywhere else. What differs, and what the marks reward, is the second question

the epilepsy forces on you: which of the effective agents is least likely to lower the seizure threshold and tip a vulnerable brain back into fits. Candidates who answer only the first question, prescribing as though the patient had no epilepsy, miss the point of the question entirely.

Two boundaries keep this topic clean. The property that ranks the agents, the dose-dependent lowering of the seizure threshold and clozapine's place at the extreme of it, is the substance of its own dedicated topic; here that ranking is taken as given and applied to a choice. And clozapine's full haematological and cardiac profile, with its monitoring, belongs to the Schizophrenia: management, antipsychotics and adverse effects notes. What is owned here is the decision itself: which agent, at what dose, introduced how, and with what antiepileptic cover.

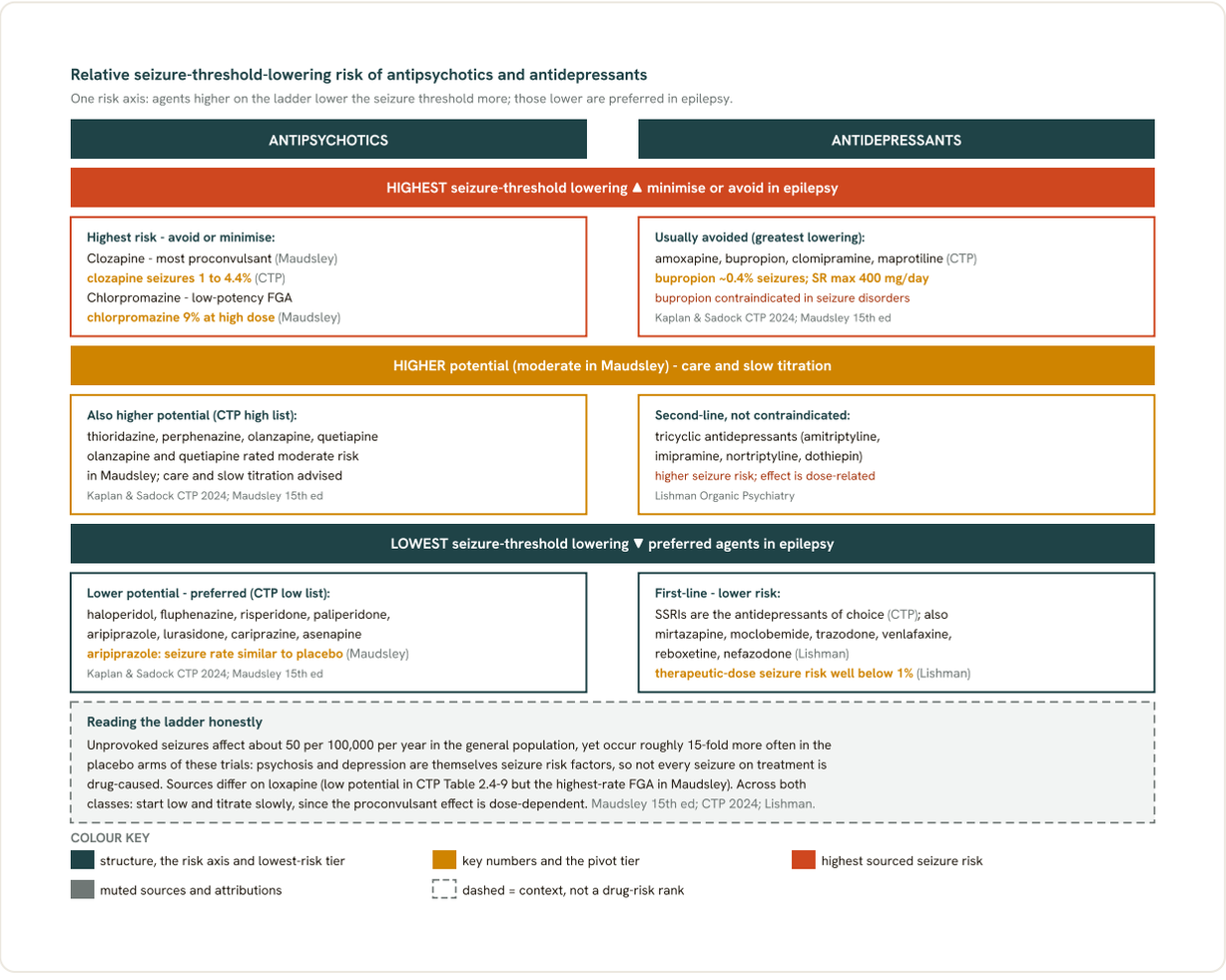


Fig 20.1 **Relative seizure-threshold-lowering risk of antipsychotics and antidepressants.** On a single risk axis, clozapine (the most proconvulsant antipsychotic, seizures in 1 to 4.4 per cent), high-dose chlorpromazine (9 per cent above 1 g per day) and the usually-avoided antidepressants amoxapine, bupropion, clomipramine and maprotiline sit highest, whereas high-potency first-generation antipsychotics, risperidone, paliperidone, aripiprazole and the SSRIs lower the threshold least and are preferred in epilepsy. Attributions per box (Kaplan and Sadock CTP 2024, Maudsley 15th ed, Lishman).

20.1 Why the choice is a threshold decision, not an efficacy contest

Efficacy is a wash; safety on the seizure threshold is the whole question. The starting fact, and the one candidates most often get wrong, is that there is no convincing difference in antipsychotic efficacy between the **first-generation antipsychotics** and the **second-generation antipsychotics** when they are used to treat the psychoses of epilepsy. If both classes bring the psychosis under control to a broadly similar degree, then efficacy cannot be the variable that

decides the prescription. The variable that can is the agent's propensity to lower the **seizure threshold**, and it is precisely this that becomes the primary selection criterion. The reasoning is worth stating explicitly because it inverts the habit of general adult psychiatry, where the choice usually turns on the efficacy and side-effect profile for the psychosis alone. In the epileptic patient the question is reframed: not which antipsychotic is most effective, but which effective antipsychotic is least likely to provoke a seizure in a brain that is already predisposed to them. Everything else in the topic, the preferred agents, the dosing, the method of introduction and the special problem of clozapine, is downstream of this single reorientation. The mechanism that generates the ranking, why some agents disturb the threshold more than others and why the effect is dose-related, is developed in its own topic and is not the subject here; the working clinician needs the ranking itself and the prescribing discipline that follows from it.

RULE **Select the antipsychotic by its effect on the seizure threshold, then introduce it low and slow.** Because the agents do not differ meaningfully in how well they treat the psychosis, the choice is made on seizure safety, and even the safest agent is made safer still by starting at a low dose and titrating gradually rather than loading.

20.2 The preferred agents at the safe end of the ranking

A small set of agents disturbs the threshold least and forms the default choice. The antipsychotics that lower the seizure threshold least, and are therefore preferred as first-line agents in a patient with epilepsy, are haloperidol, risperidone, paliperidone and aripiprazole. This is the set to reach for when an epileptic psychosis needs treating and there is no overriding reason to do otherwise. Among them, **aripiprazole** deserves separate emphasis, because it not only rarely lowers the seizure threshold but produced a seizure incidence comparable to placebo in randomised controlled trials, which is about as reassuring a signal as the evidence offers for any antipsychotic in this setting. That safety record is why it recurs on every low-risk list. The practical message is that a clinician does not need an exotic agent to prescribe safely here: two widely available high-potency options and two commonly stocked second-generation agents cover most situations, and their standing as preferred choices rests on seizure safety rather than on any claim to superior antipsychotic effect. Holding this short list in mind converts an anxious, open-ended decision into a quick one, which matters when the psychosis is florid and a drug must be started promptly.

MNEMONIC

HARP

The four preferred, least threshold-lowering antipsychotics in epilepsy. **H**aloperidol, **A**ripiprazole, **R**isperidone, **P**aliperidone. Each is an agent this topic names as a first choice on seizure-safety grounds; reach inside this set before you look outside it.

20.3 Grading the field: low risk against use with care

A working ranking sorts the antipsychotics into a safe tier and a care-required tier. The four preferred agents sit inside a slightly broader low-risk grouping that a clinician can draw from, and

it is useful to see that group set against the agents that call for caution. The Maudsley Prescribing Guidelines provide exactly this contrast in their antipsychotic table for people with epilepsy. On the safe side sit the low-risk good choices; on the other, agents at **moderate seizure risk** where more care is required, with a further pharmacokinetic wrinkle for quetiapine. The accompanying table lays the two tiers side by side.

RISK TIER	ANTIPSYCHOTICS	PRACTICAL NOTE
Low risk (good choices)	amisulpride or sulpiride, aripiprazole, ziprasidone, high-potency first-generation agents such as haloperidol and fluphenazine, and risperidone	preferred where an antipsychotic is genuinely needed
Care required (moderate risk)	olanzapine and quetiapine	quetiapine additionally carries a high risk of interaction with antiepileptic drugs

A practical two-tier grouping of antipsychotics for people with epilepsy: the low-risk agents to prefer, and the two that can be used but demand closer attention to dose and seizure control.

Reading the table the right way matters more than memorising it. The low-risk tier is not a licence to ignore the threshold; it identifies the agents least likely to cause trouble when the prescribing discipline described below is followed. The care-required tier does not forbid its agents either; olanzapine and quetiapine can be used, but they demand closer attention to dose and to seizure control, and quetiapine's high liability for interaction with antiepileptic drugs layers a pharmacokinetic hazard on top of the pharmacodynamic one. The agents that sit at the genuinely seizure-provoking end of the ranking, with clozapine the paradigm, are handled in their own topic and in the special case set out below, because they change the whole calculus of prescribing rather than sitting on a simple scale of caution.

20.4 Introducing the drug: low, slow and to a defined dose target

How the drug is started matters as much as which drug it is. Even a low-risk agent can precipitate a seizure if it is introduced carelessly, because rapid introduction and high dosing are the principal provokers of fits with these medicines. The prescribing rule is therefore to begin at a low dose and titrate upward slowly, letting the patient settle at each step rather than pushing to a target in a hurry. The dose that is actually needed is conventionally expressed in **chlorpromazine-equivalent** terms, which lets agents of very different milligram potencies be compared on a common scale. For a mild-to-moderate psychotic episode the usual requirement is **300 to 1,000** mg/day chlorpromazine-equivalent, reached by that same gradual titration rather than by front-loading, and in epilepsy the aim is the lower end of that range that still controls the psychosis. Dosing above the usual range is exceptional high-dose treatment, resorted to only when standard doses have genuinely failed and only under specialist supervision with intensified antiepileptic-level and EEG monitoring, because it is at the high end that seizure liability climbs. Severity of illness does not by itself license a higher target: the figures describe a range, not a licence to push the dose up quickly. The same total dose is far safer approached in small increments than imposed at once, which is the entire reason the method is emphasised in the

same breath as the number: a correct target reached by an incautious route can still provoke the seizure the choice of agent was meant to avoid. The dose range is also a reminder that undertreatment is its own failure; a psychosis left under-dosed out of excessive threshold anxiety is not managed, merely neglected, and the answer is a safe agent taken to an adequate dose by a safe route rather than a good agent kept ineffectually low.

20.5 Prescribing under surveillance: antiepileptic levels and the EEG

The titration is carried out watched, not blind. Adding an antipsychotic to an established antiepileptic regimen is a two-way pharmacological event, and it is monitored on two fronts. As the antipsychotic is introduced and titrated, antiepileptic drug levels and the EEG should both be followed. The drug-level check exists because antipsychotics and antiepileptics interact: enzyme induction or inhibition can move an antiepileptic concentration out of its effective range and undo seizure control silently, and the detailed pharmacokinetics of those enzyme-induction and inhibition interactions are set out in their own topic rather than reopened here. The EEG check exists because the threshold effect can announce itself electrographically before it declares itself as a convulsion, so a worsening record is an early warning that the balance is tipping while there is still time to act. Together the two turn an otherwise blind escalation into a monitored one, and they give the clinician somewhere concrete to look when deciding whether a rising dose is safe to continue or whether it has begun to cost seizure control. The same surveillance frames the response to a breakthrough seizure: it is not automatically a reason to abandon the antipsychotic, but it is a reason to reconsider the whole balance of the regimen.

- Pick an agent from the low-risk tier unless there is a compelling reason not to.
- Start below the eventual target and titrate upward slowly.
- Follow antiepileptic drug levels as the antipsychotic is added.
- Watch the EEG for early threshold change before it becomes a fit.

20.6 When clozapine is unavoidable: the antiepileptic pairing

The one agent you least want in epilepsy is sometimes the only one that works. Treatment-resistant psychosis can leave **clozapine** as the only effective option even in a patient with epilepsy, and although clozapine sits at the seizure-provoking end of the ranking, refusing it outright may not be tenable when nothing else has worked. Its position on that ranking and the mechanism behind it are the business of the dedicated seizure-threshold topic, and its full haematological and cardiac profile with the associated monitoring belongs to the Schizophrenia: management, antipsychotics and adverse effects notes; neither is re-taught here. What this topic owns is the co-prescribing decision, and it has a clear answer. When clozapine must be used in epilepsy, **lamotrigine** is the antiseizure medication of choice to pair with it, and **carbamazepine** should be avoided. Carbamazepine is the wrong partner for two converging reasons: in combination it raises the risk of blood dyscrasias, stacking a haematological hazard onto an agent that already demands haematological vigilance, and it lowers clozapine levels, threatening to undercut the very efficacy that forced the choice of clozapine in the first place. Lamotrigine's own antiepileptic titration and rash are taught in their own topic, and its separate mood-stabiliser role

belongs to the Mood disorders: pharmacotherapy notes; here it is simply the safer antiepileptic companion for a difficult but sometimes unavoidable antipsychotic.

PITFALL

A common error when starting clozapine in a patient with epilepsy is to reach for carbamazepine as the antiepileptic cover, on the loose reasoning that it is also a familiar mood stabiliser and will do double duty. That choice doubles the blood-dyscrasia hazard and simultaneously drops clozapine levels, degrading safety and efficacy at the same time. Pair clozapine with lamotrigine instead, and keep carbamazepine well away from it.

20.7 Putting the choice into a management sequence

What to actually do, and in what order. The prescribing decision sits inside a broader management logic that is anchored, for the epilepsy itself, in the ILAE and NICE epilepsy guidelines, and for the psychiatric prescribing, in the epilepsy-specific psychiatric-management literature and the Maudsley Prescribing Guidelines. The governing principle comes first and is easy to state: optimise seizure control before any psychotropic is started, and if a seizure occurs after one has been introduced, either change the psychotropic or optimise the antiseizure medication. Before committing to an antipsychotic at all, the reversible and mimicking causes of the disturbance should be excluded, because a peri-ictal or iatrogenic psychosis is treated by addressing its cause rather than by adding a threshold-lowering drug on top of it.

The **LOCUS** of care follows the severity and the risk. A stable patient with a mild presentation can be managed as an outpatient with careful titration and review; a florid psychosis carrying behavioural risk warrants inpatient admission where dosing and seizures can be watched closely; an acutely disturbed patient may first present to an emergency setting. The **focus** shifts by phase. The acute target is control of the psychosis with the least threatening effective agent at a low starting dose. The short-to-mid-term target is titration to the chlorpromazine-equivalent dose the severity requires, carried out under antiepileptic-level and EEG surveillance. The long-term target is durable seizure control, which is the best protection against both further fits and recurrence of the psychosis, together with periodic review of whether the antipsychotic is still needed at all. The **modus** spans every mode of treatment. The pharmacological mode is the core of this topic: an agent chosen from the low-risk tier, introduced low and slow, with clozapine reserved and specifically paired with lamotrigine when it cannot be avoided. The procedural mode is the monitoring itself, the antiepileptic levels and the EEG that make a safe escalation possible, alongside optimisation of the antiepileptic regimen as the durable lever on the disorder. The social and psychological modes carry the rest: securing adherence, educating the patient and family about the interaction between the two treatments, and coordinating with the treating neurologist so that seizure control and psychiatric control are not pursued at cross purposes.

IN INDIA

Access shapes the choice as much as pharmacology does. Several of the preferred and low-risk agents, haloperidol and risperidone in particular, are inexpensive, widely stocked and familiar across public and private settings, which makes safe prescribing achievable even where budgets are tight; newer agents such as aripiprazole and paliperidone are available but cost more, so the low-cost members of the low-risk tier often become the practical default. Clozapine can be used but demands the haematological monitoring infrastructure that is easier to guarantee in tertiary centres than in district practice, so the threshold for choosing it, already high on seizure grounds, is higher still where reliable monitoring is hard to sustain. The pragmatic answer, a low-cost high-potency or low-risk second-generation agent, started low and titrated slowly with close attention to seizure control, fits Indian practice well and rarely requires anything exotic.

GOOD TO REMEMBER

When an epileptic psychosis needs an antipsychotic, default to one of the preferred agents, start at a low dose and titrate slowly, keep the seizures optimally controlled first, and monitor antiepileptic levels and the EEG as you go. Do not assume a second-generation drug is automatically the safer choice on the threshold, and never pair clozapine with carbamazepine.

TRAP **Assuming newer means safer on the seizure threshold.** Candidates reflexively pick a second-generation antipsychotic as the seizure-safe option, but efficacy does not separate the classes and some second-generation agents are worse on the threshold, while a high-potency first-generation agent such as haloperidol is among the safest. The generation of the drug does not predict its threshold liability; its specific place in the ranking does.

300 to 1,000	EEG + levels	near placebo
MG/DAY CPZ-EQUIVALENT, MILD TO MODERATE EPISODE	MONITOR DURING ANY ESCALATION, ABOVE ALL AT HIGH DOSE	ARIPIPRAZOLE SEIZURE INCIDENCE IN TRIALS

In epileptic psychosis the antipsychotics do not differ meaningfully in efficacy, so choose on the seizure threshold: prefer haloperidol, risperidone, paliperidone or aripiprazole, start low and titrate slowly under antiepileptic-level and EEG monitoring, optimise seizure control first, and if clozapine is unavoidable pair it with lamotrigine and avoid carbamazepine.

21 · Antipsychotic-induced lowering of seizure threshold (clozapine and others)

The drug that quiets the psychosis can uncover the seizure. Every clinician who starts an antipsychotic in a person with epilepsy, or in a person whose psychosis has arisen out of epilepsy, works against a built-in tension: the molecules that suppress positive symptoms also nudge the cortex toward its convulsive threshold. This topic is the mechanism behind that tension. The phenomenon is antipsychotic-induced lowering of the seizure threshold, and its hallmark is a dose-dependent seizure liability. It sets out what it means to say a drug lowers the **seizure threshold**, how far the effect depends on how the drug is given rather than on the drug alone, and which agents carry the liability most heavily. It earns marks because it is the single pharmacological principle that governs a whole cluster of downstream prescribing decisions, and

because the figures attached to the worst offenders are exactly the kind of discrete fact an examiner can ask for by name. The seizure threshold is a shorthand for the level of excitability at which a synchronised discharge becomes self-sustaining; anything that tips the balance of cortical excitation and inhibition can lower it, and most antipsychotics do so to some degree.

Two boundaries frame the discussion before it opens. First, this is the mechanism, not the prescription: which antipsychotic to actually select for a patient with epileptic psychosis, and how the ranking is weighed at the bedside, is developed in its own topic and is not reopened here. Second, where clozapine is concerned this account covers only its effect on the seizure threshold; its agranulocytosis, myocarditis and the blood monitoring that surrounds it belong to the Schizophrenia: management, antipsychotics and adverse effects notes. Keeping those two lines drawn stops the topic sprawling into the two that flank it and keeps the mechanism itself in sharp focus.

21.1 What lowering the seizure threshold actually means

A graded, dose-driven effect, not an on-off switch. The central fact of the whole topic is that antipsychotics lower the seizure threshold in a **dose-dependent** way, with the danger greatest when the drug is introduced quickly and driven to high doses. The distinction that carries the marks is that proconvulsant liability is not a fixed, all-or-nothing property of a molecule. The same agent given at a low dose reached slowly may sit almost inertly at the threshold, while the identical agent pushed rapidly to a high dose becomes meaningfully epileptogenic. Mechanistically the effect reflects a shift in the working balance between cortical excitation and inhibition, and it shows itself on the electroencephalogram before it shows itself as a fit: many antipsychotics produce dose-related background slowing, and at higher concentrations some provoke frank epileptiform discharges in susceptible brains. Kaplan and Sadock's Comprehensive Textbook of Psychiatry frames the whole class this way, as a graded liability rather than a categorical one, and the practical corollary is the reason the topic matters. If the effect scales with dose and with the speed of introduction, then a meaningful share of the risk is created by the way the drug is prescribed and not only by the drug that was chosen. That is what turns an apparently fixed pharmacological hazard into something the clinician can partly control. It also explains why two prescribers using the same agent in the same patient can generate quite different seizure risk, and why a blanket statement that a given antipsychotic is safe or unsafe, stripped of any reference to dose and titration, is close to meaningless.

21.2 The determinants you can change, and the ones you cannot

Rate and concentration are the modifiable levers. Drilling into the dose-dependence gives the practically useful version of the principle. The two determinants that a clinician can move are **rapid dose escalation** and the resulting high plasma concentration, and these are where the modifiable risk lives. Everything else is comparatively fixed: the intrinsic proconvulsant ranking of the particular agent, and the patient's own underlying epilepsy or predisposition. Reading the risk this way, split into what can be changed and what cannot, is what lets the pharmacology be turned into safer prescribing without needing any further numbers.

- Modifiable: how fast the dose is escalated, since a slow, stepwise build carries less risk than an abrupt loading schedule.
- Modifiable: the target dose and the plasma level it produces, since risk climbs with concentration.
- Fixed: the intrinsic liability of the chosen agent, which sets the floor of risk before any titration decision.
- Fixed: the patient's own seizure disorder, which is the substrate the drug acts upon.

RULE **Rate and dose, not the drug alone.** The seizure-threshold effect of an antipsychotic is dose-dependent and is worst with rapid introduction at high dose. The single safest manoeuvre with any agent is therefore to titrate slowly to the lowest effective dose, which is a lever that applies before the question of which molecule to use is even asked.

MNEMONIC

Dose, Rate, Drug, Disease

Four things set the seizure risk of an antipsychotic, and each is a fact this topic states. **D**ose - the higher the dose, the greater the risk. **R**ate - the faster the escalation, the greater the risk. **D**rug - clozapine leads the field for proconvulsant liability. **D**isease - a poorly controlled epilepsy is where the effect stops being academic.

21.3 The attribution problem: not every seizure is the drug

A seizure on treatment is not proof the treatment caused it. Before any antipsychotic is blamed for a fit, a confound has to be reckoned with, and it is one of the more sophisticated points the topic can be asked to make. The background incidence of **unprovoked seizures** in the general population runs at about **50 per 100,000 person-years**, but in the placebo arms of antipsychotic and antidepressant trials the seizure rate is roughly **15-fold higher** than that baseline, as the Maudsley Prescribing Guidelines set out. The people being treated with these drugs are, in other words, already at elevated risk before a single tablet is swallowed, because psychosis and depression are themselves seizure risk factors rather than neutral backgrounds. The implication for attribution is direct and clinically important: a proportion of the seizures that occur in patients on antipsychotics would have occurred anyway, driven by the illness and its correlates rather than by the medication. This does not exonerate the drugs, whose dose-dependent contribution is real, but it does mean that a seizure appearing during treatment cannot be read as automatic proof of drug causation. The honest position is that the drug adds to a risk that was already raised, and separating the two contributions in an individual patient is genuinely difficult.

PITFALL

The reflex when a patient has a seizure on an antipsychotic is to indict the drug and stop it. Because psychosis itself raises seizure risk well above the population baseline, that reflex over-attributes: some of these seizures would have happened without the medication, and abruptly withdrawing an effective antipsychotic to chase a seizure it may not have caused can leave the patient both psychotic and no safer. Weigh the dose, the titration speed and the underlying illness before pinning the event on the drug.

21.4 The proconvulsant ladder

A ranking from high liability to low. The antipsychotics can be arranged along a gradient of seizure-threshold-lowering potential, and the accompanying figure lays this ladder out from the highest-risk agents down to the lowest. Kaplan and Sadock's table gives the two ends explicitly. The high-potential tier gathers chlorpromazine, clozapine, thioridazine, perphenazine, olanzapine and quetiapine, agents that between them include the low-potency phenothiazines and the dibenzo-structured compounds. The low-potential end gathers fluphenazine, haloperidol, risperidone, paliperidone, aripiprazole, lurasidone, cariprazine and asenapine, along with loxapine, molindone and pimozide, a group weighted toward the high-potency first-generation drugs and most of the newer second-generation agents.

TIER ON THE LADDER	ANTIPSYCHOTICS	STRUCTURAL FLAVOUR
High seizure-threshold-lowering potential	chlorpromazine, clozapine, thioridazine, perphenazine, olanzapine, quetiapine	low-potency phenothiazines and dibenzo compounds cluster here
Low seizure-threshold-lowering potential	fluphenazine, haloperidol, risperidone, paliperidone, aripiprazole, lurasidone, cariprazine, asenapine (with loxapine, molindone and pimozide)	high-potency first-generation agents and most newer drugs cluster here

The seizure-threshold-lowering ladder as set out in Kaplan and Sadock's table, from highest to lowest potential; the tiers, not a precise rank order within each tier, are what carry the teaching point.

The ladder is worth committing to memory as a shape rather than as a rote list, because its two ends behave differently and because the placement of certain agents is exactly where the sources part company. The structural pattern helps: the drugs at the high end are dominated by the sedating, low-potency and tricyclic-shaped compounds, while the high-potency agents and most of the newer molecules sit lower. That heuristic is a scaffold for recall, not a substitute for the table, and the exceptions to it are the substance of the next two sections.

21.5 Clozapine: the agent at the top

The most proconvulsant antipsychotic, and dose tells the tale. If a single agent has to be named as the seizure-threshold offender, it is clozapine, which the Maudsley Prescribing Guidelines identify as the **most proconvulsant** antipsychotic in use. The quantitative anchor is that clozapine induces seizures in **1% to 4.4%** of the patients who take it, and the spread of that range is itself instructive: the risk sits toward the lower bound with careful, gradual dosing and

rises toward the upper bound when the dose is increased rapidly. Clozapine thus reproduces the whole topic in one molecule. It is the drug whose intrinsic liability is highest, and it is also the drug in which the dose-dependence and the danger of quick escalation are most vividly demonstrated, which is why its titration is conventionally slow and stepwise. Two cautions attach to teaching it. The first is a boundary: the seizure-threshold angle is all that is owned here, and clozapine's fuller adverse-effect profile, its haematological monitoring and its other hazards, belongs to the Schizophrenia: management, antipsychotics and adverse effects notes and should be cross-referred rather than repeated. The second is that a seizure on clozapine is not by itself a reason to abandon the drug in a patient who needs it; it is a reason to review the dose, the plasma level and the speed of the recent titration, since those are the modifiable determinants the mechanism has already identified.

21.6 Chlorpromazine and loxapine: the first-generation flags

Two older agents that punch above their apparent place. Among the first-generation antipsychotics two names deserve to be flagged in their own right. Chlorpromazine, the archetypal low-potency phenothiazine, carries a clear dose-threshold effect: doses above **1 g per day** are associated with a seizure incidence of **9%**, a figure high enough that high-dose chlorpromazine is best avoided in anyone with a seizure tendency. Loxapine is the more interesting teaching case, because it is where two authorities pull apart. On Kaplan and Sadock's ladder loxapine sits in the low-potential tier, yet the Maudsley singles it out as the first-generation antipsychotic carrying the **highest seizure rate** of its class. Both statements come from reputable sources and both are worth knowing; the resolution is not to decide which is right but to recognise that loxapine, structurally a dibenzoxazepine and so a chemical cousin of the high-risk dibenzo compounds, is an agent about which the sources genuinely disagree, and to treat it with corresponding caution. The general lesson the two drugs teach together is that structural family and dose, not the drug's marketing generation, predict seizure liability.

IN INDIA

Cost and availability keep the older low-potency antipsychotics, chlorpromazine foremost among them, in far heavier routine use in much of Indian practice than in settings where newer agents are the default. That matters directly for this topic, because it is precisely these low-potency phenothiazines that occupy the high end of the seizure-threshold ladder, and because high total daily doses are still encountered where a cheaper drug is pushed rather than a more expensive one substituted. The **9%** seizure incidence attached to chlorpromazine above **1 g per day** is therefore not a historical curiosity here but a live prescribing consideration. A related caution is that co-prescribed antiepileptic drugs alter antipsychotic plasma levels through enzyme effects, a pharmacokinetic interaction that is covered in its own topic and that further complicates the dose-driven risk in exactly the patients most likely to be on both.

21.7 The second-generation gradient and why the ladder is source-dependent

The ranking is a guide, not a law. The newer antipsychotics do not lower the seizure threshold uniformly, and the same discrepancy that surfaced with loxapine reappears among them, which is what makes this the most examinable subtlety of the topic. Olanzapine and quetiapine sit at the

high end of Kaplan and Sadock's ladder, yet the Maudsley, weighing the randomised-trial data, judges their overall proconvulsant risk to be **low-to-moderate**, with the olanzapine evidence frankly mixed, since both anticonvulsant and proconvulsant reports exist for it. The point is not that one source is mistaken. It is that seizure-threshold rankings are assembled from heterogeneous evidence, case reports, trial adverse-event data and pharmacological reasoning, and different authorities weight those sources differently, so the same agent can legitimately appear in a high tier on one ladder and a lower band on another. A candidate who has memorised a single ranking and recites it as though it were fixed will be caught out by exactly these agents. The defensible position is to know the shape of the ladder, to know its firm anchors, clozapine at the top and the high-potency first-generation drugs low down, and to treat the placement of olanzapine and quetiapine as genuinely source-dependent rather than settled.

TRAP **Reciting one ladder as gospel.** The classic error is to learn a single seizure-threshold ranking and answer as though every agent's place were fixed. The sources disagree at specific rungs: loxapine is low-potential on one ladder but the highest-risk first-generation drug on another, and olanzapine and quetiapine sit high on one and low-to-moderate on the other. Name the disagreement rather than picking a side, and anchor the answer on the points that do not move.

21.8 How much it matters in the patient with epilepsy

Usually subclinical, decisive in the poorly controlled. The final calibration is the one that stops the topic being alarmist. For most patients the seizure-threshold-lowering effect of an antipsychotic is real but not clinically significant; it is a measurable pharmacological property that never translates into an actual fit. Where it changes from a footnote into a genuine hazard is in the patient whose epilepsy is already **poorly controlled**, in whom the drug's contribution is added to a threshold that is already low and a brain already prone to discharge. Kaplan and Sadock make exactly this qualification, that the effect is usually subclinical but can reach clinical significance in the setting of poorly controlled epilepsy. That single distinction organises the whole clinical stance. In a person with no seizure history and well-controlled or absent epilepsy, the threshold effect rarely dictates practice on its own; in a person with active, poorly controlled seizures it becomes one of the real constraints on treatment, and it is the reason the choice of agent and its titration are handled with such care in that population. The mechanism laid out across this topic, dose-dependence, the modifiable levers of rate and concentration, the ladder with clozapine at its head, and the attribution confound, all converges on this practical endpoint: the same drug is a minor consideration in one patient and a major one in another, and it is the state of the underlying epilepsy that decides which.

1 to 4.4%	9%	~50/100k/yr	~15-fold
CLOZAPINE SEIZURE INCIDENCE	CHLORPROMAZINE ABOVE 1 G/DAY	BASELINE UNPROVOKED-SEIZURE RATE	HIGHER IN TRIAL PLACEBO ARMS

GOOD TO REMEMBER

When you add an antipsychotic to someone with epilepsy or a prior seizure, the most useful thing you control is not always the molecule but the manner: build the dose slowly to the lowest effective level, since the effect is dose-dependent and worst with rapid escalation. Keep clozapine and high-dose low-potency phenothiazines in mind as the high-liability end, remember that a seizure on treatment may reflect the illness as much as the drug, and leave the actual agent selection for the epileptic-psychosis patient to the topic that owns it.

Antipsychotics lower the seizure threshold in a dose-dependent way that is worst with rapid escalation and high doses; the effect is greatest with clozapine and with high-dose low-potency agents, is usually subclinical, and becomes clinically important mainly in poorly controlled epilepsy.

Mood, anxiety and suicide in epilepsy

22 · Depression in epilepsy: prevalence, phenomenology and screening

Depression is the disorder that most often accompanies epilepsy, and the one most often overlooked. Of all the psychiatric problems a person with epilepsy may carry, depression is at once the most frequent, the most damaging to the very quality of life that seizure treatment is meant to protect, and the most easily written off as an understandable reaction to a difficult illness. Together with anxiety it is one of the two most frequently encountered interictal psychiatric disorders of epilepsy, the anxiety half of that pair being developed in its own topic in this chapter. The examinable material falls into three blocks that map cleanly onto what a competent clinician actually does: knowing how common depression really is and why the published figures scatter so widely, recognising that its presentation is frequently atypical and therefore does not announce itself, and screening for it deliberately rather than waiting for the patient to complain.

This topic earns marks out of proportion to its apparent simplicity because almost every part of it runs against a clinician's first instinct. The rates are higher than intuition suggests, the phenomenology is stranger than the textbook portrait of major depression, and the condition erodes daily functioning more than the fits themselves, yet it is the fits that dominate the consultation. The wider two-way traffic between epilepsy and psychiatric illness, in which each raises the risk of the other, is set out in its own dedicated topic in this chapter and is assumed here; this topic narrows to the depression that arises in the interictal state, drawing on Lishman's Organic Psychiatry and Kaplan and Sadock's Comprehensive Textbook of Psychiatry.

22.1 The disorder that owns the mood picture

Commonest, and the single heaviest drag on quality of life. The reason depression sits at the head of this band is not only that it is frequent but that it is disproportionately disabling. In patients whose seizures continue, depression is the single most important predictor of poor

quality of life, weighing more heavily on how the patient actually lives than seizure frequency, seizure severity or the burden of medication. That fact reorganises the whole clinical priority list. Epilepsy services are built to chase seizure control, and seizure freedom is the outcome the clinic measures itself by, yet a patient who has been rendered seizure-free can remain profoundly unwell if a coexisting depression goes unrecognised, while a patient with occasional seizures and good mood may function far better than the seizure count predicts. The tragedy compounded onto this is that the depression is routinely neither recognised nor treated, precisely because its atypical form keeps it below the clinician's threshold of suspicion. The mood disorder that does the most damage is therefore also the one most likely to be missed, and that pairing is the core of why the topic is worth teaching and worth examining.

RULE **Depression is the commonest interictal psychiatric disorder in epilepsy and the strongest single determinant of poor quality of life, yet it is routinely under-recognised because it seldom looks like textbook major depression.** Every clinical move that follows, from active screening to treating mood as seriously as seizures, descends from that one sentence. Seizure control is necessary but not sufficient for a good outcome.

22.2 How common it is, and why the figures scatter

The rate you quote depends entirely on how depression was defined and where the sample came from. No single prevalence figure for depression in epilepsy is defensible, and a candidate who offers one is usually offering a wrong one. The reported rates swing across an enormous range, and the swing is not noise but information: it tracks three things that must be held apart. The first is the level of the phenomenon being counted, since **depressive symptoms** on a self-report scale are far more common than a diagnosable **major depression** confirmed at structured interview. The second is the sampling frame, which climbs from unselected community cohorts, through neurology and specialist epilepsy clinics, to the highly selected presurgical populations of patients with medically refractory temporal lobe epilepsy, each tier concentrating the sickest and most treatment-resistant patients and so lifting the rate. The third is the diagnostic instrument and the window of time it interrogates. The accompanying table lays these out so the ascending gradient is visible rather than mysterious. Read that way, the figures are coherent: symptom-level counts in the community sit low and wide, clinic-based major depression sits in the middle, and lifetime rates in presurgical refractory disease sit highest, with major depressive episodes across pooled series reaching **up to 20 per cent**. The lesson to carry into the exam is comparative and mechanistic, not a single memorised percentage.

POPULATION AND SETTING	HOW DEPRESSION WAS DEFINED	REPORTED RATE
Community-based samples	depressive symptoms (not a diagnosed disorder)	4 to 37 per cent
Specialist epilepsy clinics	ICD or DSM major depression	17 to 21 per cent
Presurgical, medically refractory TLE	**lifetime prevalence** of major depression	24 to 35 per cent
Mixed epilepsy series (point and period)	DSM major depression	3 per cent over the past month, rising to 22 per cent over the previous year
Pooled across studies	any depression	7.5 to 34 per cent

Prevalence of depression in epilepsy sorted by the definition used and the population sampled; the spread is driven by measure, definition and sampling, not by genuine disagreement about the disease.

MNEMONIC

The three P's

Depression in epilepsy is **P**revalent (the commonest interictal psychiatric disorder), **P**leomorphic (atypical, frequently short of standard criteria), and **P**icked up only if you screen (it is rarely volunteered and easily missed). Each of the three is a fact this topic actually establishes, and together they are the spine of a complete answer on prevalence, phenomenology and detection.

22.3 It is unipolar depression, not bipolarity

The mood disorder of epilepsy is depressive; bipolar disorder is not over-represented. A neat and examinable negative sits alongside the prevalence data, and it is worth stating explicitly because intuition and older limbic theorising both pull the other way. Given the temporolimbic disturbance that underlies so much of the psychiatry of epilepsy, a candidate might expect an excess of bipolarity, but the evidence does not support it: bipolar disorder is not over-represented in epilepsy, and even in the enriched presurgical series where every psychiatric rate is inflated, its prevalence has been reported at **below 1 per cent**. The practical consequence is that a patient with epilepsy who presents with low mood should be worked up as a unipolar depression until there is specific evidence otherwise, and the reflexive reach for a mood-stabiliser framing on the strength of the epilepsy alone is unjustified. The corollary, that peri-ictal and interictal mood phenomena can nonetheless look labile and episodic, belongs to the phenomenology below and to the separate peri-ictal topics of this chapter, and must not be confused with a primary bipolar diathesis.

22.4 Depression tracks seizure control

In community samples, depression rises steeply as seizure control worsens. One of the most robust and most useful findings in this area is a dose-like relationship between how well the epilepsy is controlled and how much depression the population carries. In community-based work using the **Hospital Anxiety and Depression Scale**, on which each of the two seven-item subscales runs **0 to 21** with 0 to 7 read as non-case, 8 to 10 as borderline and 11 to 21 as a probable case, the proportion with significant depression climbs across bands of worsening

seizure frequency, and a second independent community series reproduced the same gradient with different absolute figures, which is exactly the kind of replication that turns an association into a fact worth teaching. The gradient makes intuitive sense: continuing seizures mean continuing unpredictability, injury, restriction and loss of autonomy, and the reactive burden mounts accordingly, while the same uncontrolled disease may also reflect the more extensive brain disturbance that independently generates mood symptoms. There is an important qualification, however, and it is examinable. The gradient is a feature of community and unselected samples; in tertiary referral populations it is not seen, because such samples are so concentrated with refractory, severely affected patients that the range of seizure control collapses and the relationship can no longer show itself. The accompanying table gives the community gradient.

SEIZURE CONTROL	SIGNIFICANT DEPRESSION
Seizure-free	4 per cent
Fewer than monthly seizures	10 per cent
More frequent seizures	21 per cent

Significant depression by seizure frequency in a community series (Hospital Anxiety and Depression Scale); a second community series reproduced the same rising gradient with figures of about 6, 11 and 33 per cent. The gradient is not seen in tertiary referral samples.

22.5 Who is most at risk

Focal temporolimbic epilepsy and poor seizure control carry the highest mood-disorder rates. Beyond the seizure-control gradient, a set of correlates identifies the patients in whom depression is most likely and in whom the clinician should look hardest. The strongest structural association is with **temporolimbic** epilepsy: focal impaired-awareness seizures of mesial-temporal origin, together with poor seizure control, are associated with higher rates of mood disorder. A left-hemisphere focus has been linked to greater depression, but this must be stated with the caution the source literature attaches to it, namely that any laterality effect is small and inconsistent, so it is a soft correlate rather than a rule to hang a diagnosis on. The seizure-type and localisation details themselves are taught in the temporal lobe and seizure-classification topics of this chapter and are named here only as risk markers. The correlates are best held as a short list, because in practice they braid together and each points to a different lever in formulation.

- Poor seizure control, the single most consistent and modifiable correlate.
- A temporolimbic, mesial-temporal focal onset, the strongest structural association.
- A left-hemisphere focus, a weak and inconsistent correlate not to be overstated.
- Antiepileptic drug treatment itself, an iatrogenic contribution whose detail belongs to the topic on drug-attributable mood and suicidality in this chapter.
- The psychosocial weight of stigma, unpredictability and lost independence, the reactive strand that no drug will reach.

22.6 Phenomenology: atypical and pleomorphic

It rarely reads as textbook major depression. The defining clinical feature of depression in epilepsy, and the one most likely to be tested, is that its presentation is frequently **atypical** and **pleomorphic**, so that a large proportion of cases fall short of the standard diagnostic criteria for a major depressive episode. Estimates that some **30 to 70 per cent** of depressive presentations in epilepsy show atypical features capture how far this departs from the classical picture. Instead of the sustained, criterion-complete syndrome of persistent low mood, anhedonia and neurovegetative disturbance, the mood disturbance of epilepsy is more often intermittent and fluctuating in its course, coloured by prominent irritability, and marked by endogenous somatic symptoms that shift and recur rather than settling into a single sustained episode. Because the syndrome is broken up and irregular, it slips through diagnostic instruments built to detect a continuous two-week episode, and it is easy for both patient and clinician to treat each fragment as a passing bad patch rather than a disorder. There is a recognised, named syndrome that formalises this atypical, chronic and pleomorphic interictal mood picture, interictal dysphoric disorder, but it is developed in full in its own dedicated topic in this chapter and is named here only to mark that the atypicality has been given a distinct diagnostic identity; its criteria and mechanism are not reopened here.

TRAP **Expecting the textbook major depressive episode and dismissing the diagnosis when it is not there.** Candidates and clinicians alike anchor on the DSM or ICD picture of a sustained two-week syndrome, and because the depression of epilepsy is intermittent, irritable and criterion-incomplete, they conclude the patient is not depressed. The examinable point is the opposite: an atypical, fluctuating, irritable mood disturbance that does not meet full criteria is the characteristic form here, not evidence against the diagnosis.

PITFALL

A common clinical error is to attribute the patient's low mood, fatigue, poor concentration and irritability to the epilepsy itself, to the sedating effect of antiepileptic drugs, or to the understandable distress of living with fits, and therefore to observe rather than treat. Each explanation may hold a grain of truth, but used as a reason not to act it leaves a treatable and disabling disorder to run. Name the depression before you explain it away.

22.7 Screening and detection

Because it is missed, it must be screened for, and there is an epilepsy-specific tool to do it. Everything above converges on a single practical conclusion: a disorder this common, this disabling and this atypical will not be caught by waiting for the patient to volunteer it, so detection has to be active and systematic. Two features make routine screening especially valuable in epilepsy. The atypical, intermittent phenomenology means the diagnosis is easily overlooked in an ordinary consultation dominated by seizures and medication, and the somatic overlap between depressive symptoms, seizure aftermath and drug side effects muddies clinical judgement. A structured screen cuts through both. The instrument developed for exactly this purpose is the **Neurological Disorders Depression Inventory for Epilepsy**, a brief screening tool designed for and validated in people with epilepsy, and constructed so that its items are not

confounded by the somatic and cognitive effects of seizures and antiepileptic drugs in the way generic depression scales can be. It is a six-item scale scored **6 to 24**, and a total **above 15** is a positive screen for major depression, a threshold chosen to be highly specific, at about 90 per cent, at acceptable sensitivity. A positive screen is a prompt to a full diagnostic assessment, not a diagnosis in itself. Once depression is confirmed, two onward questions arise that this topic hands to their owners: which antidepressant to choose against the drug's effect on the seizure threshold, which is the subject of its own dedicated topic in this chapter, and the assessment of the heightened suicide risk that depression in epilepsy carries, which likewise has its own dedicated topic. This topic ends at reliable detection, which is where the marks for depression in epilepsy are concentrated.

GOOD TO REMEMBER

Screen every patient with epilepsy for depression at routine review, not only those who look low, because depression predicts how the patient lives more powerfully than seizure frequency does. A seizure-free patient can still be doing badly, and the only way to know is to ask in a structured way rather than to infer mood from the seizure chart.

IN INDIA

In high-volume neurology and psychiatry clinics, where a consultation may last only a few minutes and is understandably consumed by seizures and drug supply, depression in epilepsy is especially easy to miss, which is exactly where a short, epilepsy-specific screen earns its place. Detection matters all the more because access to structured psychological therapy is limited outside metropolitan and tertiary centres, so recognising the disorder and initiating treatment often falls to the treating physician. Stigma compounds the picture: the social consequences of epilepsy for employment and marriage feed low mood directly, and a patient may present the depression as a somatic complaint rather than name it. The iatrogenic contribution of older, widely used antiepileptic drugs to mood is real but belongs to the drug-attributable topic of this chapter and is only flagged here.

22.8 Pulling the picture together

One disorder, higher than you think, stranger than you expect, and quiet unless you ask. The topic resolves into a short chain that is easy to reproduce under pressure. Depression is the commonest interictal psychiatric disorder in epilepsy and the strongest single predictor of poor quality of life, so it deserves the clinical priority that instinct gives to the seizures. Its prevalence appears to scatter wildly only until the scatter is decoded as the product of measure, definition and sampling, at which point the ascending gradient from community symptoms to presurgical major depression becomes orderly and the seizure-control gradient becomes intelligible. The mood disorder is unipolar, not bipolar, and it is frequently atypical and pleomorphic rather than criterion-complete, which is the fact that both explains why it is missed and warns against dismissing it when full criteria are absent. And because it is missed, the clinical answer is to screen actively, with an epilepsy-specific instrument built to see past the somatic noise, handing the confirmed case onward to the treatment and suicide-risk topics that own those questions.

commonest	17 to 21%	24 to 35%	top predictor
INTERICTAL PSYCHIATRIC DISORDER	MAJOR DEPRESSION, EPILEPSY CLINICS	LIFETIME, PRESURGICAL TLE	OF POOR QUALITY OF LIFE

Depression is the commonest and most disabling interictal psychiatric comorbidity of epilepsy, is unipolar rather than bipolar, is frequently atypical and criterion-incomplete rather than textbook major depression, and is missed unless it is actively screened for with an epilepsy-specific instrument such as the Neurological Disorders Depression Inventory for Epilepsy.

23 · Suicide risk in epilepsy and contributing factors

People with epilepsy take their own lives more often than the general population does, and the whole of this topic is an exercise in holding that fact firmly without inflating it. Suicide is one of the genuine hazards of living with epilepsy, and the excess appears to be **intrinsic to epilepsy** itself rather than merely a by-product of the drugs used to treat it: the risk is raised above that of the general population, a burden the disease carries in its own right, though the reported rates vary widely with the population studied. That last distinction is where marks are won and lost, because a separate and quite different suicidality signal attaches to the antiseizure drugs as a class, and it is developed under its own dedicated topic in this chapter. This topic owns the disease-intrinsic risk; the drug-attributable warning is not reopened here, and a reader who leaves with the two collapsed into one has missed the central discrimination the examiner is looking for.

The examinable material divides into three questions that a competent clinician actually asks. The first is how large the excess really is, which turns out to be one of the most contested figures in the whole of epilepsy psychiatry, with reputable estimates ranging from a several-fold increase down to none at all. The second is where suicide sits within the wider excess mortality of epilepsy, which is best read through an all-cause mortality statistic rather than any single suicide figure. The third, and the most clinically useful, is which patients actually carry the risk, and the striking, repeatedly examined answer is that it is features of the person and their history, not features of the epilepsy, that predict it. The source throughout is Lishman's Organic Psychiatry, and the honest posture is to quote the few established figures precisely and to reason carefully around the uncertainty rather than paper over it.

23.1 A real risk, and why it belongs to the disease

Suicide is a bona fide cause of premature death in epilepsy, and the excess is the disease's own. Before arguing about the size of the risk it is worth being clear that the direction is not in doubt. People with epilepsy are, taken as a whole, more likely to die by suicide than their peers without epilepsy, and this holds even when the contribution of any particular drug is set aside, which is what it means to call the risk intrinsic. Several strands plausibly converge to produce it. Epilepsy carries a heavy load of psychiatric comorbidity, with depression the commonest interictal disorder and a powerful driver of hopelessness, and that mood picture is developed in

its own dedicated topic in this chapter. The temporolimbic disturbance that underlies so much of the psychiatry of epilepsy may itself lower the threshold for despair and impulsive self-harm. Layered onto the neurobiology is a formidable psychosocial burden: the unpredictability of the fits, the loss of autonomy and driving, the constraints on employment and marriage, and the stigma that still attaches to the diagnosis, all of which feed a reactive demoralisation that no anticonvulsant reaches. The clinical consequence is that suicide risk in epilepsy cannot be reasoned about as though it were purely reactive or purely organic; it is both, and the assessment has to look at the person as well as the seizures.

RULE **Suicide risk is genuinely elevated in epilepsy and the elevation is intrinsic to the disease, but the size of the excess is contested and the risk is carried by the patient's psychiatric history, not by any feature of the epilepsy itself.** Every clinical move that follows descends from that one sentence: assess the person, not the seizure type, and keep the disease-intrinsic risk separate from the drug-attributable signal owned elsewhere.

23.2 How large the excess is - the figures that will not agree

The reported magnitude swings from a large multiple to nothing, and the swing is information, not noise. No single relative-risk figure for suicide in epilepsy is defensible, and a candidate who offers one confidently is usually offering a wrong one. The disagreement in the literature is real and instructive. An early and much-cited clinic-based review put the excess at **roughly fivefold**, rising to as much as 25-fold in the most highly selected samples such as specialist referral and presurgical populations. Set against that, two **population-based** mortality studies found no excess suicide risk at all. The two sets of numbers are not so much contradictory as answers to different questions. Estimates built from selected clinic and referral cohorts concentrate the sickest, most refractory and most psychiatrically comorbid patients, and in that enriched group the suicide rate is genuinely high; population-based samples dilute those patients back into the whole epilepsy population and the signal shrinks, sometimes to invisibility. The accompanying table sets the two poles side by side. The lesson to carry into the examination is comparative and mechanistic: name the range, explain that selection drives it, and do not commit to a single multiplier as though the field had settled on one.

SOURCE OF THE ESTIMATE	REPORTED SUICIDE RISK VERSUS THE GENERAL POPULATION
Early clinic-based and selected-sample reviews	about fivefold, up to 25-fold in the most highly selected samples
Two population-based mortality studies	no increase over the general population

The two poles of the suicide-risk literature in epilepsy. The discordance is driven by how the sample was assembled, not by genuine disagreement about the disease: selected clinic cohorts concentrate the highest-risk patients, while population-based studies dilute them.

23.3 Suicide within the excess mortality of epilepsy

Read the burden through the all-cause mortality statistic, and keep suicide as one contributor among several. A cleaner way to situate suicide is to step back to the overall mortality of epilepsy, which is unambiguously raised. Expressed as a **standardised mortality**

ratio, the death rate of people with epilepsy runs at about **2 to 3** times that expected in the general population. Crucially, that figure is all-cause, and suicide is only one of several contributors to the excess mortality, alongside the underlying cause of the epilepsy, accidental injury, status epilepticus and sudden unexpected death in epilepsy. Holding the statistic in that form does two useful things. It anchors the reality of premature death in epilepsy in a robust, population-level number rather than the wobbly suicide multiples of the previous section, and it forces the discipline of attribution: the raised overall mortality is not a suicide figure, and the raised suicide risk is not the whole of the raised mortality. Status epilepticus and sudden unexpected death in epilepsy are substantial causes in their own right and are not developed here. The examinable point is proportion and provenance: suicide is a real and consistent slice of the excess mortality, but it is a slice, and the standardised mortality ratio is the frame that keeps the slice honest.

TRAP **Quoting the standardised mortality ratio of 2 to 3 as if it were the suicide risk.** It is not. That ratio is the all-cause excess mortality of epilepsy, of which suicide is one contributor among several. Candidates who have memorised the number attach it to whatever the question asks about, and here they misread an all-cause statistic as a suicide-specific one. The two are different measures and the examiner is testing whether you know which is which.

23.4 A history of attempted suicide is commoner too

Non-fatal self-harm shadows the completed-suicide picture, with the same caveats about how it was counted. The excess is not confined to completed suicide. A past history of attempted suicide is about four times more common in people with epilepsy than in the general population, which matters clinically because a previous attempt is one of the most powerful predictors of an eventual completed suicide in any population and, as the next section shows, in epilepsy specifically. The figure has to be handled with the same care as the completed-suicide multiples, because it comes from survey data with real selection-bias limitations: only about one-third of the questionnaires were returned, and non-responders in a survey of this kind may differ systematically from responders in ways that pull the estimate up or down. The direction is nonetheless the clinically important message, and it is consistent with everything else in this topic. A raised prevalence of prior attempts tells the clinician that self-harm is part of the lived texture of epilepsy for a meaningful minority, and it flags the single item of history that most deserves to be asked about at every review, because it converts an abstract population risk into an individual one sitting in front of you.

23.5 The factors that actually carry the risk

The risk lives in the patient's mind and history, and its strongest markers are the ones that predict suicide anywhere. This is the heart of the topic and the part most worth memorising, because it converts a contested epidemiology into an actionable risk assessment. In a case-control study of suicide in epilepsy, a **psychiatric history** conferred an **about ninefold** increase in suicide risk, and when the analysis was reduced to essentials, a psychiatric history and a **previous suicide attempt** emerged as, by a wide margin, the two most important risk factors for completed suicide. A second, more epilepsy-specific marker sits alongside them: **early-onset**

epilepsy markedly raises the risk, with onset before 18 years, set against onset after 29 years, carrying a **relative risk of 16**, though with a wide confidence interval (4.4 to 58.3) that reflects how few events anchor the estimate; onset in adolescence appears particularly important. The accompanying table lays the three out together. What unifies them is that the first two are exactly the factors that predict suicide in the general population, which is a reassuring internal consistency, while the third is the one genuinely epilepsy-linked contributor and a reminder that a diagnosis made in a developing adolescent brain, at a formative point for identity and independence, carries a weight the same diagnosis made in mid-life does not.

CONTRIBUTING FACTOR	EFFECT ON SUICIDE RISK
A psychiatric history	about a ninefold increase
Onset of epilepsy before 18 years (adolescence particularly)	relative risk of 16, with a wide confidence interval
A previous suicide attempt	one of the two most important factors, with psychiatric history

The contributing factors that carry the elevated suicide risk in epilepsy. Two of the three are the classic general-population predictors of suicide; the third, early age at onset, is the epilepsy-specific contributor. The variables that do not predict suicide are set out in the following section.

MNEMONIC

PAY attention to the person, not the fits

The three factors that raise suicide risk in epilepsy are a **P**sychiatric history, a prior **A**ttempt, and a **Y**oung age at onset. All three are features of the patient and their history, which is precisely why the epilepsy's own characteristics add nothing to the prediction. If you remember to look at the person rather than the seizure chart, you have the risk assessment.

GOOD TO REMEMBER

At every review of a patient with epilepsy, ask directly about past and present psychiatric illness and about any previous suicide attempt, because those two questions, not the seizure type or the drug chart, are what actually stratify suicide risk. Documenting that you asked, and asking again when mood or circumstances change, is the single highest-yield thing this topic asks you to do on a Monday morning.

23.6 The variables that do not predict it

An examinable set of negatives: the epilepsy's own features are largely silent on suicide risk. Just as important as knowing what raises the risk is knowing what does not, because intuition and older theorising both push clinicians to over-read the seizure characteristics. The same case-control work found no consistent relationship between suicide and the type of epilepsy, its localisation or lateralisation, the presence of a neurological deficit, or any specific antiepileptic drug. A trend towards more frequent seizures and polytherapy fell short of statistical significance, so neither can be asserted as an established risk factor. The message is disciplined and counterintuitive. A left temporal focus, a refractory course, a heavy drug burden - the features a

clinician instinctively treats as ominous - do not, on this evidence, mark out the patients who will die by suicide. The seizure-type and localisation detail belongs to the classification and temporal-lobe topics of this chapter and is named here only to be set aside as non-contributory. The list below is the compact form worth reproducing under pressure.

- The type of epilepsy (focal versus generalised, and syndrome).
- The localisation or lateralisation of the focus.
- The presence of a neurological deficit.
- Any specific antiepileptic drug taken for seizure control.
- Seizure frequency and polytherapy, where a trend was seen but did not reach significance.

PITFALL

The clinical error this section guards against is assuming that good seizure control equals safety. Because suicide risk tracks the psychiatric history and prior attempts rather than the seizures, a patient rendered seizure-free can remain at substantial risk, and a clinic congratulating itself on seizure freedom may quietly stop asking about mood. Seizure control is necessary for a good outcome but is not sufficient for safety, and the risk assessment must run independently of the seizure chart.

23.7 Turning the risk picture into clinical action

Everything above converges on a single practical instruction: assess and treat the person, not the seizure type. The management of suicide risk in epilepsy is not a distinct pharmacology but a disciplined application of general suicide-prevention practice to a higher-risk group, framed by the epilepsy standards of the International League Against Epilepsy and NICE and by the epilepsy-psychiatry management literature synthesised in Lishman's Organic Psychiatry, which together set optimisation of seizure care and active attention to mental health as twin obligations. The **LOCUS** of care is overwhelmingly outpatient, woven into routine epilepsy or psychiatric follow-up, and escalates to the emergency department or inpatient admission only when active suicidal ideation with intent or a recent attempt makes immediate safety the priority, at which point management becomes that of any acute suicidality. The focus shifts by phase: the acute task is to detect and contain active ideation and to secure safety; the short and mid-term task is to treat the comorbid psychiatric disorder that is driving the risk, most often depression, and to keep the seizures well controlled; and the long-term task is to sustain vigilance across a chronic, relapsing illness rather than assess risk once and file it. The modus spans every available mode without changing the antiseizure regimen for the sake of the mood. The drug mode is the vigorous treatment of the underlying psychiatric illness, with the specific question of which antidepressant to choose against the seizure threshold handed to its own dedicated topic in this chapter. The psychological mode is structured risk assessment and collaborative safety planning. The social mode is family involvement, means restriction and reliable continuity of follow-up. The elements below are the workable core.

- Ask every patient about psychiatric history and any previous suicide attempt, and re-ask when circumstances change.
- Treat comorbid depression and other psychiatric illness actively rather than dismissing low mood as understandable.
- Restrict access to means and engage the family as partners in monitoring between appointments.
- Keep seizure control optimised while remembering it does not, by itself, reduce suicide risk.

IN INDIA

The risk assessment matters all the more where follow-up is hurried and access to structured psychiatric care is thin outside tertiary centres, so the treating physician often has to carry the mental-health task alone. Means restriction has a specific local shape: ready access to pesticides and other agricultural poisons in rural settings makes counselling the family about safe storage a concrete and potentially life-saving intervention rather than an abstraction. Stigma around both epilepsy and suicide runs deep and makes families slow to volunteer a change in mood or a past attempt, so the questions have to be asked directly, in the patient's own language, and an accessible contact route, often a phone or messaging line to the treating team, is a more realistic safety net than a distant review date.

23.8 Pulling the picture together

A real excess, an unsettled size, and a risk that sits in the person. The topic resolves into a short chain that is easy to reproduce under pressure. Suicide risk in epilepsy is genuinely elevated and the elevation is intrinsic to the disease, but it must be kept apart from the separate drug-attributable suicidality signal taught elsewhere in this chapter. The size of the excess is one of the field's most contested figures, running from a large multiple in selected clinic samples to nothing at all in population-based studies, and the discordance is explained by selection rather than by any real disagreement about the disease. The overall mortality of epilepsy is unambiguously raised, and suicide is one consistent contributor to that excess among several, best read through an all-cause statistic that must not be misquoted as the suicide risk itself. And the contributing factors that actually predict completed suicide are a psychiatric history and a previous suicide attempt, joined by early age at onset, while the type of epilepsy, its localisation, the neurological deficit and the specific drug do not predict it. Assess the person, treat the comorbidity, and never let seizure freedom stand in for safety.

Elevated	Contested	Psychiatric history	Not the epilepsy
AND INTRINSIC TO THE DISEASE	CLINIC-HIGH, POPULATION-NULL	THE DOMINANT RISK FACTOR	TYPE, FOCUS AND DRUG DO NOT PREDICT

Suicide risk in epilepsy is genuinely elevated and intrinsic to the disease, though its size is contested from a several-fold excess down to none; the risk is carried by a psychiatric history and a previous suicide attempt, joined by early age at onset, and not by the epilepsy type, localisation, neurological deficit or specific drug, and it must never be conflated with the all-cause standardised mortality ratio or the separate drug-attributable suicidality signal.

24 · Anxiety disorders in epilepsy

Anxiety is the quiet comorbidity of epilepsy. The anxiety disorders in epilepsy divide into a peri-ictal group, in which seizure-related fear is the prototype, and an interictal group. The mood-in-epilepsy literature is dominated by depression, and the psychoses draw the eye because they are dramatic, but anxiety runs alongside both and is, on the best available evidence, at least their equal in frequency. It is named far less often than it is felt, partly because its symptoms are so easily written off as an understandable reaction to an unpredictable, stigmatising illness, and partly because the whole field learned to screen for depression first. The examinable content sits in four places, and they map cleanly onto four things a good clinician actually does: knowing how common interictal anxiety really is, telling a genuine anxiety disorder apart from an ictal fear that is really a seizure, recognising the epilepsy-specific entity of seizure phobia, and treating the picture without entrenching the benzodiazepine misuse that so often complicates it.

These presentations are best sorted, as the rest of the peri-ictal material in this chapter is, by their relationship in time to the seizure. Anxiety arising between seizures is a true psychiatric comorbidity and is treated as such; anxiety that is the seizure, the sudden fear or panic that begins and ends with the discharge, is a neurological event wearing a psychiatric mask and belongs to a separate topic. Almost every mistake in this area comes from letting those two collapse into each other, so the discrimination is the spine on which the rest of the answer hangs.

24.1 How common it is, and why it is missed

At least as common as depression, and consistently under-recognised. Lishman's Organic Psychiatry, drawing together community and clinic-based studies, concludes that in the interictal state anxiety and depression are probably about equally prevalent, with several series finding anxiety symptoms to be a little more common than depressive ones. That comparison is the one worth carrying, because it corrects the instinct to treat anxiety as the lesser problem. The absolute figure is deliberately left qualitative: reported rates swing so widely with the population sampled, from community cohorts through neurology clinics to surgical series, that a solitary percentage without its denominator is a weak and probably wrong answer. The safe form of the fact is comparative, not numeric. Under-recognition has identifiable causes. Anxious symptoms are readily attributed to the epilepsy itself or to the antiepileptic drugs, or dismissed as the sensible worry of someone who never knows when the next fit will come, and so are normalised out of the picture. The comorbidity is also crowded out by the two conditions it accompanies, a depression that is actively screened for and a psychosis that demands attention, each taught in its own right elsewhere in this chapter. The cost of missing it is not trivial: untreated anxiety erodes quality of life at least as much as seizure frequency does, inflates unscheduled care, and

compounds both depression and the elevated suicide risk that epilepsy carries, each developed in its own dedicated topic. Anxiety is not a soft add-on to the mood picture but a hard, treatable target in its own right.

RULE **Anxiety is at least as frequent as depression in epilepsy, and it is treatable.** Screen for it deliberately rather than waiting for it to be volunteered, because it is routinely normalised as an understandable reaction and so goes unnamed and untreated. The comparative fact, that it equals or slightly exceeds depression, is the examinable one; a single point-prevalence figure is not.

24.2 Interictal anxiety versus ictal fear: the cardinal distinction

Is this an anxiety disorder, or is it a seizure? The first decision about any anxious presentation in epilepsy is where it sits on the seizure timeline, because two very different things look alike. An **interictal anxiety** disorder is a comorbidity arising between seizures and resembling anxiety in anyone else. **Ictal fear or ictal panic**, by contrast, is a seizure phenomenon and not an interictal anxiety disorder at all; it is a brief, stereotyped, self-limiting affective event that is the seizure, and its neurobiology and management are set out in their own topic in this chapter, so it is named here only as the thing to exclude. The distinction is not hair-splitting: it dictates the entire workup. A true anxiety disorder is managed as a comorbidity, whereas a paroxysm of ictal fear is a call to optimise seizure control, and an antidepressant will not touch it. Several bedside features do the separating work. Ictal events are brief, sudden, stereotyped from episode to episode, often accompanied by other seizure phenomena such as automatisms or altered awareness and followed by postictal features; interictal anxiety is sustained, situationally shaped, elaborated with worry and anticipation, and shows none of that paroxysmal stereotypy. The accompanying figure places the common anxiety-like presentations against the seizure to make the sorting explicit.

PRESENTATION	TIMING TO THE SEIZURE	WHAT IT ACTUALLY IS	WHERE IT IS TAUGHT
Interictal anxiety disorder	between seizures	a genuine anxiety disorder, like anxiety in the general population	here
Ictal fear or ictal panic	during the seizure	a seizure phenomenon, not an anxiety disorder	its own topic in this chapter
Seizure phobia	between seizures	fear of having a seizure, out of proportion to the real risk	here
Anticipatory anxiety	between seizures	understandable worry about an unpredictable illness, on a continuum with the above	here

Anxiety-like presentations in epilepsy sorted by timing to the seizure; three of the four are interictal comorbidities managed here, while ictal fear is a seizure named only as the differential to exclude.

TRAP **Treating a seizure as an anxiety disorder, or an anxiety disorder as a seizure.** Candidates lose the mark by starting an antidepressant for paroxysmal ictal fear, or by escalating antiepileptic drugs for a sustained interictal anxiety disorder. The tell is stereotypy and brevity: an event that is identical each time, lasts seconds to a minute or two, and carries other seizure features is ictal, not an anxiety disorder, however frightening it feels.

24.3 Which anxiety disorders predominate

Agoraphobia, generalised anxiety and social phobia lead; obsessive-compulsive disorder does not. Broken down by diagnosis, a consistent pattern emerges. Lishman, following the work of **Jones and colleagues (2005)**, reports that the most frequent categories are **agoraphobia**, **generalised anxiety disorder** and **social phobia**. The clustering makes clinical sense from the patient's side. Agoraphobia and social avoidance are the natural sequelae of an unpredictable event that may strike in public, be witnessed, and be humiliating or dangerous, so the person narrows their world to places felt to be safe; generalised anxiety is the diffuse, persistent apprehension of living with an illness that gives little warning. Against that grain sits a negative fact that is examinable precisely because it is counter-intuitive: **obsessive-compulsive disorder** is not over-represented in epilepsy. Older teaching linking temporal lobe and limbic pathology to obsessional phenomena might lead a candidate to expect an excess of it, and the discipline of the topic is to resist that expectation and state the negative. The pattern is worth holding whole: the disorders that flourish are those an unpredictable, socially exposed illness would breed, and the one that does not is the one intuition wrongly predicts. Naming the categories also sharpens management, since agoraphobic and social avoidance respond particularly well to the exposure-based psychological work discussed below, whereas diffuse generalised anxiety is the picture in which a drug is more often needed.

MNEMONIC

A-G-S, not O

The interictal anxiety disorders that cluster in epilepsy are **A**goraphobia, **G**eneralised anxiety disorder and **S**ocial phobia. The letter deliberately left out is **O** for obsessive-compulsive disorder, which is the one that is not over-represented. Each element is a fact the topic actually states, and the omission is as examinable as the inclusions.

24.4 Seizure phobia: the epilepsy-specific syndrome

When the fear of the fit becomes more handicapping than the fit. The one entity in this area with no counterpart in general psychiatry is seizure phobia, and it is the part most likely to be tested. It is best defined as a fear of having a seizure that is out of all proportion to the objective risk and that disables the patient more than the seizures themselves do. Phenomenologically it resembles agoraphobia: the dreaded outcome is a seizure in a place where it would cause injury or public embarrassment, and the response is to avoid such situations and lean on safety behaviours. That structure explains its self-perpetuating quality. Every avoided outing that passes without a fit is misread as proof that avoidance works, so the avoidance is reinforced and the world shrinks further, exactly as in any phobic disorder. The most instructive feature, and the one showing the

fear has become autonomous rather than a rational appraisal of danger, is that seizure phobia can be at its most striking after successful epilepsy surgery, in patients who are seizure-free yet remain housebound by dread of a fit the operation has abolished. The fear has outlived its object. That is the clinching argument for treating the anxiety directly rather than assuming it will melt away once seizures are controlled; it will not, because it has detached from them. Seizure phobia should therefore be actively asked about, since patients rarely volunteer a fear they regard as sensible, and treated as a phobic disorder in its own right rather than an inevitable shadow of the epilepsy.

24.5 Where the anxiety comes from

Reactive, organic and iatrogenic strands, usually braided together. Anxiety in epilepsy is rarely traceable to a single cause, and thinking in strands helps both formulation and treatment. The reactive strand is the most visible: living with an illness that strikes without warning, carries stigma, can injure, and constrains driving, employment and independence generates a realistic apprehension that can escalate into a disorder. The organic strand reflects the same limbic and cortical disturbance that underlies the epilepsy, so that in some patients the anxiety is a direct expression of the brain's dysfunction rather than a response to it, on the neurobiological substrate this chapter develops in its dedicated mechanism topic. The iatrogenic strand is treatment itself, since antiepileptic drugs have their own behavioural and psychiatric adverse effects, the detail of which is owned by a separate topic in this chapter. In practice the strands coexist, and the value of separating them is that each points to a different lever.

- The unpredictability and perceived dangerousness of seizures, which drives anticipatory anxiety and phobic avoidance.
- Stigma and the social consequences of a witnessed fit, which feed social phobia and agoraphobic constriction.
- The direct neurobiological disturbance shared with the seizure disorder, which can generate anxiety autonomously.
- The psychiatric adverse effects of antiepileptic drugs, an iatrogenic contribution that is always worth reviewing.

Separating the strands is not a formality: a reactive, phobic picture calls first for psychological work, a predominantly iatrogenic one for a hard look at the drug regimen, and an organic anxiety yoked to poor seizure control for better seizure control above all. Most patients need attention to more than one strand, which is why the management below spans several modes at once.

24.6 The benzodiazepine complication

The picture is frequently muddied by benzodiazepine dependence. A specific and common complication deserves its own place because it changes the whole management calculus: the anxiety of epilepsy is often layered over, and worsened by, **benzodiazepine dependence**. Two routes lead there, both easy to walk into. In the first, rescue benzodiazepines legitimately prescribed for seizure clusters come to be taken for supposed prodromes, so a drug meant for an emergency is used pre-emptively and repeatedly for a warning that may be nothing more than

anticipatory anxiety. In the second, the patient reaches for the same drugs as self-medication for the anxiety itself, finding short-term relief that narrows into dependence. Benzodiazepine use and anxiety then drive each other: inter-dose and withdrawal anxiety mimics and amplifies the underlying disorder, escalating use looks like worsening illness, and the sedation and tolerance that follow do nothing for the phobic core. Recognising the loop matters because the correct move, a graded withdrawal alongside proper treatment of the anxiety, runs against the patient's instinct and often against the path of least resistance in a busy clinic. An anxious person with epilepsy who is escalating benzodiazepines is not usually one who needs more of them.

PITFALL

The reflex response to a distressed, anxious patient with epilepsy is to reach for a benzodiazepine, or to intensify the antiepileptic regimen, when the actual problem is a phobic anxiety disorder or seizure phobia that needs psychological treatment. Each reflex entrenches the wrong thing: benzodiazepines breed dependence and antiepileptic escalation adds adverse effects, while the fear that is doing the disabling goes untreated. Name the anxiety disorder before you medicate the seizure.

24.7 Management

Treat it as anxiety, and treat the epilepsy as epilepsy. The governing principle is that anxiety in epilepsy is managed broadly as anxiety is in general psychiatry, with the epilepsy-specific overlays of seizure phobia, the ictal-fear differential and the benzodiazepine trap on top. The work is framed by the epilepsy standards of the International League Against Epilepsy and NICE, which make optimised seizure control the foundation, together with the epilepsy-psychiatry literature synthesised in Lishman's Organic Psychiatry and Kaplan and Sadock's Comprehensive Textbook of Psychiatry for the anxiety itself. The **LOCUS** of care is overwhelmingly outpatient, since interictal anxiety and seizure phobia are chronic, ambulatory problems; the exceptions that pull care into a more intensive setting are a difficult planned benzodiazepine withdrawal and the rare presentation severe enough to threaten safety or function. The **focus** shifts by phase: the acute task is to contain distress and, critically, avoid entrenching benzodiazepine use; the short-to-mid-term task is to treat the specific anxiety disorder or seizure phobia to remission; and the long-term task is relapse prevention through durable seizure control, a benzodiazepine-sparing regimen and attention to driving, work and stigma.

The **modus** spans every available mode. Psychological treatment leads, and it is where seizure phobia in particular shines, since it responds well to **cognitive-behaviour therapy**, the approach established for it by **Newsom-Davis and colleagues (1998)**; the therapy challenges the catastrophic cognitions and schema the patient holds about their epilepsy and dismantles the safety and avoidant behaviours that keep the phobia alive. Pharmacologically, anxiety in epilepsy is treated as elsewhere, with an SSRI as first-line; where an SSRI has not delivered, pregabalin is a reasonable alternative, having shown an antianxiety effect in epilepsy patients with generalised anxiety disorder, though on the limited evidence of a small open-label study rather than anything definitive. The choice of any antidepressant against its effect on the seizure threshold is a

decision handled in its own dedicated topic in this chapter and is not reopened here. The procedural and social modes complete the plan: optimising antiepileptic treatment as the organic lever, withdrawing benzodiazepines where dependence has taken hold, and addressing the driving, employment and family realities that sustain the reactive anxiety. Running through all of it is the caution that the drugs offering the quickest relief are the ones most likely to make the long-term picture worse.

IN INDIA

Two features sharpen this topic in Indian practice. First, benzodiazepines, and clonazepam in particular, are widely and easily accessible, so the slide from rescue or as-needed use into dependence is an everyday risk and the benzodiazepine-sparing discipline above is not theoretical. Second, trained cognitive-behaviour therapists are scarce outside metropolitan and tertiary centres, a real problem precisely because seizure phobia and the phobic-avoidant disorders respond so well to that treatment; the practical answer is often SSRI treatment supported by whatever structured psychological input can be mustered, alongside direct work on the stigma around epilepsy that, given employment and marriage prospects, does much of the driving here.

GOOD TO REMEMBER

Ask every patient with epilepsy two questions the notes rarely capture: how much do you fear the next seizure, and how often do you take a benzodiazepine for the fear. The first uncovers seizure phobia and the phobic-avoidant disorders that dominate the picture; the second uncovers the dependence that quietly worsens it. Both are missable, both are common, and both are treatable.

24.8 Pulling the picture together

One axis, three real disorders, one impostor. The whole area resolves if the anxious presentation is first placed against the seizure. Everything interictal, the generalised, agoraphobic and social anxiety that cluster in epilepsy, the seizure phobia that can outlast the fits it fears, and the ordinary anticipatory dread of an unpredictable illness, is a genuine, treatable psychiatric problem. The one impostor is ictal fear, the paroxysm that is the seizure rather than a reaction to it, named here only to be sent to its own topic. Hold that distinction, remember that anxiety at least matches depression in frequency and is routinely under-treated, recall that obsessive-compulsive disorder is the exception that does not cluster, and keep one hand on the benzodiazepines, and the topic answers itself.

≥ depression	Agora / GAD / Social	OCD not raised	CBT + SSRI
INTERICTAL ANXIETY FREQUENCY	COMMONEST ANXIETY DISORDERS	NOT OVER-REPRESENTED	FIRST-LINE TREATMENT

Interictal anxiety is at least as common as depression in epilepsy and is genuinely treatable; agoraphobia, generalised anxiety and social phobia predominate while obsessive-compulsive disorder does not, the epilepsy-specific syndrome is seizure phobia (fear of the fit that outstrips the fit and can persist after successful surgery), and the two governing cautions are to separate a true anxiety disorder from ictal fear and to keep the anxiety from becoming benzodiazepine dependence.

25 · Antidepressant choice and seizure-threshold safety in epilepsy

Depression is the commonest psychiatric comorbidity of epilepsy, it is treatable, and it is repeatedly left untreated for the wrong reason. The reason is an old anxiety that antidepressants lower the seizure threshold and will therefore make an already seizure-prone brain worse. That anxiety is not baseless, because some antidepressants genuinely do lower the seizure threshold, but it has been inflated far beyond the evidence, and the price of the inflation is a patient with disabling, sometimes lethal, depression who is denied an effective drug. The marks in this topic are not for reciting a list of dangerous agents. They are for holding two facts at once: that at ordinary treatment doses the seizure risk of a well-chosen antidepressant is very small, and that the choice of agent nonetheless matters, because a few specific drugs carry a real and avoidable liability.

This is a prescribing topic, so it is examined as a decision rather than as a description. The prevalence, phenomenology and screening of depression in epilepsy are developed in their own topic, as is the elevated suicide risk that makes under-treatment dangerous; here the question is what to prescribe once depression has been identified, how to start it, when a physical treatment is preferable, and what to check before the first prescription. The mechanism by which drugs lower the seizure threshold is itself taught separately, in the dedicated topic on drug-induced lowering of the seizure threshold; this topic applies that principle to pick an agent rather than re-deriving it.

25.1 The first principle: treat the depression

The proconvulsant fear has been overstated, and the untreated illness is the greater danger. The historical reluctance to give antidepressants in epilepsy rested on case reports of seizures and on the true observation that these drugs can lower the seizure threshold, but when the actual incidence is examined the picture is reassuring. Lishman's Organic Psychiatry, drawing the figure from non-epileptic populations at therapeutic doses, puts the seizure incidence at **well below 1%**, a rate that has to be set against the substantial morbidity, and the mortality, of an epilepsy patient whose depression goes untreated. The clinical inference is direct: clinicians should not hesitate to prescribe an antidepressant when depression is present and otherwise warrants treatment, because the seizure hazard at ordinary doses is small and the illness being treated is not. Kaplan and Sadock's Comprehensive Textbook of Psychiatry frames the same reassurance mechanistically. The more recently introduced antidepressants are only marginally proconvulsant, and at therapeutic doses the tricyclics, the newer agents and the SSRIs do not usually promote seizures at all; it is **overdose**, not the therapeutic dose, that is the main seizure

trigger. That single distinction dissolves most of the historical worry, because it locates the danger in intoxication rather than in treatment, and it reframes the prescribing task as one of choosing sensibly and starting cautiously rather than of avoiding the class outright.

RULE **Do not withhold an antidepressant from a depressed patient because they have epilepsy.** At therapeutic doses the seizure risk of a well-chosen agent is very low, and untreated depression carries its own serious morbidity and mortality. The live decision is which drug and how to start it, not whether to treat.

25.2 Choosing the agent: SSRIs first

Comparable efficacy means the decision is driven by safety and tolerability. Across the antidepressant classes the efficacies for depression are broadly similar, so in a patient with epilepsy the choice is settled less by potency than by which agent is least likely to provoke a seizure and best tolerated. On that logic the **SSRIs** are the antidepressants of choice and stand first-line, because they combine the usual antidepressant efficacy with a relatively low seizure liability. Beyond the SSRIs a wider group of newer agents is regarded as carrying a comparably low risk and is drawn on where an SSRI is unsuitable: the **lower-seizure-risk antidepressants** named by Lishman's Organic Psychiatry are citalopram, escitalopram, fluoxetine, paroxetine and sertraline among the SSRIs, with mirtazapine, moclobemide, reboxetine, trazodone, venlafaxine and nefazodone beyond them. Because efficacy does not separate them, the practical selection within this group turns on a small number of considerations.

- Seizure-threshold safety, the property that removes the highest-liability agents from consideration altogether.
- The side-effect fit for the individual, since tolerability is what efficacy-equivalence leaves to decide.
- The interaction profile with the patient's antiepileptic drugs.
- Safety in overdose, which weighs more heavily here because depression in epilepsy carries a raised suicide risk.

TIER	CLASS	SEIZURE-THRESHOLD LIABILITY	PRACTICAL CAVEAT
First-line	SSRIs and the wider lower-risk group	lowest	still start low and titrate slowly
Second-line	tricyclics	higher, dose-related	higher risk but not contraindicated
Usually avoided	the highest-liability agents	greatest threshold lowering	bupropion is formally contraindicated in seizure disorders

The antidepressant selection ladder in epilepsy: efficacy is broadly comparable across the tiers, so the ordering is by seizure-threshold liability and practical caveat rather than by antidepressant potency.

25.3 Tricyclics: second-line, not forbidden

A higher, dose-related risk that still leaves them usable. The **tricyclic antidepressants** sit second-line in epilepsy. They carry a higher risk of seizure exacerbation than the SSRIs, and that risk is dose-related, but, and this is the examinable subtlety, they are not contraindicated. A tricyclic remains a legitimate choice when the depression has not responded to safer first-line agents, provided the dose is kept modest and raised slowly. The group named in this second-line position includes amitriptyline, clomipramine, dothiepin, imipramine, nortriptyline, trimipramine and lofepramine. Clomipramine deserves a separate flag, because it is the tricyclic with the greatest proconvulsant liability, and for that reason it appears not only in this second-line list but also among the agents that are usually avoided outright; the same molecule can be both a permissible second-line tricyclic in general use and a drug to steer around specifically in epilepsy. The general lesson of the tricyclic tier is that "second-line" is a real and useful category sitting between "preferred" and "avoid": it says that these drugs are more hazardous than the SSRIs and should not be a first reach, without pretending that they are unusable in a patient who genuinely needs them. Reading the tier correctly, as higher risk, dose-related, and still available, is worth more than memorising the roster, because it is the reasoning an examiner is testing.

25.4 The agents to avoid, and bupropion in particular

A short list of genuine offenders, headed by one that is contraindicated. A small number of antidepressants lower the seizure threshold enough that they are usually avoided in epilepsy altogether: **amoxapine, bupropion, clomipramine and maprotiline**. These are the agents whose threshold-lowering is greatest, and in a patient who already has seizures the sensible default is simply not to use them when an equally effective, safer alternative exists. Bupropion is the one to know in most detail, because its seizure liability is both dose-dependent and, at the top of that list, a formal contraindication in a seizure disorder. Seizures have been reported in about **0.4%** of bupropion-treated patients, and because the effect is dose-related the risk is minimised by using the sustained-release form, by titrating slowly from a low start of **100 mg** daily, and by respecting the sustained-release maximum of **400 mg** daily. In a patient with epilepsy, however, those mitigations are beside the point: the drug is contraindicated in seizure disorders, and in eating disorders where the seizure risk is likewise raised, so the correct action is to choose something else rather than to prescribe it carefully. The principle that unites the four is the one applied throughout this topic, to pick against seizure-threshold liability, taken to its logical end for the agents where that liability is highest.

MNEMONIC

ABCM - the four to avoid

The antidepressants usually avoided in epilepsy, by first letter: **A**moxapine, **B**upropion, **C**lomipramine, **M**aprotiline. Bupropion is the one that is not merely avoided but contraindicated in a seizure disorder. Each name is an agent this topic actually places on the avoid list.

PITFALL

Bupropion reaches patients by routes that bypass the epilepsy history. It is chosen as an activating antidepressant when fatigue or sexual side effects dominate, and it is prescribed for smoking cessation by clinicians who are not thinking about seizures at all. In a person with epilepsy either route is a mistake, because the drug is contraindicated in seizure disorders. Screen for a seizure history before starting bupropion for any indication, not only when treating mood.

25.5 How to start: low dose, slow titration

Because the proconvulsant effect is dose-related, even the safe agents are started cautiously. Within the preferred group the way an antidepressant is introduced still matters, and the governing fact is that the proconvulsant effect of these drugs is dose-related. Lishman's Organic Psychiatry translates that into a simple prescribing rule, to **start low and go slow**, beginning at a low dose and up-titrating slowly, so that the plasma level, and with it any threshold-lowering effect, rises gradually rather than in a jump the brain has to absorb at once. The rule applies across the classes, not only to the second-line tricyclics, because dose-dependence is a general property; it is simply most consequential where the baseline liability is highest. The corollary concerns overdose, and it is the point at which the prescribing decision loops back to the reason for treating in the first place. Therapeutic doses of the tricyclics, the newer agents and the SSRIs do not usually promote seizures, but overdose does, and depression in epilepsy carries a raised risk of self-harm. That combination is a specific argument for the safer agents and for attention to the quantity dispensed, because the very patient in whom a therapeutic dose is low-risk may be at risk of taking far more than the therapeutic dose. Starting low and going slow protects against the therapeutic-dose hazard; choosing an agent that is safer in overdose, and limiting supply, addresses the larger one.

TRAP **Writing that antidepressants are contraindicated in epilepsy.** The blanket statement is wrong and loses the mark. Only a few named agents are usually avoided, and only bupropion carries a formal contraindication in seizure disorders; the SSRIs are first-line and even the tricyclics are second-line rather than forbidden. The examiner wants the graded answer, not a class-wide ban.

25.6 ECT for severe depression

An effective and safe option, and paradoxically unproblematic in a seizure disorder. When depression in epilepsy is severe, whether psychotic, life-threatening through suicidality or refusal to eat and drink, or unresponsive to drugs, **electroconvulsive therapy** is an effective and safe treatment, and it is worth stating plainly because clinicians hesitate over the apparent contradiction of deliberately inducing a seizure in someone whose problem is seizures. Lishman's Organic Psychiatry settles the worry: ECT is effective and safe in this population, and it usually has no discernible effect on the frequency of the patient's own epileptic seizures. Practically, a therapeutic seizure can generally be induced without needing to reduce the patient's antiepileptic drugs first, which removes the main logistical objection to giving it. The reasoning is that the controlled, brief seizure of a treatment session is a different event from the disorder's spontaneous seizures and does not destabilise seizure control in the way intuition fears. This

makes ECT a genuine and sometimes preferable route in exactly the patients where the stakes are highest and where waiting for a slowly titrated drug to work is least acceptable, the severely depressed, the acutely suicidal, and those who have already failed pharmacotherapy. It should therefore be held not as a last resort forced by the failure of everything else, but as a first-rank option for severe depression in epilepsy on its own merits of speed and safety.

GOOD TO REMEMBER

When depression in an epilepsy patient is severe or the patient is acutely suicidal, do not defer electroconvulsive therapy on the assumption that it will worsen the epilepsy. It is effective, it is safe, and it usually does not change seizure frequency, and a treatment seizure can generally be induced without first cutting back the antiepileptic drugs.

25.7 Check interactions before you prescribe

Few matter in practice, but the check is not optional. Before writing the prescription, look for interactions between the chosen antidepressant and the patient's antiepileptic drugs, and in particular the **enzyme-inducing antiepileptic drugs**, whose induction of hepatic metabolism can lower the plasma concentration of a co-prescribed antidepressant. Lishman's Organic Psychiatry is reassuring about the scale of the problem, since clinically significant interactions are few in practice, but reassurance about frequency is not the same as permission to skip the step, because the interactions that do matter can undo treatment by keeping the antidepressant sub-therapeutic. The detailed pharmacokinetics of these interactions, which antiepileptic induces or inhibits which enzyme and how the psychotropic level is affected, are set out in their own dedicated topic on the pharmacokinetic interactions between antiepileptic drugs and psychotropic agents; they are named here only to place the check in the prescribing sequence, not to be reworked. The practical posture is one of routine vigilance rather than alarm: assume most combinations will be fine, confirm that the specific pair in front of you is one of them, and be ready to use a higher antidepressant dose, or to prefer an agent less affected by induction, when an enzyme-inducing antiepileptic is on board. The step costs little and occasionally prevents a treatment failure that would otherwise be misread as a non-responsive depression.

IN INDIA

The interaction check carries more weight in Indian practice than the Western default implies, because the older enzyme-inducing antiepileptic drugs, phenobarbital above all, and phenytoin and carbamazepine, remain widely used first-line agents on grounds of cost and availability. A depressed epilepsy patient is therefore more likely to be on an enzyme inducer than a patient in a setting where the newer non-inducing antiepileptics predominate, and the co-prescribed antidepressant is correspondingly more likely to be pushed toward sub-therapeutic levels. The SSRIs that lead the first-line choice are, helpfully, cheap and readily available, so the preferred agent is rarely the constraint; the discipline that matters is remembering to check the antidepressant against the inducing antiepileptic, rather than assuming, as one safely might elsewhere, that the background drug is metabolically quiet.

25.8 Putting it together: managing depression in the epilepsy patient

The pieces assemble into a single prescribing pathway. The management of depression in epilepsy is anchored in the epilepsy guidelines of ILAE and NICE for the seizure disorder itself, and in the epilepsy-specific psychiatric-management literature together with the Maudsley Prescribing Guidelines for the antidepressant decision, with textbook prose as the connective cover.

The **LOCUS** of care is usually the outpatient clinic, where most depression in epilepsy is identified and treated; inpatient admission is reserved for severe, psychotic or high-suicide-risk presentations, and the emergency department is where acute suicidality first arrives, the elevated suicide risk of epilepsy being handled as its own topic. The **focus** moves by phase: the acute task is to secure safety and begin an effective antidepressant; the short-to-mid-term task is to titrate that agent to response while watching seizure frequency; and the long-term task is maintenance to prevent relapse and continued optimisation of seizure control, since better-controlled epilepsy is itself better for mood. The **modus** spans every mode of treatment. The pharmacological mode is the spine of this topic: an SSRI first, the wider lower-risk group next, tricyclics second-line where needed, the highest-liability agents avoided and bupropion excluded outright, every drug started low and raised slowly, and interactions with enzyme-inducing antiepileptics checked first. The procedural mode is electroconvulsive therapy for severe or refractory depression, effective and safe without destabilising seizures. The psychological mode, structured psychotherapy for the depression, and the social mode, addressing the disability, stigma and loss of driving or employment that feed low mood in epilepsy, complete the plan; the choice of an antipsychotic against its seizure-threshold effect in epileptic psychosis is the sibling question this pathway sits beside.

below 1%	**0.4%**	**400 mg**	**SSRIs**
SEIZURE RISK AT THERAPEUTIC DOSE	SEIZURES ON BUPROPION	BUPROPION SR MAXIMUM (CONTRAINDICATED IN EPILEPSY)	FIRST-LINE IN EPILEPSY

In epilepsy, treat the depression: at therapeutic doses the seizure risk of a well-chosen antidepressant is well below 1%, SSRIs are first-line and tricyclics second-line but not contraindicated, amoxapine, bupropion, clomipramine and maprotiline are usually avoided with bupropion contraindicated outright, every agent is started low and titrated slowly, ECT is effective and safe for severe cases, and interactions with enzyme-inducing antiepileptics are checked before the first prescription.

26 · AED-attributable mood and suicidality (FDA warning) and management

The suicidality caution printed on every antiseizure prescription is a pharmacovigilance story, not a piece of psychopathology. The examinable core of this topic is a single regulatory event and the epidemiology that produced it. In **2008** the United States Food and Drug Administration issued a **class warning** of increased **suicidality**, meaning suicidal ideation or behaviour, for antiepileptic drugs taken as a group. The mark-earning discrimination, and the one

a good answer opens with, is that this drug-attributable signal is a different thing from the suicide risk that is intrinsic to epilepsy itself. The elevated suicide risk of the disease, expressed as its standardised mortality ratio, is a separate and in fact larger question that is worked up under its own topic in this chapter, and the reader must not leave with the two collapsed into one.

The topic tests three things at once: how a drug-safety signal is generated from pooled trials, how relative and absolute risk are read against each other, and how a caution of this kind should change prescribing. It is also a topic where confident memory is dangerous, since the exact regulatory form of the warning and the arithmetic of the risk are both routinely misremembered. The safe approach is to hold the few established facts precisely and reason around them, rather than inflate the signal into something it was never shown to be.

26.1 What the 2008 warning actually says

A class-level caution about emergent suicidality, applied to the whole drug group. The warning is best understood first for what kind of statement it is. It is not a diagnosis, not a claim that antiseizure drugs cause a depressive illness, and not a statement about any single agent. It is a **class effect**: an increase in the frequency of suicidal thoughts and suicidal behaviour observed across antiseizure drugs considered together, applying to patients treated for epilepsy and to those with psychiatric disorders alike, as summarised in Kaplan and Sadock's Comprehensive Textbook of Psychiatry. The endpoint of interest is deliberately behavioural and cognitive rather than diagnostic. It captures new or worsening suicidal ideation and suicidal acts as adverse events emerging on treatment, which is why the clinical response the warning asks for is monitoring rather than a change in diagnosis. Framing it this way also keeps the topic in its lane. The negative affective and behavioural effects that particular agents can produce, and the broader cognitive and behavioural adverse effects of antiseizure drugs, are owned by their own dedicated topics in this chapter and are only named here; what belongs to this topic is the class-wide suicidality signal and the regulatory and clinical machinery built around it. A candidate who starts reciting which individual drug is most depressogenic has drifted off the actual question, which is about a pooled safety signal and what a prescriber does with it.

26.2 The evidence base: a pooled analysis of placebo-controlled trials

The signal came from meta-analysis, not from spontaneous reports. The strength of the warning, and much of its credibility, rests on how it was generated. Rather than resting on case reports or registry noise, the FDA assembled a pooled analysis of **nearly 200 placebo-controlled trials** spanning eleven antiseizure drugs, drawing together tens of thousands of randomised patients, as tabulated in Dulcan's Textbook of Child and Adolescent Psychiatry. Pooling randomised, placebo-controlled data matters because it is the design least vulnerable to the biases that plague drug-safety questions: randomisation balances the baseline suicide risk between arms, the placebo comparator supplies the counterfactual, and prospectively captured, uniformly classified events avoid the selective recall that inflates spontaneous reporting. The scale of the pooled population is also what let a rare outcome be detected at all, since suicidal behaviour is infrequent enough that no single trial could reliably show a difference. The accompanying table sets the two arms side by side on the measures that carry the argument.

TRIAL ARM	PATIENTS POOLED	COMPLETED SUICIDES
Active antiseizure drug	about 28,000	4
Placebo	about 16,000	0

The pooled FDA programme by trial arm: a larger drug-treated population against a placebo comparator, with the completed suicides falling only in the active-drug arms. The event is rare in absolute terms, which is exactly why so large a pooled sample was needed to see it.

The completed suicides are worth holding carefully rather than over-reading. They fell only in the drug-treated groups, which is sobering; but the absolute count is very small, and a handful of events cannot by itself establish a rate. This is why the primary signal was carried by the far more common outcome of suicidal ideation and behaviour, where the numbers were large enough to compare, with the completed suicides serving as corroboration rather than the headline.

26.3 Relative versus absolute risk: reading the magnitude

Roughly doubled in relative terms, but small in absolute terms. The single most misused fact in this topic is the size of the effect, because relative and absolute framings tell very different emotional stories from the same data. On the pooled analysis, suicidal ideation or behaviour occurred **about twice as often on active drug as on placebo, roughly 0.43% versus 0.22%**. Stated as a ratio, a doubling of risk sounds alarming. Stated in absolute terms, the same finding amounts to **about two additional affected patients per 1,000 treated**, which is a modest increment set against the substantial benefit of controlling seizures. Both figures are true and neither is the whole truth; the skill the examiner is testing is the ability to hold them together. Translated into the language of number needed to harm, a great many patients must be treated before one additional suicidality event is attributable to the drug, which is why the warning was framed as a prompt to vigilance rather than a reason to withhold treatment. The point generalises: a relative risk without its baseline is a rhetorical device, and a responsible reading of any safety signal converts the ratio back to absolute terms before acting on it.

RULE **The signal is real, small in absolute terms, and spread across the drug class.** It is an argument for actively monitoring mood and suicidality after starting an antiseizure drug, not an argument for avoiding effective treatment. Convert the relative doubling into its absolute increment before you let it change a prescribing decision.

26.4 Raised across indications, and the confounding problem

The risk held across every indication and was greatest in the epilepsy patients. An obvious objection to any such signal is that antiseizure drugs are prescribed for conditions that themselves carry a raised suicide risk, so that the drugs might merely be markers of a high-risk population. This is the problem of **confounding by indication**, and it is not a hypothetical worry here: these agents are used for migraine, neuropathic pain and bipolar disorder among other indications, each of which is associated with suicidality in its own right. The mood-stabiliser role of the class in bipolar disorder, with its own mechanism and monitoring, is developed in the Mood disorders: pharmacotherapy notes and is not reopened here. The FDA analysis addressed the confounder directly by examining the signal within indication groups, and the finding that gives the warning its weight is that the raised risk was present across all the

indications studied rather than being confined to one high-risk group. Within that spread, the relative increase was highest among the patients treated for epilepsy, which is the group of greatest interest to the psychiatrist working at this interface. That the signal survived the confounding, and was strongest precisely where seizure control is least dispensable, is what makes it a genuine drug effect worth acting on rather than an artefact of who receives these drugs.

TRAP **Attributing an epilepsy patient's whole suicide risk to the drug.** The intrinsic suicide risk of epilepsy and its standardised mortality ratio is a large, separate story taught under its own topic in this chapter, and it is a heavier contributor than the drug signal. The examiner wants the two attributions kept apart: the disease raises suicide risk substantially, and the drug class adds a small further increment on top. Collapsing them, in either direction, loses the mark.

26.5 A class effect across many agents

The warning names a long list, and the class label is deliberate. The breadth of the class effect is easiest to grasp from the agents it covered. In the analysis these were carbamazepine, divalproex sodium and valproate, felbamate, gabapentin, lamotrigine, levetiracetam, oxcarbazepine, pregabalin, tiagabine, topiramate and zonisamide, a set that cuts across chemical classes, mechanisms of action and eras of introduction. That diversity is exactly the point. When a safety signal appears across agents that share little except their antiseizure indication, the parsimonious reading is a class effect rather than a property of any one molecule, and the warning was written to the class for that reason. It would be a misreading to conclude that these agents carry equal psychiatric liability in ordinary practice; individual drugs differ markedly in their propensity to produce low mood, irritability and behavioural change, and those agent-specific psychiatric side-effect profiles, along with the cognitive and behavioural adverse effects of antiseizure drugs more broadly, are the subject of their own topics in this chapter. What the class warning establishes is narrower and firmer: a pooled, class-level increase in emergent suicidality that a prescriber of any of these drugs should have in mind, whichever particular agent is chosen.

IN INDIA

The practical shape of this caution differs here. Much prescribing still rests on older, cheaper agents, follow-up is often hurried, and structured suicidality screening at each visit is far from routine, so the burden of noticing an emergent change often falls on the family. The workable response is to build the warning into psychoeducation from the first prescription: tell the patient and a family member, in plain language and in the patient's own tongue, what an emerging change in mood or thinking looks like and how to raise it quickly, since a WhatsApp message or a call to the treating team is a more realistic safety net than a distant review appointment. Stigma around both epilepsy and suicide makes families slow to volunteer these observations, which is another reason to name them explicitly.

26.6 A warning, not a black box

The regulatory form is the fact most often upgraded from memory. When the joint FDA advisory committees deliberated, they recommended that the labelling of these drugs carry **warnings and precautions** about suicidality, but they stopped short of a **black box warning**, the strongest and most restrictive form of labelled caution. The distinction matters both clinically and for the examination. A black box is reserved for hazards judged severe enough to demand the most prominent possible flag and often to constrain use directly; the committees' judgement that a small absolute excess, set against the clear benefit of seizure control, did not warrant that highest tier is itself the substance of the decision. The warning has also drawn methodological criticism, chiefly that pooling heterogeneous trials with very low event rates and non-uniform ascertainment can generate a fragile signal, and that the class framing may obscure real differences between agents. Those debates do not overturn the core, cautiously stated finding, and the sensible posture is to take the monitoring seriously while not treating the label as a reason to deny patients effective drugs.

TRAP **Calling it a black box warning.** This is the classic upgrade error. The advisory committees voted for warnings and precautions in the package insert and specifically not for a black box. Candidates, reasoning that anything about suicidality must be a black box, misremember the tier and lose an easy mark. Hold the exact regulatory form.

PITFALL

The mirror-image clinical error is over-reaction: withholding or hastily withdrawing an effective antiseizure drug because of the suicidality label, and leaving the patient with uncontrolled seizures. The excess risk is small and absolute, and it is greatest in exactly the epilepsy population who can least afford poor seizure control. The correct response to the warning is to monitor and to educate, not to stop a drug that is working.

26.7 Management

The whole of management follows from the size and shape of the risk. Because the signal is small, class-wide and expressed as emergent change, the response is surveillance and shared vigilance rather than any change in which drug is used for the seizures. The guidelines that frame the work are the epilepsy standards of the International League Against Epilepsy and NICE, which set optimisation of seizure control as the non-negotiable foundation, together with the epilepsy-psychiatry management literature synthesised in Dulcan's Textbook of Child and Adolescent Psychiatry for the handling of the suicidality caution itself. The **LOCUS** of care is overwhelmingly outpatient, since this is a monitoring task layered onto routine epilepsy or psychiatric follow-up; care escalates to emergency or inpatient settings only if active suicidal ideation or intent actually emerges, at which point the management becomes that of any acute suicidality regardless of its trigger. The focus shifts by phase. The acute and short-term task, concentrated around initiation and the early weeks of any new antiseizure drug, is to watch for newly emergent or worsening suicidal thoughts and for the mood and behavioural change that heralds them; the mid-term task is continued periodic review as treatment stabilises; and the

long-term task is to keep the caution alive across a chronic illness without letting it erode adherence to an effective regimen.

The modus spans several modes at once, though none of them is a change of antiseizure agent. The core intervention is clinical monitoring coupled with structured education: clinicians prescribing these drugs should monitor mood and behaviour for emergent suicidality and advise patients and their families to report the warning signs promptly. Making that education concrete is what turns a label into a safeguard, so the specific signs to hand to the household are worth naming.

- New or worsening depressed mood.
- Talking or thinking about self-harm.
- A preoccupation with death or dying.
- Social withdrawal or a marked, unexplained change in behaviour.

The remaining modes are supportive rather than pharmacological in the drug-substitution sense. Psychological input is the safety planning and psychoeducation that make the family an active part of monitoring; the social mode is the family vigilance and accessible contact route that catch an emerging change between appointments; and the separate question of choosing an antidepressant, weighed against its effect on the seizure threshold, is handled in its own dedicated topic in this chapter. The through-line is that the drug keeps doing its job while clinician and family together keep watch for the small, real, detectable risk it carries.

MNEMONIC

FDA: Found pooled, Doubled but small, A warning not a box

The three things to carry are that the signal was **F**ound in a pooled analysis of placebo-controlled trials and holds across the whole drug class, that it represents a **D**oubling in relative terms but only a small absolute excess, and that the regulatory outcome was **A** warning and precaution rather than a black box. The agency's own initials are the memory hook for its own decision.

GOOD TO REMEMBER

Build a one-line suicidality inquiry into the visit at which any antiseizure drug is started and into the early follow-up, the same reflex with which a clinician asks about rash after starting a new agent. Ask about mood and any thoughts of self-harm, document that you asked, and make sure one family member knows the warning signs and how to reach you. It costs a sentence and it is where an emergent change is actually caught.

26.8 Pulling the picture together

One decision, four facts, and a discrimination that must survive. The topic resolves into a compact set of things worth carrying into the examination and the clinic. A drug-safety decision was reached from a large pooled analysis of randomised trials; the risk it identified is roughly a doubling in relative terms and a small increment in absolute terms; it was a class effect across a long list of agents and held across every indication while being greatest in epilepsy; and its

regulatory form was a warning and precaution rather than the black box that memory tends to supply. Above all, the drug-attributable suicidality signal must not be conflated with the far larger, disease-intrinsic suicide risk of epilepsy that is taught under its own topic. Hold that separation, keep the absolute and relative framings side by side, and the management writes itself as monitor, educate, and do not withdraw an effective drug. The at-a-glance strip below fixes the spine.

Class-wide	Small, real	Warning, not a box	Watch, don't withdraw
ALL ANTISEIZURE DRUGS, NOT ONE AGENT	ABSOLUTE EXCESS SUICIDALITY	THE FDA REGULATORY FORM	THE MANAGEMENT STANCE

In 2008 the FDA issued a class warning, not a black box, of increased suicidality across antiseizure drugs, based on a pooled analysis showing suicidal ideation or behaviour about twice as often on drug as on placebo but only a small absolute excess; the risk held across indications and was greatest in epilepsy, and the correct response is to monitor mood and behaviour and educate patients and families, never to reflexively withhold an effective drug or to confuse this drug signal with the disease-intrinsic suicide risk of epilepsy taught elsewhere.

Personality, behaviour and cognition in epilepsy

27 · Temporal lobe epilepsy and interictal personality change

This is the topic where a century of clinical folklore meets a firm modern correction, and the marks lie in holding the tension between them. Interictal personality change refers to the enduring temperament a person with epilepsy carries in the settled periods between seizures - not the fear or déjà vu of the ictus, not the confusion of the postictal state, but the stable, cross-situational way of being that colours ordinary life. For more than a century clinicians believed that chronic epilepsy stamped a characteristic personality on those who had it, and the phrase "epileptic personality" entered the textbooks as though it named a real and unitary thing. The contemporary position is far more careful, and stating it correctly is where the examiner separates the reader who has weighed the evidence from the one who has merely memorised a trait list. The anatomy and semiology of temporal lobe epilepsy, and the named Gastaut-Geschwind cluster, are constructs of their own and taught in their own right elsewhere in this chapter; this topic is the general interictal personality change, the temperamental alteration itself, the instrument built to measure it, and the reasons the old certainty collapsed.

What follows moves from the historical idea and its modern deflation, through the Bear-Fedio inventory, the disputed laterality claim and the specificity problem that unravelled the concept, to viscosity, the social-cognitive substrate, the personality disorders that genuinely cluster with epilepsy, and the markers of risk. Kaplan and Sadock's Comprehensive Textbook of Psychiatry,

Lishman's Organic Psychiatry and Kaufman's Clinical Neurology for Psychiatrists supply the framing between them.

27.1 The idea of an epileptic personality, and why it fails as a general law

A nineteenth-century character that modern evidence dissolved. Early alienists wrote of an epileptic character - adhesive, irritable, egocentric, religiose, slow and circumstantial in thought - and treated it as a near-inevitable consequence of the falling sickness. The modern reading, set out plainly by Kaplan and Sadock, is that there is no single general or specific **epileptic personality** at all: the traits once gathered under that heading are neither universal among people with epilepsy nor uniform in those who show them, and where they do appear they cluster in a subgroup, chiefly those with temporal lobe epilepsy, rather than marking the whole population of patients. That correction does not deny that some patients with long-standing epilepsy change; it denies that the change is one thing, that it is common to all, or that it defines a syndrome. **Interictal personality change**, properly understood, is therefore a description of traits over-represented in a minority, not a diagnosis that any given patient with epilepsy can be expected to develop. The clinical and examinable danger runs in a single direction: the phrase sounds authoritative, it has a long pedigree, and it invites the unwary to expect a personality where there is only a person with a seizure disorder.

RULE **There is no single epileptic personality.** Interictal traits are neither universal in epilepsy nor a unitary syndrome; where they appear they are concentrated in a subgroup, chiefly temporal lobe epilepsy. Every error in this topic comes from promoting a minority description into a general law.

MNEMONIC

Not Universal, Not Specific, Not a Disorder

The three restraints that keep the concept honest: the interictal traits are **not universal** (they appear in a subgroup, not in every patient), **not specific** (the same traits are found in people who do not have epilepsy at all), and **not a disorder** (they are read as markers of brain change, not as a categorical personality disorder). Drop any one restraint and you have reinvented the discredited epileptic personality.

27.2 The Bear-Fedio inventory: an attempt to measure the traits

Turning a clinical impression into a questionnaire. The most influential attempt to give the putative interictal traits an objective footing was the **Bear-Fedio Inventory**, an MMPI-style instrument that Bear and Fedio devised in 1977 to score the specific behaviours long attributed to temporal lobe epilepsy. Where earlier writers had offered adjectives, the inventory offered a scale: it enumerated **18** putative traits - among them circumstantiality, deepened emotionality, a sense of personal destiny and interpersonal viscosity - and asked the patient and an observer to rate each, on the reasoning that a real behavioural syndrome should be visible to the patient and to those around them alike. In their original series of **27** patients with presumed temporal lobe epilepsy, those patients did score higher on the inventory than healthy comparison subjects, and for a time this looked like the objective confirmation the epileptic-personality tradition had

always lacked. The instrument matters for the exam less as a clinical tool - it is not used to diagnose anyone - than as the hinge of the whole debate: it made the traits measurable, and once they were measurable they could be tested against the right control groups, which is precisely how the syndrome came undone.

27.3 Laterality: an emotional right, an ideational left

The claim that the side of the focus shapes the character. One of the inventory's most quoted findings was that the trait profile seemed to differ with the **side of the temporal focus**. Patients with a right temporal focus were reported to show the more overtly emotional traits - a deepened, sometimes sorrowful emotionality and a strong moralising tendency - while those with a left temporal focus showed a more ideational, ruminative profile weighted towards religious and philosophical preoccupation, humourlessness and a sense of personal destiny. The distinction was seductive because it fitted a broader intuition that the right hemisphere is the more affective and the left the more verbal and conceptual, so that a discharging right temporal lobe might exaggerate emotional expression and a discharging left temporal lobe might exaggerate the life of ideas. It is worth carrying as a described association and a favourite of examiners, but it must be carried with its health warning: the laterality claims were later disputed and have not been robustly replicated, and they belong to the same body of findings that the specificity critique went on to undercut. The accompanying figure sets the profiles side by side; the table below holds them for revision.

SIDE OF TEMPORAL FOCUS	TRAIT PROFILE	REPRESENTATIVE FEATURES
Right temporal	overtly emotional	deepened emotionality, a sorrowful affect, hypermoralism
Left temporal	ideational and ruminative	religiosity, philosophical preoccupation, humourlessness, a sense of personal destiny

The disputed Bear-Fedio laterality contrast: emotional traits reported with right temporal foci, ideational traits with left. The association is quoted often but was not robustly replicated.

27.4 Why it is not an epilepsy-specific behavioural syndrome

The control groups that dismantled the concept. The decisive objection to the epileptic personality is one of specificity, and it is the single most examinable point in the topic. When the same rating traits were measured not only in patients with epilepsy but in psychiatric patients without epilepsy and in people with comparable physical disability but no seizures, those comparison groups showed the traits too. A pattern that appears just as readily in the non-epileptic sick and disabled cannot be a signature of epilepsy; it is a marker of chronic illness, brain injury and the psychosocial weight of a disabling condition, distributed across many populations rather than owned by one. Kaufman's is explicit that these are best understood as not an **epilepsy-specific** phenomenon - features of brain damage and its circumstances rather than a distinct, codable personality disorder - and that contemporary video-EEG-based studies have failed to corroborate a discrete epileptic personality. The reasons the concept unravelled are worth listing in order:

- the traits appear in non-epileptic psychiatric patients and in equally disabled controls, so they are not specific to epilepsy;
- they are better read as markers of brain damage and chronic illness than as a categorical personality disorder;
- the rating instruments cannot separate the effect of the epilepsy from the effect of the life lived around it;
- and modern studies with video-EEG confirmation have not reproduced a discrete syndrome.

TRAP **Reifying the trait list into an epilepsy-specific personality disorder.** The candidate who has just learnt the Bear-Fedio traits is tempted to treat them as diagnostic of temporal lobe epilepsy, or as a personality disorder caused by it. They are neither: the same traits appear in controls who do not have epilepsy, and they index brain injury and its burdens rather than a categorical disorder. The examiner is testing whether you know the concept was refuted, not whether you can recite the list.

IN INDIA

Epilepsy already carries a heavy social penalty here - it can end a marriage negotiation, bar a school place and cost a job - so an added label of "epileptic personality" attaches a second, spurious stigma to the first, and it does so on a concept the evidence has discredited. Where access to video-EEG, formal neuropsychology and unhurried longitudinal review is uneven, an interictal trait is especially easily misread as a fixed defect of character. The safer discipline is to describe the behaviour actually observed, to attribute it cautiously, and never to let a refuted label narrow a patient's social options in a setting where the social cost of the word is real.

27.5 Viscosity: the one interictal marker that endures

The trait that survives the critique. If most of the epileptic personality dissolved under scrutiny, one general interictal phenomenon has kept a more robust clinical identity: **viscosity**. Lishman describes it as a tendency to prolonged, repetitive, circumstantial and pedantic social interaction, in which the patient cannot easily bring an exchange to a close. The conversation adheres: the same preoccupations are circled again and again, detail is heaped on detail, the point is approached by a long and winding road, and the patient holds the listener past every natural ending, so that interviews run long and disengagement takes effort. What lifts viscosity above a mere manner is the way it is best understood - not as stubbornness or neediness but as a fundamental abnormality of language and social communication, a disturbance in the pragmatics of talk and turn-taking. Seen this way it overlaps with the communication style described on the autistic spectrum, where the machinery of reciprocal conversation is likewise disturbed, and that framing is more useful clinically than reading the behaviour as a character flaw. Viscosity also appears, under the same name, as one strand of the named Gastaut-Geschwind cluster taught elsewhere in this chapter; here it is the general interictal phenomenon in its own right - circumstantial, over-detailed speech that circles a restricted range of preoccupations, resists closure, and holds the listener with an adhesive, earnest quality that is effortful to leave.

27.6 The substrate: impaired social cognition and theory of mind

Where the interpersonal change may come from. A more mechanistic account of why some patients with temporal lobe epilepsy become interpersonally difficult has grown from the study of social cognition. Kaplan and Sadock report that patients with temporal lobe epilepsy show impaired social cognition and **theory of mind**, with measurable deficits in affective as well as cognitive empathy - the capacity to feel with another and the capacity to model what another is thinking. If a patient reads other minds less accurately and shares their feelings less readily, then the surface behaviour follows: reciprocity falters, conversations misjudge the listener's state, and the interpersonal texture the older literature labelled as personality can be reframed as a consequence of a disturbed social brain. The relevant machinery is the temporolimbic and prefrontal circuitry that supports social inference, sitting close to the very structures that generate temporal lobe seizures, which makes it plausible that chronic disturbance there should show up as altered social behaviour; the anatomy of the limbic system in full belongs to the Functional Neuroanatomy and Neurophysiology notes, and the general neuropsychological and cognitive effects of epilepsy are a separate topic taught in their own right elsewhere in this chapter. What this substrate adds is a kinder and more accurate lens: the behaviour is not chosen, and it is not moral failure.

> **PITFALL**
>
> The common clinical error is to read a temporal lobe epilepsy patient's poor social reciprocity, tangential over-talking and blunted attunement as a difficult or wilful personality, and to respond with irritation rather than accommodation. If impaired social cognition and empathy underlie the behaviour, that response is both unfair and unhelpful: it blames a patient for a deficit they cannot simply switch off. Recognising the social-cognitive basis shifts the clinical stance from exasperation to structured support - clearer communication, more patience with disengagement, and involvement of family who can be taught the same understanding.

27.7 Personality disorder that genuinely clusters with epilepsy

A real comorbidity, distinct from the mythical syndrome. Rejecting the epileptic personality does not mean epilepsy and disordered personality never travel together; they do, and the association is genuine. Kaplan and Sadock report that formal personality disorders are highly prevalent in epilepsy, more so than in the general population, and that the most common of them is **borderline personality disorder**, with dependent and avoidant patterns also over-represented - Lishman puts the excess of these traits at roughly **two-fold** relative to unaffected comparison groups. Keeping this straight turns on a pair of distinctions. First, a categorical personality disorder is a different object from the vague interictal trait description: borderline, dependent and avoidant disorders are recognised diagnoses with their own criteria, and finding them more often in epilepsy is an epidemiological fact, not a resurrection of the epileptic personality. Second, the enduring personality picture must be separated from the interictal dysphoric disorder of epilepsy, the episodic mood syndrome taught in its own right elsewhere in this chapter; a persistent way of relating is not the same as a fluctuating affective state, and conflating them muddles the picture. The clinical value of the comorbidity is that these disorders

carry their own management and their own risks, and they are missed if the clinician is looking only for a mythical epileptic character.

27.8 Who is at risk, and what recognition should change

The recognition changes what you understand more than what you prescribe. Some features of the epilepsy make an interictal behavioural or personality change more likely, and knowing them lets the clinician anticipate rather than discover it. Kaufman's identifies a recognisable risk profile: **early-onset epilepsy**, seizures that secondarily generalise, and bilateral EEG changes, with a superimposed psychosis or mood disorder raising the risk further still. The markers to weigh are:

- an early age at onset of the epilepsy;
- seizures that secondarily generalise rather than staying focal;
- bilateral, rather than strictly unilateral, EEG changes;
- and comorbid psychosis or a mood disorder, which compounds the risk.

There is no specific pharmacotherapy for interictal personality change itself; the point of recognising it is to interpret the patient correctly and to find the treatable disorders that hide behind it. Management therefore rests on the same footing as the rest of epilepsy-psychiatry care, framed by the ILAE and NICE epilepsy guidelines for the seizures and by the epilepsy-specific psychiatric-management literature for the behaviour. The **LOCUS** of care is overwhelmingly the outpatient clinic, a chronic pattern followed over time, reaching inpatient or emergency settings only through a comorbidity - a psychosis, a mood crisis or a safety concern - never through the trait change itself. The **focus** moves with the horizon: the acute task is recognition and differentiation, separating a described interictal trait from a categorical personality disorder, from the interictal dysphoric disorder and from an evolving psychosis; the short-to-mid-term task is to optimise seizure control and to screen for and treat the mood, anxiety and psychotic comorbidities that carry the real morbidity; the long-term task is psychosocial, supporting relationships, work and adjustment around a stable temperament. The **modus** spans every mode without aiming a drug at personality: the pharmacological lever is pointed at seizures and at any comorbid disorder; the psychological and social modes carry most of the work through psychoeducation, family involvement and attention to marital and occupational consequences; and the procedural lever is the optimisation of antiepileptic treatment that serves the epilepsy at large.

GOOD TO REMEMBER

When a patient with long-standing temporal lobe epilepsy strikes you as sticky in conversation, over-serious and hard to read socially, name it to yourself as a described interictal trait, not as an epileptic personality and not as a chosen difficulty. Then do the useful thing the label cannot do: screen deliberately for depression, anxiety and psychosis, because those are prevalent, treatable and far more dangerous than any trait, and they are exactly what a fixation on personality causes you to miss.

No single type	18	Not specific	Borderline
OF EPILEPTIC PERSONALITY	BEAR-FEDIO PUTATIVE TRAITS	TRAITS ALSO SEEN IN CONTROLS	COMMONEST PERSONALITY DISORDER IN EPILEPSY

There is no single epileptic personality: interictal traits are neither universal in epilepsy nor specific to it, are concentrated in a temporal lobe subgroup, and index brain change rather than a categorical disorder; viscosity is the one durable marker, and the clinical priority is to screen for the treatable psychiatric comorbidities the label conceals.

28 · Gastaut-Geschwind syndrome: hyperreligiosity, hypergraphia, viscosity, altered sexuality

This is the eponym examiners love and candidates over-claim. The Gastaut-Geschwind syndrome names a recognisable pattern of behaviour and personality that emerges in a subset of people with long-standing temporal lobe epilepsy - not during a seizure and not in its immediate aftermath, but in the settled periods between fits. It is one of the very few places in neuropsychiatry where a distinctive interictal behavioural signature has been tied, however loosely, to a particular epileptogenic zone, and that is exactly why it earns marks: it forces the candidate to hold two ideas at once, that the cluster is real and clinically recognisable, and that its status as a specific, universal consequence of temporal lobe epilepsy is far from settled. Hold the first half without the second and you sound credulous; hold the second without the first and you sound as if you have never met such a patient.

The syndrome is built from four cardinal features: a tendency to write, an intensified preoccupation with religious and metaphysical themes, a distinctive interpersonal and conversational stickiness, and a change in sexual behaviour that is usually a reduction of drive. Aggressiveness is sometimes added, though aggression, irritability and episodic dyscontrol in epilepsy are treated as their own topic elsewhere in this chapter. Kaplan and Sadock's Comprehensive Textbook of Psychiatry frames the cluster and its laterality, while Lishman's Organic Psychiatry supplies the original formulation and the objective evidence for the writing. What follows takes the syndrome as a whole, then each feature in turn, and closes with what recognising it should and should not change.

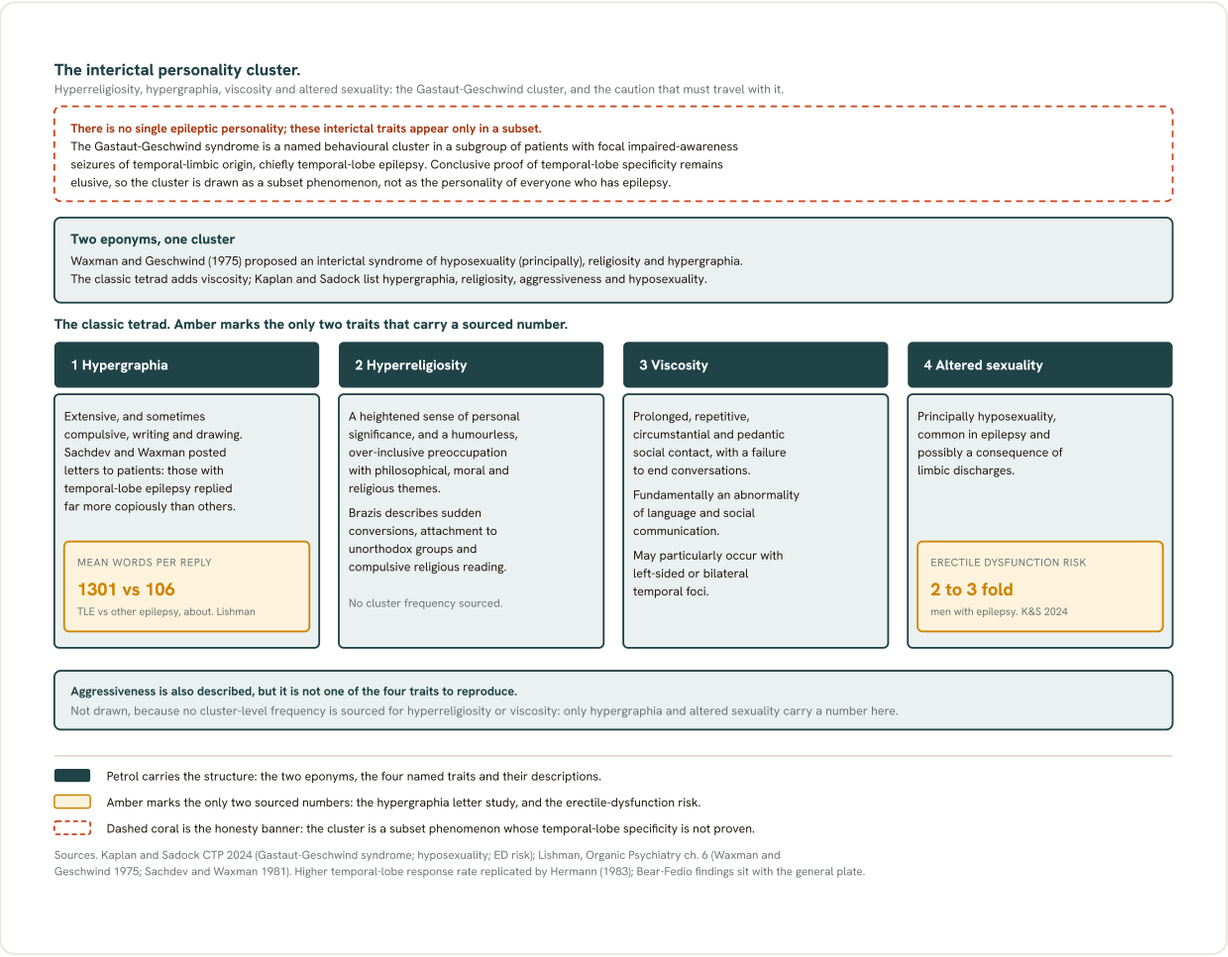


Fig 28.1 **The interictal personality cluster.** The Gastaut-Geschwind cluster is hyperreligiosity, hypergraphia, viscosity and altered sexuality (principally hyposexuality), seen in a subset of patients with focal impaired-awareness seizures of temporal-limbic origin, not in everyone with epilepsy. Only two of the four carry a sourced number: hypergraphia, shown objectively by Sachdev and Waxman's letter study (mean reply about 1301 words in temporal-lobe epilepsy against 106 in other epilepsy), and altered sexuality, where men with epilepsy carry a two- to threefold risk of erectile dysfunction. Aggressiveness is listed alongside the tetrad but is not one of the four, and conclusive proof of temporal-lobe specificity remains elusive.

28.1 A named interictal cluster, of uncertain specificity

Between the seizures, in some of the patients, from a temporal-limbic focus. The **Gastaut-Geschwind syndrome** is an interictal behavioural cluster arising in a subset of patients whose seizures are focal impaired-awareness seizures of temporal-limbic origin. Three qualifiers in that sentence carry the whole examinable weight. It is interictal: the behaviour belongs to the enduring personality of the patient between attacks, not to the ictus or the postictal state, which is what separates it from the peri-ictal syndromes that occupy much of this chapter. It is a subset phenomenon: the great majority of people with temporal lobe epilepsy never develop the full picture, so the syndrome is neither a diagnostic marker of the epilepsy nor an inevitable outcome of it. And it is tied to a temporal-limbic focus, which is the anatomical claim that makes it interesting and the anatomical claim that remains only partly proven. Kaplan and Sadock are explicit that conclusive proof of specificity to temporal lobe epilepsy remains elusive: the cluster is described most often in this population, but whether it is genuinely specific to it, or a more general accompaniment of chronic epilepsy and its psychosocial burden, has never been settled.

It is therefore best carried as a clinical gestalt with a strong association and a weak proof, not as a fixed lesion-to-behaviour law.

RULE **It is interictal, partial and associated - never ictal, universal or proven.** The Gastaut-Geschwind cluster is a feature of the personality between seizures, seen in only a fraction of temporal lobe epilepsy, and linked to that epilepsy by association rather than by demonstrated specificity. Every examinable error in this topic comes from dropping one of those three restraints.

TRAP **Treating the syndrome as a proven, near-universal stamp of temporal lobe epilepsy.** Candidates who have just learnt the four features are tempted to promote them into a specific, reliable sign - as if a religious, prolific writer must have a temporal focus, or as if temporal lobe epilepsy must produce the cluster. The source itself withholds that certainty: the features occur in a subset and the specificity is unproven, so the inference the examiner is probing runs only weakly, and never as a rule.

28.2 The paradigm: behaviour tied to localisation

An argument about the brain, dressed as a syndrome. The modern formulation belongs to Waxman and Geschwind, in 1975, who described an **interictal behavioural syndrome** whose core they took to be hyposexuality above all, together with deepened religiosity and a tendency to write. What made the proposal influential was less the list than the ambition behind it: they offered the cluster as a paradigm, a worked example of how a circumscribed change in behaviour might be read as the signature of a particular cerebral localisation. When the idea that focal brain activity could sculpt personality was still contentious, an epilepsy that seemed to leave a consistent behavioural fingerprint was powerful evidence for localisationism, and the syndrome has carried that charge ever since. This is also why it attracts as much scepticism as devotion. The very features that make it a neat demonstration of brain-behaviour linkage - that they are qualitative, that they overlap with ordinary human variation, that religiosity and volubility are common enough in people without epilepsy - are exactly what make it hard to prove that the epilepsy, rather than the life lived around it, produced them. State both the claim and its fragility in the same breath, because the examiner's follow-up is almost always whether the evidence holds.

28.3 The four cardinal features as a cluster

Four strands that tend to travel together. Taken together, the syndrome's cardinal features are hypergraphia, hyperreligiosity with a deepened metaphysical interest, viscosity, and altered sexuality that is principally a reduction of drive; Kaplan and Sadock's own enumeration lists hypergraphia, religiosity, aggressiveness and hyposexuality, so aggressiveness recurs as a fifth, less constant member. The clinically important word is cluster. The features are described as tending to co-occur and to shade into one another - the same overinclusive, weighty seriousness runs through the compulsive writing, the moral-religious preoccupation and the conversational adhesiveness - but they do not appear as a fixed tetrad in which all four are always present. A patient may show florid hypergraphia with little religiosity, or a deep religiosity with unremarkable writing. The accompanying figure sets the features around their shared temporal-

limbic substrate; for revision, a four-part memory hook holds them without implying that all four must be present at once.

MNEMONIC

Write, Worship, Weld, Wane

The four cardinal features as four W's: the patient **writes** copiously and often compulsively (hypergraphia), **worships** with intensified religious and metaphysical preoccupation (hyperreligiosity), **welds** to people and to a narrow range of topics in a sticky, hard-to-interrupt way (viscosity), and finds sexual drive **waning** (hyposexuality). The hook is a memory aid only; it asserts nothing about how many of the four any given patient will show.

CARDINAL FEATURE	HOW IT PRESENTS	THE POINT TO HOLD
Hypergraphia	extensive, sometimes compulsive writing and drawing	the one feature with objective, quantified support
Hyperreligiosity	heightened significance and moral-religious preoccupation, at times sudden conversions	hardest to separate from cultural norm
Viscosity	repetitive, circumstantial talk around a restricted range of topics	may favour a left-sided or bilateral temporal focus
Altered sexuality	principally hyposexuality, a lowered drive	has a probable neurophysiological, not only psychosocial, basis

The Gastaut-Geschwind cluster across its four cardinal features. They tend to co-occur and share a weighty, overinclusive quality, but the syndrome is a cluster, not an all-or-none tetrad.

28.4 Hypergraphia: the feature you can measure

The pen that will not stop. Of the four features, **hypergraphia** is the one with a definition sharp enough to be tested and evidence solid enough to be quoted. Lishman describes it as a tendency towards extensive and sometimes compulsive writing and drawing: the patient keeps voluminous diaries, copies out passages, annotates and re-annotates, writes long unsolicited letters, and produces the material with a driven, meticulous quality rather than for any external purpose. The content often carries the same moral or religious weight that colours the rest of the syndrome, which is one of the ways the features are said to cohere. What lifts hypergraphia above anecdote is that it has been shown objectively. In a much-cited study, Sachdev and Waxman, in 1981, sent a simple letter to patients and measured how much they wrote back: those with temporal lobe epilepsy replied far more copiously than patients with other forms of epilepsy, with a mean reply of about **1301** words against about **106** words for the comparison group. The size of that gap is the point - it is not a marginal tendency but an order-of-magnitude difference in output - and the higher response rate was afterwards replicated by Hermann, in 1983. Hypergraphia is therefore the feature to reach for when asked whether any of this can be demonstrated rather than merely asserted.

28.5 Hyperreligiosity and deepened metaphysical interest

Not a delusion, but a change of emphasis. **Hyperreligiosity** here is a heightened, humourless sense of the significance of things, an overinclusive preoccupation with philosophical, moral and religious themes that comes to dominate the patient's inner and outer life. Kaplan and Sadock describe its more florid forms:

- a pervasive sense of heightened significance attaching to ordinary events;
- humourless, overinclusive dwelling on philosophical and moral questions;
- sudden religious conversions, at times more than one over a lifetime;
- attachment to unorthodox or fringe religious groups;
- and compulsive reading of religious texts.

The clinical craft lies in not mistaking this for what it is not. It is a change in the intensity and preoccupying quality of belief, arising in a person whose reality testing is otherwise intact, and it is not, in itself, a psychotic phenomenon; treating a patient's deepened devotion as a delusion to be suppressed both misreads the sign and injures the therapeutic relationship. Equally, the feature must not be inflated into a diagnostic test. Intense religiosity is common in people who have never had a seizure, and the syndrome supplies no threshold above which faith becomes pathological.

IN INDIA

Nowhere is the boundary between the sign and the norm harder to draw than in a society where intense devotion, daily ritual, pilgrimage and scriptural reading are woven into ordinary healthy life. A patient who prays at length, keeps religious diaries and speaks constantly of moral and spiritual matters may be entirely typical of their family and community, not hyperreligious in the syndromic sense. The change that matters is a shift from the person's own baseline - a new, driven, overinclusive preoccupation that those who know the patient recognise as different - and it is read against that baseline and that culture, never against a Western yardstick of ordinary belief. Getting it wrong risks pathologising healthy faith or, in the other direction, missing a genuine change because the content looks culturally unremarkable.

28.6 Viscosity: the interpersonal and conversational stickiness

Hard to start a conversation with, harder to end one. As a feature of the cluster, **viscosity** is the tendency to talk repetitively and circumstantially about a restricted range of topics, so that speech and social contact acquire an adhesive, hard-to-disengage quality. The patient circles the same preoccupations, resists moving the conversation on, and holds the listener in a way that can feel warm and earnest but is effortful to leave; interviews run long and return, again and again, to a narrow set of concerns. Within the Gastaut-Geschwind picture, viscosity is the interpersonal correlate of the same overinclusive, weighty cognitive style that produces the writing and the religiosity. Kaplan and Sadock note a laterality association worth carrying: viscosity may fall particularly to those with a left-sided or bilateral temporal focus, in contrast to features less tied to the side of onset.

Viscosity is also the point at which this topic brushes against its neighbour. The broader interictal personality change of temporal lobe epilepsy - the general alteration in temperament of which conversational and interpersonal stickiness is one described element - is a separate subject taught in its own right elsewhere in this chapter, as is the anatomy and semiology of temporal lobe epilepsy itself. Here the concern is narrower: viscosity as one cardinal strand of the named eponymous cluster, not the general personality change of which it can also be read as a part. Keeping the two apart is what stops this account from swallowing its neighbour.

28.7 Altered sexuality: hyposexuality and its basis

Usually less, not more. The change in sexual behaviour that completes the cluster is, in the great majority, a reduction: **hyposexuality**, a lowered sexual interest and drive, common in epilepsy generally and not confined to the syndrome. What makes it examinable is the argument about mechanism. It would be easy to attribute a flattened libido in a person with chronic epilepsy entirely to the psychosocial weight of the illness, to mood, or to medication, and all of those contribute. But the feature is thought to have a genuine neurophysiological component, possibly a consequence of the same limbic discharges that drive the seizures, so that the temporal-limbic disturbance shaping the rest of the personality also reaches the circuitry of sexual drive. The evidence marshalled for a physical basis is that men with epilepsy carry about a **two- to threefold** increased risk of erectile dysfunction, a raised rate hard to explain by attitude alone and pointing instead to a physiological substrate. The limbic mechanism is named here only as the probable seat of the change; the anatomy of the limbic system in full belongs to the Functional Neuroanatomy and Neurophysiology notes.

> **PITFALL**
>
> The recurring clinical error is to file a patient's lowered libido entirely under antiepileptic side effect or low mood, adjust nothing, and move on. That misses two things: the change may have a neurophysiological basis in the epilepsy itself, and, whatever its cause, it is distressing, treatable and almost never volunteered. Patients rarely raise sexual dysfunction unprompted, so unless it is asked about directly it goes unrecorded and unaddressed. The safer habit is to enquire, to weigh the contributions of drug, mood and disease rather than assuming one, and to treat the problem as real.

28.8 Recognising the cluster and what to do with it

The syndrome changes what you understand more than what you prescribe. There is no specific pharmacotherapy for the Gastaut-Geschwind cluster, and the point of recognising it is largely to interpret a patient correctly and to avoid two opposite mistakes: medicalising a personality into a disease, and dismissing genuine, treatable distress as mere temperament. Management therefore rests on the same foundations as the rest of epilepsy-psychiatry care, framed by the ILAE and NICE epilepsy guidelines for the seizures themselves and by the epilepsy-specific psychiatric-management literature for the behavioural picture, with the accompanying textbook accounts as connective cover.

The **LOCUS** of care is overwhelmingly the outpatient clinic; this is a chronic interictal pattern followed over time, and it reaches inpatient or emergency settings only through a comorbidity - a superimposed psychosis, a mood crisis or a safety concern - rather than through the cluster itself. The **focus** shifts with the horizon. The acute task is recognition and differentiation: placing the hyperreligiosity, hypergraphia, viscosity and hyposexuality against the patient's own baseline and culture, and separating the deepened religiosity of the syndrome from a psychotic illness that would demand a different response. The short-to-mid-term task is to address what is treatable - optimising seizure control, reviewing the contribution of medication and mood to the sexual and behavioural change, and enquiring directly about the hyposexuality the patient will not raise. The long-term task is to support adjustment and relationships around a stable interictal personality, and to watch for the comorbidities that do carry specific treatment. The **modus** spans every available mode without leaning on a drug for the cluster: the pharmacological lever is aimed at seizures and at any comorbid psychiatric disorder, not at personality; the psychological and social modes carry most of the work, through psychoeducation of patient and family, validation of the distress, and attention to the marital, occupational and sexual consequences; and the procedural lever is the same optimisation of antiepileptic treatment that serves the epilepsy at large.

GOOD TO REMEMBER

When a patient with long-standing temporal lobe epilepsy strikes you as intensely religious, endlessly voluble on a few themes, a copious writer and sexually withdrawn, name the pattern to yourself as the interictal cluster it may be, then do the two useful things it points to. Ask directly about sexual function, because hyposexuality is treatable and unspoken, and hold the deepened religiosity as a change in personality rather than a delusion unless independent psychotic features say otherwise. The recognition is worth marks and, more importantly, it stops you from treating a personality as a psychosis.

Interictal	A subset	1301 vs 106	2 to 3x
WHEN THE CLUSTER APPEARS	OF TEMPORAL LOBE EPILEPSY	HYPERGRAPHIA, WORDS PER REPLY	ERECTILE DYSFUNCTION RISK IN MEN

The Gastaut-Geschwind syndrome is an interictal behavioural cluster - hypergraphia, hyperreligiosity, viscosity and hyposexuality - seen in only a subset of temporal-limbic epilepsy and linked to it by association rather than proven specificity; recognise it to interpret the patient and to treat the hyposexuality, never to convert a personality into a psychosis.

29 · Aggression, irritability and episodic dyscontrol in epilepsy

Few topics in this chapter are as misremembered as this one. The lay intuition, and a good deal of medico-legal folklore, holds that epilepsy is a disorder of sudden violence: that a seizure can seize the body into a purposeful attack and then wipe the memory of it clean. The examinable reality is close to the opposite. Aggression is certainly over-represented in people with epilepsy, but it is overwhelmingly a phenomenon of the periods between seizures rather

than of the seizure itself, and on the rare occasions it is directly seizure-related it is crude, brief and undirected. Marks are awarded for holding that distinction steadily, because it is the hinge on which both clinical management and forensic opinion turn: get it wrong and a candidate will either medicalise ordinary interpersonal violence as "epileptic" or, worse, endorse a seizure as the author of an organised crime.

The orderly way to hold the subject is to sort aggressive behaviour by its timing relative to the seizure - the same peri-ictal axis, from preictal through ictal and postictal to interictal, that organises the chapter's other syndromes - and then to set beside it two constructs that recur in the exam and the courtroom: the contested notion of an episodic dyscontrol syndrome, and the irritability that colours the phases around a fit. Kaplan and Sadock's Comprehensive Textbook of Psychiatry supplies most of the phenomenology, Kaufman's Clinical Neurology for Psychiatrists the account of interictal violence, and the forensic-psychiatry literature the vocabulary of dyscontrol and criminal responsibility.

29.1 The central point: aggression is usually interictal, not ictal

The discharge is rarely the culprit. Start from the finding that anchors the whole topic: the great majority of aggressive behaviour seen in people with epilepsy is not the direct expression of **epileptiform activity**, and does not coincide with a seizure at all. It belongs to the interictal state, and it answers to the ordinary determinants of aggression in any population - temperament, social adversity, intoxication, provocation and comorbid mental illness - rather than to the electrical event. This matters because the older literature, and popular belief, ran the other way, imagining a specifically epileptic drive to violence lodged in an abnormal personality. The interictal personality change of temporal lobe epilepsy, and the named Gastaut-Geschwind cluster, are real and are taught in their own right elsewhere in this chapter, but neither is a licence to read a given assault as a seizure equivalent. What aggression there is tends to keep company with two associations in particular: a coexisting psychotic illness, or the impulse-control diagnosis of **intermittent explosive disorder**. Kaplan and Sadock frame both points, and both push the clinician toward the same discipline. Before attributing violence to the epilepsy, look for the interictal cause that is almost always there.

RULE **Aggression in epilepsy is interictal until proven otherwise.** The default attribution for a violent act in a person with epilepsy is not the seizure but the interictal state and its comorbidities, because most such aggression bears no relation to the discharge at all. A seizure-based explanation has to be earned by the timing and the character of the act, never assumed from the diagnosis on the chart.

29.2 Ictal violence: what a seizure can and cannot do

Crude, brief and never planned. When violence genuinely is ictal, generated by the seizure itself, it takes a highly restricted form. **Ictal violence** is not organised, not premeditated and not goal-directed; what actually occurs at the onset of a focal impaired-awareness seizure is confined to **simple violent automatisms** such as spitting, the utterance of profanities and an undirected flailing of the arms, the same fragmentary motor and vocal automatisms that characterise these seizures in general. These are reflex fragments, not actions with an object. Kaplan and Sadock

make the strong version examiners want to hear: coordinated, purposeful violence has rarely if ever been convincingly shown to be a seizure. The mechanistic reason turns a fact to be memorised into one that can be reasoned: the impaired awareness, motor disorganisation and dense amnesia of the ictus are incompatible with the sustained intention, target selection and sequencing a purposeful assault demands. A person cannot at once be lost in the disorganised automatism of a focal impaired-awareness seizure, a seizure type defined in its own topic, and be executing a planned attack on a chosen victim. The clinical and forensic weight of this is large, because it is precisely the plea that "the seizure made me do it" which the examinable facts are built to test and, almost always, to defeat.

MNEMONIC

A seizure spits, it does not plan

Everything ictal violence can do sits in that line. The ictus can throw out a fragment - a reflex, a curse, an undirected swipe - but it cannot supply a target, a sequence or a plan, because the seizure state is disorganised and amnesic. Whenever a violent act was coordinated and purposeful, the discharge did not author it, and the true explanation lies elsewhere on the timeline.

29.3 Postictal violence: resistance, not assault

The struggling patient is trying to get away, not to attack. The second seizure-related window is the postictal one, and the violence that belongs to it has a distinctive quality. **Resistive violence** arises during postictal delirium: as the patient surfaces through a clouded, disoriented aftermath, the attempts by staff or family to help or to physically restrain are misread as an attack, and the patient fights back against the perceived threat. It is nondirected and defensive rather than predatory, and the archetype is the confused patient wrestling free of the hands holding them down. The postictal confusion and delirium in which this happens is itself a peri-ictal syndrome taught in its own right elsewhere in this chapter, and delirium as a general state, with its assessment, subtypes and prevention, belongs to the Stroke, TBI, delirium and focal brain syndromes notes; here the point is only that the disorientation supplies the misinterpretation that drives the struggle. A related and more sustained form of post-seizure aggression can appear as part of a postictal psychosis, the abrupt psychosis after a lucid interval that is covered separately in this chapter, where agitation and disturbed behaviour run a longer subacute course. Recognising postictal violence as fundamentally reactive rather than intentional changes how it should be met, and that is where most wards go wrong.

PITFALL

The commonest way this goes wrong is to read a postictally delirious patient's struggling as deliberate assault and to answer it with force - more hands, tighter holds, a show of physical control. That is exactly the input the disoriented brain misreads, and it escalates the very resistance it was meant to stop. The safer response is to lower stimulation, keep the patient safe with the least restraint that will do, allow the delirium to clear, and sedate rather than overpower if control is genuinely required.

29.4 Interictal violence and its correlates

Verbal and minor, and concentrated in particular patients. Away from the seizure, the aggression that people with epilepsy show is usually low-grade. Kaufman's Clinical Neurology for Psychiatrists describes **interictal violence** as consisting, in the main, of verbal outbursts and minor physical acts rather than serious assault, and, the discriminating observation, as concentrated in patients who are antisocial, psychotic or schizophrenic, or intellectually disabled. The corollary is the part examiners like to probe: violent and non-violent patients with epilepsy do not differ in their seizure type, their EEG or their antiepileptic treatment. What predicts interictal violence, in other words, is the person and the comorbidity, not any feature of the epilepsy, which is the same lesson the ictal and forensic data teach from their own directions. A consistent set of correlates travels with aggression in this population, and, tellingly, none of them is an epileptological variable:

- subnormal intelligence;
- lower socioeconomic status;
- childhood behaviour problems;
- a history of prior head injury;
- and, at the level of the brain, possible **orbitofrontal damage**.

Read as a group these are the familiar risk markers for aggression in general - developmental, social and frontal - reinforcing the message that epilepsy-related aggression largely arrives by the usual routes in a person who also happens to have epilepsy. Where a psychotic illness is the driver, its separation from a primary schizophrenic disorder rests on features developed in the Schizophrenia: aetiology, phenomenology, classification and course notes, and is not reopened here.

RELATION TO THE SEIZURE	CHARACTER OF THE VIOLENCE	ORGANISED AND PURPOSEFUL?	TYPICAL SETTING
Ictal	brief, fragmentary automatisms	No, essentially never	at the onset of a focal impaired-awareness seizure
Postictal	non-directed, resistive struggling	No, reactive to a misperceived threat	during postictal delirium or a postictal psychosis
Interictal	mostly verbal, with minor physical acts	Variable, driven by the person not the fit	in antisocial, psychotic or intellectually disabled patients

Aggression in epilepsy sorted by its timing to the seizure. The single examinable thread is that only the ictal and postictal rows are seizure-generated, both are crude and unplanned, and the organised violence a courtroom cares about fits none of the three.

29.5 Epilepsy, crime and criminal responsibility

Over-represented in prisons, but not more violent. The forensic data close the argument the phenomenology opens. Epilepsy is indeed over-represented behind bars: its prevalence among prison inmates runs at **about two to four times** that of the general population. The naive

inference, that epilepsy therefore breeds violent crime, is exactly what the same data refute. Epileptic prisoners commit no more violent offences than non-epileptic prisoners, and EEG abnormalities do not correlate with violent offending. The over-representation is better explained by the upstream company aggression already keeps - the social adversity, head injury, low intelligence and comorbid disorder catalogued among the correlates - than by any direct criminogenic effect of the seizures. The two halves together are the whole point: a raised prevalence that carries no raised violence. For the witness box this is decisive. An organised, directed violent act cannot be handed to a seizure merely because the accused has epilepsy and an abnormal EEG; the timing must place the act within a seizure or its immediate aftermath, and its character must match the crude, unplanned template that is all a seizure can produce. Absent both, the epilepsy is a comorbidity that travelled to court with the defendant, not an explanation for what they did.

TRAP **Accepting the seizure as the author of an organised crime.** The classic error is to reason from an epilepsy diagnosis and an abnormal EEG straight to ictal responsibility for a purposeful, directed act of violence. It fails on two independent counts: seizure-generated violence is only ever crude and unplanned, and abnormal EEGs do not mark out the violent from the non-violent. Demand the timing and the character before you concede an ictal explanation.

29.6 Episodic dyscontrol syndrome: a contested construct

A forensic label, not an epilepsy diagnosis. The term **episodic dyscontrol syndrome** denotes explosive episodes of sudden, uncontrolled violence for which the person is afterwards apparently amnesic. Its surface resemblance to a seizure - abrupt, stereotyped, out of character, followed by a memory gap - is exactly why it recurs in this chapter and the courtroom, and why it is handled with caution. It is a contested, largely forensic construct rather than a specific epilepsy diagnosis; the forensic-psychiatry literature, Jiloha's Forensic Psychiatry among it, places it among the labels invoked to explain violence and criminal responsibility rather than among the epilepsies proper. The candidate's task is to keep three things apart. The syndrome is not proof of an underlying seizure disorder, however seizure-like the narrative sounds. The apparent amnesia is not the genuine post-ictal amnesia of a fit, and can be hard to separate from the self-serving amnesia claimed after any violent act. And the label overlaps heavily with intermittent explosive disorder, the impulse-control diagnosis for which it often functions as an older synonym. Where the phenomenon is genuine it is usually discussed against a background of temporal lobe epilepsy, whose anatomy and semiology are set out separately in this chapter, but that association is one of clinical interest, not established causation. The sound exam stance is to treat episodic dyscontrol as a description in search of a mechanism, not a settled disease entity.

29.7 Irritability: the affective substrate

The lowered threshold behind the act. Aggression names the behaviour; irritability names the state that lowers the threshold for it, and the two are worth separating because they carry different clinical weight and are managed differently. Irritability, a heightened readiness to react with anger to ordinary provocation, is among the most common and most under-recorded neuropsychiatric symptoms in epilepsy, and it is often more corrosive to family life than the

seizures themselves, because it is continuous rather than episodic and rarely volunteered as a complaint. It is distributed across the same peri-ictal timeline that organises everything else in this topic. It clusters in the hours before a fit, as one strand of the preictal prodrome of dysphoria and irritability that heralds an attack in some patients, a prodrome taught in its own right elsewhere in this chapter. It is a defining component of the interictal dysphoric disorder of epilepsy, the pleomorphic interictal mood syndrome that likewise has its own dedicated topic and that must not be collapsed into an ordinary depressive episode. And it colours the postictal phase in many patients as the storm settles. The connecting message, rather than the syndromic detail that belongs to those owners, is this: irritability is the affective soil from which much epilepsy-related aggression grows, and because patients so rarely offer it, asking about it directly and validating it to the family is often the single most useful thing done in a review that was ostensibly about seizures.

29.8 Assessment and management

What to actually do with the aggressive patient who has epilepsy. Because most of this aggression is not ictal, management is mostly the management of the interictal cause, wrapped around the discipline of first optimising seizure control. The standard of care for the epilepsy itself is set by the ILAE and NICE epilepsy guidelines, while the behavioural disturbance draws on the epilepsy-specific psychiatric-management literature; none of the three licenses a reflex reach for sedation as the whole answer.

The **LOCUS** of care follows the risk and the phase. A florid postictal or psychotic disturbance with a real safety concern may need brief inpatient containment, and an acutely violent patient may first reach an emergency setting; established interictal aggression and irritability, by contrast, are outpatient work, built on careful assessment, treatment of the comorbidity and structured follow-up. The **focus** shifts with time. The acute target is immediate safety and de-escalation; the short-to-mid-term target is the treatable driver, the psychosis, the intermittent explosive disorder, the intoxication or the social crisis, identified by placing the act on the peri-ictal timeline and reconstructing its character; the long-term target is relapse prevention through genuine seizure control and through sustained treatment of the comorbid disorder. The **modus** spans every available mode. The pharmacological lever is aimed at the cause rather than at "aggression" as an abstraction, and where an antipsychotic is used, its selection against the seizure threshold is a decision handled in its own dedicated topic and not reopened here. The psychological and social modes carry much of the interictal work: behavioural analysis of triggers, family education about the reactive nature of postictal struggling, and attention to the adversity and substance use the correlates flag. The procedural lever is the optimisation of antiepileptic treatment itself, prophylactic against the very ictal and postictal windows in which seizure-related violence lives.

GOOD TO REMEMBER

Before you attribute any violent act to a patient's epilepsy, reconstruct two things: when it happened relative to the last seizure, and what it actually looked like. Seizure-related violence is confined to the ictus and its immediate aftermath, and it is always crude and unplanned. If the act was organised, directed and outside those windows, the epilepsy is a bystander, and the assessment should turn to the psychosis, impulse-control disorder, intoxication or social crisis that is doing the real work.

IN INDIA

Two realities sharpen this locally. Stigma still carries the old belief that epilepsy is a disorder of madness and violence, so families, and sometimes courts, over-attribute aggression to the seizures; part of the clinician's job is to correct that on the evidence, since most such aggression is interictal and the epilepsy explains little of it. At the same time the wide treatment gap means poorly controlled seizures, and the clusters that generate postictal disturbance, are common, so optimising antiepileptic treatment is often the single most neglected useful lever. Where a forensic question arises, the same discipline of timing and character applies, and the epilepsy must not become a blanket explanation for a directed crime.

Interictal	**Automatisms**	**Resistive**	**2 to 4x**
WHERE MOST AGGRESSION SITS	THE ONLY TRUE ICTAL VIOLENCE	POSTICTAL, A MISREAD RESTRAINT	EPILEPSY IN PRISON INMATES

Aggression in epilepsy is overwhelmingly interictal and driven by comorbidity, not by the discharge; genuine ictal violence is limited to simple, unplanned automatisms and postictal violence to reactive resistance, so an organised, directed violent act can almost never be laid at the door of a seizure - the single fact that governs both the ward and the witness box.

30 · Cognitive effects of epilepsy and of repeated seizures

The whole of this topic turns on a single act of attribution. A patient with epilepsy who is not thinking clearly hands the clinician three candidate explanations, and the marks here are awarded almost entirely for keeping them apart. The cognition may be impaired by the underlying brain pathology that also produced the epilepsy; by the seizures and the epilepsy syndrome themselves; or by the antiepileptic drugs. The cognitive and behavioural adverse effects of the antiepileptic drugs are taught in their own right elsewhere in this chapter and are deliberately left there. This topic owns the first two limbs, what the disease and the repeated seizures do to cognition, and the reward is for attributing correctly rather than reaching reflexively for the seizures as the author of every deficit.

The second habit to unlearn is the old notion of a specifically epileptic dementia: the belief that epilepsy is a progressively deteriorating disease of the mind, each fit leaving the brain a little poorer than it found it. Lishman's Organic Psychiatry is the anchor text, and the sober picture it assembles is that, for the great majority of people with epilepsy, this is untrue. Cognitive

impairment is real and common, but it is usually mild, usually non-progressive, and frequently present before the first seizure was ever recognised. A short list of named syndromes genuinely does dement, and holding those exceptions in mind is what keeps the general rule honest.

30.1 The myth of epileptic dementia

Uncomplicated epilepsy does not lower intelligence. The cleanest refutation comes from population data rather than clinic samples, which are always skewed toward the severe. Measured across an unselected community population of people with epilepsy, intelligence is normally distributed, and this holds even within the temporal lobe epilepsy subgroup, the very patients in whom folklore expected the greatest ruin. The classical demonstration is the **Isle of Wight study**, from which the conclusion is drawn that a lowered intelligence is not intrinsic to uncomplicated epilepsy. Everything hangs on the qualifier buried in the word "uncomplicated": where intelligence genuinely is low in a person with epilepsy, the low intelligence and the seizures are usually two products of one underlying brain lesion, not a sequence in which the epilepsy eroded a previously intact endowment. The term **epileptic dementia**, when used at all, should be reserved for the handful of syndromes that actually deteriorate, never applied to the ordinary patient whose fits are controlled and whose brain is otherwise sound. A candidate who can state that intelligence in epilepsy is normally distributed, and name the source, has dismantled the commonest misconception in the topic.

30.2 Three sources of impairment, and the task of telling them apart

Disease, seizures, drugs. Because cognition in epilepsy has more than one master, the clinician's first job is aetiological rather than descriptive. Impairment can arise from three broadly separable sources, and a given patient may carry all three at once:

- the antecedent brain pathology, the lesion, malformation or diffuse insult that generated the epilepsy and independently damaged cognition;
- the epilepsy itself, meaning the syndrome, the seizure focus and the burden of repeated seizures acting on the developing or mature brain;
- and the antiepileptic drugs, whose sedative and specific cognitive costs are the subject of their own dedicated topic in this chapter and are not reopened here.

Examiners linger here because the three dissociate on the axes that matter. The drug contribution is dose-related and largely reversible, responding to rationalising the regimen; the disease contribution tends to be fixed or slowly evolving; the antecedent contribution was present from the outset and will not move whatever is done to the seizures. Treating the picture as one thing, and in particular as all the fault of the seizures, is how a reversible drug load or a treatable comorbid depression gets missed while the epilepsy is blamed for damage it never did.

> **RULE** **Cognitive impairment in epilepsy is usually mild, usually non-progressive, and must be attributed before it is treated.** The default is not a dementing process. The clinical task is to apportion the deficit between the antecedent brain pathology, the epilepsy and the drugs, because only that apportionment tells you what, if anything, is reversible and what is fixed.

30.3 The impairment often precedes the first seizure

The most decisive evidence in the topic. If seizures were the engine of decline, cognition should be intact at diagnosis and fall thereafter. The paediatric-onset data show the opposite and are worth carrying verbatim. Children presenting with new-onset epilepsy have academic difficulties far more often than healthy controls, on the order of **about 26 per cent** against roughly **4 per cent**, and these problems are usually identified before the first recognised seizure. That temporal ordering is the whole argument: a deficit that is already present when the epilepsy declares itself cannot have been produced by the seizures that follow. It points instead to a shared antecedent, an early neurodevelopmental disturbance expressing itself both as the seizure disorder and as the learning difficulty, so the two are siblings rather than cause and consequence. This finding, associated with the work of Hermann and colleagues, is the empirical spine of the modern view that epilepsy-related cognitive problems are, in large part, not seizure-driven at all. It also reframes the conversation with families, who assume the fits are damaging the child's schooling and can be reassured that the relationship is more tangled and less bleak than that.

30.4 Impairment is broad, mild and present early in both syndrome types

Not a late complication of refractory disease. The antecedent story is matched at the other end by what is found shortly after onset. Careful neuropsychological study of recently diagnosed patients shows broad but mild cognitive impairments present in both generalised and localisation-related epilepsy close to seizure onset, when patients were assessed on average about ten months after onset. Two features earn marks. First, the impairment is not confined to the chronic, drug-resistant patient of the old textbooks; it is measurable near the beginning of the illness, before years of seizures or polypharmacy could accumulate. Second, it is not the exclusive property of the localisation-related, and specifically temporal, epilepsies; the idiopathic generalised epilepsies, long regarded as cognitively benign, carry their own mild diffuse burden. Taken with the antecedent data, this establishes the shape of the ordinary case: a modest, early, fairly stable impairment reflecting the brain's baseline state rather than a mounting toll. The pattern is a plateau, not a slope, and recognising it stops the clinician over-reading a stable deficit as an unfolding catastrophe.

30.5 The temporal lobe memory signature

Where the epilepsy does leave a disease-intrinsic fingerprint. None of this means epilepsy is cognitively silent, and the clearest disease-intrinsic signature belongs to temporal lobe epilepsy, whose anatomy and semiology are set out in their own topic and not reworked here. Its characteristic neuropsychological profile is one of **selective episodic memory** deficits, a difficulty laying down and retrieving new autobiographical and event memory that stands out against relatively better-preserved function elsewhere. The word selective must not be over-read: memory is to the fore, but impairments can occur across all cognitive domains, so a patient is not guaranteed normal on everything except memory. The emphasis is one of mechanism, the mesial temporal substrate of memory being the tissue most implicated, rather than a promise of isolated amnesia. This deficit is a property of the disease and its substrate, distinct in origin from the diffuse dulling the drugs impose, which is why disentangling the two at the bedside is both

possible and necessary. The interictal personality change that can accompany temporal lobe epilepsy is a separate construct with its own topic and is not a cognitive deficit in this sense.

30.6 Material-specific memory and lateralisation

Which memory fails tracks which side is affected. The temporal memory signature carries a refinement examiners love, because it links neuropsychology to laterality. Memory impairment in temporal lobe epilepsy is **material-specific**: a left-sided focus tends to impair verbal episodic memory, while a right-sided focus tends to impair non-verbal, visuospatial memory. The logic is the standard hemispheric one, verbal material handled by the language-dominant hemisphere and non-verbal by its partner, so the epileptogenic temporal lobe embarrasses the class of memory its side ordinarily serves. Two honest caveats belong with the rule. The verbal side of the dissociation is robust and reproducible; the non-verbal, right-sided side is less consistent and should be stated with more reserve. And the pattern is modulated by language dominance, so the confident left-equals-verbal mapping assumes the typical left-dominant brain and can invert in atypical dominance. Clinically the pattern is more than a curiosity: it helps lateralise the seizure focus in the neuropsychological work-up, a verbal-memory deficit pointing toward the left temporal lobe and a visuospatial one toward the right, complementing the imaging and electrophysiology.

> **PITFALL**
>
> A young person with epilepsy who complains of failing memory and poor concentration is easily written up as showing early evidence of a brain being progressively damaged by seizures. That reading skips the two commonest and most treatable contributors. A sedating or high antiepileptic drug load is dose-related and largely reversible and belongs to its own topic in this chapter; and a comorbid depression, which is frequent in epilepsy and taught separately here, can present as a pseudodementia of poor effort and slowed thinking. Rationalise the drugs and treat the mood before you conclude the epilepsy itself is eroding the brain.

30.7 Repeated seizures and status epilepticus

Does the seizure burden itself accumulate? The title asks specifically about repeated seizures, and this is where the disease-intrinsic account meets its hardest question. The theoretical case that recurrent seizure activity could injure the brain is real: sustained or repeated discharge is metabolically punishing and potentially excitotoxic, and the limbic structures that generate temporal lobe epilepsy are also thought to be the ones most vulnerable to it. The bridging theory here, the kindling model and the neurobiology linking epilepsy to behaviour, is developed as the mechanism step of this chapter and is named rather than reworked, and the full limbic anatomy on which it rests belongs to the Functional Neuroanatomy and Neurophysiology notes. Set against that plausibility is the evidence already assembled: if much of the impairment antecedes the seizures and is measurable near onset, a model in which every fit subtracts a little more cognition is hard to sustain for the ordinary patient. The examinable balance is cautious. Prolonged convulsive **status epilepticus** is a neurological emergency with a genuine capacity to leave lasting injury, on a different footing from a scatter of brief seizures. Transient postictal cognitive impairment, the temporary clouding after a fit, is a distinct peri-ictal phenomenon

owned by the postictal-confusion topic in this chapter, and delirium as a general state belongs to the Stroke, TBI, delirium and focal brain syndromes notes; neither is permanent decline.

TRAP **Reading any cognitive impairment in epilepsy as proof of a progressive, seizure-driven dementia.** The classic error runs straight from "impaired cognition" to "the seizures are damaging the brain", and it fails on the evidence: much of the deficit predates the first seizure, most of it is mild and stable rather than mounting, and a large slice of what does fluctuate is the reversible drug effect or a treatable mood disorder. Progression is the exception, confined to named syndromes, not the rule.

30.8 The syndromes where cognition truly deteriorates

The exceptions that keep the rule honest. A rule stated this firmly needs its exceptions named precisely, because in the examination they are where the discriminating marks sit. Two groups genuinely break the pattern of stable, non-progressive cognition. The first is the epileptic encephalopathies, in which the epileptic activity itself drives a regression: the syndrome of continuous spike-wave activity in slow-wave sleep, whose acquired language variant is **acquired epileptic aphasia**, or **Landau-Kleffner** syndrome, produces specific cognitive deficits including a loss of speech comprehension, and its electrographic hallmark is an almost **continuous spike-and-wave discharge during slow-wave sleep**. The second is the rare progressive myoclonic epilepsies, and here the deterioration is neurodegenerative rather than purely epileptic: **Unverricht-Lundborg disease** and **Lafora body disease** are both associated with a progressive ataxia and dementia, and they stand as the explicit exception to the general rule that epilepsy does not cause progressive decline. The unifying teaching is that when a patient with epilepsy is genuinely and progressively losing cognitive ground, the differential narrows toward this small, named set rather than a vague epileptic deterioration.

MNEMONIC

Two encephalopathies, two dystrophies of the brain: Landau, Unverricht, Lafora

When epilepsy truly dements, the answer is almost always one of these named syndromes rather than the seizures at large. Landau-Kleffner regresses through continuous spike-wave in sleep and takes speech comprehension with it; Unverricht-Lundborg and Lafora body disease march on with ataxia and dementia. Learn the three names as the standing exception to the rule that epilepsy does not progress.

SETTING	COGNITIVE PICTURE	COURSE	ATTRIBUTION
Uncomplicated epilepsy, temporal lobe subgroup included	intelligence normally distributed	static	not intrinsic to the epilepsy
New-onset childhood epilepsy	academic difficulty present before the first seizure	antecedent	shared neurodevelopmental origin
Recent-onset epilepsy, either syndrome type	broad but mild impairment near onset	early, plateau	disease-related, not late
Temporal lobe epilepsy	selective episodic memory deficits, left verbal and right non-verbal	relatively stable	disease-intrinsic substrate
Landau-Kleffner syndrome	loss of speech comprehension	can regress	the epileptic activity itself
Progressive myoclonic epilepsy	progressive ataxia and dementia	progressive	the true exception

Cognition in epilepsy sorted by setting. The single examinable thread is that the top four rows are mild, static or antecedent and are not a dementia, while only the bottom two genuinely deteriorate, and they do so as named syndromes.

30.9 Assessing cognition and what to do about it

Attribution is not an academic exercise; it dictates the next move. Because the reversible contributors are the drugs and the mood, the assessment of the cognitively complaining patient with epilepsy is built to find those first. The standard of care for the epilepsy itself is set by the ILAE and NICE epilepsy guidelines, while the neuropsychiatric layer draws on the epilepsy-specific psychiatric-management literature; none of the three sanctions a fatalistic shrug at "epileptic deterioration".

The **LOCUS** is overwhelmingly the outpatient clinic, where cognition is tracked, the drug regimen reviewed and the family counselled; inpatient and neuropsychology-unit assessment is reserved for the presurgical work-up, a suspected epileptic encephalopathy, or a genuinely progressive picture that raises the named degenerative syndromes. The **focus** shifts with the time frame: the acute task is to exclude and treat the reversible contributors, the sedating drug load and the comorbid depression, both handled in their own topics; the mid-term task is formal characterisation, including material-specific memory testing where lateralisation matters; the long-term task is protecting cognition through good seizure control and the least burdensome effective regimen, with educational and vocational support. The **modus** spans every mode. The pharmacological lever is rationalising rather than adding, trimming the offending agents whose cognitive cost belongs to the antiepileptic-drug topic. The procedural lever is seizure control and, in selected refractory temporal cases, the surgical pathway whose cognitive and psychiatric consequences are covered separately in this chapter. The psychological and social levers carry cognitive rehabilitation, memory strategies, family education that the deficit is usually stable rather than doom-laden, and accommodation at school and work.

GOOD TO REMEMBER

When a patient with epilepsy seems to be getting cognitively worse, walk the reversible causes before the fixed ones. Review the antiepileptic drugs for a sedating or excessive load, screen for and treat depression, and confirm seizures are actually controlled. Only when those are addressed and the decline is still advancing should you escalate toward the small differential of genuinely progressive epileptic syndromes and formal neuropsychological and imaging assessment.

IN INDIA

The folk model that epilepsy is a progressive madness rotting the intellect remains widespread and feeds a stigma touching schooling, employment and marriage. The clinical counter is exactly the evidence of this topic: uncomplicated epilepsy does not lower intelligence, and most cognitive impairment is mild and non-progressive. Two local realities sharpen the work. Access to formal neuropsychological testing is limited outside tertiary centres, so a careful history of function and a few bedside measures often stand in for a full battery. And the treatment gap means many arrive after a long duration of untreated seizures, so optimising control and reviewing an inherited drug regimen for cognitive cost are among the highest-yield things a clinician can do.

Normally distributed IQ IN UNCOMPLICATED EPILEPSY	**26% vs 4%** ACADEMIC PROBLEMS BEFORE FIRST SEIZURE	**Antecedent** IMPAIRMENT OFTEN PREDATES SEIZURES	**Rarely progressive** OUTSIDE THE NAMED ENCEPHALOPATHIES

Cognition in epilepsy is usually mildly impaired, often before the first seizure, and rarely progressive; the exam task is to attribute the deficit between the antecedent brain disease, the seizures and the reversible drug load, and to reserve genuine decline for the named exceptions - Landau-Kleffner and the progressive myoclonic epilepsies - rather than blaming the seizures for a dementia they do not cause.

31 · Cognitive and behavioural adverse effects of AEDs

Every antiepileptic drug is a bargain. It buys seizure control, and it pays for that control in some coin of cognition or behaviour. This topic owns the cognitive adverse effects of AEDs and the behavioural adverse effects of AEDs, and the attribution logic that separates them from the disease. The examinable skill in this topic is not memorising a toxicology list but learning to read the bargain: when a person with epilepsy loses mental sharpness or becomes irritable, you must separate what the drug is doing from what the disease and its seizures are doing. That act of attribution is the whole point. The cognitive effects of the epilepsy itself and of repeated seizures are taught as their own topic, and the disease-intrinsic aggression, irritability and episodic dyscontrol that arise from the condition rather than from its treatment belong to the behaviour-in-epilepsy topic. Here we own only what the drugs do.

Two adverse-effect streams run in parallel, and the trap is to assume they run together. One is cognitive: psychomotor slowing, inattention, memory difficulty and word-finding trouble. The other is behavioural and psychiatric: dysphoria, irritability, aggression and, less often, frank

psychosis. The agents that head the cognitive list are not the agents that head the behavioural list, and a clinician who carries only one of the two maps will keep swapping a patient off a cognitively heavy drug onto a behaviourally worse one and wonder why nothing improves. Our concern is the cognition-and-behaviour lens and the attribution logic behind it.

31.1 The attribution problem: disease, seizures, or drug

Three possible authors, one presentation. A person with epilepsy who reports poor concentration, mental slowing or a shorter fuse presents a single clinical picture with at least three possible authors, and telling them apart is the examinable core of this topic. The first author is the epilepsy itself, with its underlying lesion and the cumulative effect of repeated seizures, a contribution owned by its own topic and not reopened here. The second is the disease-linked change in temperament and impulse control, the interictal personality change and disease-intrinsic irritability that spring from the condition rather than its treatment, both taught elsewhere in this chapter. The third, and the only one this fragment teaches, is the drug. Because these authors produce overlapping phenotypes, you cannot reason safely from the symptom to its cause; the reasoning has to run the other way, from the history of the treatment to the symptom in front of you.

The tools for that reasoning are temporal and pharmacological rather than diagnostic: a genuine drug effect is time-locked to prescribing changes, dose related and reversible, whereas a disease-driven deficit tracks seizure burden and the underlying pathology. A short structured enquiry sorts most cases:

- did the change begin or worsen when a drug was started or its dose was raised;
- does it rise and fall with the dose;
- does it ease when the drug is lowered or stopped;
- and does its character fit the known profile of the agent in question.

No single answer is decisive, and baseline deficits from the epilepsy muddy every one of them, but taken together they let you assign a probable author and act on it. The discipline matters because the drug is the one author you can often simply remove.

RULE **Before you blame the epilepsy or the personality, account for the drug.** A cognitive or behavioural change in a treated patient is drug-attributable until the timeline says otherwise, because the drug's contribution is dose related, time-locked to prescribing changes and, crucially, reversible. The disease and its seizures are the harder authors to modify; the treatment is the one you can actually test by adjusting it.

31.2 Cognitive adverse effects and how the agents rank

Older is usually heavier, with sharp exceptions. As a broad generalisation the established antiepileptic drugs impose more of a cognitive load than the newer agents, mostly through sedation, psychomotor slowing and dulled attention rather than through any focal amnestic effect. Within that pattern there is a clear worst offender. **Phenobarbital** is the agent most consistently held to carry the worst cognitive effects, a reputation earned through its marked

sedative action and its blunting of arousal, attention and processing speed, and it is this profile that has driven its retreat from first-line use wherever alternatives can be afforded. Ranged behind it, and broadly similar to one another rather than cleanly separable, sit the other long-established agents: phenytoin, carbamazepine, valproate and clonazepam share a comparable middle-ground profile, so that swapping among them for cognitive reasons alone rarely buys much. At the favourable end, **two** of the newer agents stand out. **Lamotrigine** and **gabapentin** carry a comparatively clean cognitive profile against the older drugs, which is one reason they are reached for when preserving cognition is a priority. Newer does not mean cognitively free, though, as the next agent shows.

AGENT(S)	COGNITIVE PROFILE	PRACTICAL NOTE
Phenobarbital	the heaviest cognitive burden of the group	sedation, slowed attention and processing
Phenytoin, carbamazepine, valproate, clonazepam	broadly similar to one another	the older-agent middle ground; little to gain by switching among them for cognition alone
Lamotrigine, gabapentin	comparatively favourable	preferred where protecting cognition matters

The cognitive-burden ranking of the antiepileptic drugs owned here. Topiramate is deliberately left out because its cognitive signature is distinctive rather than a matter of degree, and it is taken up in its own right below.

IN INDIA

This ranking is not an academic ordering here, because the agent with the worst cognitive profile is also among the cheapest and most available. Phenobarbital remains in wide first-line use across resource-limited Indian practice, sustained by its low cost, once-daily dosing and secure supply, long after richer systems have pushed it down the list precisely for its effect on cognition. A dulled, underperforming patient on long-standing phenobarbital is thus a common and correctable sight, the saving on the tablet quietly paid for in schooling, work and daily function. Where an affordable alternative exists, the cognitive burden is a legitimate reason to move.

31.3 Topiramate: a cognitive signature of its own

Not sedation, but words that will not come. Topiramate deserves separate treatment because its cognitive adverse effect is qualitatively distinctive rather than simply severe. **Topiramate** can produce a marked and characteristic disturbance dominated by word-finding difficulty and a loss of verbal fluency, with impairment of attention and memory layered on top. Patients and their families describe it vividly: the sentence that stalls halfway, the ordinary noun that will not surface, the sense of having become slow and inarticulate. This pattern is distinctive enough that an experienced clinician can often suspect the drug from the complaint alone, and it separates topiramate from the diffuse sedative dulling of the barbiturate-type effect. The breadth of the agent's pharmacology, acting on several neuronal systems at once, is the usual explanation for why language and executive function are hit so selectively.

The redeeming feature is that the effect is strongly bound to how the drug is introduced. The word-finding and fluency disturbance is substantially minimised by very slow titration, so that

the same total dose reached gradually is much better tolerated than the same dose reached quickly. That turns a drawback into a prescribing instruction: start low, climb slowly, and a large part of the cognitive cost can be avoided. It also means a patient already impaired on topiramate is not necessarily condemned to lose the drug, since dose reduction and patience may recover a good deal of the lost ground.

PITFALL

The avoidable error is to titrate topiramate quickly, provoke the word-finding and fluency disturbance, and then abandon the drug as intolerable. Much of that impairment is a function of the speed of introduction rather than of the molecule as such, so a rapid climb both manufactures the problem and forfeits an otherwise useful agent. Where the drug is genuinely needed, the answer to emerging cognitive complaints is usually a slower schedule and a lower rate of increase, not immediate withdrawal, and the patient should be warned of the effect in advance so that it is reported early rather than silently endured.

31.4 Behavioural and psychiatric reactions: the agents and the spectrum

A different list from the cognitive one. The behavioural and psychiatric adverse effects of the antiepileptic drugs are governed by a different set of culprits from the cognitive effects, which is exactly why the two maps must be held apart. **Four** agents recur at the head of the behavioural list: levetiracetam, tiagabine, topiramate and vigabatrin are the ones most frequently implicated in adverse psychiatric and behavioural reactions. Two of them, **tiagabine** and **vigabatrin**, earn their place chiefly through behavioural rather than cognitive trouble, while topiramate manages to appear on both lists at once. The essential caveat, and a frequent examination point, is that this list is not a whitelist for the rest: no antiepileptic drug is truly free of psychiatric adverse effects, and the named four are simply where the risk concentrates. The systematic per-drug account of these psychiatric effects across individual agents is developed in the antiepileptic-pharmacology material; the point owned here is the behavioural dimension itself and its place beside the cognitive one.

The severity of these reactions runs along a spectrum, and knowing its shape prevents both complacency and alarm. The great majority of behavioural reactions sit at the milder end, taking the form of **dysphoria**, irritability and anxiety, unpleasant and corrosive to daily life but not dangerous and usually reversible on dose adjustment. The more severe end, including frank psychotic reactions, is genuinely less common, so that a florid drug-induced psychosis is the exception rather than the rule even with the agents most prone to cause trouble. An antiepileptic class effect on mood and suicidality, and the regulatory warning attached to it, is a separate topic in this chapter and must not be folded into the ordinary behavioural spectrum described here.

MNEMONIC

L-T-T-V: the four to watch

The agents most frequently blamed for psychiatric and behavioural reactions line up as **L**evetiracetam, **T**iagabine, **T**opiramate and **V**igabatrin. The letters are worth holding, but so is the caution that comes with them: because no antiepileptic is truly free of behavioural risk, remembering the four must not breed false reassurance about everything left off the list.

TRAP **Assuming the cognitive villains and the behavioural villains are the same drugs.** They are largely not. The agent with the worst cognitive profile is not the agent most associated with behavioural disturbance, and several of the drugs kindest to cognition still carry real behavioural risk. Candidates who collapse the two into a single ranking of good and bad antiepileptics get the individual attributions wrong; the disciplined answer keeps a cognitive map and a behavioural map, overlapping only where a drug genuinely sits on both.

31.5 Levetiracetam: irritability, aggression and mood lability

The behavioural agent every candidate must know. Of the behavioural offenders, levetiracetam is the one that turns up most often in vignettes and in clinics, and its profile is worth learning in detail. **Levetiracetam** is particularly associated with a rise in irritability and overt aggression together with mood lability, a cluster distinctive enough that its emergence in a newly treated patient should point straight at the drug. The disturbance can range from a shorter temper and increased argumentativeness to genuine hostility, and it tends to appear early in the course of treatment. The single most important conceptual point is one of attribution: this is a drug-attributable phenomenon, mechanistically and clinically distinct from the disease-intrinsic aggression, irritability and episodic dyscontrol that arise from the epilepsy itself and are taught as their own topic in this chapter. Conflating the two is a live error, because the two demand opposite responses. Disease-driven aggression is managed by treating the epilepsy and its comorbidities; drug-driven aggression is managed by addressing the drug.

That distinction has direct therapeutic teeth. When irritability and aggression emerge in step with starting or up-titrating levetiracetam, the rational move is to reconsider the drug rather than to escalate it or reach reflexively for an antipsychotic to sedate the behaviour, and the disturbance frequently settles once the offending agent is reduced or replaced. The trap is to read a levetiracetam-induced temper as the patient's underlying nature or an expression of their epilepsy, and so leave the true cause untouched.

GOOD TO REMEMBER

When a patient with epilepsy becomes newly irritable, aggressive or emotionally volatile, the first question is not what is wrong with this person but what changed in the drug chart, and when. Map the behaviour against every recent start and dose increase before ascribing it to temperament or to the epilepsy. If the change tracks a new or up-titrated agent, especially one of the recognised behavioural offenders, the most useful intervention is usually to the prescription itself, not a tranquilliser laid on top of it.

31.6 What amplifies the harm: drug load, titration and folate

The dose and the number of drugs matter as much as the choice. The identity of the agent is only part of the story; the way it is prescribed does much to decide whether its cognitive and behavioural potential is ever realised. Three modifiable amplifiers stand out. The first is total drug load: the more antiepileptic drugs a patient carries and the higher the cumulative dose, the greater the cognitive and behavioural cost, so that a polytherapy regimen assembled over years of incremental additions becomes a burden in its own right, independent of any single agent's profile. The second is the speed of titration, already met with topiramate but general in principle, since a fast climb to a target dose provokes adverse effects the same dose reached gradually may not. The third is a metabolic route worth knowing: antiepileptic drugs can deplete **folate**, and because folate status bears on mood and cognition, that depletion is one more thread tying long-term antiepileptic treatment to low mood and mental sluggishness.

Two boundaries keep this honest. The wider pharmacokinetics of how enzyme-inducing antiepileptics interact with other drugs, folate among the substrates affected, is the province of the antiepileptic-drug interaction topic in this chapter and is not reworked here; the point retained is only that folate depletion is a recognised and relevant consequence, offered as a mechanism to be aware of rather than as a licence to assert a supplementation regimen the owned rows do not carry. What the reader should carry away is that load, speed and folate are levers, and that each is adjustable in a way the underlying epilepsy is not.

31.7 Recognising and reversing it: the management logic

The most reversible harm in the chart. Managing the cognitive and behavioural adverse effects of antiepileptics is unusually rewarding precisely because, unlike most of the neuropsychiatry of epilepsy, the cause is one you put there and can take away. The relevant standards are the ILAE and NICE epilepsy guidelines for the seizure disorder itself, read alongside the epilepsy-specific psychiatric-management literature for the behavioural dimension; none of them endorses tolerating an avoidable drug effect as the price of seizure control.

The **LOCUS** of care is overwhelmingly the outpatient clinic, where recognition, dose review and rationalisation of therapy proceed over successive visits; only a severe behavioural reaction, such as a frank drug-induced psychosis with a safety concern, pushes care briefly toward an inpatient or emergency setting. The focus shifts by phase. The acute task is recognition and attribution, placing the symptom on the drug timeline; the short-to-mid-term task is to act on the offending agent by lowering the dose, slowing an over-rapid titration, or switching from a heavy agent to a cognitively favourable one; the long-term task is to keep the regimen as lean as seizure control allows and to monitor cognition and mood over time; the adverse-event burden of the regimen can be tracked formally with the **Liverpool Adverse Events Profile**, a **19-item** self-report scored **19 to 76** in which a higher total means a heavier burden and which carries no single validated clinical cut-off, so it is read as a continuous measure of change rather than as a threshold. The modus spans every available mode. The pharmacological levers are dose reduction, substitution toward a kinder agent, and attention to folate status where relevant. The single most powerful lever is deprescribing: the **withdrawal of unnecessary antiepileptic**

drugs commonly improves cognition and behaviour, and rationalising an overloaded regimen is among the most gratifying interventions in all of neurorehabilitation. The psychological and social modes wrap around this: forewarning patients of a known effect so that it is reported early, psychoeducation for families who may be mislabelling a drug effect as character, and attention to the driving, work and schooling consequences that cognitive dulling quietly imposes.

Cognition	Behaviour	Favourable	Reversible
WORST ON PHENOBARBITAL	LEVETIRACETAM NOTORIOUS	LAMOTRIGINE, GABAPENTIN	WITHDRAW UNNEEDED DRUGS

The cognitive and behavioural adverse effects of the antiepileptic drugs form two separate maps that do not overlap, with phenobarbital heading the cognitive one and levetiracetam the behavioural, and because the cause is a drug you can adjust this is often the most reversible part of the whole clinical picture: before attributing decline to the epilepsy or the personality, account for the treatment.

32 · Psychiatric outcomes and de novo psychiatric disorders after epilepsy surgery

Epilepsy surgery is offered to stop seizures, but whether the patient counts the operation a success is decided as much by what happens to the mind afterwards as by the seizure diary. This topic owns the psychiatric and cognitive outcome of resective epilepsy surgery, and in particular the **de novo psychiatric disorders** that appear for the first time after operation in a person who did not carry them before. The marks reward a balanced ledger rather than a slogan. On one side, removing an epileptogenic focus, most often by **temporal lobectomy**, tends to reduce psychiatric morbidity across the operated population, most convincingly in those whose seizures are abolished. On the other, a recognisable set of new problems - a short-lived depression, an occasional psychosis, a change in sexual behaviour, and losses in language and memory - can arise after operation, and some of them arise whether or not the seizures were controlled. Holding both halves at once is the examinable skill.

Two framing errors are worth naming at the outset: that a good seizure result guarantees a good psychiatric result, and that any postoperative disorder proves the operation went wrong. Neither survives the data assembled below, drawn from Lishman's Organic Psychiatry and from the neuropsychiatric-epilepsy account in Kaplan and Sadock's Comprehensive Textbook of Psychiatry. Psychiatric and seizure outcome are related but not identical, and the months after operation demand their own psychiatric vigilance.

32.1 The two-sided ledger of surgical outcome

Net benefit, greatest with seizure freedom. The honest headline is favourable. Taken as a whole, temporal lobectomy lowers the psychiatric burden a refractory epilepsy population carries, and the benefit is concentrated in those rendered seizure-free. This makes sense, because much of the depression, anxiety and social disability of chronic epilepsy is downstream of the seizures and of a life organised around them, so abolishing the seizures lifts the load. A candidate must therefore not describe surgery as psychiatrically hazardous on balance; it is, on balance, psychiatrically

helpful, and the strongest predictor of psychiatric improvement is the same variable that defines surgical success, freedom from seizures. The gain is never automatic and never uniform. Superimposed on the population-level improvement is a scatter of new disorders the operated patient did not have before, and these de novo phenomena are the substance of this topic. They do not cancel the net benefit but they qualify it, and they are where the discriminating marks sit. The posture that follows is neither triumphalism nor gloom but structured surveillance: expect most patients to do better, and watch for the minority in whom a new disorder declares itself, often in a predictable window and sometimes despite an excellent seizure result.

RULE **Temporal lobectomy reduces psychiatric morbidity overall and most when seizures are abolished, yet distinct de novo disorders can appear after operation independently of the seizure result.** The net direction is benefit, driven by seizure freedom; but a new depression in particular occurs whether or not seizures recur, so the operation's psychiatric and seizure outcomes must be judged on separate axes.

32.2 De novo postoperative depression, the commonest new disorder

Early, self-limiting, and easy to miss. The single most important de novo syndrome is a **de novo postoperative depression**. About **one-third** of temporal-lobectomy patients develop a depressive disturbance in the early weeks after operation, and its texture is characteristic: low mood coloured by anxiety and by **emotional lability**, the affect brittle and tearful rather than uniformly flat. The timing is itself a teaching point. This is not the slow accretion of a chronic mood disorder but an early, phasic disturbance surfacing while the patient is still adjusting to the operation. It is usually self-limiting, tending to resolve over the following months, with most cases settling by around six months. There is also a lateralising hint examiners like: the disturbance is commoner after right-sided lobectomy, which sits against the naive assumption that dominant-hemisphere surgery must be riskier for mood. Because the depression is early, transient and arrives amid an expected convalescence, it is disarmingly easy to fold into a narrative of understandable adjustment and to leave unmonitored, which is exactly the wrong move given what the next section adds about risk.

MNEMONIC

One in three, in the first few weeks, whether the seizures come back or not

The de novo postoperative depression in one line. Roughly a third of temporal-lobectomy patients, arising early in convalescence, with anxiety and emotional lability, usually self-limiting, a little commoner on the right, and, the sting in the tail, occurring independently of whether the seizures were controlled. Learn it as a disorder to be looked for on a schedule, not stumbled upon.

32.3 Why postoperative depression happens, and who is at risk

A biological event, not merely a disappointment. The mechanism dictates whom to watch. Postoperative depression behaves as a biological consequence of temporal-lobe surgery rather than a psychological reaction to a poor result, and the decisive observation is that it occurs equally in patients with and without seizure recurrence. If it were simply demoralisation at a failed operation, it would cluster in those whose seizures returned; it does not. That dissociation

is the empirical heart of the topic. Risk is not evenly spread: the patient at greatest risk is the one with a prior history of depression, so a careful psychiatric history before operation identifies much of the vulnerable group in advance. Crucially, though, the disorder can arise de novo in a patient with no previous psychiatric history at all, which is what makes universal postoperative monitoring, rather than monitoring only the forewarned, the safe policy. The stakes are raised by mood's dangerous companion: an early, under-recognised depression carries a suicide risk, and early recognition is therefore important. The elevated suicide risk that attends epilepsy more broadly is developed in its own topic and not reworked here, but its shadow falls squarely on this postoperative window. A depression that is expected, time-limited and biologically driven is not a reason to withhold treatment; it is a reason to detect it promptly.

TRAP **Assuming postoperative depression only follows a failed operation, or that seizure freedom protects mood.** Candidates instinctively tie psychiatric outcome to seizure outcome and expect the depressed patients to be those whose fits returned. The examinable fact is the opposite: de novo postoperative depression occurs independently of seizure recurrence, so a seizure-free patient is not thereby a mood-safe patient and must still be screened.

PITFALL

The commonest bedside error is to hear early low mood in a recovering surgical patient, judge it a reasonable adjustment to a big operation, and file it as expected without arranging follow-up. Because the disorder is biological and carries a suicide risk, that charitable reading can be lethal. Treat early postoperative low mood as a disorder to be assessed and tracked, and ask specifically about hopelessness and self-harm rather than accepting a smiling reassurance at a busy clinic.

32.4 De novo and persistent psychosis after temporal lobectomy

Uncommon, but it can surface late. Psychosis is the more feared, though less frequent, de novo outcome. In temporal-lobectomy series a psychosis occurs in about **7 to 8 per cent** of patients, and the feature that earns marks is its timing: it can appear or persist even long after the seizures have been arrested. This defeats the tidy expectation that removing the focus should retire the psychiatric risk with the seizures. A **de novo psychosis** emerging months or years into a seizure-free course is a real entity and a reason that psychiatric follow-up after epilepsy surgery cannot be a brief affair closed once the seizures settle. Three boundaries must be kept clear so the reader does not conflate distinct constructs:

- it is not the schizophrenia-like psychosis of epilepsy, the chronic interictal psychosis that has its own topic;
- it is not forced normalisation or alternative psychosis, where disturbance emerges as seizures and the electroencephalogram paradoxically improve, again its own topic;
- and at the bedside it must be separated from a primary psychotic illness, whose phenomenology and course belong to the Schizophrenia: aetiology, phenomenology, classification and course notes.

What this topic owns is the observation that resecting a temporal focus does not immunise against psychosis and may, in a small minority, be followed by one - a reason for sustained rather than perfunctory surveillance.

32.5 Sexual outcome after temporal lobectomy

An outcome that usually improves, if the seizures are controlled. Sexuality is the outcome most often mishandled in the exam because it runs against expectation. The baseline is that most patients with temporal lobe epilepsy are **hyposexual** before operation, that is, they have reduced sexual interest and drive as part of the interictal picture; the altered sexuality of the interictal personality, and its place in the Gastaut-Geschwind cluster, is taught in its own topic and is only the pre-surgical starting point here. What surgery does is, on balance, favourable. After temporal lobectomy, **nearly one-third** of patients show an increase in libido, an unshackling of a previously suppressed drive, and this gain is contingent: it is seen provided the seizures are controlled. That conditionality is the whole teaching. The libido increase is not a random side effect but part of the general improvement that follows seizure freedom, so it belongs with the net-benefit story rather than with the de novo hazards. The practical implication is to counsel patient and partner that sexual interest may rise after successful surgery, a change usually welcome but occasionally destabilising for a relationship organised around a hyposexual partner.

32.6 Cognitive outcome: memory and word-finding

The preoperative brain writes the postoperative report. Cognition is where the surgical trade-off is most explicit, because the tissue removed to stop the seizures is the tissue that serves memory and, on the dominant side, language. The governing principle is that memory outcome is determined primarily by the preoperative state. A significant new memory deficit follows operation in about **5 per cent** of patients in controlled study, and the counter-intuitive refinement examiners prize is who those patients are: the deficit is more likely in those with high preoperative memory function. The logic is that a well-functioning temporal lobe still doing real memory work has more to lose when resected, whereas a diseased, already failing lobe was contributing little, so its removal costs less and can even release the healthier contralateral side. Consistent with that, improvement is possible in those with low preoperative function. The material-specific structure of memory in temporal lobe epilepsy, the verbal-versus-nonverbal dissociation, is a property of the disease taught in its own topic and not reopened here; what this topic owns is the surgical prediction rule that the preoperative baseline, not the operation alone, forecasts the memory result.

Language carries its own postoperative signature. Minor **word-finding difficulty** is relatively common after temporal lobectomy, concentrated after dominant-hemisphere operations, which is unsurprising given that the resection encroaches on language-serving cortex. This is usually a mild anomia rather than a gross aphasia, but frequent enough that it should be named in the consent conversation for a dominant-side procedure rather than sprung on the patient afterward. Serious neurological complications, by contrast, are rare: a major event such as a stroke or a large visual-field defect occurs in under 2 per cent of operations, so the honest framing is of a common

minor cognitive cost, a memory trade-off keyed to the patient's own baseline, and a small but real chance of a major complication.

DE NOVO OR ALTERED OUTCOME	FREQUENCY OR DIRECTION	TIMING AND DEPENDENCE ON SEIZURE RESULT	KEY PREDICTOR
Postoperative depression	about one-third	early weeks, self-limiting; independent of seizure recurrence	prior depression; can be de novo
De novo or persistent psychosis	about 7 to 8 per cent	may appear long after seizures arrested	distinct from schizophrenia-like psychosis
Sexual outcome	nearly one-third increase libido	postoperative, only if seizures controlled	preoperative hyposexuality lifts
New memory deficit	about 5 per cent	determined by preoperative state	high preoperative function at risk
Word-finding difficulty	relatively common	after operation, persistent but minor	dominant-hemisphere resection
Major neurological complication	under 2 per cent	peri-operative	stroke or large field defect

Outcomes of temporal lobectomy sorted by type. The examinable thread is the middle column: depression and psychosis are independent of the seizure result, whereas the libido gain is contingent on seizure control, and the cognitive costs are keyed to the preoperative brain.

32.7 The burden of normality and psychosocial adjustment

Getting better can be its own crisis. Beyond the discrete disorders lies a subtler outcome that connects them. A patient whose seizures vanish must rebuild an identity and a set of relationships that had been organised for years around being a person with epilepsy, and this readjustment can be genuinely destabilising even when, by every clinical measure, the operation succeeded. The phenomenon is sometimes called the **burden of normality**: the paradoxical difficulty of adapting to health, in which a cured patient struggles with new expectations to work, to drive, to take on adult responsibilities and to occupy a well role that family dynamics may resist ceding. It is not a distinct diagnosis and carries no number to memorise, but it is the connective tissue that makes the de novo depression, and the sexual and vocational changes, intelligible as parts of one adjustment rather than unrelated complications. Recognising it shifts the clinician's stance from surprise to anticipation: the postoperative task is not only to abolish seizures but to accompany the patient through the reorganisation of a life, and to normalise the fact that improvement itself can feel like loss.

32.8 Presurgical assessment and postoperative management

The whole topic converges on a single service design: screen before, and watch after. Because the most dangerous de novo outcome is an early depression that is biological, common and independent of the seizure result, the management of psychiatric outcome is built around prediction and surveillance rather than reaction. Three guideline sources anchor the approach without prescribing a numerical protocol: the epilepsy standards of the International League

Against Epilepsy and of the National Institute for Health and Care Excellence set the frame for comprehensive presurgical evaluation and follow-up, and the epilepsy-specific psychiatric-management literature supplies the neuropsychiatric layer neither epilepsy guideline fully covers. Within that frame the LOCUS -> FOCUS -> MODUS structure organises the work.

The **LOCUS** of care is overwhelmingly the specialist epilepsy-surgery centre and its outpatient follow-up clinic, with two flanking settings: before operation, the multidisciplinary presurgical assessment unit, where a formal psychiatric history identifies the prior-depression group at greatest risk and documents any active disorder; and, for a severe postoperative depression with suicidal intent or a frank psychosis, escalation to inpatient or emergency care. The **focus** tracks the phase. The presurgical task is prediction and counselling, mapping vulnerability and preparing patient and family for the burden of normality and the realistic cognitive trade-offs. The acute and short-term task, across the vulnerable early weeks, is active screening for and treatment of the de novo postoperative depression and vigilance for suicidal thinking. The mid-term task is characterising any new cognitive deficit, particularly memory and word-finding, and supporting adjustment. The long-term task is a follow-up long enough to catch a psychosis that may surface well after the seizures have gone. The **modus** spans every mode: the pharmacological mode treats an emergent depression or psychosis on its merits, with the attention to seizure threshold developed in the antidepressant and antipsychotic topics of this chapter; the psychological mode carries pre-operative preparation, monitoring and support through the re-adjustment; and the procedural and social modes are the surgery itself, consented with its psychiatric and cognitive outcomes made explicit, plus the vocational, driving and family support that make the seizure result liveable.

GOOD TO REMEMBER

Two habits change outcomes. First, take a proper psychiatric history before operation, because a documented past depression flags the highest-risk patient and lets you plan follow-up rather than react to a crisis. Second, schedule active mood screening in the first postoperative weeks for every patient, not only the forewarned, and specifically ask about suicidal thinking, because the de novo depression is common, biological and blind to how well the seizures were controlled.

IN INDIA

Comprehensive epilepsy surgery is concentrated in a handful of tertiary centres, and full presurgical evaluation, with prolonged video-electroencephalography, imaging and neuropsychology, reaches only a small fraction of patients with drug-resistant epilepsy. The consequence is twofold. Most eligible patients never reach operation, so the treatment gap sits upstream of the outcomes discussed here; and among those operated, structured psychiatric follow-up can be patchy once the patient returns to a distant district. A realistic plan makes the presurgical psychiatric assessment non-negotiable while the patient is still at the centre, and hands the family and the referring clinician a clear written schedule for postoperative mood monitoring, since the early depressive window will otherwise pass unobserved far from the surgical team.

~one in three	7 to 8%	~one in three	~5%
DE NOVO POSTOPERATIVE DEPRESSION	PSYCHOSIS AFTER TEMPORAL LOBECTOMY	LIBIDO RISES, IF SEIZURES CONTROLLED	SIGNIFICANT NEW MEMORY DEFICIT

Temporal lobectomy reduces psychiatric morbidity overall and most when seizures are abolished, yet a de novo postoperative depression in about a third of patients arises early and independently of the seizure result, an occasional psychosis can surface long after the fits have stopped, libido tends to rise only when seizures are controlled, and cognitive outcome is written mainly by the preoperative brain - so screen before, and watch after.

Psychogenic non-epileptic seizures

33 · PNES: definition, terminology and classification within functional neurological disorder

A name is a clinical act. Few conditions in neuropsychiatry have been renamed as often, or as anxiously, as the paroxysmal episodes that look like epileptic seizures yet arise without the electrical signature of epilepsy. The words a clinician writes in the notes shape how the patient understands the illness, whether the referral is taken seriously, and whether treatment ever engages the psychological mechanism at the root of the problem. For the examination this is the deceptively simple part of the syndrome, the ground on which everything else is built: what these events are, what to call them, and where they sit in the two diagnostic manuals. It rewards precision because it is precisely where loose thinking shows. A candidate who cannot define the entity cleanly, who reaches for a discredited label, or who cannot say whether the disorder is coded as conversion or as dissociation, has signalled that the rest of the answer will be shaky too.

The scope here is deliberately narrow. This topic covers the definition, the terminology and the classification of the syndrome within the functional neurological disorders. The bedside features that separate these attacks from epileptic seizures, the confirmatory investigations, the psychiatric comorbidity and predisposing factors, the delicate task of delivering the diagnosis with its stepped management, and the difficult patient who has both dissociative and epileptic attacks at once are each developed in their own right elsewhere in this chapter. The broad category of functional neurological and conversion disorder, of which this is one presentation, is taught in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes; here the concern is only where the seizure form of that category belongs and what to call it.

33.1 What the syndrome is: the working definition

A seizure-like event with no epileptiform correlate, presumed to be psychological.

Psychogenic non-epileptic seizures are sudden, involuntary episodes of altered movement, sensation or awareness that outwardly resemble epileptic seizures but are not produced by the abnormal, hypersynchronous cortical discharge that defines epilepsy, and that are presumed to arise from a psychological process. The definition has three moving parts, and each carries clinical weight. The first is the resemblance: the events mimic seizures closely enough to be

mistaken for them, which is exactly why they generate referrals to epilepsy and psychiatry services and why the bedside distinction becomes such a demanding skill. The second is the critical negative: there is no ictal epileptiform activity accompanying the attack, so the event is not an electrographic seizure however convulsive it looks. The third is the positive attribution: the mechanism is presumed psychological, understood in contemporary models as a dissociative one, an involuntary disruption of the normal integration of movement, sensation and awareness rather than a performance the patient chooses to stage. The word presumed is doing honest work here, because the mechanism is inferred rather than directly observed, and the word involuntary is the pivot of the whole topic. What follows from these three parts is that the diagnosis is a positive identification of a functional disorder, not merely the empty space that remains once epilepsy has been excluded.

RULE **This is a positive diagnosis, not a diagnosis of exclusion.** The syndrome is a definable functional disorder in its own right, with its own presumed mechanism and its own place in the classifications, not simply the residue of a negative epilepsy workup. Framing it as an absence, as merely not epilepsy, is the conceptual error that then generates a cascade of downstream problems, from a demoralised patient to a stalled referral.

33.2 A contested vocabulary: from hysteria to neutral terms

The old labels were abandoned because they insulted the patient. No entity in this chapter has attracted more terminological baggage, and the reason is historical. The syndrome descends conceptually from the nineteenth-century notion of hysteria, and its oldest clinical labels carry that inheritance. The terms **pseudoseizures** and hysterical seizures are now regarded as pejorative and have been largely abandoned, and the objection to them is not merely one of fashion. The prefix pseudo implies that the events are false, feigned or somehow not real, which both misrepresents the involuntary nature of the disorder and communicates contempt to a patient who is genuinely suffering. Hysterical carries an even older weight of dismissiveness and a gendered connotation that alienates patients and colours the clinical encounter before it begins. Against this backdrop the field has converged on neutral alternatives. The syndrome is now treated as synonymous with the terms **dissociative seizures** and functional seizures, and the two roots emphasise different things. Dissociative foregrounds the presumed mechanism, aligning the label with the dissociative model of an involuntary interruption of integrated function. The other root emphasises disturbed function of a structurally intact nervous system, sidestepping any statement about mechanism at all. Both were chosen precisely because they describe the disorder without demeaning the person who has it, and both mark a deliberate move away from a vocabulary that pathologised the patient rather than the pathology.

33.3 Why the negative label is unsatisfactory

Defining a thing by what it is not tells the patient nothing. Even the ostensibly neutral term at the head of this topic has a weakness that is worth naming, because it is a recurring source of trouble. The labels built on non-epileptic define the condition entirely by what it is not, and a patient who is told only that the attacks are non-epileptic may reasonably conclude that the doctors have ruled out epilepsy but still do not know what is actually wrong. That reading

corrodes the therapeutic relationship at the very moment it needs to form. For this reason the term **functional seizures** is increasingly preferred as a neutral description that patients find acceptable and that does not prejudge whether the process is conscious or unconscious. The problems with a purely negative framing can be set out plainly:

- it defines the disorder by negation, so it names an absence rather than a presence;
- it reads to the patient as a non-diagnosis, as if the assessment has ended in a shrug;
- it says nothing about the mechanism, and therefore offers no foothold for explanation or for treatment;
- it can be heard as an accusation that the attack was not a real seizure, reviving the very insult that the neutral vocabulary was meant to retire.

The practical resolution, which the wider neuropsychiatric literature has increasingly endorsed, is to lead with a positive functional formulation rather than an exclusionary one: to say what the disorder is and how it works, in a vocabulary that neither blames nor mystifies the patient.

33.4 Events and spells: softening the descriptor further

Sometimes the safest early move is to drop the word seizure altogether. Some clinicians go a step beyond the neutral synonyms and avoid the word seizure entirely in the earliest phase of assessment. Describing the phenomena as **events or spells** is a way of reducing the confusion that arises when a patient hears the word seizure and immediately assumes epilepsy, with all the associations of brain disease, driving restrictions and lifelong medication that the word carries. This descriptive language is most useful while the diagnosis is still provisional, before video-electroencephalography or the settled clinical picture has fixed the label, because it lets the clinician talk about the attacks factually without committing prematurely to a mechanism or importing the wrong frame. It is a small linguistic device, but it reflects the same principle that runs through the whole of this topic: the words come first, and they either open a door to understanding or quietly close one.

> **GOOD TO REMEMBER**
>
> In letters, discharge summaries and the case notes, prefer functional seizures or dissociative seizures as the working term, and reserve the non-epileptic phrasing for the specific context of contrasting these attacks with epilepsy. While the diagnosis is still being settled, events or spells is a safe, honest descriptor. Retire pseudoseizures and hysterical seizures from your vocabulary entirely, including from the notes, because patients read what is written about them and a careless label can undo months of careful work.

33.5 Locating the syndrome in the classifications

Conversion in one manual, dissociation in the other. The two major diagnostic systems agree that this is a functional neurological disorder but reach it by different routes, and knowing both, along with the codes, is a reliable examination point. In DSM-5 the syndrome is classified as a subtype of **functional neurological symptom disorder**, the diagnosis also known as conversion disorder, which sits among the somatic symptom and related disorders. DSM-5-TR keeps that

placement and marks the seizure presentation with a symptom-type specifier, coded **F44.5** and designated **with attacks or seizures**. ICD-11 takes the alternative conceptual route, classifying the disorder among the dissociative rather than the somatic-symptom disorders: it appears as a **dissociative neurological symptom disorder**, with non-epileptic seizures, under the code **6B60.4**. The divergence is not trivial pedantry. It reflects a genuine and unresolved conceptual tension over whether the syndrome is best understood as a conversion phenomenon or a dissociative one, a tension that the two neutral synonyms in common use quietly mirror. Services and examinations still working with ICD-10 will find the entity among the dissociative and conversion disorders of that older system. The general construct of conversion and functional neurological disorder, its mechanism and its wider family, belongs to the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes; the point to hold here is simply where the seizure subtype is filed in each manual and under what code.

F44.5	6B60.4	late teens to early 20s
DSM-5-TR CONVERSION DISORDER, WITH ATTACKS OR SEIZURES	ICD-11 DISSOCIATIVE NEUROLOGICAL SYMPTOM DISORDER, WITH NON-EPILEPTIC SEIZURES	TYPICAL AGE OF ONSET

33.6 Not conscious simulation: the boundary with feigning

The single boundary most often blurred is the one that matters most. The defining feature of the syndrome, and the source of its commonest misunderstanding, is that the attacks are not deliberately produced. This is what separates it sharply from the conditions of conscious feigning, and the separation runs along an axis of awareness and motivation. In **factitious disorder** the illness is wilfully and consciously simulated, the patient deliberately producing or feigning symptoms without any external reward, the motivation lying in the psychological need to occupy the role of the sick person. In **malingering** the feigning is equally deliberate but is driven by a tangible external gain, such as financial compensation, avoidance of work or duty, or the evasion of a legal consequence. The dissociative attack sits apart from both, because the events are not consciously generated at all: the patient is not choosing to produce them, and there is no external incentive to be uncovered. It is useful to think of a spectrum of conscious awareness, with the deliberate deceptions at one end and the involuntary dissociative attack at the other, and with self-deception occupying the uneasy middle ground where a patient is neither fully feigning nor fully aware. The accompanying table sets the three conditions side by side on the axes that separate them.

AXIS	DISSOCIATIVE (FUNCTIONAL) SEIZURES	FACTITIOUS DISORDER	MALINGERING
Production of the episode	Involuntary, not deliberately generated	Consciously and wilfully simulated	Consciously and deliberately feigned
Conscious intent to deceive	Absent	Present	Present
Motivation	Psychological, outside awareness	To occupy the patient role, no external reward	An external, tangible gain

The conscious-awareness axis distinguishing dissociative seizures from the two disorders of feigning; the essential point is that the dissociative attack alone is not consciously produced.

TRAP **Equating a dissociative attack with malingering or putting it on.** Candidates, and colleagues on a busy ward, slide from not epileptic to not real to putting it on, and so treat the patient as a feigner. That is a category error: the attacks are involuntary, and conflating the disorder with factitious illness or malingering both fails the examination and, in the clinic, insults and abandons a genuinely unwell person.

33.7 Who develops it, and where this topic hands over

A young-adult onset, though the disorder respects no age. The syndrome most characteristically begins in the **late teens or early twenties**, the years in which the developmental, interpersonal and psychological stresses that predispose to it tend to converge. That said, onset is not confined to this window, and the attacks can begin across the lifespan, in older adults and in children as well as in the young-adult peak. This demographic sketch is where the present topic deliberately stops. The psychiatric comorbidities that so often accompany the disorder and the predisposing and precipitating factors that make a particular person vulnerable are the substance of a separate topic, as are the mechanics of confirming the diagnosis, the way it should be communicated and managed, and the prognosis, including the outlook for patients who carry both dissociative and epileptic seizures together. Naming those boundaries matters, because a great deal of examinable and clinically important material about this syndrome lives just outside the definition, and the disciplined answer teaches the definition and terminology fully while pointing, rather than straying, to the rest.

33.8 Why the terminology carries the whole case

Get the name right and the treatment has somewhere to stand. It can look, from a distance, as though the argument over what to call this disorder is a semantic quarrel of interest only to nosologists. It is not. The name is the interface between an invisible mechanism and a frightened patient, and it determines whether the explanation that follows will be believed. A label that implies fakery invites denial and disengagement; a label built on an absence invites the conclusion that nobody knows what is wrong; a neutral, mechanistic term that the patient can accept opens the door to a positive formulation and to the psychological work that actually helps. The whole of this topic, the clean definition, the retirement of the pejorative vocabulary, the preference for a functional over a negative term, the correct placement in the classifications, and the firm line against the language of feigning, converges on a single practical goal: to hand the patient an account of the disorder that is both true and bearable. The detail of how that account is delivered and how treatment is then structured belongs to a separate topic; the foundation it rests on is laid here, in the words.

IN INDIA

These attacks are among the commonest functional presentations seen in Indian general and neurology clinics, where they are widely known by lay terms such as fits and are frequently understood by families through idioms of possession, spirit affliction or the older language of hysteria. Many patients reach a psychiatrist only after a long circuit through faith healers, general practitioners and neurologists, often already committed to antiepileptic drugs and to an epilepsy identity. The stigma attached both to epilepsy and to any psychiatric attribution is heavy, and it bears directly on marriage prospects, employment and standing in the family, which makes the choice of a neutral, non-pejorative, family-acceptable term not a courtesy but a clinical necessity. Language that blames or belittles the patient, in a setting where a psychiatric label is already feared, will simply end the consultation.

PITFALL

Recording the diagnosis as non-epileptic or, worse, as pseudoseizures, and stopping there. An exclusionary label committed to the notes travels with the patient through every future referral, propagating an absence in place of a diagnosis and quietly reasserting the old insult. The remedy is to commit a positive functional formulation to the record, in neutral terms, so that the next clinician inherits a diagnosis rather than a negation.

The syndrome is an involuntary, seizure-like event with no epileptiform electroencephalographic correlate and a presumed psychological mechanism; call it functional or dissociative seizures rather than the discredited pseudoseizures, classify it as a conversion or dissociative neurological symptom disorder, and never mistake it for conscious feigning.

34 · Clinical features differentiating PNES from epileptic seizures

Getting this distinction wrong commits a patient to the wrong illness for years. A large proportion of people with psychogenic non-epileptic seizures carry an epilepsy label and swallow antiepileptic drugs for a long time before anyone questions it, and the reverse error, dismissing a genuine seizure as merely functional, is every bit as damaging. The reference standard that settles the matter is prolonged video-electroencephalographic monitoring, which captures an attack together with its brain electrical correlate, but that resource sits in tertiary units and is rarely at the bedside where the decision first has to be made. So the examinable skill, and the daily clinical one, is reading the attack itself: knowing which features of onset, movement, eyes, awareness and recovery push the odds toward a dissociative attack and which toward an epileptic one, and holding firmly to the discipline that not one of them decides the case on its own.

What follows is only the clinical differentiation. What a psychogenic non-epileptic seizure actually is, the terminology it travels under and its place among the functional neurological disorders belong to the companion topic and, more broadly, to the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes. The investigations that confirm it, video-EEG capture and serum prolactin among them, are treated in their own right, as are the psychiatric

comorbidities and predisposing factors that make a person vulnerable, the delicate business of communicating the diagnosis and its stepped management, and the genuinely difficult patient who has both dissociative and epileptic seizures at once. Here we stay with semiology: the phenotype of the event as a witness sees it unfold.

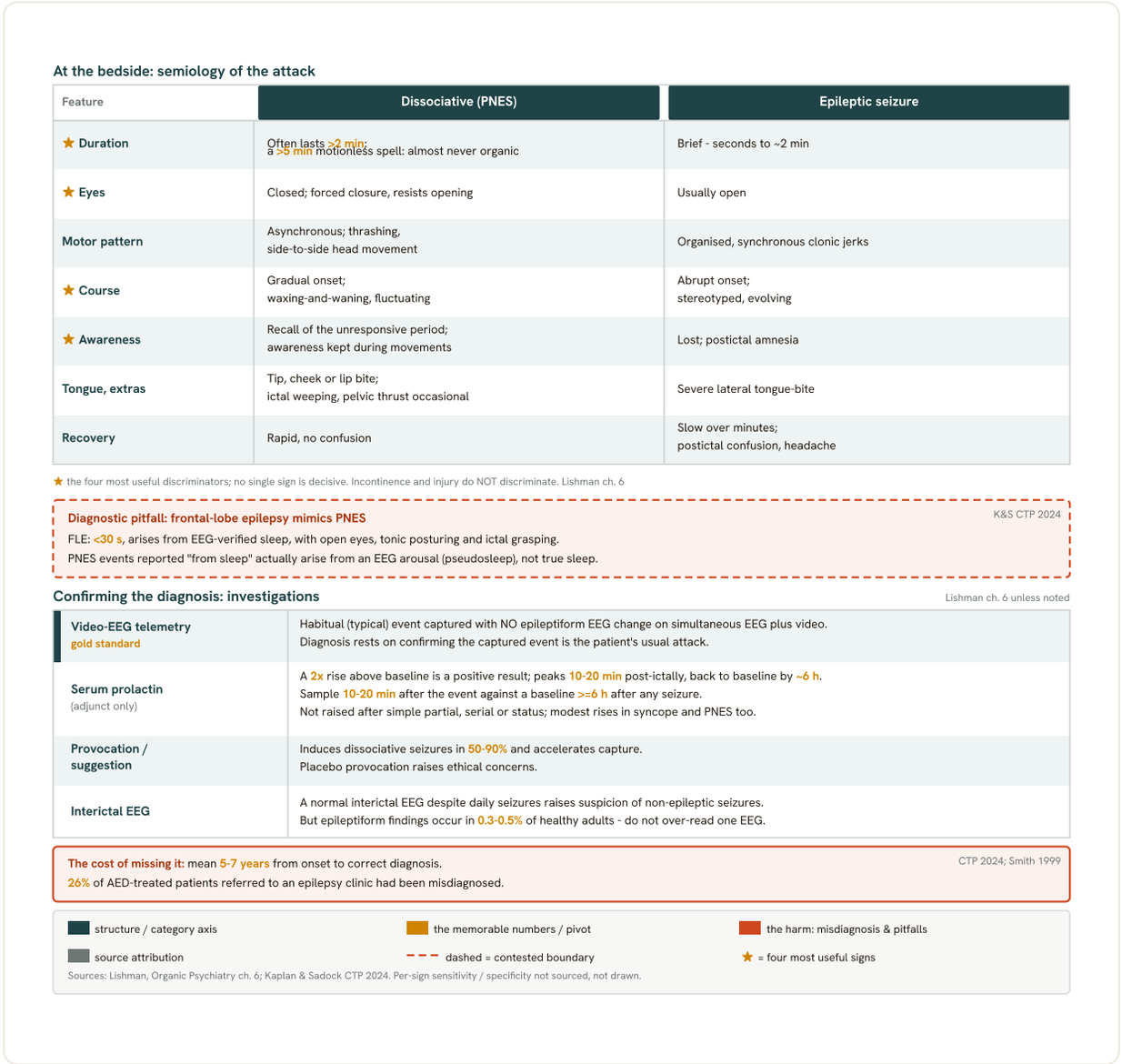


Fig 34.5 **PNES against epileptic seizures.** The four most useful bedside discriminators are long duration, eye closure, a fluctuating course and recall following unresponsiveness, yet no single sign is decisive. Video-EEG telemetry that captures the habitual event without epileptiform change is the gold standard, with serum prolactin and suggestion as adjuncts.

34.1 The governing principle: probability, not proof

Every sign shifts the odds; none closes the case. The single most important idea to carry into any answer on this topic is that no individual symptom or sign discriminates dissociative from epileptic seizures with certainty. Each feature behaves like a likelihood ratio, not a verdict. Epileptic seizures are electrically and clinically diverse, and frontal attacks in particular can be brief, bizarre and bilaterally motor while barely disturbing the scalp trace, so any single textbook feature of a dissociative attack can turn up in genuine epilepsy, and the occasional atypical dissociative event borrows the look of a convulsion. The clinician therefore reasons the way a

good diagnostician always does, by accumulating features that point the same way until the constellation becomes compelling, and by weighting what is observed against the reference standard rather than trusting a favourite sign. According to Lishman's Organic Psychiatry the most useful discriminators cluster into a small, memorable group, and it is their co-occurrence in one attack, corroborated by a reliable eyewitness or by a recording, that carries diagnostic weight. This is why a careful account from someone who watched the whole event, ideally more than one observer, is worth more than any laboratory result short of a captured attack.

RULE **Diagnose on the pattern, not on the single sign.** Because no one feature is fully discriminating, a confident bedside diagnosis rests on several concordant features in the same attack plus a trustworthy witness account. A resident who stakes the answer on one dramatic sign has missed the whole method, and will meet the counter-example in the viva.

34.2 Duration, onset and the tempo of the attack

Long, gradual and fluctuating all point away from epilepsy. Epileptic seizures are, as a class, brief and stereotyped: a convulsion runs its course in a couple of minutes and a focal seizure is usually shorter. A dissociative attack tends to last longer, and an event that continues **beyond two minutes** is common in the dissociative form and distinctly unusual in an epileptic one. Tempo matters as much as total length. Epileptic events begin abruptly and evolve through a fixed sequence, whereas a dissociative attack more often shows a **gradual onset** and a fluctuating, waxing-and-waning course, the motor activity rising, subsiding and rising again over the episode rather than marching through the ordered progression of a generalised fit. The most striking version of this is the patient found lying motionless and unresponsive for a long stretch who then recovers intact: a period of motionless unresponsiveness lasting **more than five minutes**, from which the person comes back without residual deficit, almost never has an organic basis. The reasoning behind these signs is mechanistic. An epileptic seizure is the behavioural expression of a self-limiting electrical storm that the brain terminates through its own inhibitory machinery within a tight window; a dissociative attack is not bound by that physiology, so it can be prolonged, can wax and wane with the patient's state and the surroundings, and can build up gradually rather than switch on.

34.3 The character of the movements

Disorganised and thrashing versus organised and rhythmic. When there is prominent motor activity, its character is one of the richest sources of discrimination. Epileptic convulsive movements are organised: bilateral clonic jerking is coarse, rhythmic and roughly synchronous, and it slows in frequency as the seizure exhausts itself. Dissociative motor activity tends instead to be **asynchronous**, with the limbs moving out of phase, and to take the form of thrashing, side-to-side head movement and violent flailing rather than the patterned jerking of a true convulsion. Two further movements are worth naming because they are classically taught even though they are neither common nor decisive: **pelvic thrusting** and truncal arching into **opisthotonus**, the arched posture sometimes called arc de cercle. These occur occasionally in dissociative attacks and are rare in epileptic ones, but they are not pathognomonic, and a candidate who calls pelvic thrusting proof of a functional attack will be caught out, because it can

arise in frontal seizures. The underlying logic again helps the memory. Synchronous, rhythmic, decrementing clonus reflects a single electrical rhythm driving both sides of the body from a common generator; asynchronous, variable, shifting movement has no such unifying driver, which is exactly why it looks disorganised and why its tempo can fluctuate through the attack.

34.4 The eyes and the face

Closed, resisting eyes and weeping favour a dissociative attack. The state of the eyes is one of the most reliable and easily observed signs. In the great majority of epileptic seizures the eyes are open, often deviated or staring; in dissociative attacks they are characteristically shut, and the finding that carries real weight is **forced eye closure** with active resistance when an examiner tries to prise the lids apart. That resistance implies a degree of motor control and engagement with the environment that a genuine ictal state does not permit, which is why it points so firmly toward a dissociative mechanism. Emotional expression during the attack tells a similar story. **Ictal weeping**, crying or distressed vocalisation during the event, occurs occasionally in dissociative attacks and is very rare in epileptic seizures, again because sustained, contextually coherent emotional behaviour is hard to reconcile with a generalised discharge that suspends organised cortical function. Neither sign stands alone, but eye closure in particular earns its place among the highest-yield discriminators and should be sought deliberately, and documented, in any witnessed or recorded attack rather than left to chance recall.

34.5 Awareness, responsiveness and recall

Being present during a supposedly generalised attack is the giveaway. Perhaps the most conceptually important discriminator concerns whether the patient was genuinely absent. In a generalised convulsive seizure consciousness is lost and the event is not remembered. In dissociative attacks patients commonly retain **preserved awareness** during bilateral motor activity and can afterwards recall the period in which they appeared unresponsive, and some respond to a bystander's intervention while the attack is running. Retained recall of a spell of apparent unresponsiveness, and any evidence that the patient was registering and reacting to the surroundings while both sides of the body were moving, are strong pointers, because bilateral motor activity with intact awareness is physiologically incongruent with a generalised epileptic discharge, which by definition disrupts consciousness. This is where a skilled history pays off. Gently asking the patient what they experienced during the attack, and asking witnesses whether the person seemed to respond to them or to a touch, extracts information that no single physical sign provides. It is also the feature most often overlooked, because clinicians assume that a dramatic motor event must have abolished consciousness and so never think to ask about it at all.

34.6 How the attack ends

Quick and clear-headed versus slow and confused. The recovery phase is as informative as the attack. After a generalised epileptic seizure the patient typically surfaces slowly over several minutes through a period of **postictal confusion**, disoriented, drowsy and not fully accessible. A dissociative attack characteristically ends with rapid, full recovery and a return to clear consciousness without that clouded aftermath. The contrast reflects the biology once more: an epileptic discharge leaves the cortex temporarily exhausted and inhibited, and that neuronal

aftermath takes time to clear, whereas a dissociative attack imposes no such metabolic debt and can therefore switch off cleanly. One important caution belongs here. Postictal tiredness and headache do not discriminate, because they are common after both kinds of attack and are driven as much by the physical exertion and the emotional aftermath as by any specific mechanism. It is the presence or absence of genuine confusion, the clouding of consciousness rather than mere fatigue, that carries the diagnostic signal, and conflating the two is a common way to misread the recovery phase and lose the point.

34.7 The features that do not help

Tongue-biting, incontinence and injury are widely over-trusted. Some signs enjoy a folk reputation as proof of epilepsy that the evidence does not support, and knowing their real value is a favourite examination point. Tongue-biting is the instructive case. Severe biting of the side of the tongue, **lateral tongue-biting**, is genuinely useful, being common in epileptic seizures and very rare in dissociative ones, but its worth is specific to that location: in dissociative attacks any bite is more likely to fall on the tip of the tongue, the cheek or the lip, so a report that the patient bit the tongue, without asking where, is nearly worthless. **Incontinence** of urine and physical injury are even less reliable. Incontinence is occasional in dissociative attacks and common in epilepsy but occurs in both, and injuries, including those from falls, are sustained in dissociative as well as epileptic events. Because these signs feel dramatic and consequential, they are habitually over-weighted, and treating them as clinching evidence for epilepsy is one of the surest routes to the wrong diagnosis.

TRAP **Taking incontinence, injury or a bitten tongue as proof of epilepsy.** These are the features candidates and clinicians most often over-read. Incontinence and injury occur in both kinds of attack, and only severe biting of the side of the tongue carries weight; a bite to the tip or the cheek does not. Marks, and diagnoses, are lost by presenting any of them as decisive.

34.8 The mimic that catches everyone: frontal lobe seizures

The dangerous error is the opposite one, calling a frontal seizure functional. The discriminators above are built around the generalised convulsion, and against it they work well. The great trap of the whole field is **frontal lobe epilepsy**, whose attacks are the principal epileptic imitators of dissociative seizures and the commonest reason a genuine seizure is wrongly labelled psychogenic. Frontal seizures can be brief, bizarre, hypermotor and emotionally florid, with thrashing and pelvic movements that look functional, and, crucially, they may produce little or no change on the scalp electroencephalogram, so even a recorded attack can mislead the unwary. Kaplan and Sadock's Comprehensive Textbook of Psychiatry sets out the features that pull the diagnosis back toward frontal epilepsy:

- a very short duration, often **under thirty seconds**, against the typically longer dissociative attack;
- onset directly out of electroencephalographically verified sleep, which dissociative attacks do not show;
- open rather than closed eyes during the event;

- tonic posturing of the limbs;
- ictal grasping and other stereotyped automatisms.

The lesson is one of humility. A stereotyped, very brief, nocturnal attack arising from confirmed sleep, with tonic posturing and open eyes, should raise frontal lobe epilepsy however functional the flailing looks, and this is precisely the situation in which the definitive investigation, prolonged video-EEG monitoring covered in its own topic, earns its keep. The wider behavioural and psychiatric presentation of frontal lobe epilepsy is developed separately; here it matters only as the great differential to hold in mind before committing to a functional label.

PITFALL

Brief, stereotyped, night-time hypermotor attacks with thrashing and pelvic movements are the ones most often dismissed as functional, and withdrawing antiepileptic drugs on that basis can be dangerous. When attacks are very short, arise from confirmed sleep and carry tonic posturing, treat frontal lobe epilepsy as the leading possibility and seek monitoring before you relabel the diagnosis.

34.9 The highest-yield cluster and reading the whole attack

Assemble the features and let them vote. Pulled together, the discriminators that Lishman's Organic Psychiatry rates as most useful form a compact set: a long attack duration, eye closure, fluctuating motor activity, and recall of the period of unresponsiveness. When several of these appear in one attack, corroborated by a good witness, the probability of a dissociative mechanism becomes high; when they are absent and the event is a brief, organised, amnesic convulsion with slow confused recovery, epilepsy is far more likely. The accompanying table lays the individual features side by side so that the whole phenotype, rather than any one line of it, can be weighed at a glance.

MNEMONIC

FRED

The four features that shift the odds hardest toward a dissociative attack: **F**luctuating, waxing-and-waning motor activity; **R**ecall of the unresponsive period; **E**ye closure; and a long **D**uration. The point of grouping them is that they carry weight together, not one at a time, and each is a discriminator this topic actually states.

FEATURE	FAVOURS DISSOCIATIVE (PNES) ATTACK	FAVOURS EPILEPTIC SEIZURE
Attack duration	Often prolonged	Brief, self-limited
Onset and course	Gradual, waxing and waning	Abrupt, stereotyped sequence
Prolonged motionless spell with full recovery	Suggestive, rarely organic	Distinctly unusual
Motor pattern	Asynchronous, thrashing, side-to-side head movement	Organised, roughly synchronous clonus

FEATURE	FAVOURS DISSOCIATIVE (PNES) ATTACK	FAVOURS EPILEPTIC SEIZURE
Pelvic thrusting, opisthotonus	Occasional, not pathognomonic	Rare
Eyes	Closed, often forcibly resisting opening	Usually open
Ictal weeping	Occasional	Very rare
Awareness and recall	May be preserved, with later recall	Lost, not remembered
Recovery	Rapid, clear-headed	Slow, with postictal confusion
Tongue-biting	Tip, cheek or lip, if any	Severe, to the side
Incontinence and injury	Non-specific, can occur	Non-specific, can occur

A feature-by-feature comparison of dissociative and epileptic attacks; the diagnosis is read from the balance of the whole column, since no single row is decisive on its own.

over 2 min	over 5 min	under 30 s
ATTACK DURATION COMMON IN PNES	MOTIONLESS YET RECOVERS: RARELY ORGANIC	FRONTAL LOBE SEIZURE, THE MIMIC

IN INDIA

In much of Indian practice the diagnosis has to be made on semiology alone, because prolonged video-EEG monitoring is concentrated in a few tertiary centres and is out of reach for most patients. This raises the premium on a meticulous eyewitness history, increasingly from a smartphone recording of the attack that families can be coached to capture. The older label of hysteria still colours how these presentations are received, and the stigma attached both to epilepsy and to a psychiatric attribution means patients frequently arrive already committed to antiepileptic drugs and reluctant to accept a functional explanation. Getting the clinical differentiation right, gently and alongside the family, matters most exactly where confirmatory testing is scarce.

GOOD TO REMEMBER

When you cannot get an attack on video, chase the account instead: how long it lasted, whether the eyes were shut and resisted opening, whether the movements were rhythmic or thrashing, whether the person remembers the spell or responded to anyone during it, and how quickly they came round afterwards. Ask a witness to record the next event on a phone. Several concordant soft signs beat one dramatic sign every time.

No single sign is decisive: the diagnosis of a dissociative attack rests on a cluster of long duration, eye closure, fluctuating asynchronous movement, preserved awareness with later recall and a rapid clear recovery, while very brief nocturnal attacks with tonic posturing must always raise frontal lobe epilepsy as the great mimic.

35 · Video-EEG, serum prolactin and other investigations in PNES diagnosis

The whole weight of a confident diagnosis here falls on investigation, because the bedside is never quite enough. The features of the attack itself, set out in the companion topic on the clinical features that differentiate the two events, shift the odds but never close the case, and both errors that follow from stopping at the bedside are costly: a person committed to antiepileptic drugs for an illness they do not have, or a genuine seizure waved away as functional. What settles the question is a recorded event, and the marks in this area cluster around a single disciplined idea, that the diagnosis is made positively by capturing the attack, not negatively by a normal routine test. This topic walks the investigative pathway in the order a clinician actually uses it: the reference standard of simultaneous video and electroencephalography, the interictal trace and its two opposite traps, serum prolactin and its narrow window, provocation, the sleep-onset discriminator, and the sobering delay that makes getting all of this right a matter of years.

What a dissociative attack actually is, and the shifting terminology it has travelled under, belongs to its own companion topic and, more broadly, to the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes. The general fundamentals of the electroencephalogram, and the distinction between ictal and interictal discharges, are developed in their own right and are used here only as they bear on this one diagnostic question. Delivering the diagnosis to the patient and its stepped management, the psychiatric comorbidities and predisposing factors, and the genuinely difficult patient who has both dissociative and epileptic seizures at once are each treated separately. Here we stay with the tests: what each one can prove, what it cannot, and how they are sequenced.

35.1 The reference standard: ictal video-EEG telemetry

Capture the attack with its brain trace and the argument is over. The single investigation that settles the diagnosis is **video-EEG telemetry**, prolonged simultaneous recording of the electroencephalogram alongside time-locked video of the patient, and per Lishman's Organic Psychiatry it is the accepted gold standard for the condition. Its power lies in recording the clinical event and the brain's electrical activity at the same instant, so that semiology and cortical rhythm can be read against each other rather than inferred separately. The diagnostic move it permits is a positive one. According to Kaplan and Sadock's Comprehensive Textbook of Psychiatry, the diagnosis is established when the patient's habitual attack is captured and the trace shows no accompanying epileptiform change during the event: the typical spell unfolds on camera while the electroencephalogram stays free of any ictal discharge. Two conditions have to hold for this to mean anything. The recorded event must be confirmed by the patient or a relative as their usual attack and not some lesser or atypical spell, because a normal trace during an event nobody recognises proves nothing about the attacks that matter. And the trace reflects cortical seizures visible at the scalp, so deep or very small discharges can escape surface electrodes, which is why the method leans on capturing the full, typical semiology rather than a fragment. Held to those conditions, a captured habitual attack with a clean ictal trace is as close to certainty as this field offers.

RULE **The diagnosis is positive, not an absence.** Non-epileptic attacks are confirmed by recording the patient's own typical event and showing that no ictal electrographic change accompanies it, with the event verified as habitual. A normal test in the gaps between attacks is not the same thing and never carries the same weight.

35.2 The interictal EEG: two opposite traps

A normal trace does not rule epilepsy out, and an abnormal one does not rule it in. Between attacks the electroencephalogram is far weaker evidence than most people assume, and it misleads in both directions. On one side, a completely normal **interictal EEG** in a patient reporting seizures every day is genuinely suggestive of non-epileptic attacks, because uncontrolled daily epilepsy usually leaves some interictal footprint. Yet that inference is soft, not hard, because roughly one-third of people with epilepsy show no interictal abnormality at all when awake, so a clean trace is entirely compatible with real epilepsy and can never exclude it. On the other side lies the more dangerous error. Interictal and even frankly epileptiform discharges can appear in people whose only attacks are dissociative, and epileptiform findings turn up in about **0.3 to 0.5 per cent** of entirely healthy adults, so a sharp wave on the trace is not a licence to diagnose epilepsy and start drugs. Lishman's Organic Psychiatry is explicit that the interictal record has to be read in the light of the history and the semiology and never in isolation. The practical upshot is that the routine EEG, ordered and interpreted on its own, is one of the commonest sources of diagnostic error in this whole area, capable of manufacturing a false epilepsy label out of a benign blip or false reassurance out of a normal study. It is a supporting tool, not an arbiter.

TRAP **Reading a normal interictal EEG as proof of non-epileptic attacks, or a minor epileptiform blip as proof of epilepsy.** Both are examination favourites. A substantial minority of people with genuine epilepsy have normal traces between seizures, and a small fraction of healthy people carry epileptiform discharges, so the interictal study confirms neither diagnosis. Candidates who let it decide the case lose the mark.

35.3 Serum prolactin: the physiology and its narrow window

A generalised discharge that reaches the hypothalamus drives prolactin up, but only briefly. The rationale for the **serum prolactin** test is that a generalised epileptic discharge spreading through hypothalamic pathways transiently releases prolactin from the anterior pituitary, a hormonal signature that a purely psychological event should not produce. The kinetics are the whole test and must be known precisely. Following a tonic-clonic seizure the serum level climbs and reaches its peak at about **10 to 20 minutes**, then falls back, returning to baseline after roughly 6 hours, as characterised by Lishman's Organic Psychiatry. Everything that makes the test useful and everything that makes it fail follows from that curve. Because the rise is short-lived, a sample drawn too late catches a level already on its way down or back to normal, which is then read, wrongly, as a negative result. Because the release depends on a discharge large enough to engage the hypothalamus, seizures that stay small or focal may never move the hormone at all. And because prolactin also responds to physiological stress, the specificity of any single raised

value is limited. The accompanying figure sketches this brief postictal rise and its return; the sampling rules that follow simply try to catch the hormone at the top of that curve.

35.4 Sampling prolactin correctly and reading the result

One value proves nothing; the test is a timed pair. A prolactin result is interpretable only as the relationship between a well-timed postictal sample and the patient's own baseline, and getting either wrong wastes the test. Lishman's Organic Psychiatry sets out the protocol:

- draw the postictal sample at the top of the curve, within that early peak window and not later;
- take a paired baseline sample at least 6 hours after any seizure, when the hormone has certainly returned to rest;
- match that baseline to approximately the same time of day, because prolactin has its own diurnal rhythm.

Only against that baseline does the postictal figure mean anything, and the threshold conventionally taken as a significant, positive result is a rise to **at least twice the baseline** value, although the exact cut-off has varied between studies and is not a single agreed number. A doubling or more, sampled at the right moment against a proper resting level, supports a preceding generalised or complex partial seizure; a value below that threshold is informative only if you are confident the sample was drawn inside the window and the event was one that should have raised the hormone.

> **GOOD TO REMEMBER**
>
> If you are going to send a prolactin at all, send it properly or not at all: one tube at the peak of the rise, as soon as possible after the attack, and a second, paired baseline tube drawn well clear of any seizure and matched for time of day. A lone postictal value with no baseline, or one drawn too late, is uninterpretable, and an uninterpretable test is worse than no test because it invites a false conclusion.

35.5 Why prolactin is an adjunct, not a rule-out test

Too many epileptic seizures fail to raise it, and too many other events do. The limitations of the test are as examinable as its rules, because they define exactly what a normal or a raised value is allowed to mean. Per Lishman's Organic Psychiatry, serum prolactin is not reliably raised after several kinds of genuinely epileptic activity, and it rises in some events that are not epileptic at all. On the false-negative side, it is typically not elevated after simple partial seizures, after serial seizures and after **status epilepticus**, and it is a less dependable marker after complex partial seizures than after a full tonic-clonic convulsion. On the false-positive side, modest rises occur in **syncope** and in dissociative attacks themselves. So a failure to rise never proves an event was non-epileptic, and a modest rise never proves it was epileptic.

MNEMONIC

Simple, Serial, Status

The three epileptic situations after which serum prolactin is characteristically *not* raised, so a normal value cannot be trusted to exclude a seizure: **S**imple partial seizures, **S**erial seizures and **S**tatus epilepticus, with the complex partial seizure a fourth, less reliable, case. Each is a situation this source names explicitly.

PITFALL

The classic clinical mistake is to read a normal postictal prolactin as evidence that an attack was psychological, when in fact it followed a simple partial or complex partial seizure, or was drawn outside the window. The mirror-image error is to treat a modest rise as proof of epilepsy when syncope, or even a dissociative attack, can produce one. Prolactin helps sort a probable tonic-clonic seizure from a probable functional event; it does nothing else safely.

Where the test still earns its keep is captured by the formal verdict on it. According to Lishman's Organic Psychiatry, the **American Academy of Neurology** judged postictal serum prolactin a useful adjunct for separating generalised and complex partial seizures from psychogenic events, while insisting that it is an adjunct and not a stand-alone test. With prolonged video-EEG now more widely available the assay has fallen out of routine favour, but a negative result after an apparently convulsive attack retains real practical value, and in settings without ready telemetry that modest role is not to be dismissed.

35.6 Provocation and induction: bringing an attack on

If attacks are too infrequent to catch, you can try to precipitate one. Prolonged telemetry only works if an event obligingly occurs while the patient is wired up, and many people do not attack often enough for that. The response is **provocation**, the deliberate use of induction to precipitate a habitual attack under recording so that its nature can be settled on the trace. Per Lishman's Organic Psychiatry, the non-specific activation procedures already built into a routine study, **hyperventilation** and **photic stimulation**, together with verbal suggestion, will induce a dissociative attack in a large proportion of susceptible patients, quoted across studies as somewhere between **50 to 90 per cent**, and they shorten the time to a captured event. Simple suggestion alone, without any elaborate placebo, provoked a typical event in about two-thirds of patients in one series and cut the need for prolonged telemetry in nearly half. The obvious efficiency of this has to be weighed against its ethics. Techniques that rely on active deception, for instance injecting saline described as a drug that will bring on a fit, raise real problems of consent and of the very trust on which the subsequent conversation about a functional diagnosis depends, and many units now prefer transparent activation and honest suggestion to any frank placebo. However it is elicited, the captured event still has to be confirmed as the patient's usual attack to count.

35.7 Pseudosleep and the sleep-onset discriminator

An attack that seems to come out of sleep may be coming out of wakefulness. One elegant discriminator emerges only because video and electroencephalography run together, and it turns on what "sleep" actually means on the trace. Patients with dissociative attacks not uncommonly report that their events arise from sleep, which sounds reassuringly organic, but the recording tells a different story. According to Lishman's Organic Psychiatry, these attacks in fact begin from a state of **pseudosleep**: the patient appears to be asleep, yet the trace shows an arousal to wakefulness in the moments before the attack starts, so the event is really arising from the waking brain behind closed eyes. This matters because it is precisely the feature that the main epileptic mimic does not share. Frontal lobe epilepsy, the seizure disorder that most often imitates dissociative attacks, can genuinely arise directly out of electrographically documented sleep, so an attack that emerges from a verified sleep trace points toward epilepsy while one that emerges from pseudosleep points away from it. The wider behavioural and psychiatric presentation of frontal lobe epilepsy is developed in its own right; here it matters only as the organic counterpoint that makes the pseudosleep sign useful, and the finding is available only when the attack is caught with simultaneous video and electroencephalography.

35.8 Sequencing the tests, and the cost of getting it wrong

The tests form a hierarchy, and the reason to climb it quickly is measured in years. No single investigation is used in isolation; they form a ladder, and the accompanying table lays out what each rung can and cannot prove. Beyond these, neuroimaging and neuropsychological assessment have supporting roles in the wider work-up without confirming or excluding the diagnosis, and are named here only to be placed. The stakes in getting up this ladder are high, because the natural history of a missed diagnosis is long. According to Kaplan and Sadock's Comprehensive Textbook of Psychiatry the mean latency from the first manifestation to the correct diagnosis runs to **5 to 7 years**; Lishman's Organic Psychiatry records that such events are commonly treated as epileptic for more than 3 years, and that among antiepileptic-drug-treated patients referred on to a specialist epilepsy service, 26 per cent had in fact been misdiagnosed. Those years are not benign: unnecessary drugs with their own adverse effects, psychological treatment not given, and a diagnostic identity that hardens the longer it stands. That is the real argument for reaching the reference standard early rather than accumulating inconclusive routine tests.

INVESTIGATION	WHAT A POSITIVE OR TYPICAL RESULT SHOWS	MAIN LIMITATION
Ictal video-EEG telemetry	Habitual attack captured with no ictal EEG change: the positive diagnosis	Needs an event during recording; deep discharges can escape scalp electrodes
Interictal (routine) EEG	A normal trace despite frequent daily attacks is suggestive of non-epileptic events	Normal in much genuine epilepsy; epileptiform blips occur in healthy people
Serum prolactin (paired, timed)	A sufficient postictal rise supports a generalised or complex partial seizure	Not raised after several seizure types; rises in syncope and dissociative attacks
Provocation and suggestion	Precipitates a habitual attack under recording, accelerating capture	Deceptive placebo raises consent and trust concerns; event must be confirmed habitual
Video-EEG in apparent sleep	Pseudosleep arousal before onset points away from epilepsy	Requires simultaneous video and EEG; interpretation is specialist

The investigative ladder in psychogenic non-epileptic attacks, from the confirmatory reference standard down to the supporting adjuncts; no rung below the captured attack is diagnostic on its own.

10 to 20 min	**at least 2x**	**50 to 90%**	**5 to 7 years**
POSTICTAL PROLACTIN PEAK	RISE COUNTED AS POSITIVE	ATTACKS PROVOKED BY SUGGESTION	TYPICAL DELAY TO DIAGNOSIS

IN INDIA

Prolonged video-EEG telemetry sits in a handful of tertiary centres, so across much of the country the diagnosis has to lean on the cheaper, more available adjuncts, and this is exactly where they are most often misused. A single unpaired serum prolactin, drawn whenever the patient happens to reach the laboratory, is ordered widely and then interpreted as if a normal value excluded a seizure, which it cannot. The disciplined answer where telemetry is scarce is a properly timed, paired prolactin used only to support a probable convulsion, a good home or smartphone recording of a typical attack, and pragmatic activation during a routine study, with frankly deceptive placebo methods best avoided given how heavily the later therapeutic relationship depends on the patient's trust.

The diagnosis of non-epileptic attacks is made positively, by capturing the patient's own habitual event on video-EEG with no accompanying ictal discharge; the interictal EEG, serum prolactin and provocation only support that finding, no normal result ever excludes epilepsy, and a properly timed twofold prolactin rise means a probable generalised seizure and nothing more.

36 · Psychiatric comorbidity and predisposing factors in PNES

Vulnerability is the frame here, not causation. A psychogenic non-epileptic seizure does not arise from nowhere; it grows on a psychiatric and developmental substrate, and the marks in this topic sit on knowing what that substrate is, who carries it and how it is assembled into a

formulation. Examiners return to two things again and again: the comorbidity profile that separates these patients from those with epilepsy, and the discipline that no amount of comorbidity, however striking, actually establishes the diagnosis. A candidate who can list the predisposing, precipitating and maintaining factors, weight them correctly, and still say that the diagnosis rests elsewhere has understood the whole topic. The clinical stakes are the same as the examination ones, because a formulation of vulnerability is what turns a confirmed diagnosis into a treatment plan the patient can accept.

This account stays with the who and the why. What a **dissociative seizure** actually is, the terminology it travels under and its place within the family of functional neurological disorders belong to the companion topic and, for the general construct, to the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes. How these attacks are told apart from epileptic seizures at the bedside, how they are confirmed with video-electroencephalographic capture and serum prolactin, how the diagnosis is communicated and managed in stepped fashion, and the genuinely difficult patient who has both dissociative and epileptic seizures at once, are each developed in their own right. Here the material is narrower: the epidemiology of the vulnerable patient, the psychiatric disorders that cluster around these attacks, the childhood and illness-behaviour history that predisposes, and the biopsychosocial architecture that holds it all together.

36.1 The vulnerable patient: who develops dissociative seizures

Young, disproportionately female, and sitting in the epilepsy clinic already. The demographic signal is consistent enough to be examinable. According to Kaplan and Sadock's Comprehensive Textbook of Psychiatry, dissociative seizures affect women in their reproductive years at approximately a **3:1 female-to-male** ratio, are most frequent in young adults, and yet occur across the whole lifespan, so neither an older nor a male patient can be excluded on demographic grounds alone. The second half of the epidemiology matters more for daily practice, because it locates these patients where the neurologist and the psychiatrist actually meet them. Far from being a rarity, dissociative seizures account for up to **30 per cent** of admissions to specialist epilepsy monitoring units, and about one in five of the patients referred for evaluation of apparently intractable epilepsy prove, on capture, to have dissociative seizures rather than a drug-resistant epilepsy. The lesson embedded in those figures is that the population at risk is not a separate psychiatric caseload but a large fraction of the very patients being worked up as difficult epilepsy. A young woman whose seizures are not responding to escalating antiseizure medication is, on base rates alone, a patient in whom the possibility should already be alive, and the reproductive-age preponderance is why the disorder so often intersects with pregnancy and the social pressures around young women.

36.2 The comorbidity profile that separates PNES from epilepsy

Trauma, PTSD and personality disorder discriminate; depression does not. The single most testable idea in the comorbidity of these attacks is a comparative one, and it is easy to get half-right and lose the mark. Both patients with dissociative seizures and patients with epilepsy carry a heavy psychiatric burden, so the discriminating question is not whether a disorder is present but whether it is present more often than in epilepsy. Kaplan and Sadock's Comprehensive

Textbook of Psychiatry draws the line cleanly: prior traumatic experiences, **post-traumatic stress disorder** and **personality disorder** are more frequent in dissociative seizures than in epilepsy, whereas **mood disorder** is frequent in dissociative seizures but not significantly more so than in epilepsy. The direction of that last clause is the whole examination point, and reversing it is the classic slip. Depression is common in both groups because chronic, frightening, socially disabling paroxysmal disorders are depressing whatever their mechanism, so a high rate of low mood does nothing to separate the two. It is the trauma-spectrum burden, the post-traumatic and personality pathology, that is genuinely over-represented in the dissociative group, which is why the trauma history is worth taking with particular care even though it will never, by itself, decide the case. The accompanying table lays the comparison out so that the direction of each row can be read at a glance.

PSYCHIATRIC DISORDER	IN DISSOCIATIVE SEIZURES, COMPARED WITH EPILEPSY
Prior traumatic experiences	More frequent than in epilepsy
Post-traumatic stress disorder	More frequent than in epilepsy
Personality disorder	More frequent than in epilepsy
Mood disorder (depression)	Frequent, but not significantly more than in epilepsy

The comparative comorbidity profile after Kaplan and Sadock's Comprehensive Textbook of Psychiatry; only the trauma-spectrum rows discriminate, and the mood-disorder row is the one candidates most often invert.

36.3 Childhood adversity and abuse as the developmental substrate

The recurring theme is early adversity, above all abuse. Behind the adult comorbidity lies a developmental story that Lishman's Organic Psychiatry treats as a common underlying theme rather than a universal law. Adverse and traumatic childhood experiences run through these histories, with a particularly highlighted association with **childhood sexual, physical and emotional abuse**. The mechanistic reading, which the exam rewards over rote recall, is that early abuse and adversity shape the very capacities that dissociative seizures exploit: an ingrained tendency to dissociate under threat, a body that has learned to express distress somatically, and an attachment and coping style forged in an environment where direct expression of need was unsafe or futile. What the candidate must hold alongside this, though, is the caveat the source itself insists on. This developmental adversity is not unique to dissociative seizures; it is shared with the other somatoform and dissociative disorders, so a childhood abuse history raises the prior probability of a functional mechanism in general without pointing specifically at seizures and without proving anything on its own. The two errors to avoid are opposite in direction. One is to treat a disclosed abuse history as confirmatory; the other, subtler and commoner, is to treat its absence as exclusionary, forgetting both that adversity is a theme and not a criterion and that abuse is chronically under-disclosed, especially at a first meeting and where cultural shame is heavy.

PITFALL

Do not read a negative trauma history as evidence against the diagnosis, and do not go hunting aggressively for buried abuse to shore one up. Childhood adversity is a common theme, not a necessary ingredient, and disclosure is often delayed by shame and by the very dissociative style that predisposes to the attacks. Pressing a reluctant patient to produce a trauma narrative early can rupture engagement and reads, wrongly, as making the diagnosis contingent on a confession.

36.4 A history of medically unexplained symptoms

The seizure is usually not the first functional presentation. One of the most useful pieces of history in these patients is not about seizures at all. Dissociative seizures tend to arrive in a person with an established pattern of somatic distress, and Lishman's Organic Psychiatry records that up to **80 per cent** of patients with these attacks have a history of previous **medically unexplained presentations**. The attack, in other words, is often the latest and most dramatic expression of a longer somatising trajectory rather than a bolt from a clear sky, and a careful review of past consultations frequently turns up earlier functional symptoms, repeated investigations that came to nothing, and a relationship with medical services already shaped by unexplained complaints. This matters clinically in two ways. It supports the formulation, because a lifelong tendency to somatise is the diathesis on which a dissociative seizure disorder is built, and it warns against the reflex of investigating each new symptom afresh as though the slate were blank, which entrenches illness behaviour and risks iatrogenic harm. It also softens the disclosure that comes later, because a patient who can be shown the pattern of their own history is often more able to accept a functional account than one confronted with the seizures in isolation. The figure is worth carrying, but it describes a tendency and a prior probability, not a gate through which every patient must pass.

36.5 The biopsychosocial formulation: predisposing, precipitating, maintaining

Do not list the factors; assemble them across three time-frames and three domains. The mature way to hold everything above is the formulation that Lishman's Organic Psychiatry sets out, in which vulnerability is organised along two axes at once: a temporal axis of predisposing, precipitating and maintaining factors, and a domain axis of biological, psychological and social contributions. The predisposing factors are the standing vulnerabilities that make a person the kind of person in whom these attacks can develop, spanning all three domains: a biological substrate such as **learning difficulty**; the psychological traits of somatising and dissociative tendency, **avoidant coping** and an underlying personality or mood disorder; and the social conditions of poor family functioning and the modelling of attacks on others with epilepsy in the patient's environment. The precipitating factors are what tips a vulnerable person into the first attacks, and Lishman's Organic Psychiatry names adverse life events and acute episodes of panic or syncope here. The maintaining factors are what keeps the attacks going once begun, and they are where treatment often has most leverage: the benefits of the **sick role**, agoraphobic avoidance and safety behaviour, and the confused or anxious reactions of the patient and of carers to the diagnosis itself. Reading them together shows why the disorder is self-perpetuating: the

precipitant lights the fire, and the sick-role and avoidance keep feeding it long after the original trigger has passed.

MNEMONIC

Three Ps, three domains

Formulate every case on a grid. Down the side, the three Ps: **P**redisposing (standing vulnerability), **P**recipitating (what triggered the first attacks) and **P**erpetuating or maintaining (what keeps them going). Across the top, the three domains: biological, psychological and social. The predisposing row fills all three domains; the value of the grid is that it forces you to place each factor in both its time-frame and its domain rather than reciting an undifferentiated list.

RULE **Vulnerability is formulated, not merely listed.** The examinable move is to sort every factor into predisposing, precipitating or maintaining, and into biological, psychological or social. A flat list of risk factors earns little; a formulation that separates the standing diathesis from the trigger and from the perpetuating loop shows the reasoning and tells you which factors are actually modifiable.

TIME-FRAME	DOMAIN	FACTORS (AFTER LISHMAN'S ORGANIC PSYCHIATRY)
Predisposing	Biological	Learning difficulty
Predisposing	Psychological	Somatising and dissociative traits; avoidant coping; personality or mood disorder
Predisposing	Social	Poor family functioning; modelling of attacks on others with epilepsy
Precipitating	Psychological or physical	Adverse life events; an acute episode of panic or syncope
Maintaining	Psychological	Benefits of the sick role; agoraphobic avoidance and safety behaviour
Maintaining	Social	Confused or anxious reactions of the patient and of carers to the diagnosis

The predisposing, precipitating and maintaining formulation; the standing vulnerability spreads across all three domains, while the trigger and the perpetuating loop are where the timeline earns its keep.

36.6 Pre-ictal autonomic arousal and the panic overlap

Most attacks are preceded by a surge of autonomic symptoms. One phenomenological feature earns its place among the predisposing and precipitating material because it links the physiology of arousal to the attack itself. Lishman's Organic Psychiatry records that about **two-thirds** of patients with dissociative seizures report symptoms of **autonomic arousal** preceding their seizures, a pre-ictal build-up that typically includes:

- hyperventilation;

- palpitations;
- paraesthesiae;
- a dry mouth;
- perspiration.

The clinical significance of this cluster is that it overlaps almost completely with a panic attack and with presyncope, which is exactly why acute panic and syncope appear among the precipitating factors and why the boundary with an anxiety disorder is so blurred. A person prone to panic who then dissociates as the arousal peaks has a plausible route from autonomic surge to attack, and for many patients the seizure functions as an escape from an intolerable state of arousal. This is where the topic touches, without re-teaching, the ictal fear and panic phenomena of epilepsy and the interictal anxiety disorders that are developed elsewhere; the point owned here is that the arousal is a common pre-ictal experience and a maintaining engine, not that it is a diagnostic sign. It also has a practical yield, because a patient who recognises the arousal prodrome can be taught to intervene at that moment, which is the entry point for several of the psychological treatments described in the management topic.

36.7 Comorbidity raises probability, it does not make the diagnosis

The psychiatric history is context, not proof. Everything in this topic is a statement about probability, and the cardinal error is to let it harden into a diagnosis. Kaplan and Sadock's Comprehensive Textbook of Psychiatry is explicit that clinicians should not assume a dissociative-seizure diagnosis on the basis of comorbid psychiatric disorder alone, such as post-traumatic stress disorder or a personality disorder. The reasoning is straightforward and worth being able to state. Trauma, post-traumatic stress disorder and personality pathology are common in the general population and commoner still among people with epilepsy, so a patient can carry every one of these and still have entirely epileptic seizures; conversely, a patient with none of them can have dissociative seizures, since the risk factors are enrichments of probability, not necessary conditions. The diagnosis is made where it has always been made, on the attack itself, from a captured event and its electrical correlate and the clinical semiology, all of which are developed in the semiology and investigations topics. The comorbidity and predisposing factors do real work in raising suspicion, in shaping the formulation and in guiding treatment, but they sit on the wrong side of the line to establish the diagnosis, and the most dangerous version of the error runs the other way, dismissing a patient's genuine seizures as functional because their psychiatric history is florid.

TRAP **Diagnosing dissociative seizures from the psychiatric history.** The classic error is to reason that a young woman with an abuse history, post-traumatic stress disorder and a personality disorder whose seizures resist medication must have functional attacks. Every clause raises the prior, and none of them is diagnostic. The examiner wants you to say that comorbidity informs suspicion but the diagnosis is made on the captured attack, and that reversing this has sent patients home with untreated epilepsy.

36.8 Formulating vulnerability in the individual patient

The purpose of all of this is a formulation the patient can accept. Pulled together, the material assembles into one working picture: a young, often female patient who carries a trauma-spectrum burden heavier than that seen in epilepsy, a developmental history in which adversity and abuse are a recurring theme, a long track record of medically unexplained symptoms, and attacks heralded by a surge of autonomic arousal that shades into panic. Laid on the predisposing, precipitating and maintaining grid, these stop being a list of risk factors and become a shared explanation of how this person came to have these attacks and what keeps them going, which is the account that makes the diagnosis credible and the treatment intelligible when the time comes to deliver it. The formulation is where comorbidity earns its clinical keep, and it is also where the discipline of the topic is honoured, because a formulation is offered as an understanding of vulnerability, never smuggled in as the evidence for the diagnosis.

3:1	**up to 30%**	**up to 80%**	**~two-thirds**
FEMALE-TO-MALE, REPRODUCTIVE YEARS	OF EPILEPSY MONITORING UNIT ADMISSIONS	WITH PRIOR UNEXPLAINED SYMPTOMS	REPORT PRE-ICTAL AUTONOMIC AROUSAL

IN INDIA

The reproductive-age, predominantly female profile lands on a specific social reality. Presentations still often reach services under the older idiom of hysteria, and the attacks frequently carry a heavy load of family and marital distress, dowry pressure and constrained agency for young women, which functions both as precipitant and as maintaining context. Childhood adversity and abuse are under-disclosed at the best of times and more so where shame and family honour are in play, so a negative first-visit history is especially weak evidence. The stigma attached both to epilepsy and to a psychiatric attribution means many patients arrive already committed to antiseizure drugs and to a physical explanation, and the formulation has to be built gently, often with the family in the room, if it is to be accepted at all.

GOOD TO REMEMBER

Take the trauma history and the illness-behaviour history with real care, because they build the formulation and soften the eventual disclosure, but write them into the record as vulnerability and context, never as the basis of the diagnosis. The two histories that most repay the effort are the map of past medically unexplained presentations and the account of the pre-ictal arousal, because both feed directly into treatment: the first reframes the pattern for the patient, and the second gives them a moment to intervene.

Trauma, post-traumatic stress disorder and personality disorder are over-represented in dissociative seizures while mood disorder is not, childhood adversity and a long history of medically unexplained symptoms are recurring predisposing themes, and the whole picture is formulated as predisposing, precipitating and maintaining factors across biological, psychological and social domains, yet none of it makes the diagnosis, which is read from the captured attack.

37 · Communicating the diagnosis and stepped psychological and pharmacological management of PNES

The consultation in which you tell the patient is itself the first dose of treatment. Once the investigations have settled that the attacks are **psychogenic non-epileptic seizures** and not epilepsy, the clinical problem changes shape. It stops being a diagnostic puzzle and becomes a therapeutic one, and the pivot on which everything after it turns is the conversation in which the diagnosis is handed over. Handled badly, that conversation loses the patient before treatment has begun, sometimes for good; handled well, it can begin to reduce the attacks on its own. The marks in this topic sit almost entirely in that transition: how to say what has been found, how to keep an easily alienated patient engaged, and how to build the stepped plan that follows the news.

This account picks the story up at the point of disclosure. How the diagnosis is reached and confirmed, including video-EEG and serum prolactin, belongs to the investigations topic; how these attacks are told apart from epileptic seizures at the bedside, the psychiatric comorbidity and predisposing factors that cluster around them, and where the disorder sits within the family of **functional neurological disorder** are each developed elsewhere, the general construct of conversion and functional neurological symptoms in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes. What is owned here is narrower and more practical: the delivery of the news, the non-confrontational stance it demands, the safe withdrawal of unnecessary antiepileptic drugs, and the stepped, multidisciplinary treatment in which psychological therapy, not medication, does the definitive work.

37.1 Delivering the diagnosis is the pivotal step

Engagement is the treatment's first hurdle and its most fragile. The most important and often the most difficult task in the whole management of these patients is engaging them in treatment at all, and the vehicle for that engagement is the way the diagnosis is communicated. This is why the disclosure is treated as a management step in its own right rather than as a formality that precedes the real work. Lishman's Organic Psychiatry, drawing on the observations of Shen and Mellers, makes the strong version of the point: the **communication of the diagnosis** is potentially therapeutic in itself and can reduce the frequency and severity of the attacks, an effect that is most marked in recent-onset cases where the pattern has not yet consolidated. The mechanism is intuitive once stated. A patient who has been investigated repeatedly, often labelled with refractory epilepsy, and treated with escalating antiseizure drugs to no effect, has usually accumulated fear, uncertainty and a sense that something dangerous is being missed. A clear, credible account that names the condition, explains what the attacks are and are not, and offers a route out of the impasse removes much of the ambiguity that has been feeding the symptom. The clinical implication is direct: the encounter deserves unhurried time, a private setting and a prepared clinician, because it is doing more than conveying a result. It is beginning the intervention, and the early consultation is a window that closes as the disorder becomes entrenched.

37.2 The non-confrontational imperative

The stance governs the content. The single principle that governs the entire encounter is that it must be **non-confrontational**. Patients with these attacks are extremely sensitive to any suggestion, however oblique, that they are producing the events deliberately or putting them on, and the merest hint of that reading will end the therapeutic relationship. The vulnerability is not perverse; it is structural. In lay understanding the idea of a psychological seizure sits uncomfortably close to an accusation of faking, and a patient who hears the diagnosis as a charge of fabrication will feel disbelieved, dismissed and, quite reasonably, angry. Because the symptoms are involuntary and genuinely experienced, treating them as if they were feigned is both wrong and destructive. The practical consequence is that every element of the explanation has to be framed positively and explicitly around genuineness. The clinician states plainly that the attacks are real, that the patient is believed, and that the diagnosis is not a verdict of malingering. Only once that ground is secured does the rest of the conversation become possible, which is why the stance is logically prior to the content rather than a matter of bedside manner laid over it.

RULE **The delivery must be non-confrontational above all else.** Because these patients are acutely sensitive to any implication that they are feigning, the diagnosis is delivered as a real, involuntary, believed condition, never as an unmasking. Get the stance wrong and no amount of correct information will land; get it right and the explanation becomes therapeutic.

TRAP **Confronting the patient with the psychological nature of the attacks.** The classic error is to announce that the tests are normal, that there is nothing physically wrong, and that the problem is psychological, phrasing the patient hears as an accusation of pretending. It ruptures engagement at the exact moment it was most needed. The exam answer is that reassurance of genuineness comes first and the word feigning never appears.

37.3 What a good explanation contains

Two complementary templates, one shared skeleton. The content of the disclosure has been formalised into overlapping frameworks that a candidate can reproduce. Kaplan and Sadock's Comprehensive Textbook of Psychiatry organises it into a **four**-element **framework**: reassure the patient that the symptoms are genuine and not fabricated; explain how the diagnosis was reached and give the condition its names; offer the hypothetical mechanisms, including a neurobiological model, together with any identified risk factors; and review the treatments that are effective. Lishman's Organic Psychiatry sets out a closely matched non-confrontational explanation template that runs along the same axis but foregrounds the concept of **dissociation** as the model offered to the patient: explain why the events are not epilepsy and what the patient does have; reassure the patient that they are not suspected of putting the attacks on and that the disorder is very common; discuss the causes and maintaining factors; and outline the treatment. Read side by side, the two are one method described twice, and holding them together lets the reader see both the structure and the substance of a competent explanation.

BEAT OF THE EXPLANATION	COMPREHENSIVE TEXTBOOK OF PSYCHIATRY FRAMEWORK	LISHMAN'S EXPLANATION TEMPLATE
Establish genuineness	reassure the symptoms are real and not fabricated (element one)	reassure the patient is not suspected of putting on the attacks, and that the disorder is very common
Name and account for it	explain how the diagnosis was reached and give the condition its names	explain why it is not epilepsy and what the patient does have, described as dissociation
Offer a mechanism	give hypothetical mechanisms including a neurobiological model, plus identified risk factors	discuss the causes and maintaining factors
Point to the way out	review the effective treatments	outline the treatment

The two authoritative templates for delivering the diagnosis, aligned beat for beat; they are one non-confrontational method, with the Comprehensive Textbook of Psychiatry naming four discrete elements and Lishman foregrounding dissociation as the model given to the patient.

Two features of the shared skeleton are worth drawing out because they carry the therapeutic load. The first is that naming the condition is not optional politeness but an active ingredient: telling the patient the names of what they have, and that it is common, converts a frightening private experience into a recognised entity with a known course and a treatment. The second is the mechanism step. Offering a neurobiological model, a way of understanding how distress and dissociation can produce a physical event without conscious intent, gives the patient a face-saving and biologically respectable account that neither blames them nor mystifies the symptom. This is what makes the explanation non-accusatory in substance rather than only in tone.

MNEMONIC

The four R's

The Comprehensive Textbook of Psychiatry explanation in four beats. **R**eal: reassure the symptoms are genuine and not fabricated. **R**eached: explain how the diagnosis was made and give the condition its names. **R**eason: offer the hypothetical mechanisms and a neurobiological model, with any risk factors. **R**emedy: review the treatments that work. Each beat is something the framework actually states, and the order is deliberate because genuineness has to be settled before anything else is heard.

37.4 Withdrawing antiepileptic drugs safely

The drugs come off, but they come off carefully. Many of these patients arrive already taking one or more antiseizure medications, prescribed empirically over years for what was assumed to be refractory epilepsy. A logical consequence of the diagnosis is that treatment aimed at epilepsy is unnecessary and can be stopped, but this cannot be done abruptly. Patients must be cautioned that any **antiepileptic drug withdrawal** should be gradual and that the drugs should not be stopped suddenly. There are two reasons, and both should be made explicit in the consultation. First, the original diagnosis may not have been secure, and abrupt cessation risks precipitating genuine seizures or, at worst, status epilepticus if any true epileptic tendency is present or

coexists. Second, several antiseizure drugs carry their own withdrawal phenomena and are unsafe to discontinue quickly regardless of the indication. The tapering is therefore a medical decision made with the neurologist rather than a message to act on alone, and it is best framed to the patient as a planned, supervised reduction rather than as stopping useless tablets. Carers should be brought in early, with the patient's consent, both because they often witness the attacks and manage them and because they will be central to supporting the withdrawal and the psychological work that follows.

GOOD TO REMEMBER

When you tell a patient their events are not epilepsy, do not let them walk out and stop their antiseizure medication that evening. Spell out that the withdrawal will be gradual and supervised, arrange it with the neurologist, and involve a carer early with the patient's consent. The safe-taper message is part of the diagnostic conversation, not an afterthought to it.

37.5 Psychological therapy is the mainstay

The definitive treatment is a talking treatment. Once engagement is secured, the mainstay of treatment is psychological therapy, and the evidence base points most clearly to **cognitive behavioural therapy**. Kaplan and Sadock's Comprehensive Textbook of Psychiatry and Lishman's Organic Psychiatry both place structured psychotherapy at the centre of management, with randomised controlled trials, including multicentre trials, supporting cognitive behavioural therapy through improvements on measures of functioning and symptom severity. The therapy is not generic. The protocols used have been modelled on the cognitive behavioural approaches developed for panic disorder and for post-traumatic stress disorder, which is coherent with the phenomenology: the attacks often have the abrupt, fear-driven, dissociative quality of a panic or trauma response, and the therapeutic work addresses the triggers, the catastrophic appraisals and the avoidance that maintain them. The clinical message is that referral for a specific, evidence-based psychological therapy is the definitive step after diagnosis, not a vague onward disposal, and that the diagnosis should be delivered in a way that sets up and motivates that referral. Naming cognitive behavioural therapy as the treatment during the disclosure itself is one of the ways the explanation is made to point toward a way out rather than merely closing a door.

37.6 The limited and specific place of medication

Drugs treat the company the disorder keeps, not the disorder itself. The counterpart to the primacy of psychotherapy is a genuinely restricted role for pharmacology. There is no convincing evidence that pharmacological treatment reduces the frequency or severity of the attacks themselves, and so psychopharmacology should be reserved for treating comorbid psychiatric conditions rather than aimed at the seizures. Where depression, an anxiety disorder or post-traumatic stress disorder coexists, and comorbidity is common, treating it on its own merits is appropriate and may indirectly help; the psychiatric comorbidity and predisposing factors that accompany these attacks are set out in their own topic. But the drug is being given for the depression or the anxiety, not as an anticonvulsant-by-another-name for the non-epileptic attacks. This distinction is examinable precisely because it runs against the reflex to reach for a

tablet when a patient keeps having what look like fits, and because it protects the patient from an accumulating and useless polypharmacy layered on top of the antiseizure drugs that are already being withdrawn.

> **PITFALL**
>
> The instinct to control the attacks with medication, swapping one antiseizure drug for another or adding a sedative or an antipsychotic to suppress the events, is both futile and harmful. It has no evidence behind it for the attacks themselves, it reinforces the patient's belief that the problem is a drug-responsive brain disease, and it delays the psychological treatment that actually works. Prescribe only for a diagnosed comorbid condition, and be able to say why.

37.7 Stepped, multidisciplinary management

What actually has to be organised around the patient. The elements above assemble into a stepped, **multidisciplinary** model in which definitive care is shared across **two** clinical limbs that must stay coordinated. The relevant standards of care are the epilepsy guidelines of the International League Against Epilepsy and of NICE, which frame the medical management and the withdrawal of unnecessary treatment, together with the epilepsy-psychiatry reference literature, Lishman's Organic Psychiatry and Kaplan and Sadock's Comprehensive Textbook of Psychiatry, which frame the communication and the psychological treatment. Around those, the plan can be set out on the management spine.

The **LOCUS** of care is predominantly outpatient and community-based, because this is a disorder managed through a planned sequence of consultations and a course of therapy rather than through admission; the acute presentation of an attack may bring the patient to an emergency setting, but the emergency is not where the disorder is treated, and the task there is recognition and avoidance of iatrogenic harm rather than definitive care. The **focus** moves by phase: the acute target is the diagnostic conversation itself and safe handling of any attacks, the short-term target is securing engagement and beginning the supervised withdrawal of unnecessary antiseizure medication, and the mid-to-long-term target is the course of psychological therapy and the treatment of any comorbidity, with attention throughout to function, work and driving. The **modus** spans every treatment mode. The psychological mode is the definitive one and is described above. The pharmacological mode is deliberately narrow, confined to comorbidity. The medical and procedural mode belongs to the neurologist. The social mode runs through all of it: educating and enlisting carers with consent, and addressing the practical fallout for employment and driving that the diagnosis carries.

- The **neurologist** manages any comorbid neurological conditions, tapers the unnecessary antiseizure medication, and advises the patient on work and on driving.
- The **mental health clinician** delivers the definitive psychological treatment and manages comorbid psychiatric disorder.
- Both share responsibility for the consistent, non-confrontational message, since a contradiction between the two limbs is enough to undo the engagement the disclosure achieved.

IN INDIA

Several realities sharpen this in Indian practice. Stigma around the attacks is heavy, and families may read them as possession or as deliberate misbehaviour, so the non-confrontational, genuineness-first delivery matters even more, and a diagnosis heard as faking can damage marriage prospects and standing in ways that outlast the attacks. Trained cognitive behavioural therapists are scarce outside tertiary and metropolitan centres, which means the psychiatrist's own explanation carries a larger share of the therapeutic load and a longer follow-up may substitute for formal therapy where none is accessible. Because undiagnosed fits are frequently treated empirically with antiseizure drugs, often phenobarbital, many patients arrive on medication that must be withdrawn gradually and under supervision, making the safe-taper counselling a routine and important part of the consultation. Advice on driving and on manual or shift work has real economic weight and should be given concretely.

Four	**First-line**	**Comorbidity only**	**Two limbs**
ELEMENTS OF THE DIAGNOSTIC EXPLANATION	PSYCHOLOGICAL THERAPY, CHIEFLY CBT	THE LICENCE FOR MEDICATION	NEUROLOGIST AND MENTAL HEALTH CLINICIAN

The way the diagnosis is communicated is the pivotal, potentially therapeutic step: deliver it non-confrontationally and genuineness-first, taper unnecessary antiseizure drugs gradually rather than stopping them, make evidence-based psychological therapy the mainstay while reserving medication for comorbidity, and run the care as a coordinated collaboration between neurologist and mental health clinician.

38 · Coexisting PNES and epilepsy and prognosis of PNES

Two clinical problems fold into a single examination topic here. The first is what to do when a patient proves to have both epileptic and **dissociative seizures** at once, a combination that is neither rare nor academic; the second is what becomes of the patient with psychogenic non-epileptic seizures over the years that follow the diagnosis. Both are tested because both are routinely mishandled at the bedside. The coexistence is missed whenever a confirmed diagnosis of one seizure type is allowed to explain every event the patient has, and the outcome is misjudged whenever a falling seizure count is read as a recovering patient. This is the topic in which a candidate shows they can hold two diagnoses in one patient and can keep the fate of the seizures separate from the fate of the person.

Coexistence and prognosis are linked, not merely filed together. Comorbid epilepsy is itself one of the features that darkens the outlook of the dissociative seizures, so the patient who has both is, almost by definition, in the harder group. What belongs to neighbouring topics is deliberately left to them: the definition and classification of psychogenic non-epileptic seizures within the functional neurological disorder family, and the broader conversion construct developed in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes; the clinical features that separate a dissociative from an epileptic seizure; the video-EEG and other investigations that confirm the diagnosis; the psychiatric comorbidity and predisposing factors that underlie it; and

the way the diagnosis is delivered together with its stepped psychological and pharmacological management. What is owned here is the overlap with epilepsy and the long-term outcome.

38.1 How often the two coexist, in both directions

The overlap runs both ways, and at a similar rate. The single most useful fact to fix first is that epilepsy and dissociative seizures are not mutually exclusive diagnoses between which a clinician must choose, but conditions that frequently sit in the same patient. Kaplan and Sadock's Comprehensive Textbook of Psychiatry puts electrographically and clinically confirmed **comorbid epilepsy** at **about 10 to 20 per cent** of adults with psychogenic non-epileptic seizures, and adds that the proportion is higher still in children. Strikingly, the traffic runs the other way at much the same rate: a comparable 10 to 20 per cent of adults attending epilepsy services also have dissociative seizures. The symmetry is worth memorising, because in either clinic a substantial minority of patients carries the other diagnosis as well, and the label already in the notes is no guarantee that it accounts for the events now being described.

The direction matters because it changes what the clinician is prone to miss. In the epilepsy clinic, a patient with a secure diagnosis of epilepsy who begins to have atypical or treatment-resistant events invites the assumption that the epilepsy has worsened, when some of those events may be dissociative. In the seizure-disorder clinic the mirror error is to assume that a confirmed dissociative-seizure diagnosis rules out an epileptic fit, so that a genuinely epileptic event is waved away as psychogenic. Neither denominator, on its own, tells the clinician which mechanism produced the event in front of them. That has to be worked out event by event, which is why the coexisting group is treated as its own problem rather than as a straightforward instance of either disorder.

RULE **One seizure type never excludes the other.** A documented epilepsy does not mean that every event is epileptic, and a confirmed dissociative-seizure diagnosis does not mean the patient can never have an epileptic fit. In the coexisting patient each event must be assigned to its own mechanism on its own evidence, not deduced from the diagnosis already on file.

38.2 When the epilepsy comes first

An epileptic template that the dissociative seizures later imitate. In most patients the two conditions are simply concurrent, but in a minority the epilepsy is the older diagnosis and the dissociative seizures appear later. Lishman's Organic Psychiatry records that a pre-existing history of epilepsy precedes the onset of the dissociative seizures in **fewer than one in five** patients, a genuine minority but a clinically instructive one. The sequence matters because it hints at a mechanism. A person who has lived with epilepsy holds an intimate, embodied knowledge of what a seizure looks and feels like, and that experience can furnish the template from which a dissociative seizure is unconsciously constructed. The dissociative event is not feigned; it is modelled, without the patient's awareness, on a form they already know from the inside.

Two practical consequences follow. First, the patient with prior epilepsy whose new events do not quite match the old ones deserves careful re-appraisal rather than a reflexive increase in

medication, because the new events may be dissociative copies of the familiar fit. Second, the resemblance is precisely what makes these dissociative seizures hard to separate from the patient's own epileptic ones, since they are drawn from the same original. The discrimination of dissociative from epileptic events on their clinical features is developed in its own topic and is not repeated here; the point that belongs to this one is that a shared history gives the two event types a shared appearance, which is the deep reason the coexisting patient is so hard to read.

38.3 Why the coexisting patient is the hardest to treat

Two disorders in one patient, and a target that keeps moving. Lishman's Organic Psychiatry singles out patients with coexistent epilepsy as the most difficult group of all to manage, and the difficulty is diagnostic before it is therapeutic. Before any single event can be treated it has to be attributed to the right disorder, and in this group that attribution will not stay fixed. Showing the patient and their carers a recording of a captured attack is, in Lishman's account, invaluable, because it lets everyone see the seizure rather than argue about it; but the **semiology** of a given person's events changes over time, so a recording that settled the question a year ago may no longer describe the events happening now, and the **video-EEG telemetry** may have to be repeated. The clinician is therefore not making one diagnosis but maintaining two, and re-checking which is which as the picture shifts.

Management is guideline-anchored on the epilepsy side by the ILAE and NICE epilepsy guidelines, while the dissociative seizures draw on the epilepsy-specific psychiatric-management literature and Lishman's Organic Psychiatry; the way the diagnosis is communicated and the stepped psychological and pharmacological management of the dissociative seizures are set out in their own topic and are not reopened here. The **LOCUS** of care is principally outpatient, and for this group ideally a joint neurology and psychiatry clinic; the events themselves are best characterised on an inpatient monitoring unit when clinic assessment cannot resolve them, and the acutely misread attack may first present to the emergency department. The **focus** shifts by phase: the acute priority is safety and the avoidance of treating a dissociative event as status epilepticus; the short-to-mid-term work is the event-by-event sorting of epileptic from dissociative seizures; and the long-term aim is to keep both diagnoses simultaneously in view, so that the epilepsy is not undertreated while the dissociative seizures are not chased with ever-larger doses of medication. The **modus** spans every mode of care at once: antiepileptic optimisation for the epileptic seizures, the psychological treatment that belongs to the dissociative ones, and the shared work of showing patient and carers their own recorded events.

- Events have to be sorted one at a time, because no single investigation labels the whole patient as one disorder or the other.
- Telemetry often has to be repeated rather than done once, which is costly and not always available.
- Both disorders are managed in parallel, so undertreating the epilepsy and overtreating the dissociative seizures are simultaneous risks.

PITFALL

The commonest way to go wrong with the coexisting patient is to treat every event as refractory epilepsy. Dissociative seizures mistaken for worsening fits attract escalating antiepileptic doses and added second and third agents, and when an event is read as status epilepticus they draw benzodiazepine loading and intensive-care admission. The result is drug toxicity and iatrogenic harm inflicted for events that no antiepileptic could ever have stopped. When a patient with established epilepsy stops responding to sensible dose increases, ask whether some of the events are dissociative before reaching for the next drug.

IN INDIA

The tool Lishman leans on, repeated video-EEG telemetry, is concentrated in a handful of tertiary and academic centres and is out of reach for most patients, so in practice the dual diagnosis usually has to be built from careful serial history and witness accounts rather than confirmed on a monitoring unit. Add the long distances, the cost of prolonged monitoring and the stigma that keeps dissociative seizures from being named openly, and events are commonly mislabelled and mistreated for years before the coexistence is recognised. Because outcome turns heavily on how early the diagnosis is made, this delay is not a bureaucratic inconvenience but a direct prognostic loss.

38.4 The natural history: this does not simply pass

The prognosis is soberer than the reassurance implies. Once the diagnosis is secure and the frightening possibility of uncontrolled epilepsy excluded, it is tempting to treat psychogenic non-epileptic seizures as a benign problem that will resolve with explanation. The naturalistic outcome data do not support that optimism. In the cohort reported by Reuber and Elger in 2003, the seizures were still occurring beyond three years in about **two-thirds** of patients. For the majority, in other words, the disorder is not self-limiting; left to its own course after diagnosis it runs on for years. This is the fact that justifies active treatment rather than reassurance alone, and it is the counterpoint to the relief that so often follows the exclusion of epilepsy.

The persistence also reframes the goal. If most patients still have events years on, the meaningful questions are not whether the seizures will vanish of their own accord, for most they will not, but which patients do better and whether stopping the seizures is even the outcome that matters most to the patient's life. The long persistence is the background against which every prognostic factor should be read: the factors do not carry a patient from certain cure to certain failure, they shift the odds within a disorder that, untreated, tends to endure.

38.5 The single strongest predictor: duration of illness

Catch it early, or watch it entrench. Across the outcome literature, one variable does more work than any other. As Couprie and colleagues showed in 1995, and consistent with what is seen in other somatoform disorders, the single most important predictor of a good outcome in psychogenic non-epileptic seizures is a short **duration of illness** at the time the diagnosis is made. The longer a patient has been having dissociative seizures before anyone names them

correctly, the poorer the outlook, and the effect is strong enough that it outranks most of the other factors that intuitively seem important.

The reason is best understood as consolidation. A disorder caught soon after onset has not yet reorganised the patient's life around it: work, relationships, benefits and self-concept are not yet built upon the illness. Once those structures have formed, the seizures become load-bearing, and dismantling them is far harder than it would have been early on. This is why the strongest prognostic factor is also the most actionable one for the clinician: duration is the one variable that a prompt, confident diagnosis can still influence, whereas age at onset or the presence of comorbid epilepsy cannot be changed. The practical corollary is that delay is itself harmful, not merely a postponement of care, and that the effort spent reaching a firm diagnosis early is repaid directly in outcome. It also sharpens why the coexisting patient, in whom the diagnosis is genuinely hard and therefore often late, tends to do badly: the very difficulty of the diagnosis lengthens the illness before it is finally named.

GOOD TO REMEMBER

Of everything that shapes the outcome of dissociative seizures, the one lever still in the clinician's hand is the time to diagnosis. A patient whose events are correctly named within months of onset has a materially better prospect than one carried for years under the wrong label. Treat a confident, early diagnosis as a therapeutic act in its own right, not merely as a piece of administrative labelling.

38.6 The wider prognostic profile

Duration leads, but it does not stand alone. Around the dominant effect of illness duration sits a cluster of demographic, clinical and social factors that push the outcome one way or the other. Kaplan and Sadock's Comprehensive Textbook of Psychiatry groups them into a poor-prognosis set and a good-prognosis set, and the two are worth holding side by side, because they are in part mirror images of one underlying dimension: how entrenched, and how supported, the patient is. Which psychiatric comorbidities occur, and why they predispose to dissociative seizures, is the business of a neighbouring topic; what matters here is only that their presence worsens the outlook.

CORRELATES OF A POORER OUTCOME	CORRELATES OF A BETTER OUTCOME
comorbid epilepsy	higher educational attainment
comorbid psychiatric diagnoses	a shorter duration of illness
a dramatic seizure semiology	being in employment
a younger age at onset	**acceptance of the diagnosis** and social support

The poor-prognosis and good-prognosis correlates read as near mirror images: the more entrenched and less supported the patient, the worse the course; the better educated, employed, supported and accepting, the better.

Read across the two columns and one theme keeps returning. Comorbid epilepsy and a younger age at onset mark a more biologically and developmentally embedded problem; a dramatic semiology and a longer illness mark one more woven into the patient's identity and relationships;

while education, employment, social support and an accepting stance toward the diagnosis all describe a person with resources to draw on and a reason to recover. The list is not a scoring system but a way of forming a considered expectation, so the clinician neither promises a cure the data do not support nor writes off a supported, recently diagnosed patient with every reason to do well.

MNEMONIC

Better with EASE

The good-prognosis cluster in four beats. **E**ducation, the higher the better. **A**cceptance of the diagnosis by the patient. **S**upport, meaning social support around them. **E**mployment, being in work. Sitting above all four is the single strongest factor of all, a short duration of illness at the time of diagnosis, which the mnemonic keeps in reserve as the point to state first and forget last.

38.7 When the seizures stop but the patient does not recover

Seizure outcome and life outcome come apart. The sharpest and most examinable idea in the prognosis of dissociative seizures is that the two outcomes a clinician cares about do not move together. Kaplan and Sadock's Comprehensive Textbook of Psychiatry, echoed by Lishman's Organic Psychiatry, records that participation in psychotherapy is associated with an improvement in seizure frequency in the **one to two years** after diagnosis; the seizures, in other words, can be treated and often do get better. And yet the rates of disability do not fall over the same years, and even among patients whose seizures remit, many remain out of work and dependent on state support. The **functional outcome** lags behind, and sometimes never catches up with, the seizure outcome.

This dissociation carries a hard clinical lesson. The seizures are, in one sense, only the surface of the disorder; beneath them lie the psychological and social difficulties that generated and maintained them, and those do not dissolve merely because the visible events have stopped. A treatment plan that measures success by seizure counts alone will record a victory while the patient's life stays as constrained as before. It follows that stopping the seizures, though a real and worthwhile goal, is not the same as restoring the person, and that the rehabilitation of function, meaning work, relationships and a way out of the sick role, has to be pursued as a target in its own right rather than expected to follow once the events cease.

TRAP **Reading seizure remission as recovery.** Candidates and clinicians alike equate a fall in seizure frequency with a cure, and so predict that the patient whose events have stopped will resume an ordinary life. The outcome data say otherwise: disability persists and many remain unemployed and dependent even after the seizures have remitted. State the dissociation explicitly, that the seizures can improve while function does not, and the mark, like the patient, is secured.

10 to 20% EPILEPSY COEXISTING WITH PNES IN ADULTS	**under 20%** EPILEPSY PRECEDES THE DISSOCIATIVE SEIZURES	**two-thirds** STILL FITTING BEYOND THREE YEARS	**1 to 2 y** SEIZURE GAINS AFTER PSYCHOTHERAPY

Epilepsy and dissociative seizures coexist in a substantial minority of patients in either direction, and this dual group is the hardest to treat because each event must be assigned to its own mechanism against a semiology that drifts; the disorder usually persists for years, the strongest predictor of a good outcome is a short duration of illness at diagnosis, and seizure remission must never be mistaken for functional recovery.

Antiepileptic drugs: the psychiatric interface

39 · AEDs used as mood stabilisers: valproate, carbamazepine, lamotrigine indications and evidence

Three molecules, two clinics. Valproate, carbamazepine and lamotrigine are the drugs a candidate meets first in the epilepsy clinic and again in the mood clinic, and the examiner's favourite manoeuvre is to see whether the two encounters have been kept apart. This topic takes the antiepileptic lens on purpose. It asks which of these agents is chosen for which kind of seizure, what the controlled evidence actually says about that choice, and which adverse-effect signatures they share when they are used as anticonvulsants. The **AEDs used as mood stabilisers**, with their bipolar indications and evidence - why a psychiatrist reaches for them in bipolar illness, how their plasma levels are followed, what they do to the ovary in that setting - are set out in full in the Mood disorders: pharmacotherapy notes and are not rehearsed here. The systematic, class-level account of the anticonvulsants as psychotropic agents belongs to the **systematic psychopharmacology notes**, which in turn defer back to this chapter for the seizure-selection material.

Why should a psychiatrist care how a neurologist picks an antiepileptic drug? Because almost every decision that follows rides on that choice. The agent already on the prescription decides which psychotropics can be added without a pharmacokinetic collision, it colours mood, behaviour and cognition in ways that are easy to misattribute to the epilepsy or to a primary psychiatric disorder, and in a woman of childbearing potential it forces a conversation about pregnancy long before any pregnancy is planned. Reading the logic backwards, from the drug in hand to the seizure type it implies, is one of the more reliably rewarded skills in this part of the paper, and it starts from a single rule about how the choice is made.

39.1 Selection is anchored to the seizure type

The organising principle of antiepileptic prescribing is that the drug is matched to the seizure type and syndrome, not to the patient's psychiatric label. The framework that underwrites this, the grouping of seizures into focal, generalised and unknown onset, is the ILAE seizure classification developed in this chapter's account of how seizures are classified; the selection rules simply hang off it. The practical division a candidate must carry is between **broad-spectrum** agents, which suppress several seizure types at once, and narrow-spectrum agents, which are effective against one mechanism and may be useless, or even aggravating, against another. A broad-spectrum drug is the safe default when the syndrome is uncertain or when more than one

seizure type coexists in the same patient; a narrow-spectrum drug is a precision instrument that demands a confident diagnosis before it is deployed.

SEIZURE TYPE	FIRST-LINE ANTIEPILEPTIC	WHY IT IS CHOSEN
Focal (localisation-related epilepsy)	**carbamazepine**, with **lamotrigine** as a co-first-line option	Long-established control of focal and secondarily generalised seizures; lamotrigine holds equivalent first-line standing with a different tolerability profile
Generalised tonic-clonic seizures	**valproate**	Broad-spectrum cover across the generalised seizure types makes it the drug of choice for generalised epilepsy
Absence seizures	**ethosuximide**	Highly effective for absence, but narrow in spectrum, so it is an alternative rather than a universal generalised-epilepsy drug

First-line choice reads off the seizure type: the same three principal drugs, sorted by the kind of seizure each one is best matched to.

MNEMONIC

Focal - CBZ/LTG; Generalised - VPA; Absence - ESM

Read the spine as a coverage ladder rather than a list. Carbamazepine, with lamotrigine alongside it, answers focal onset; valproate answers the generalised syndrome by covering it broadly; and ethosuximide answers pure absence. Hold the three pairings and you can reconstruct the first move in almost any seizure-selection question without reaching for anything the fragment has not already stated.

Only once the seizure type has fixed the shortlist do the second-order considerations come into play, and these are what separate two otherwise seizure-appropriate agents from one another:

- tolerability and the everyday side-effect burden the patient has to live with;
- the pharmacokinetic interaction load the drug imposes on co-prescribed psychotropics;
- its effect on mood, behaviour and cognition;
- reproductive constraints in a woman of childbearing potential.

RULE **Match the drug to the seizure type first; let the psychiatric picture fine-tune, never overturn, that choice.** The seizure type sets the shortlist of agents that will actually control the epilepsy, and only within that shortlist do the second-order factors above decide between the candidates. A drug chosen for its psychiatric convenience but mismatched to the seizure type protects nobody, because uncontrolled seizures are the primary harm the prescription exists to prevent.

39.2 Focal epilepsy: carbamazepine, with lamotrigine alongside

Focal epilepsy, the commonest pattern of adult-onset seizures, is the setting in which carbamazepine earned its reputation as a drug of first choice, and it remains a reference agent against which newer drugs are measured. Its strength is dependable suppression of focal seizures and of the secondarily generalised convulsions that arise from them. What complicates its use is not efficacy but everything that surrounds it: it is a potent modifier of hepatic metabolism, and the pharmacokinetic interactions between antiepileptics and psychotropics that this creates, through enzyme induction and inhibition, are the province of a dedicated topic in this chapter and are only named here. For a psychiatrist, the practical upshot is that a patient arriving already on carbamazepine comes with a built-in constraint on what can be safely added.

Lamotrigine sits beside it as a genuine co-first-line agent for focal onset, chosen not because it controls seizures more forcefully but because it is generally the gentler drug to live with, better tolerated across mood and cognition in many patients. That trade-off, robust seizure control from the older agent against tolerability from the newer one, is exactly what the controlled evidence set out to quantify. Lamotrigine's own mechanism, its obligatory slow titration and the rash that titration is designed to avoid are taught where lamotrigine is covered in its own right; the point that belongs here is simply its standing as a first-line choice for focal epilepsy, level with carbamazepine on the selection map.

39.3 Generalised epilepsy: valproate and the value of breadth

Where the epilepsy is generalised, valproate is the drug of choice, and the reason is written into the word broad-spectrum. A single agent that suppresses generalised tonic-clonic seizures while also covering absence and myoclonic seizures is worth a great deal in the idiopathic generalised epilepsies, where more than one seizure type frequently coexists in the same patient and where a narrow drug aimed at only one of them would leave the others unguarded. Breadth also buys tolerance of diagnostic uncertainty: when the precise syndrome is not yet settled, a broad-spectrum drug is far less likely to aggravate an unrecognised seizure type than a precision agent chosen on an incomplete diagnosis.

That advantage is why valproate has been so hard to displace despite the difficulties attached to it, and it sets up the central tension of the whole topic, because the population with idiopathic generalised epilepsy skews young and includes many women for whom valproate is the very drug that reproductive considerations argue against. The evidence and the dilemma that flow from this are taken up in the sections that follow; here the point to fix is the pharmacology, that valproate's breadth is a real clinical asset and not merely a default. A broad-spectrum agent earns its place precisely in the patient whose seizures do not fit a single tidy category.

39.4 Absence seizures and the narrow-spectrum trap

Absence seizures are the clearest illustration of what narrow spectrum means at the bedside. Ethosuximide is a first-line drug for absence and a highly effective one, but its usefulness stops at the edge of that single seizure type. It is the textbook example of a precision agent: excellent for the target it is designed for, and no help at all outside it. The clinical consequence follows directly. In a patient whose generalised epilepsy comprises absence alone, ethosuximide is an entirely

rational and often preferable choice. In a patient who has absence together with generalised tonic-clonic seizures, the same drug controls only half the problem, and the convulsive seizures it does not touch are the more dangerous half.

When absence and convulsive seizures coexist, the broad-spectrum agent that covers both is usually the sounder single-drug choice, and ethosuximide is reserved for the pure absence picture or added to a regimen that already protects against convulsions. The exam reward here is not the name of the drug but the recognition that spectrum, not potency, decides whether a given agent is adequate as sole therapy for a given patient.

PITFALL

The recurring clinical error is to treat ethosuximide as a general-purpose generalised-epilepsy drug because it is filed under generalised seizures. It is effective for absence but does not cover generalised tonic-clonic seizures, so a patient who has both, prescribed ethosuximide alone, is left unprotected against the convulsive seizures that carry the greater risk of injury. Before ethosuximide is chosen as monotherapy, the question is always whether any convulsive seizures coexist; if they do, a broad-spectrum agent, not a narrow one, is the safer spine of the regimen.

39.5 The controlled evidence for AED selection

Selection rules are only as good as the trials behind them, and the reference study for these first-line choices is the large pragmatic comparison known as **SANAD**, run in the United Kingdom to test standard against newer antiepileptic drugs in the seizure types where a clinician genuinely hesitates. In the arm addressing focal epilepsy, carbamazepine performed well on control of the epilepsy itself, showing a better time to a sustained **12-month remission**, although that particular advantage reached statistical significance only against gabapentin among the comparators. Lamotrigine, by contrast, did better on a different and arguably more clinically honest endpoint, achieving a longer time to treatment failure.

The distinction between those two endpoints is the whole lesson of the trial and worth spelling out. Time to remission asks only whether the drug stops seizures; time to treatment failure is a composite that captures both whether the drug works and whether the patient can tolerate staying on it, since a drug abandoned for side effects has failed just as surely as one that never controlled the seizures. Lamotrigine's edge on that composite, together with its being judged cost-effective and well tolerated, is precisely why it rose from a newer alternative to a co-first-line agent standing level with carbamazepine. The examinable message is that the older drug can look stronger on raw seizure control while the newer drug wins on the measure that combines efficacy with tolerability, and real prescribing lives in that gap.

39.6 Valproate in women of childbearing potential

The single most important qualification to everything said about valproate is that its status as drug of choice for generalised epilepsy does not survive intact in women of childbearing potential. Its cosmetic and its reproductive effects together create a genuine treatment dilemma: the drug that best controls the seizures is also the drug that a woman who might become pregnant has the strongest reasons to avoid. This is not a small caveat; it actively drives selection

toward alternatives across a large and clinically common group of patients, sometimes at a real cost to seizure control. The full account of valproate's teratogenicity, the formal contraception requirements it now carries and the detail of prescribing it in women is the subject of a dedicated topic in this chapter and is not pre-empted here; what belongs to selection is the dilemma itself and the way it reshapes the first-line choice.

IN INDIA

The dilemma bites harder in Indian practice, and for structural reasons rather than clinical ones. Valproate and carbamazepine are cheap, familiar and widely stocked, so they are often the pragmatic first reach for generalised epilepsy, while the newer agents that would sidestep the reproductive problem can be costlier or less reliably available outside larger centres. The counselling and contraception apparatus that is meant to accompany valproate in a woman of childbearing potential, set out in the topic on prescribing valproate in women, is unevenly implemented, and many women are never told that their antiepileptic carries reproductive implications at all. The workable stance is to raise the question of pregnancy at the point of prescribing, not years later, and to make the selection with that conversation already in view rather than as an afterthought.

39.7 The idiosyncratic reactions that shape selection

Every one of these drugs can turn on the skin. A shared adverse-effect signature runs across the aromatic anticonvulsants and it bears directly on how they are chosen and monitored. Carbamazepine, phenytoin and phenobarbital, and more commonly lamotrigine, all carry a risk of an idiosyncratic allergic rash, occurring in **about 2 to 4 per cent** of patients on the drug and appearing characteristically early in treatment rather than after long exposure. Most such rashes are benign and settle on withdrawal, but the same agents are the ones implicated when a drug eruption escalates into its dangerous forms, **Stevens-Johnson syndrome** and **Lyell's syndrome**, the latter also known as toxic epidermal necrolysis. Because the reaction is a class phenomenon, a serious rash on one aromatic agent casts a shadow of cross-reactivity over the others, which is itself a selection constraint when a substitute has to be found.

For selection and monitoring the implications are concrete. The higher rash risk attached to lamotrigine is the reason its introduction is governed by the slow-titration rule taught where that drug is covered in its own right; here the point is the class effect and its early timing. Any patient starting one of these drugs is warned to report a rash at once, is reviewed while the risk window is still open, and has the drug stopped rather than continued through an evolving eruption.

2 to 4%	12 months
ALLERGIC RASH, EARLY ON THE AROMATIC AEDS	SANAD SUSTAINED-REMISSION ENDPOINT

GOOD TO REMEMBER

Treat any new rash in the first weeks of an aromatic antiepileptic as a stop-and-review event, not a wait-and-see one. The benign rashes and the earliest stages of the dangerous ones look alike at onset, and the safe habit is to assume the worst until the drug is off and the patient reviewed. It costs little to stop and re-choose within the seizure-appropriate shortlist; it costs a great deal to continue through the rash that turns out to be the start of Stevens-Johnson syndrome.

39.8 Choosing, switching and sharing the prescription

Antiepileptic selection is a management problem, and it is best organised the way any management problem is. The standards that anchor it are the epilepsy guidelines of the International League Against Epilepsy (ILAE) and the National Institute for Health and Care Excellence (NICE), which govern first-line choice by seizure type, together with the epilepsy-specific psychiatric-management literature that addresses the mood, behavioural and reproductive considerations layered on top of the seizure diagnosis.

The **LOCUS** of this care is overwhelmingly the outpatient interface between neurology and psychiatry, where drugs are started, cross-titrated and swapped, and where the real hazard is that two prescribers do not see each other's changes; it moves inpatient when an unstable epilepsy or a complex switch is safest under observation, and to the emergency setting when a blistering rash or a serious reaction has already declared itself. The **focus** shifts by phase: in the acute phase the target is simply matching a seizure-appropriate agent to the confirmed seizure type; across the medium term it is tolerability and the emerging mood, behavioural and cognitive footprint, which are the concern of the topics on individual-AED psychiatric side effects and on AED-attributable mood change; and in the long term it is the patient whose circumstances change, above all the woman moving toward pregnancy, for whom the selection made years earlier must be revisited. The **modus** spans every lever available: the pharmacological lever of first-line choice and rational switching within the seizure-appropriate shortlist, with the interaction pharmacokinetics, lamotrigine's titration and valproate's use in women each handled by their own topics; the procedural options for drug-resistant epilepsy, whose psychiatric consequences are covered under outcomes after epilepsy surgery; and the social and educational levers of adherence support, driving and employment counselling, and honest communication between prescribers so that a change on one chart is known to the other.

TRAP **Importing the mood-stabiliser evidence to justify the antiepileptic choice.** Because these three drugs are also mood stabilisers, candidates reach for the bipolar evidence to defend a seizure-selection decision, or the reverse. The two literatures are separate: a drug's standing in bipolar illness, developed in the Mood disorders: pharmacotherapy notes, says nothing about whether it matches a given seizure type, and the antiepileptic evidence says nothing about mood prophylaxis. Keep the streams apart and cross-refer them cleanly; conflating them is the error the paired framing is designed to expose.

Antiepileptic choice follows the seizure type, not the psychiatric label: carbamazepine or lamotrigine for focal epilepsy, valproate for generalised epilepsy, ethosuximide for absence alone, with the mood-stabiliser role of these same drugs a separate lens taught elsewhere.

40 · Psychiatric side effects of individual AEDs (levetiracetam, topiramate, phenobarbital, vigabatrin and others)

Every antiepileptic drug that quietens the cortex can also unsettle the mind, and the pattern of that disturbance is rarely random. This topic is the per-agent map of psychiatric adverse effects: which individual antiepileptic drug produces which behavioural or affective syndrome, in whom, and how often. The marks are won in the vignette where a named drug is started and a named psychiatric picture follows, the previously equable adult who turns explosively short-tempered, the child transformed into an agitated whirlwind, the patient who develops a frank psychosis within weeks of a new prescription. The examiner wants the drug identified, its signature recognised, and that recognition converted into the right clinical move. The presiding fact, worth stating at the outset, is that the offending molecule is usually the single part of the whole neuropsychiatry of epilepsy that the clinician put there and can simply take away.

Two boundaries keep this topic honest. The shared attribution problem, separating a drug effect from the disease, ranking the agents by cognitive load and holding the cognitive map apart from the behavioural one, belongs to the cognitive and behavioural adverse effects of the antiepileptic drugs, taught as their own topic in this chapter; it is invoked here, not rebuilt. The class-wide suicidality caution attached to the antiepileptic drugs as a group, with its pooled-trial evidence and regulatory history, is likewise a separate topic and is named here only to be set against the agent-specific signal this fragment genuinely owns. The mood-stabiliser reading of valproate, carbamazepine and lamotrigine, their bipolar indications, mechanism of mood action and monitoring, lives in the Mood disorders: pharmacotherapy notes and is not reopened. What remains, and what is ours, is the individual drug and the particular way it disturbs thought, mood and behaviour.

40.1 Why each agent carries its own psychiatric signature

The phenotype tends to follow the pharmacology. An antiepileptic drug does not disturb behaviour at random; the flavour of the disturbance is shaped by how the molecule alters cortical and limbic signalling. Agents that push hard on inhibitory tone can blunt affect and, paradoxically, disinhibit behaviour; agents that touch glutamatergic excitation can drive irritability and aggression; agents with a broad, multi-system pharmacology tend to produce a diffuse mix of slowing and low mood. Some of these mechanisms are firmly established for the individual drugs and worth stating precisely: **vigabatrin** acts as an irreversible GABA-transaminase inhibitor, **perampanel** is a selective AMPA-receptor antagonist, and **topiramate** is, among its several actions, a weak carbonic anhydrase inhibitor. The point of the frame is not to memorise receptor targets but to make the psychiatric signature predictable, so a clinician can reason from the drug to the symptom rather than be surprised by it.

Across the class these reactions share a common grammar more useful than any single drug fact: they appear early, are frequently dose related, and are above all reversible, settling when the offending agent is reduced or withdrawn. That grammar separates a drug reaction from the disease-driven psychopathology of epilepsy and gives the clinician a lever the underlying condition rarely offers.

RULE **A new psychiatric symptom in a treated epilepsy patient is drug-attributable until the timeline says otherwise.** Antiepileptic psychiatric adverse effects are typically early, dose related and reversible, so they are testable in a way the disease is not: map the symptom against every recent start and dose change, and if it tracks the drug, the most powerful intervention is usually to the prescription rather than a tranquilliser laid on top of it.

40.2 Levetiracetam: irritability, aggression and behavioural activation

The behavioural agent every candidate must know. Of all the modern antiepileptic drugs, **levetiracetam** is the one most reliably tied to emergent behavioural trouble, a cluster informally known as levetiracetam rage that spans **aggression**, agitation, anger, anxiety, apathy, depressed mood, emotional lability, hostility and irritability. In adults treated for epilepsy, these behavioural adverse effects were reported in **13.3% against 6.2% on placebo**, roughly a doubling over background, as documented in Kaplan and Sadock's Comprehensive Textbook of Psychiatry. They characteristically arise early, often within the first month of treatment, which is exactly when the drug timeline is easiest to reconstruct. Most cases are irritability and a shortened fuse rather than dangerous behaviour, but the reaction is real enough that it drove discontinuation for behavioural reasons in 1.7% of adults, and suicide attempts, though rare, were recorded in 0.5% against none on placebo. The teaching point is one of attribution: this is a drug-attributable phenomenon, mechanistically and clinically distinct from the disease-intrinsic aggression, irritability and episodic dyscontrol of epilepsy that is taught elsewhere in this chapter, and the two demand opposite responses, one aimed at the prescription and the other at the illness.

That distinction has therapeutic teeth. When irritability or overt aggression emerges in step with starting or up-titrating levetiracetam, the rational move is to reconsider the agent, not to reach reflexively for an antipsychotic to sedate it, because the disturbance frequently settles once the drug is lowered or replaced. The recognised amplifier is a personal history of mood or behavioural disorder; the recognised mitigation is a slow introduction, the same total dose reached gradually being far better tolerated than the dose reached in a hurry.

40.3 Levetiracetam psychosis and the paediatric amplification

Rarer psychosis, and a much louder problem in children. Beyond the behavioural cluster sits a smaller but harder-edged signal. Levetiracetam-associated psychosis occurred in **0.7% of treated adults against 0.2% on placebo**, an uncommon reaction but a genuine one, and an emergent psychosis within weeks of starting the drug should raise it as a first hypothesis rather than being ascribed reflexively to the epilepsy. What matters clinically is that a drug-induced psychosis of this kind is expected to remit on withdrawal, which sets it apart from the enduring interictal psychoses of epilepsy that are taught as their own topics in this chapter.

The behavioural liability is markedly worse in children, which is the fact most often examined and most often forgotten. In paediatric epilepsy the behavioural adverse effects, spanning agitation, depression, emotional lability, hostility and hyperkinesia, were reported in **37.6% against 18.6% on placebo**, roughly triple the adult figure, and the reaction was severe enough to force discontinuation in 10.9% of children. A drug merely irritating in an adult can therefore be genuinely disabling in a child, warranting a frank warning to the family and a lower threshold for switching. The accompanying figure sets the adult and paediatric burdens side by side, and the contrast is the whole lesson.

GOOD TO REMEMBER

Prescribe the behavioural offenders defensively from the first tablet. Start low and climb slowly, name the likely reaction to the patient and one family member in advance so it is reported early rather than silently endured, and set an explicit review point in the first few weeks when the risk is highest. In children the warning is not optional: the behavioural burden is far heavier than in adults, and a forewarned family reports the change that an unwarned family mislabels as naughtiness or as the illness.

40.4 Topiramate: cognitive slowing, low mood and rare psychosis

A distinctive drag on cognition, with a mood cost behind it. Topiramate is unusual in appearing on both the cognitive and the behavioural registers at once. Its best-known effect is neuropsychological: a psychomotor and cognitive slowing together with a characteristic word-finding and memory difficulty that patients describe vividly as the sentence that stalls and the ordinary noun that will not surface. That cognitive signature is developed in full as part of the cognitive and behavioural adverse effects of the antiepileptic drugs, taught as its own topic in this chapter; what concerns us here is topiramate's specifically psychiatric liability, which is real if less famous. Alongside the slowing, topiramate can produce depression and mood problems, and, less commonly, a frank psychosis, as tabulated in Kaplan and Sadock's Comprehensive Textbook of Psychiatry. The broad, multi-target pharmacology of the drug is the usual explanation for why cognition, language and mood are all touched rather than any one domain in isolation.

The practical consequence is a low threshold of suspicion: a patient who becomes slow, inarticulate and low in mood after topiramate is introduced is usually showing the drug, not a new primary depressive illness. Because that low mood is easily attributed to the burden of epilepsy itself, the discipline is to check the drug timeline before reaching for an antidepressant, since lowering or replacing the topiramate may resolve the picture without a second agent.

40.5 Phenobarbital: depression, irritability and the paradoxically hyperactive child

The old drug with a young patient's paradox. **Phenobarbital**, one of the oldest agents still in wide use, carries a psychiatric profile that spans depression, irritability, aggression, impaired cognition and attention and sedation, so that a dulled, low, short-tempered patient on long-standing phenobarbital is a common and correctable sight. The examinable twist is age-dependent. Where the drug sedates the adult, in children it characteristically produces the opposite, a **paradoxical hyperactivity** and behavioural activation, so that the child becomes

restless, overactive and hard to manage rather than drowsy. This paradoxical reaction is a classic teaching point precisely because it inverts the expected effect and is so readily misread. The mechanism is not the point the examiner tests; the recognition is.

IN INDIA

Phenobarbital remains a common first-line antiepileptic in children across resource-limited Indian practice, kept there by its low cost, once-daily dosing and secure supply. That makes its paradoxical behavioural activation a live problem rather than a museum fact. A previously settled child who turns overactive, inattentive and irritable after starting phenobarbital is often mislabelled as naughty, as having attention-deficit hyperactivity disorder, or as expressing the epilepsy, and is disciplined or medicated for it while the true cause sits in the drug chart. Ask when the behaviour began relative to the prescription; where it tracks the phenobarbital, revisit the agent, and where an affordable alternative exists the behavioural cost is a legitimate reason to move.

40.6 Vigabatrin: precipitated psychosis and irreversible visual-field loss

Two liabilities, and one of them you cannot take back. Vigabatrin sits among the agents most likely to precipitate psychiatric decompensation, and it precipitated psychotic and affective symptoms in between 3% and 10% of patients, a reaction made considerably more likely by a significant past psychiatric history, as set out in the Companion to Psychiatric Studies. That prior history is the screening question, since it marks the patient in whom vigabatrin most readily unmasks a psychosis. The drug's second and defining liability is not psychiatric at all but limits its use so severely that no account of it is complete without it. Vigabatrin causes a progressive, bilateral **visual field constriction** that is irreversible, and it is this vision loss, more than any behavioural effect, that has pushed its use to the margins, so that it is now largely restricted to infantile spasms, particularly those of tuberous sclerosis, as described in Goodman and Gilman's Pharmacological Basis of Therapeutics.

MNEMONIC

Vigabatrin: two things you cannot reverse

The drug irreversibly blocks GABA-transaminase and it irreversibly narrows the visual fields, and it can precipitate a psychosis on top, most readily in the patient with a psychiatric past. Hold the pairing of an irreversible enzyme action and an irreversible field defect and both halves of why the agent is used sparingly fall into place.

PITFALL

The avoidable harm is to keep a patient on vigabatrin for its undoubted efficacy while never arranging or repeating visual field assessment, so that peripheral vision is quietly and permanently lost before anyone notices. The field defect is asymptomatic until it is advanced, and it does not recover once established, which is exactly why baseline and periodic perimetry, not a wait for the patient to complain, is the standard of care wherever the drug is used.

40.7 Perampanel, zonisamide, gabapentin and the wider field

The signatures continue past the headline drugs. Perampanel earns particular attention because its behavioural liability is cleanly dose dependent, which makes it both predictable and manageable. Hostility and aggression reactions rose with dose, reported in about **12% at the 8 mg daily dose and 20% at 12 mg daily, against 6% on placebo**, generally emerging within the first six weeks of treatment, and the drug carries a boxed warning for serious neuropsychiatric reactions, as documented in Kaplan and Sadock's Comprehensive Textbook of Psychiatry. That boxed warning is agent-specific and is a different regulatory object from the class-wide suicidality caution named earlier; here it flags this drug's own propensity to hostility and aggression. Because the effect climbs with the dose, the management is written into the prescription: keep the dose as low as seizure control allows. **Zonisamide** carries its own mixed neuropsychiatric profile of agitation, depression, psychosis, impaired cognition and somnolence, a reminder that the sulphonamide-related agents are not behaviourally silent. **Gabapentin**, often thought of as benign, shows a behavioural signal in children, with hostility and aggressiveness in 5.2% against 1.3% on placebo and emotional lability or behavioural problems in 6% against 1.3%, per its prescribing information, so that even the gentler agents earn a caveat in the paediatric setting.

AGENT	SIGNATURE PSYCHIATRIC ADVERSE EFFECT	PRACTICAL NOTE
Levetiracetam	irritability, aggression and emotional lability; rarer psychosis	worst in children; early onset
Topiramate	depression and mood change; rare psychosis, alongside cognitive slowing	slow titration mitigates
Phenobarbital	depression and irritability; paradoxical hyperactivity in children	the paradox inverts by age
Vigabatrin	precipitated psychosis and affective symptoms	worse with a psychiatric past; watch the visual fields
Perampanel	dose-dependent hostility and aggression	boxed warning; keep the dose low
Zonisamide	agitation, depression, psychosis, cognitive impairment, somnolence	a mixed profile
Gabapentin	hostility and behavioural problems in children	less benign than assumed in the young

The individual-agent psychiatric signatures owned here. The point of the table is discrimination: the drug most associated with rage is not the one most associated with cognitive slowing, and the one whose worst liability is not psychiatric at all sits in the middle.

40.8 The agent-specific suicidality signal and managing the reactions

Not every antiepileptic carries the same self-harm risk, and that is the point. Set against the flat, class-wide suicidality caution taught under its own topic in this chapter, there is a sharper, agent-specific finding that belongs here. A British case-control analysis found that the antiepileptic drugs whose clinical-trial depression rate exceeded 1% carried an increased self-

harm liability, and for those agents the risk of self-harm or suicidal behaviour was raised roughly **three-fold**, with no such association for the remaining drugs, as reported by Andersohn and colleagues in 2010 and summarised in Kaufman's Clinical Neurology for Psychiatrists. The high-depression-risk agents that carried the signal were:

- levetiracetam;
- tiagabine;
- topiramate;
- vigabatrin.

The discrimination is the mark: the self-harm risk is not spread evenly across the class but concentrates in the agents most prone to lower mood, which is exactly what one would predict and which the class-level caution alone would obscure.

Managing these reactions is the most rewarding work in the neuropsychiatry of epilepsy, because the cause is one the clinician can adjust. The relevant standards are the epilepsy guidelines of the International League Against Epilepsy and of NICE for the seizure disorder itself, read alongside the epilepsy-specific psychiatric-management literature for the behavioural dimension. The **LOCUS** of care is overwhelmingly the outpatient clinic, where recognition, dose review and rationalisation proceed across visits; only a severe reaction, a frank drug-induced psychosis or emergent suicidality with a safety concern, briefly pushes care toward an emergency or inpatient setting. The focus shifts by phase. The acute task is recognition and attribution, placing the symptom on the drug timeline; the short-to-mid-term task is to act on the offending agent by lowering the dose, slowing an over-rapid titration, or switching to a behaviourally kinder drug; and the long-term task is to keep the regimen lean and to monitor mood and behaviour over time, and for vigabatrin to keep the visual fields under review. The modus spans every mode: dose reduction and substitution pharmacologically, with simple restraint in the target dose for dose-dependent offenders such as perampanel; and around these the psychological and social work of forewarning patients and families, psychoeducating households that mislabel a drug effect as character or illness, and attending to the schooling, work and driving consequences of behavioural change. The choice of an antidepressant where genuine depression persists, weighed against its effect on the seizure threshold, is handled in its own dedicated topic in this chapter.

TRAP **Reading a drug-induced psychosis as the epilepsy's own psychosis.** When a patient becomes psychotic within weeks of starting levetiracetam or vigabatrin, the tempting error is to file it as an interictal psychosis of epilepsy or as forced normalisation and to add an antipsychotic, leaving the true cause in place. The tell is the timeline: a drug-induced psychosis is time-locked to the prescription and expected to remit on withdrawal, whereas the chronic psychoses of epilepsy are not. Account for the drug before you diagnose the disease.

Levetiracetam	Vigabatrin	Perampanel	Reversible
IRRITABILITY AND RAGE	PSYCHOSIS PLUS VISION LOSS	DOSE-DEPENDENT HOSTILITY	RECOGNISE AND REDUCE

Each antiepileptic drug has its own psychiatric signature: levetiracetam drives irritability and aggression, with florid behavioural activation and rarer psychosis especially in children; topiramate brings cognitive slowing with low mood; phenobarbital sedates adults but paradoxically activates children; vigabatrin precipitates psychosis and, if missed, irreversible visual-field loss; and perampanel produces dose-dependent hostility under a boxed warning. Because every one of these reactions is early, dose related and reversible, the decisive intervention is almost always to the prescription, not a sedative laid on top of it.

41 · AED-psychotropic pharmacokinetic interactions (enzyme induction and inhibition)

Two drugs, one liver, and a level that will not sit still. This topic earns marks out of proportion to its apparent dryness because it is where the neurologist's prescription and the psychiatrist's prescription silently collide. The older antiepileptic drugs are among the most powerful modifiers of hepatic drug metabolism in the whole formulary, and the psychotropics a patient with epilepsy so often also needs are cleared down exactly the pathways those antiepileptics disturb. Get the direction of the interaction wrong and a stabilised patient relapses on an unchanged dose, or a quietly accumulating drug climbs into toxicity, with nothing in the prescription itself to show what happened. The examinable core is not a list of pairings to be memorised but a single organising question asked of every co-prescription: does this antiepileptic induce the enzymes that clear its partner, or inhibit them, and therefore does the partner's level fall or rise?

Several of these same drugs are also used as mood stabilisers, a role developed in the Mood disorders: pharmacotherapy notes; here the lens is purely pharmacokinetic, concerned with what one drug does to another's concentration and not with what either does to mood. Their own psychiatric side effects, likewise, are a separate topic in this chapter. The interaction runs in both directions and reaches well beyond psychiatry, so the safe prescriber holds the mechanism rather than a memorised table of pairs.

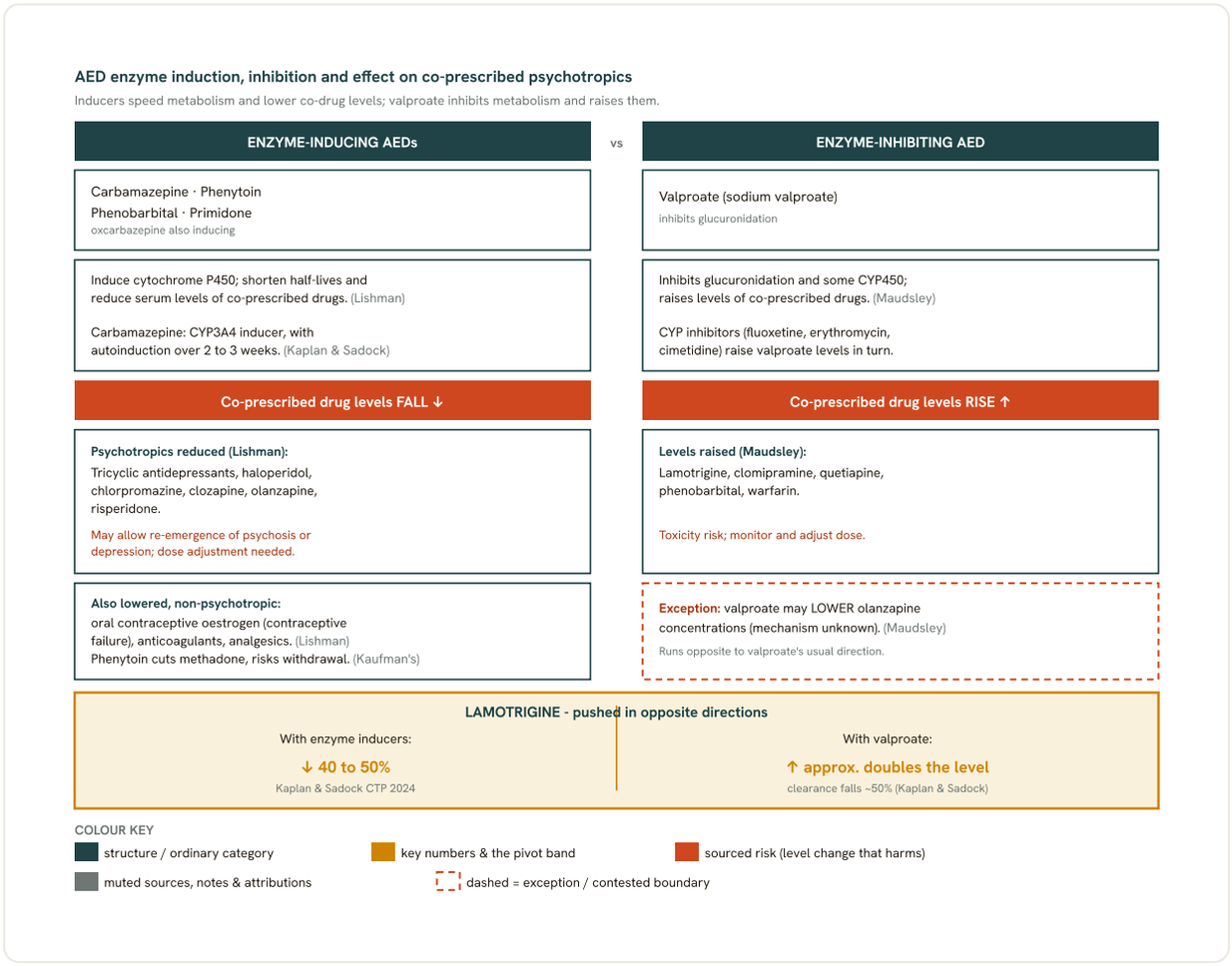


Fig 41.1 AED enzyme induction and inhibition and the effect on co-prescribed psychotropics. Enzyme-inducing AEDs (carbamazepine, phenytoin, phenobarbital, primidone) lower serum levels of many co-prescribed drugs, risking loss of efficacy, while the inhibitor valproate raises them, risking toxicity; lamotrigine is the pivot, cut 40 to 50 per cent by inducers yet roughly doubled by valproate. Attributions per box (Lishman, Kaplan & Sadock CTP 2024, Maudsley, Kaufman's).

41.1 One mechanism, two directions

Everything downstream follows from induction versus inhibition. Hepatic and gut drug metabolism runs largely through the **cytochrome P450** enzyme system, and a co-prescribed drug is only ever a substrate being processed by it. **Enzyme induction** means the antiepileptic increases the quantity of enzyme the liver manufactures, so the partner drug is metabolised faster, its half-life shortens and its steady-state serum concentration falls. **Enzyme inhibition** is the mirror image: the antiepileptic blocks the enzyme, the partner is cleared more slowly, and its level rises. The two processes even differ in tempo in a way that matters at the bedside. Induction is slow because it depends on the synthesis of new enzyme protein, so its effect builds gradually over the first weeks of co-treatment and, just as importantly, unwinds over a similar period when the inducer is withdrawn. Inhibition is comparatively rapid, appearing as soon as the inhibiting drug reaches an effective concentration. This is precisely why the whole subject collapses into one question rather than a grid to be learnt by rote: establish whether the drug in front of you is an inducer or an inhibitor, and the direction in which every one of its partners will move is already decided. The drug names are the easy part; the direction is the mark.

RULE **Inducers lower, inhibitors raise; always ask which.** Before adding, stopping or swapping any drug in a patient on antiepileptics, decide whether the antiepileptic induces or inhibits the enzymes that clear the other agent. An enzyme-inducing drug drives co-prescribed levels down and risks loss of effect; an enzyme-inhibiting drug drives them up and risks toxicity. The direction of the interaction, not the identity of the particular pair, is what you are being tested on and what keeps the patient safe.

MNEMONIC

Induce reduces, inhibit inflates

The whole interaction in four words. An enzyme-**induc**ing antiepileptic **reduc**es the level of the drug given with it; an enzyme-in**hib**iting antiepileptic in**flat**es it. Attach the four classic strong inducers, carbamazepine, phenytoin, phenobarbital and primidone, to the first half and valproate to the second, and the direction of nearly every examinable pairing falls out without a memorised table.

41.2 The enzyme-inducing antiepileptic drugs

A short, high-yield list of powerful inducers. The strong enzyme-inducing antiepileptics are almost all the older, first-generation agents, and for examination and prescribing purposes they behave as a single class with one shared consequence.

- **Carbamazepine**
- **Phenytoin**
- **Phenobarbital** and the barbiturates
- **Primidone**, which is itself metabolised to phenobarbital

Taken together, Lishman's Organic Psychiatry describes how these agents accelerate the clearance and reduce the serum levels of many co-administered drugs, an effect that spares almost nothing else the patient is taking: other antiepileptics, oral contraceptives, anticoagulants, analgesics and the psychotropics that are this chapter's concern. The clinical signature of induction is a quiet one. It is not a new symptom but the return of an old one, because the partner drug is now being cleared before it can work. A patient whose depression or psychosis had been controlled begins to slip, the antiepileptic and the psychotropic doses are both unchanged, and the cause stays invisible unless the prescriber is thinking pharmacokinetically. The breadth of that list matters as much as any single pairing, which is why the safe habit is to treat the addition of an enzyme-inducing antiepileptic as a standing reason to review every other drug on the chart.

41.3 Carbamazepine and autoinduction

The inducer that induces its own metabolism. Carbamazepine deserves a paragraph of its own because it does something the others do not do so dramatically: it induces the very enzyme that clears itself. Kaplan and Sadock's Comprehensive Textbook of Psychiatry identifies it as a **CYP3A4** inducer that undergoes **autoinduction**, a process developing over the first **2 to 3 weeks** of treatment. The practical consequence is counter-intuitive and repeatedly tested. A dose that produced a good level in the first week yields a lower level a fortnight later, because the liver

has caught up and is now metabolising the drug faster, so slightly higher doses are often needed once autoinduction is complete. There is a second lesson folded into this. Because carbamazepine's own enzyme induction takes those first weeks to reach full strength, every interaction it has with other drugs is time-dependent in the same way: its power to suppress a partner's concentration is not fully expressed on the first day but grows over the same window. A level checked or a clinical judgement made too early therefore reads falsely reassuring, and the true effect on both carbamazepine and its partners is only seen once the enzyme population has finished expanding.

GOOD TO REMEMBER

When you start carbamazepine, do not judge the steady state from the first days. The level you measure early will drift down as autoinduction proceeds, and so will the levels of anything else the patient is taking that carbamazepine induces. Recheck the drug level and the patient's mental state once the induction has settled, not while it is still building, and warn the patient that a dose which felt right at first may need a modest increase.

41.4 What induction does to psychotropic levels

The stabilised patient who relapses on an unchanged dose. The interaction that bears most directly on a psychiatrist is the fall in plasma antipsychotic and antidepressant concentrations that enzyme-inducing antiepileptics produce. Lishman's Organic Psychiatry documents the effect across both drug classes and across generations of antipsychotic, from the older agents such as haloperidol and chlorpromazine to the second-generation drugs, and across the tricyclic antidepressants. The consequence the source draws out is the clinically important one: as the concentrations drop, a previously controlled psychosis or depression can re-emerge, and the psychotropic dose may need to be raised to compensate. This is a silent relapse mechanism, because nothing about the presentation announces that a level has fallen; the illness simply returns while the prescription looks unchanged and adherence looks perfect. The correct response is not to escalate treatment blindly but to recognise the pharmacokinetic cause, and either raise the psychotropic dose against a target level or, where the choice is still open, prefer an antiepileptic that does not induce. Therapeutic drug monitoring, where the drug can be assayed, converts an invisible fall into a measured one.

41.5 Lamotrigine, the drug pulled both ways

One partner, two opposite interactions: the clearest illustration of the whole principle. No drug demonstrates the bidirectionality of antiepileptic interactions better than lamotrigine, because it is a substrate for both an inducing and an inhibiting antiepileptic and moves in opposite directions with each. Enzyme inducers drive it down. Kaplan and Sadock's Comprehensive Textbook of Psychiatry records that co-administration with inducers such as carbamazepine, phenytoin, phenobarbital or primidone lowers the lamotrigine plasma concentration by **40 to 50 per cent**. Valproate does the reverse, and does it powerfully. As an inhibitor of the glucuronidation pathway that clears lamotrigine, valproate **approximately doubles** the lamotrigine concentration. Set those two facts side by side and the same nominal

lamotrigine dose can represent twice or half the effective exposure depending purely on the company it keeps. The stakes of reading that direction correctly are high, because lamotrigine's dosing is dominated by the need to titrate slowly to avoid a serious rash, and that slow-titration rule and its rash risk are taught where lamotrigine is covered in its own right in this chapter. The pharmacokinetic fact to carry from here is its simplest summary: valproate roughly doubles the level while inducers roughly halve it, so the direction of the adjustment is set entirely by which partner the lamotrigine shares the chart with.

41.6 Valproate: the inhibitor on the antiepileptic bench

The antiepileptic that raises levels instead of lowering them. Valproate is the important counter-example to the inducing antiepileptics, and holding it firmly as an inhibitor is the second half of the whole topic. Rather than inducing cytochrome P450, it inhibits **glucuronidation**, so the drugs paired with it are cleared more slowly and their concentrations climb. The Maudsley Prescribing Guidelines note that valproate raises the levels of a spread of co-prescribed drugs: lamotrigine most strikingly, but also tricyclic antidepressants such as clomipramine, quetiapine, phenobarbital and warfarin. The last two make the point that this is no psychiatric curiosity, since a rising phenobarbital level brings sedation and a rising warfarin effect brings a bleeding risk. The traffic also runs the other way. Other enzyme inhibitors, and the same source names erythromycin, fluoxetine and cimetidine, in turn raise valproate's own concentration, so valproate is both a perpetrator and a victim of inhibition. And then the exception that tests real understanding: contrary to its usual level-raising direction, valproate significantly lowers the plasma concentration of olanzapine, by a mechanism that remains unknown. That single reversal is the detail most likely to catch a candidate who has learnt valproate as a drug that only ever pushes concentrations up.

TRAP **Assuming valproate raises every level.** Because valproate is the textbook enzyme inhibitor and famously doubles lamotrigine, candidates generalise it into a drug that can only push concentrations upward. It cannot. It significantly lowers the plasma concentration of **olanzapine**, by an undetermined mechanism, and that exception is exactly the kind of item an examiner uses to separate rote recall from a grasp of the pharmacology. The direction of an interaction is a property of the specific pair, not a fixed label stamped on one drug.

41.7 Interactions beyond the psychotropics

The induction that defeats a pregnancy plan, an anticoagulant or an opioid substitution. The enzyme-inducing antiepileptics do not confine their reach to psychiatric drugs, and three non-psychiatric interactions are high-yield because each carries a discrete and serious harm. Lishman's Organic Psychiatry notes that induction reduces the efficacy of oral contraceptives, oral anticoagulants and analgesics through the same acceleration of metabolism. The first of these is the reproductive one. Kaplan and Sadock's Comprehensive Textbook of Psychiatry warns that carbamazepine, and enzyme-inducing antiepileptics generally including oxcarbazepine, lower estrogen levels and can cause contraceptive failure, so higher-dose estrogen preparations or a reliable non-hormonal method are needed. The distinct and much larger problem of valproate's teratogenicity and the formal contraception requirements it imposes on women of childbearing

potential belong to the topic on prescribing valproate in women covered elsewhere in this chapter; the point here is only the pharmacokinetic one, that induction can silently disarm an oral contraceptive.

The second is opioid substitution. Kaufman's Clinical Neurology for Psychiatrists describes how phenytoin's induction reduces the activity of **methadone** and can precipitate opioid withdrawal, which is why the methadone dose should be raised in anticipation before phenytoin is started rather than after the patient becomes unwell. The third, anticoagulation, follows the same arithmetic: a patient stabilised on warfarin can drift to a subtherapeutic anticoagulant effect when an inducing antiepileptic is added, and back toward a bleeding risk if that antiepileptic is later withdrawn and the induction unwinds.

IN INDIA

These pharmacokinetics are not academic in Indian practice, because the cheap and widely available first-line antiepileptics are precisely the strong inducers: carbamazepine, phenytoin and, in resource-limited settings where it remains a genuine first-line drug, phenobarbital. A large share of patients with epilepsy are therefore on an enzyme inducer, so the induction interactions dominate the ground: reduced psychotropic efficacy in a population with high psychiatric comorbidity, and **contraceptive failure** in women who may never have been told that their antiepileptic can defeat the pill. Where the budget allows, a non-inducing antiepileptic sidesteps the whole problem at once. Where it does not, the workable answer is active review of co-medication and explicit contraceptive counselling, rather than assuming the two prescriptions are independent of each other.

PITFALL

The interaction does not only appear when an inducing antiepileptic is started; it reverses, with equal clinical force, when one is stopped. Reduce or discontinue an enzyme-inducing antiepileptic in a patient who is also on a psychotropic, an anticoagulant or a contraceptive, and the co-drug that had been cleared too fast is now cleared normally again, so its level climbs over the following weeks toward, and sometimes past, the toxic range. Clinicians who remember to adjust doses when adding an inducer routinely forget to readjust them when withdrawing one. Every induction interaction is a two-way street, and the return journey is the one that is missed.

ANTIEPILEPTIC AND EFFECT	PARTNER DRUG LEVEL	REPRESENTATIVE PARTNERS AND THE RESULTING RISK
Inducers on psychotropics (induction)	falls	antipsychotics and tricyclic antidepressants become subtherapeutic; silent relapse
Inducers on other drugs (induction)	efficacy falls	oral contraceptives, oral anticoagulants and analgesics lose effect
Carbamazepine on contraception (induction)	estrogen falls	contraceptive failure; use higher-dose estrogen or a non-hormonal method
Phenytoin on methadone (induction)	activity falls	opioid withdrawal; raise methadone before starting phenytoin
Valproate on most partners (inhibition)	rises	lamotrigine, quetiapine, warfarin, phenobarbital; toxicity and bleeding
Valproate on olanzapine (exception)	falls	the one direction reversal to remember

A direction map of the examinable interactions: read the middle column first, because the direction of the level, not the identity of the pair, is what the answer turns on.

41.8 Prescribing safely around the interactions

The mechanism is only useful if it changes the prescription. There is no single "treatment" of a pharmacokinetic interaction; the management is a disciplined prescribing method, and it is best organised the way any management problem is. The standards that anchor it are the epilepsy guidelines of the International League Against Epilepsy (ILAE) and the National Institute for Health and Care Excellence (NICE), which govern antiepileptic selection and the preference for non-inducing agents where a co-prescription makes that sensible, together with the Maudsley Prescribing Guidelines for the psychotropic side of the same chart.

The **LOCUS** of this care is overwhelmingly the outpatient clinic, where medications are added, stopped and cross-titrated, and where the real danger is that the two prescribers, often a neurologist and a psychiatrist, do not see each other's changes. It moves to the inpatient setting when a complex cross-titration is safest under observation, and to the emergency department when an interaction has already declared itself as frank toxicity or as withdrawal. The **focus** shifts by phase. At the moment of any change, the target is anticipation: predict the direction of the interaction and adjust pre-emptively rather than reactively. Across the first weeks, the target is the slow-building nature of induction, so a level or a mental state assessed too early gives false reassurance and must be repeated once the effect is fully expressed. In the longer term, the target is vigilance at every subsequent change to either drug, because each addition or withdrawal reopens the interaction afresh. The **modus** spans all the levers available: the pharmacological lever of dose adjustment in the predicted direction, or the cleaner option of selecting a non-inducing antiepileptic so the interaction never arises; the procedural lever of therapeutic drug

monitoring, which turns an invisible interaction into a measured quantity for the drugs that can be assayed; and the social and educational levers of explicit counselling, about contraception, adherence and never stopping a drug abruptly, and above all communication between prescribers so that a change on one chart is known to the other. Managed this way, the interaction becomes predictable arithmetic rather than an ambush.

2 to 3 weeks	40 to 50%	about double
CARBAMAZEPINE AUTOINDUCTION DEVELOPS	FALL IN LAMOTRIGINE WITH INDUCERS	LAMOTRIGINE LEVEL WITH VALPROATE

Enzyme-inducing antiepileptics (carbamazepine, phenytoin, phenobarbital, primidone) lower the levels of the drugs given with them, while valproate, an inhibitor, raises them, with olanzapine the notable exception, so the direction of the interaction, not the drug's name, must drive every dose decision.

42 · Lamotrigine: mechanism, titration, rash and bipolar maintenance role

The antiepileptic whose dose has to be earned slowly. Lamotrigine is the antiepileptic a psychiatrist meets more often than almost any other, because it sits at the crossing point of two clinics: it is a first-line drug for focal epilepsy and, separately, a familiar name in the mood clinic. This topic takes the antiepileptic lens deliberately. It asks how the drug works against seizures, why its introduction is governed by an unusually strict titration schedule, and why that schedule exists at all, which is to say the rash that has shaped every prescribing rule attached to the molecule. The maintenance targets below are the representative destinations of that slow titration, not a substitute for the age-specific and product-specific escalation steps of the current label. The maintenance mood-stabiliser role in bipolar disorder, for which lamotrigine is equally well known, is set out in full in the Mood disorders: pharmacotherapy notes and is only named here; none of that mood-stabiliser evidence is rehearsed in this account.

The marks on this topic are almost entirely practical and almost entirely about safety. An examiner is rarely interested in whether a candidate can recite that lamotrigine blocks a sodium channel; the interest is in whether the candidate can start the drug without harming the patient. That means knowing how the starting dose and the speed of escalation change with whatever else the patient is already taking, and knowing the one adverse reaction that turns a well-tolerated drug into a dangerous one. Everything below is organised around that single clinical spine.

42.1 Antiepileptic mechanism of action

A membrane-stabilising sodium channel blocker that restrains glutamate release. Lamotrigine's anticonvulsant action is that of a **voltage-gated sodium channel blocker**. Kaplan and Sadock's Comprehensive Textbook of Psychiatry describes it binding preferentially to the channel in its inactivated state, so that neurons which are firing rapidly and repetitively, the pattern of a seizure, are the ones whose membranes are most effectively stabilised, while normal background firing is comparatively spared. The downstream consequence, and the part most

often asked, is presynaptic: by damping this pathological high-frequency firing the drug reduces the release of the principal excitatory neurotransmitter, **glutamate**. That combination, a use-dependent sodium channel action expressed as a fall in excitatory transmission, is the mechanistic sentence to carry. It is worth holding apart from the quite different set of ideas invoked to explain why the same molecule stabilises mood, which belong to the notes named above; here the concern is purely the antiepileptic one, and the sodium channel is where it begins.

42.2 The serious rash and its boxed warning

The reaction that defines the drug. The reason lamotrigine cannot simply be started at a therapeutic dose is a risk of serious, potentially life-threatening skin reaction grave enough to carry a **boxed warning**. In premarketing trials the incidence of serious rash was about **1 per cent** in children under sixteen years and about 0.3 per cent in adults, the higher paediatric figure being one reason for particular caution at the younger end. The reactions that give the warning its weight are the severe cutaneous drug eruptions, **Stevens-Johnson syndrome** and, at its most extreme, **toxic epidermal necrolysis**, and rash-related deaths have been reported. These are not common events, but they are catastrophic when they occur, and their possibility is what converts an otherwise well-tolerated drug into one whose introduction demands a specific discipline. The examinable point is less the exact figure than the category of risk: a drug that can, rarely, kill through the skin, and that therefore has to be started in a way that keeps that risk as low as it can be made.

42.3 Benign rash and the stop-at-first-sign rule

Common, usually harmless, but never to be watched. Most rashes on lamotrigine are not the dangerous kind. A benign rash occurs in **up to 10 per cent** of patients, typically a simple maculopapular eruption appearing early in treatment, and the great majority of these settle uneventfully once the drug is withdrawn. The difficulty is that at their onset the trivial rash and the first hours of a severe one can look alike, and there is no reliable way at the bedside to be certain which is beginning. Kaplan and Sadock's Comprehensive Textbook of Psychiatry gives the resulting rule its characteristically cautious form: the drug should ordinarily be discontinued at the first sign of any rash, unless the rash is clearly not drug-related. That default toward stopping is deliberately conservative. It accepts that many benign rashes will lead to a discontinuation that was, in hindsight, not strictly necessary, because the cost of that caution is small and the cost of continuing through the one rash that turns out to be Stevens-Johnson syndrome is not. In practice the patient is told before ever taking the first tablet to report any rash at once, rather than wait to see whether it worsens.

GOOD TO REMEMBER

Before you write the first lamotrigine dose, find out what else the patient is already taking, because that single question decides the whole starting regimen. A patient on valproate needs a much lower, slower start; a patient on an enzyme-inducing antiepileptic needs a higher one, taken up more quickly. Choosing the starting dose without knowing the co-medication is the commonest way to get a lamotrigine prescription wrong before the patient has swallowed anything.

42.4 The timing window of the dangerous rash

When the risk is live, and when it has largely passed. The serious rash is not a hazard spread evenly across the life of the prescription; it is concentrated early. Nearly all life-threatening rashes appear within the first weeks of treatment, the great majority falling **between 2 and 8 weeks** after the drug is started, with only isolated cases reported later. This early clustering is what makes the titration period the critical window, and it explains why review is front-loaded into the first weeks rather than spread evenly across the year. It also gives the reassurance that matters for the long-term patient: a person who has climbed slowly through the escalation schedule and reached a stable maintenance dose without a rash has passed through the interval of greatest danger, and the risk on continued steady dosing is very much lower. The corollary, developed below, is that anything which effectively returns the patient to the beginning of treatment, most importantly a break in dosing, reopens that early window and has to be treated as a fresh start rather than a simple continuation.

42.5 Why the rash happens: risk factors and the slow-titration rule

The rash is a rate phenomenon, not a dose phenomenon. The single idea that unifies lamotrigine prescribing is that the risk of a serious rash tracks the speed at which the serum concentration is allowed to rise far more than the dose eventually reached. A high maintenance dose arrived at gently is safer than a modest dose reached in a rush. Three circumstances raise the risk, and each is a way of making the level climb too fast:

- escalating the dose more rapidly than the recommended schedule allows
- starting above the recommended initial dose
- **concurrent valproate**, which pushes the lamotrigine level up and so amplifies any given rate of increase

From this single mechanism the entire prescribing discipline follows. Because the danger is the rate of rise, the protection is a slow rise, and Kaplan and Sadock's Comprehensive Textbook of Psychiatry together with Lishman's Organic Psychiatry make **gradual, stepwise introduction** the defining rule for starting the drug. It is the reason lamotrigine takes weeks rather than days to reach a useful dose, an inconvenience that is not a shortcoming of the schedule but its entire purpose.

RULE **Introduce lamotrigine slowly; the rate of rise, not the final dose, sets the rash risk.** A gradual dose introduction is the crucial manoeuvre for attenuating the risk of a serious rash, and it is the one prescribing rule that must never be compromised for the sake of reaching a therapeutic effect sooner. Every other adjustment, for co-medication or for a missed-dose restart, is a variation on protecting that slow rise.

MNEMONIC

Low, slow, and stop at the first rash

The whole safety of the drug in five words. Start **low**, climb **slow**, and **stop** at the first sign of any rash rather than watching it. The first two protect against the rash by keeping the rate of rise gentle; the third protects against it by refusing to gamble on a given rash being the benign kind. Nothing in the sentence has to be memorised as a number, which is exactly why it survives the pressure of a real clinic.

42.6 Titration and target doses by co-medication

The starting dose is a function of the company the drug keeps. Because the rash risk is set by the rate at which the level rises, and because other drugs powerfully change that level, lamotrigine has no single correct starting dose or target; both depend on the co-medication. Lamotrigine is cleared chiefly by hepatic **glucuronidation**, and it is this pathway that its partners disturb, so its elimination half-life shifts markedly with what else is on the chart. The detailed pharmacokinetics of enzyme induction and inhibition are the subject of the topic on antiepileptic and psychotropic interactions and are not reworked here; the point that belongs to lamotrigine itself is the practical consequence for how it is started and where it is aimed.

With valproate the level roughly doubles, so the standard response, drawn from Lishman's Organic Psychiatry and Kaplan and Sadock's Comprehensive Textbook of Psychiatry, is to halve the usual start and climb more gently still. With an enzyme-inducing antiepileptic the opposite holds, and the drug can be both begun higher and taken up more briskly. The accompanying table sets the three situations side by side.

CO-MEDICATION CONTEXT	STARTING APPROACH AND TITRATION	TARGET MAINTENANCE DOSE	APPROXIMATE HALF-LIFE
Lamotrigine monotherapy	standard gradual introduction	200 mg/day	about 28 hours
With valproate	halved start, 25 mg on alternate days, slower climb	100 mg/day	about 56 hours (roughly doubled)
With enzyme-inducing antiepileptics	start 50 mg/day and titrate up more quickly	400 mg/day	about 14 hours (roughly halved)

Read the table across a single row: the co-medication decides the start, the target and the half-life together, which is why the first clinical question is always what else the patient takes.

PITFALL

The commonest serious prescribing error with lamotrigine is to start it at the ordinary dose in a patient already taking valproate. Valproate roughly doubles the lamotrigine concentration, so a standard start becomes, in effect, a rapid escalation, driving the level up exactly as fast as the rash risk fears. The valproate combination is one of the named risk factors for a serious rash for precisely this reason, and it demands a halved, slower start rather than the usual one. The mirror error, under-dosing the patient on an enzyme inducer and then wrongly concluding the drug does not work, is far less dangerous but wastes months of treatment.

42.7 Restarting after an interruption

A break in treatment is a return to the beginning. One of the most clinically important and most often forgotten rules attaches to the patient who has stopped the drug for a while, whether through non-adherence, a supply problem, or an admission in which doses were missed. The safe accommodation that the slow titration built up is lost when the drug is absent, so restarting at the previous maintenance dose reproduces exactly the rapid rise in level that the original slow titration was designed to avoid. Kaplan and Sadock's Comprehensive Textbook of Psychiatry gives the operational rule: if the drug has not been taken for **5 days or more**, it should be reintroduced using the full, gradual initial escalation regimen from the start, not resumed at the dose the patient was previously on. Serious rashes have followed a rapid restart, which is why this rule is not a bureaucratic formality but a genuine safety instruction.

TRAP **Resuming at the old maintenance dose after a gap.** The instinct, when a patient who was stable on lamotrigine returns after missing it, is to restart them on the dose they were taking. If the interruption has lasted beyond that short threshold, that instinct is dangerous: the drug must be re-titrated from the beginning of the escalation schedule, because the accommodation built by the original slow climb is gone and a full-dose restart reopens the early window of serious-rash risk. Examiners favour this item precisely because the reflex answer is the wrong one.

IN INDIA

Lamotrigine is a more expensive antiepileptic than the older first-line agents that dominate Indian practice, and its obligatory slow titration adds a second, less obvious cost: it takes weeks of graded dosing, and therefore several clinic contacts, before the drug reaches a useful level. In a system where follow-up can be irregular and paid out of pocket, this creates real temptations to shortcut the schedule, to restart at the old dose after a lapse rather than begin again, or to abandon a drug that seems to be doing nothing during its long run-in. Each of those shortcuts is the specific behaviour the rash rules exist to prevent. Where lamotrigine is genuinely indicated, the counselling that goes with it, that the slow start is the safety and not a delay, and that a rash means stop and call, is as much a part of the prescription as the tablets themselves.

42.8 Prescribing lamotrigine safely

The mechanism only helps if it changes the prescription. There is no separate treatment of the lamotrigine rash beyond preventing it and responding decisively when it appears, so the management here is a disciplined prescribing method. The standards that anchor it are the

epilepsy guidelines of the International League Against Epilepsy (ILAE) and the National Institute for Health and Care Excellence (NICE), which govern antiepileptic selection and safe use, together with the drug's own boxed-warning prescribing information, which is the primary authority on the rash and its titration schedule.

The **LOCUS** of care is overwhelmingly the outpatient clinic, where the drug is started and titrated over weeks and where the whole safety of the exercise is decided; it moves to the inpatient setting only when an unstable epilepsy or a complex cross-titration is safest under observation, and to the emergency and dermatology setting the moment a severe rash or a systemic reaction declares itself, at which point the drug is stopped and the patient managed as a dermatological emergency. The **focus** shifts by phase. In the acute phase, the first weeks, the target is simply to climb the escalation schedule slowly and to keep review close, because this is the interval in which the dangerous rash lives. Across the medium term the target is reaching an effective maintenance dose appropriate to the co-medication without ever sacrificing the slow rate of rise. In the long term the targets are adherence and the correct handling of any interruption, since a break resets the patient to the beginning. The **modus** spans every available lever: the pharmacological lever of a starting dose and titration matched to the co-medication, halved and slowed for valproate and raised for enzyme inducers; the procedural lever of immediate discontinuation and supportive dermatological care should a serious rash appear; and the social and educational levers of counselling the patient to report any rash at once, never to restart at the old dose after a lapse, and to understand that the slow introduction is the very thing keeping them safe.

about 1%	up to 10%	2 to 8 weeks	5 days+
SERIOUS RASH, CHILDREN UNDER 16 IN TRIALS	BENIGN RASH ON LAMOTRIGINE	WINDOW FOR THE LIFE-THREATENING RASH	GAP THAT FORCES RE-TITRATION FROM THE START

Lamotrigine is all about the rash: start low and titrate slowly, halve the starting dose with valproate and raise it with enzyme inducers, stop at the first sign of any rash, and re-titrate from the beginning after a break of 5 days or more, because the rate at which the serum level rises, not the final dose, decides the risk of a life-threatening reaction.

43 · Valproate teratogenicity, contraception requirements and prescribing in women

No prescribing decision in the epilepsy clinic is more heavily freighted than whether to place, or to leave, a woman of childbearing potential on sodium valproate. For a given patient valproate may be the most effective drug she has for her seizures, and yet it is the most teratogenic antiepileptic in routine use, harming a developing fetus in ways both structural and behavioural. That combination, high efficacy against seizures set against high risk to the fetus, is precisely what makes the topic a perennial examination favourite and a live regulatory concern. The woman with epilepsy cannot be managed as though the drug could simply be withdrawn the moment a pregnancy test turns positive: uncontrolled seizures carry their own maternal and fetal

dangers, so the clinician is forced into a genuine risk-against-risk calculus rather than a simple instruction to stop.

The examinable core has four parts, and a strong answer holds them apart: the size and shape of the teratogenic signal, its dependence on dose, the neurodevelopmental teratogenicity that no anomaly scan can exclude, and the framework of contraception, preconception optimisation and antenatal surveillance that now governs prescribing. The mood-stabiliser and polycystic-ovary aspects of valproate belong to the Mood disorders: pharmacotherapy notes and are only named here; what follows is the epilepsy-facing account.

The most economical way to see valproate's position is to compare drugs on a single broad endpoint. Taking **any adverse outcome**, meaning either a major congenital malformation or fetal death, the exposed-pregnancy rates separate the agents unambiguously, and valproate stands far above the alternatives:

20.3%	10.7%	8.2%	1.0%
VALPROATE, ANY ADVERSE OUTCOME	PHENYTOIN	CARBAMAZEPINE	LAMOTRIGINE

43.1 The teratogenic signal and where valproate sits among the antiepileptics

A class risk with a wide internal gradient, and valproate at the wrong end of it. Antiepileptic drugs as a group raise the baseline risk of major congenital malformation. Pooled across the older agents, Lishman's Organic Psychiatry puts the malformation rate in exposed pregnancies at about **5 to 9 per cent**, roughly twice the background-population rate, so a woman on any antiepileptic begins pregnancy with an elevated absolute risk before the specific molecule is chosen. The clinically decisive point, and the one an examiner is testing, is that this class figure conceals a broad gradient between drugs, and valproate occupies the unfavourable extreme.

Prospective pregnancy registers have made that gradient concrete. In **the UK Epilepsy and Pregnancy Register**, drawn on by Lishman's Organic Psychiatry, the major-malformation rate on valproate monotherapy was substantially higher than on the common alternatives, and the ordering is the pattern to carry into the exam:

MONOTHERAPY	MAJOR MALFORMATION RATE
Valproate	6.2 per cent
Lamotrigine	3.2 per cent
Carbamazepine	2.2 per cent

On the same register, denominator and endpoint, valproate roughly doubles the risk seen with lamotrigine and nearly trebles that with carbamazepine.

Two features matter beyond the digits. The ordering is consistent across datasets and endpoints: whether the outcome is a narrow count of major malformations or the broader composite shown at the head of this account, valproate is the outlier and the alternatives sit well below it. And register figures describe monotherapy under controlled conditions, so real-world risk is pushed higher still by two further variables, dose and combination, taken in turn below. The lamotrigine

and carbamazepine numbers appear only as comparators that fix valproate's position; their own prescribing stories are told elsewhere in this chapter.

RULE **Valproate carries the highest teratogenic risk of the drugs in routine antiepileptic use, and that risk is dose-dependent.** Every downstream decision, whether to start it at all, how to counsel, what dose to accept and how intensively to screen, follows from these two facts held together. Miss either one and the management falls apart.

43.2 Structural malformations: neural tube defects and the wider pattern

The signature lesion is a defect of neural-tube closure. The malformation most specifically associated with intrauterine valproate is the **neural tube defect**, classically **spina bifida**, and the risk in valproate-exposed pregnancies is of the order of about **1 to 2 per cent**. That figure must be read against the natural history of the defect. The neural tube closes very early in the first trimester, ordinarily before a woman knows she is pregnant, which is the single most important reason that prevention before conception matters far more than anything done once a pregnancy is confirmed. By the time the drug's risk is discussed in the antenatal clinic, the window in which the defect is determined has usually closed.

The anomaly profile is broader than the neural tube alone. Kaplan and Sadock's Comprehensive Textbook of Psychiatry records that valproate exposure also predisposes to cardiac and to skeletal malformations, alongside a recognisable pattern of dysmorphic features, so the structural picture spans several systems rather than one:

- neural tube defects, above all spina bifida, the lesion most tightly linked to valproate
- cardiac malformations
- skeletal and limb anomalies, together with the characteristic facial dysmorphology of the exposed infant

Why valproate does this is only partly understood. The molecule interferes with folate metabolism, inhibits histone deacetylase and generates oxidative stress, each of which can disturb the tightly timed processes of neural-tube closure, cardiac septation and neuronal migration. None is established as the sole cause, but together they explain the drug's reach across several developmental systems and why folate supplementation, addressed under management, has a rational place even though it cannot abolish the risk.

43.3 Dose-dependence and the special danger of polytherapy

Teratogenicity that scales with exposure, not an all-or-nothing property of the molecule. The harm valproate does is not a fixed toll levied on every exposed pregnancy equally; it rises with the amount of drug the fetus sees. Risk climbs with the daily maternal dose, and the inflection examiners look for is a threshold in the region above **1000 mg per day**, beyond which the malformation rate increases steeply. This dose-dependence is among the most reliably examined facts in the area, because it converts a blunt warning into a graded instruction: if valproate cannot be avoided, the objective is the lowest dose that still controls the seizures, since a lower dose is genuinely less dangerous even though no dose is safe.

Combination therapy compounds the problem. A regimen containing valproate alongside other antiepileptics carries a malformation rate near 9 per cent, higher than valproate monotherapy and than most other polytherapies, so a valproate-containing combination is the pattern to be most reluctant to create or to leave in place in a woman who might conceive. The two levers point the same way: keep the dose as low as seizure control allows and the drug alone rather than in company, since simplifying away from a valproate combination is among the most valuable single changes that can be made before a pregnancy.

43.4 Behavioural teratogenicity and the fetal valproate syndrome

The harm that a normal scan cannot exclude. The most important idea in the topic, and the one candidates most often miss, is that valproate damages the developing brain in ways that have nothing to do with any visible malformation. This **behavioural teratogenicity** is the core of what is termed the **fetal valproate syndrome**, and it is present in children who were structurally normal at birth and who passed every anomaly scan. Kaplan and Sadock's Comprehensive Textbook of Psychiatry sets out a consistent and worrying picture across three overlapping outcomes.

The first is a loss of general cognitive ability. Prenatal valproate is associated with a mean fall in childhood IQ of about **10 points**, and exposed children scored roughly 9 points below lamotrigine-exposed children when assessed at three years of age, with some falling into the range of intellectual disability. The second is confirmed in population data: a nationwide **Danish birth cohort** showed roughly a 4.5-fold increase in intellectual disability, rising to about a 6-fold increase when delayed developmental milestones were counted alongside it. The third is a raised risk of **autism spectrum disorder**, with a many-fold higher risk of childhood or lifetime autism reported and supported by animal models. Taken together these define a neurodevelopmental toll that unfolds over years:

- lowered global cognitive ability, sometimes reaching the range of intellectual disability
- frank intellectual disability and delayed developmental milestones
- autism spectrum disorder and autistic traits

The clinical force here is a single asymmetry. A structural defect such as spina bifida can, in principle, be detected before birth; a lowered IQ, an intellectual disability or an emerging autism cannot. Behavioural teratogenicity is not screenable and declares itself only as the child grows, so it is the part of valproate's harm that surveillance can never reach and that only prevention can prevent.

TRAP **Treating valproate teratogenicity as a structural problem that a reassuring anomaly scan can rule out.** The neurodevelopmental harm, reduced IQ, intellectual disability and autism, occurs in children with no visible malformation and cannot be found antenatally, so a normal scan does not mean a normal outcome. Candidates who equate teratogenicity with spina bifida forfeit the point the examiner is actually testing.

43.5 Why the woman with epilepsy cannot simply stop the drug

The dilemma that puts this topic in an epilepsy chapter at all. A woman prescribed valproate for a mood indication can often be moved to a different agent without much loss, but the woman with epilepsy is frequently the one for whom valproate is the drug that actually stops her seizures, and stopping it is not a neutral act. A generalised tonic-clonic seizure in pregnancy risks maternal injury, hypoxia and status epilepticus, each threatening the fetus in its own right, so the clinician weighs a quantified fetal risk from the drug against a real, if less easily counted, risk from uncontrolled seizures, and neither side of the balance can be waved away.

There is a further subtlety drawn out by Kaplan and Sadock's Comprehensive Textbook of Psychiatry: the birth-defect and developmental-delay risks appear more prominent in **women with epilepsy** taking valproate than in those given it for bipolar disorder. The interpretation calls for caution, since the epilepsy population differs in dose, in the frequency of polytherapy, in the disorder itself and possibly in genetic liability, so this is not a clean drug-versus-drug contrast. The practical message is unambiguous: the population in which valproate is most useful is also the one in which its risks are most evident, which is why the decision belongs before conception, taken deliberately with the woman, not in the crisis of an unplanned pregnancy.

> **PITFALL**
>
> Abruptly stopping valproate the instant a pregnancy is confirmed feels protective and is often the reverse. By the time most women know they are pregnant the neural tube has already closed, so the impulsive withdrawal cannot undo the exposure that has already happened, while the sudden loss of seizure control can precipitate breakthrough seizures or status epilepticus that endanger both mother and fetus. The correct response to an unplanned pregnancy on valproate is an urgent specialist review, not a unilateral stop.

43.6 Contraception and the Pregnancy Prevention Programme

Because the greatest harm precedes recognition, contraception becomes a condition of prescribing. The logic of everything above is that the damage is largely done before a pregnancy is known, so the only reliable protection is to prevent an unintended pregnancy while the drug is taken. The regulatory response, set out in the Maudsley Prescribing Guidelines, is formal: valproate should not be used in a girl or woman of childbearing potential unless a **Pregnancy Prevention Programme** is in place, effective contraception is mandatory throughout treatment, pregnancy is to be actively avoided while the drug is taken, and some authorities go further and advise against valproate in women under fifty altogether. This reframes the prescription: valproate in a woman who could conceive is not a routine start but a decision that has to be justified and that comes bundled with a contraceptive plan.

Which method is used is a clinical judgement favouring reliable, ideally long-acting contraception, and the interaction between enzyme-inducing antiepileptics and hormonal contraception is a real but separate consideration belonging to the pharmacokinetics of antiepileptic interactions rather than to valproate teratogenicity as such. What stays with this

topic is that the contraceptive conversation is not a one-off at the first prescription but a standing part of treatment for as long as the drug and the possibility of pregnancy coexist.

MNEMONIC

No Programme, no prescription

The single sentence that governs valproate in women of childbearing potential. The drug is not started, and ideally not continued, in a woman who could become pregnant unless a formal Pregnancy Prevention Programme with effective contraception is in place. Nothing in the rule is a number to be memorised, which is exactly why it survives the pressure of a real clinic.

GOOD TO REMEMBER

At every review of a woman of childbearing potential who is taking valproate, re-ask about contraception and about any plan or wish for pregnancy, and document that you did. Teratogenic risk is a conversation to be renewed, not a consent form signed once and filed, because contraception fails and intentions change, and the one review at which the question is skipped is the one before the unplanned conception.

IN INDIA

Valproate remains widely and cheaply prescribed to women in Indian practice, often where the formal apparatus of a Pregnancy Prevention Programme is not implemented and where the drug was started by a non-specialist before any discussion of pregnancy. Many women present already pregnant on valproate, sometimes soon after marriage, at a point where the exposure has already occurred. The workable response is opportunistic rather than regulatory: raise contraception and preconception planning at every contact with a woman of childbearing potential on the drug, prefer a less teratogenic agent wherever seizure control allows, and ensure access to folate for those who continue it, since the ideal of a pre-planned switch is frequently overtaken by events.

43.7 Prescribing when valproate cannot be avoided: preconception optimisation and antenatal surveillance

When a woman's seizures respond only to valproate, the task changes from avoidance to damage limitation, best carried out as a deliberate, staged plan. The standards that frame it are the Medicines and Healthcare products Regulatory Agency (MHRA) valproate pregnancy-prevention requirements, which set the contraceptive and consent conditions; the epilepsy guidance of the National Institute for Health and Care Excellence (NICE); and the recommendations of the International League Against Epilepsy (ILAE) on women of childbearing age. Together they turn the evidence above into a routine.

The **LOCUS** of care is shared and mostly ambulatory: the outpatient neurology or epilepsy clinic, alongside the psychiatric or primary-care setting where the drug may first have been started, with preconception counselling ideally completed before any pregnancy and specialist obstetric care taking over once a woman conceives; the emergency setting enters only when a woman presents already pregnant or with breakthrough seizures, and its rule is urgent specialist review rather than abrupt withdrawal. The **focus** shifts by phase: before conception, switch to a less teratogenic

agent where possible or else reduce valproate to the lowest effective dose and simplify away from polytherapy, with folate begun in advance; at the discovery of pregnancy, continue surveillance without a dangerous abrupt stop; across the antenatal months, structural screening; and in the long term, developmental follow-up of the child, because behavioural teratogenicity emerges only with time. The **modus** spans every mode. The pharmacological mode is the lowest effective valproate dose, avoidance of valproate-containing combinations, and periconceptional folic acid at **at least 1 mg** per day with vitamins B6 and B12. The psychological mode is a documented, unhurried discussion of the risks. The procedural mode is the antenatal surveillance set out below. The social mode is secure access to contraception, to folate and to planned rather than crisis-driven care.

Surveillance, where exposure has occurred or continues, rests on **fetal anomaly ultrasound**, maternal serum screening and, where indicated, amniotic fluid sampling, timed across the pregnancy as follows:

INVESTIGATION	TIMING	PURPOSE
Fetal anomaly ultrasound	18 to 20 weeks	detect neural tube and other structural defects
Maternal serum alpha-fetoprotein	mid-trimester	raised in the presence of an open neural tube defect
Amniocentesis (amniotic fluid sampling)	16 weeks, if indicated	confirm a suspected open defect

The surveillance addresses structural defects only: useful for the neural tube but, as above, unable to detect or predict the behavioural teratogenicity that is the drug's least reversible harm.

The whole plan is front-loaded toward the decision made before pregnancy. Folate and ultrasound are worth doing, but they mitigate a risk best managed by never incurring it, which is why the contraceptive framework and the preconception switch carry more weight than any antenatal test.

> Valproate is the most teratogenic antiepileptic in common use, its harm is dose-dependent and both structural and neurodevelopmental, and it must not be given to a woman of childbearing potential unless a Pregnancy Prevention Programme with effective contraception is in place; where unavoidable, use the lowest effective dose alone, add periconceptional folate, and remember that the behavioural harm cannot be screened for at all.

Consolidate and apply

44 · Worked vignettes

A worked case is where this chapter stops being a catalogue and becomes a clinical skill. Every syndrome in the preceding sections was defined in isolation, but no patient arrives pre-

labelled. What the examiner hands you, on paper and far more so in the viva, is a short clinical picture and a demand that you place it, attribute it, and exclude what it is not before you commit to a name. These vignettes rehearse exactly that traffic between a presentation and a diagnosis. They deliberately withhold the numbers, intervals and eponyms that belong to the individual topics, because the point of a worked case is not to re-state a fact you have read but to practise the reasoning that retrieves it at the right moment.

Read each vignette as a closed problem first. Resist the pull to answer the surface, and instead run the disciplined sequence the chapter installed at the outset: place the symptom in time, weigh whether the disease, its drug or a non-epileptic process is its author, and name the imitator you have excluded. The cases below are chosen because each one baits a fast, wrong, surface answer and rewards a slower, structured one, which is precisely the discrimination that separates a pass from a distinction.

- Each turns on a single resolving datum, a timing or a drug change or a recording, rather than on the overall impression.
- Each pairs a syndrome with a look-alike the examiner expects you to exclude by name.
- Each withholds the owned specific, so that what is practised is the retrieval and not the recitation.

44.1 The man who was lucid, then was not

A cluster of convulsions, a clear interval, then a florid disturbance. A young man with long-standing epilepsy is brought to the emergency department by his family after a run of tonic-clonic seizures more frequent than usual. He recovered his orientation between and after the fits. Then, after a symptom-free interval in which he was awake, coherent and correctly oriented, he became floridly psychotic: grandiose and religiose, agitated, with paranoid conviction and disturbed sleep. On examination now there is none of the clouding of consciousness that follows a fit directly, and the family fear he has lost his mind for good.

The disciplined reading places the disturbance on the **peri-ictal axis** before it reaches for a psychiatric label. The presence of a lucid, oriented interval between the seizure cluster and the onset of psychosis is the single most valuable datum in the history, because it is what separates a **postictal psychosis** from the confusion and delirium that come on immediately after a fit with no clear-headed gap. This is not a new, unrelated psychotic illness, and it is not the chronic interictal psychosis that develops on a settled seizure baseline; it is a peri-ictal event, characteristically self-limiting, that follows a flurry of seizures across a lucid gap. The length of that interval, the usual duration of the episode and its management are developed where postictal psychosis is taught and should be recalled from there.

GOOD TO REMEMBER

A psychosis that erupts after a lucid gap following a seizure cluster is, until shown otherwise, a postictal phenomenon, and the immediate task is not a lifelong antipsychotic commitment but safety: the patient can be grandiose, agitated and at real risk to himself during a self-limiting episode. Secure him, treat the acute disturbance, coordinate with the seizure team, and let the course declare itself before you reclassify a peri-ictal event as a standing psychotic illness.

44.2 The seizures stopped and the trouble started

Good control on paper, a disturbed patient in the room. A patient whose epilepsy had been difficult for years is finally brought under tight control after a change of regimen. The seizure diary is clean, the treating team is pleased, and the recordings that were once busy with discharges have quietened. Precisely as this happens, the patient becomes psychotic, or in other presentations irritable, dysphoric and behaviourally disturbed. A clinician who has read the seizures as the whole problem is baffled: the fits are better than they have ever been, so why is the patient worse?

The counter-intuitive answer this chapter has already named is **forced normalisation**: the emergence of psychiatric disturbance, classically a psychosis, as seizure activity is suppressed and the record normalises. Its clinical cousin is the notion of an alternative psychosis, in which fits and psychosis seem to alternate rather than to coincide. The examinable move is to recognise that improving seizure control is itself on the differential for the new disturbance, so that the correct response is to rethink the regimen with the neurologist rather than to treat the psychosis as an unrelated event and drive the antiseizure drugs harder. The historical eponym attached to this phenomenon, and the mechanism proposed for it, belong to the section that develops it.

44.3 Two kinds of fear that are not the same disorder

The same emotion, one a seizure and one an anxiety disorder. Two patients describe fear. The first has brief, stereotyped, out-of-the-blue surges of intense dread that arrive without a trigger, last a short and remarkably constant time, and are sometimes accompanied by a rising epigastric sensation and a momentary lapse of full awareness with automatisms, after which she is briefly muddled. The second has longer episodes of fear that build in feared situations, come with catastrophic thoughts about dying or losing control, and leave no clouding of awareness behind them.

The temptation is to call both panic and be done. The disciplined reading uses timing and form to separate an **ictal fear**, which is the seizure itself arising from limbic structures, from an interictal panic phenomenon, which is an anxiety disorder in the settled periods between seizures. The ictal event is brief, stereotyped, unprovoked and frequently stamped with the other features of a focal seizure, the epigastric aura, the depersonalisation, the automatisms and the postictal muddle, whereas the interictal attack is longer, situationally shaped, cognitively elaborated and followed by clear consciousness. Getting this apart matters because one is treated as epilepsy and one as an anxiety disorder, and the ictal amygdala phenomenon itself is developed where ictal fear and the ictal affective states are taught.

44.4 The events that monitoring could not catch as seizures

"Drug-resistant epilepsy" that resists because it is not epilepsy. A young woman is referred with frequent, dramatic episodes labelled drug-resistant seizures despite escalating antiseizure medication. The events are prolonged, wax and wane, involve side-to-side head movements and asynchronous limb thrashing, her eyes are firmly closed and resist opening, and afterwards she recalls parts of the episode and is not deeply confused. There is a heavy load of psychological adversity in the background. Prolonged recording captures a typical event with no accompanying electrical seizure.

This is the great imitator of the epilepsy clinic, a **psychogenic non-epileptic seizure**, a functional event that mimics a fit and is confirmed not by any single bedside sign but by weighing the semiology, the recording and the psychiatric context together. The features that point away from an epileptic seizure, and the confirmatory role and limits of video-electroencephalography and the timed serum prolactin, are developed where the diagnosis is taught, as is the stepped way the news is delivered and the event is managed. As a general functional neurological disorder, it belongs within the family described in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes. The vignette's lesson is narrower and vital: escalating antiseizure drugs against events that are not seizures does harm, and the possibility of a functional event belongs on the differential of every apparently refractory case, not only after every drug has failed.

IN INDIA

Functional seizure disorders in Indian practice frequently present late and heavily stigmatised, framed by the family in spiritual or moral terms and taken first to faith healers rather than to a clinic, so the diagnosis lands on a patient already convinced her body has failed her. Definitive video-electroencephalography sits in tertiary centres alone, so much of the work of separating a functional event from a true seizure is done from a careful history, and the manner in which the diagnosis is explained carries even more weight than in the settings where the evidence base was written.

44.5 Low mood, and two different stories about suicide

A common, treatable, under-recognised depression, and a risk that arrives from two directions. A patient with established epilepsy is low, anhedonic and irritable, sleeping poorly, with a mood picture that does not keep tidily to the shape of a classical depressive episode and that fluctuates in a way the family put down, reasonably enough, to the strain of living with fits. He is not being screened.

Two examinable points collide in this case. The first is that depression is among the commonest and most missed psychiatric comorbidities of epilepsy, that its phenomenology can depart from the textbook episode, and that it warrants active screening with an instrument designed for the epilepsy setting. The second is **attribution** applied to suicide risk, which in epilepsy runs along two separate tracks that must never be merged: a risk intrinsic to the disease, elevated on its own account against the general population, and, quite separately, a class-level signal that the antiseizure drugs may raise suicidality, flagged by a drug regulator and demanding its own

vigilance and counselling. The magnitude of the intrinsic risk, the identity of the epilepsy-specific screen, and the regulator behind the class warning belong to the sections that own them.

PITFALL

The habitual error here is to read low mood in a patient with epilepsy as an understandable reaction to a hard life and to stop there, screening no further. Depression in epilepsy is common, treatable and frequently atypical, and the suicide risk sits above that of the general population before any drug is even considered, so "of course he is low, look at what he lives with" is precisely the sentence that lets a treatable and dangerous condition go unaddressed. Normalising the mood is not the same as assessing it.

44.6 Is it the epilepsy, or is it the tablet?

The question that recurs more often than any other in this field is whose fault a symptom is. A patient becomes irritable, low and cognitively slowed some time after a change to the antiseizure regimen. The family attribute it confidently to the epilepsy, to more seizures, or to the brain disease itself, and ask for reassurance. The cross-sectional picture cannot settle it: apathy, slowed thinking and mood change look the same whether the disease or the drug produced them.

Working the case means holding both authors open and interrogating the one the family have not suspected. The temporal link to a recent drug change or dose increase, the known behavioural and cognitive profile of the particular agent, and the response to adjusting it are what break the tie, because several antiseizure drugs carry recognised psychiatric and cognitive adverse effects that are dose-related and reversible. The individual offending agents and their signature effects, and the cognitive burden attributable to the epilepsy and to repeated seizures themselves, are deliberately taught as two separate topics, so that the reader must assign the picture to disease or to drug rather than to a single comfortable cause. A woman of childbearing potential complicates it further, because the agent most implicated in mood questions may also be the one whose use in pregnancy is most fraught; the mood-stabiliser lens on these drugs, their bipolar indications and monitoring, belongs to the Mood disorders: pharmacotherapy notes, while this chapter keeps only the antiseizure lens.

44.7 The diary, the deity and the sticky conversation

A personality, changed slowly, that the examiner wants named. A patient with long-standing temporal lobe epilepsy is described by his family as profoundly changed over years rather than in any single episode. He writes compulsively and at length, filling notebooks with detailed private reflections; his life has acquired an intense religious and philosophical preoccupation; his conversation has become circumstantial and hard to bring to a close, so that visitors cannot easily leave; and his sexual interest has altered. He is not psychotic and not acutely disturbed; this is a durable interictal change of the person.

The vignette is a portrait of the eponymous interictal behavioural cluster associated with temporal lobe epilepsy, the excessive writing, the deepened religiosity, the stickiness of thought and social engagement, and the altered sexuality, which a minority of such patients develop and which the examiner expects you to recognise from the description and name. It sits against the

broader, commoner **interictal personality change** of which **viscosity**, that same stickiness of thought and difficulty disengaging, is the marker. The two are held apart as a general phenomenon and a named syndrome, and the anatomy of the temporal lobe that underlies both is developed where temporal lobe epilepsy is taught. Naming the cluster is the mark here; reconstructing the eponym from memory, rather than recalling it from the section that owns it, is where candidates stumble.

44.8 Working the next one: turning cases into a habit

The unseen vignette is the same problem in new clothes. The cases above were chosen to cover the peri-ictal syndromes, the psychoses, mood and suicide, the functional imitator and the drugs, but their value is not as a handful of facts to memorise. It is that they share a single method, and once that method is a reflex the unseen case becomes tractable, because it is only ever a fresh surface over one of a small set of discriminations. The accompanying table indexes the worked cases by the surface answer each baits and the disciplined move that resolves it; read it as a rehearsal, not as a summary of the syndromes themselves, which are owned and developed by their own sections.

THE PICTURE PRESENTED	THE FAST, SURFACE ANSWER	THE DISCIPLINED MOVE THAT RESOLVES IT
Florid psychosis after a seizure cluster	"a new psychotic illness"	find the lucid interval and place it as a postictal event
Disturbance as the fits come under control	"an unrelated psychosis, push the drugs"	consider forced normalisation and rethink the regimen
Sudden surges of intense fear	"panic disorder"	separate an ictal fear from an interictal anxiety by timing and form
Dramatic, drug-resistant "seizures"	"escalate the antiseizure drugs"	weigh a functional event and confirm it with monitoring
Low mood and thoughts of self-harm	"an understandable reaction"	screen actively and separate disease-intrinsic from drug-attributed risk
Irritability and slowing after a drug change	"the epilepsy is worsening"	interrogate the drug before blaming the disease
A slowly, deeply changed personality	"he has simply aged"	recognise the interictal cluster and name it

An index of the worked cases: the resolving move is the examinable skill; the specifics it retrieves are owned and developed by the individual topics and are not restated here.

Two errors recur across all of these, and a single mnemonic then gathers the discriminations the cases turned on.

RULE **Reason the case; do not recognise it.** A vignette rewards the clinician who runs a sequence, timing then attribution then exclusion, over the one who pattern-matches the surface to the nearest remembered syndrome. Pattern recognition is fast and, in this field, reliably wrong, because the examiner builds the case so that its surface points one way and its resolving datum points another. The mark is in the datum, not the impression.

TRAP **Answering the phenomenology instead of the diagnosis.** Candidates name what the picture looks like, a psychosis, a panic attack, a depression, a seizure, and stop, when the examiner has deliberately made the surface appearance and the true diagnosis diverge. The fear that is a seizure, the psychosis that is a postictal event, the seizure that is functional: each is missed by the reader who answers what it resembles rather than what, once placed and attributed, it actually is.

MNEMONIC

Every case is a PAIR to prise apart

The vignette payoff in one word: each classic question hides two look-alikes that must be separated. **P**ostictal states, the delirium that follows a fit directly against the psychosis that follows across a lucid gap. **A**ttribution, the disease against its drug, in mood, cognition and suicidality alike. **I**ctal versus interictal, the fear that is a seizure against the anxiety that is not. **R**eal versus functional, the epileptic seizure against its psychogenic imitator. Prise the pair apart and the case is answered; every letter names only a discrimination this chapter has already drawn.

Do not answer what the vignette looks like; find the one datum, the timing to the seizure, the drug chart, the recording, that tells you what it is, and name that.

Lucid gap	**Drug chart**	**Last fit**	**The recording**
POSTICTAL PSYCHOSIS, NOT DELIRIUM	THE DRUG, NOT THE DISEASE	WHERE THE SYMPTOM SITS IN TIME	A SEIZURE, OR A FUNCTIONAL EVENT

45 · Consolidation and quick review

This closing section teaches nothing new; it makes what came before retrievable. A chapter on epilepsy and psychiatry is unusually easy to read and unusually hard to recall, because its facts arrive as a long series of syndromes that look alike on the surface and separate only on detail. The examiner exploits exactly that. Questions rarely ask you to define a single entity in isolation; they hand you a patient and ask you to place the disturbance, exclude the imitator, and decide who is to blame, the disease or the drug. What follows is built for that skill. It gathers the chapter into one reasoning frame, then hands you an answer-free set of prompts to test against, so revision becomes active retrieval, not a comfortable re-reading that flatters the memory.

The prompts here deliberately withhold their answers. Each names a construct developed earlier and asks you to supply the number, interval, criterion or eponym that belongs to it; a prompt you cannot answer is a gap to close before the examination, and far better found here than in the hall. Use this section last, once the individual topics are read.

45.1 How to use this review: test, do not re-read

The method matters as much as the material. The strongest predictor of what survives to the examination is not how many times a passage has been read but how many times it has been retrieved from a blank page. **Active recall**, the effortful pulling of an answer from memory before checking it, builds durable access in a way that re-reading, which feels productive precisely because it is easy, does not. Pair it with **interleaving**: rather than drilling all the psychoses together and then all the mood syndromes, shuffle the prompts so a postictal question is followed by a valproate question is followed by a personality one, because the examination itself is interleaved and the skill tested is discrimination across categories, not recall within one.

Spaced, repeated passes compound the gain: a syndrome retrieved today, again after a few days and again after a week, is far more secure than one crammed the night before. The prompts below are short and answer-free.

- Cover the rest of the page and attempt the prompt aloud or on paper before you look anything up.
- For every number you cannot produce, mark it and return to the owning topic, not to this list.
- Shuffle the domains between passes so that no answer is primed by the one before it.
- Re-test the items you missed after a short delay, not immediately, since immediate success is mostly short-term memory.

RULE **Retrieve before you review.** Reading a fact again and recognising it is not the same as being able to produce it unprompted, and the examination only rewards production. Treat every prompt in this section as a closed-book attempt first; the checking comes second. A fact you had to fight for is a fact you will keep.

45.2 The one frame that ties the chapter together

Almost every question in this field is a variation on three questions asked in order. Faced with a psychiatric presentation in a patient known to have epilepsy, the disciplined clinician does not begin with the psychiatric differential at all. The first question is *when*: where does the disturbance sit in relation to the seizure? Placing it on the **peri-ictal axis**, the temporal framework introduced at the front of this chapter and owned there in full, narrows a sprawling differential to a short candidate list, because each position on that axis carries only a handful of syndromes. The second is *whose*: the epilepsy itself, an **attribution** to the antiseizure drug, or an independent psychiatric disorder that happens to co-occur? That one decision separates the cognitive slowing of the disease from that of the medication, and the mood change of the illness from the class-effect signal of the drug, and a large share of marks turns on it. The third is *what else*: what imitator must be excluded before the label is accepted, whether a **psychogenic non-epileptic seizure** masquerading as a fit or a primary psychiatric disorder masquerading as an interictal syndrome?

Held together, these three questions are the spine of the chapter, and the individual topics are their elaborations: a postictal psychosis is a *when* answer, the class warning on the antiseizure drugs is a *whose* answer, and the separation of a seizure from a functional event is a *what else*

answer. Learned as a list of syndromes the chapter feels endless; learned as three questions with named answers it becomes examinable.

MNEMONIC

SEIZE the timeline

The whole diagnostic approach, held as one word. **S**ituate the symptom in time, its relation to the seizure, on the peri-ictal axis. **E**xclude the imitator: a functional event for a fit, a primary disorder for an interictal state. **I**dentify the phenomenology: fear, psychosis, dysphoria, personality change or cognitive decline. **Z**ero in on attribution: the epilepsy, the drug, or an independent disorder. **E**nsure safety and treat: gauge the suicide risk and choose agents with the seizure threshold in mind. Each letter names only a step this chapter has already taught.

In a patient with epilepsy, no psychiatric symptom is fully described until you have fixed its timing to the seizure and decided whether the disease, the drug, or an independent disorder produced it.

45.3 The peri-ictal axis: what you must be able to recall

The spine of the chapter, tested position by position. The peri-ictal band is where the most easily confused material lives, because the syndromes clustered around the seizure share a phenomenology and separate mainly on their timing, their duration, and the presence or absence of a symptom-free gap after the fit. The prompts walk the axis from before the seizure to the long interictal baseline; supply the withheld specifics yourself, since each belongs to a topic developed earlier and is deliberately not repeated here.

- State the temporal windows of the peri-ictal axis in order, and place each named syndrome on it without looking.
- Give the duration over which preictal dysphoria and irritability typically build before a seizure.
- For postictal psychosis, recall the length of the characteristic lucid interval that precedes it, how long the episode usually lasts, and the seizure pattern that most often precedes it.
- Distinguish postictal confusion and delirium from postictal psychosis on the single feature of the intervening lucid period.
- Recall the phenomenology of ictal fear and ictal panic and the one feature that separates them from an interictal panic attack.
- Name the interictal mood syndrome of epilepsy characterised by an intermittent, pleomorphic dysphoria, and list its component symptoms.

TRAP **Treating the psychoses of epilepsy as one entity.** Candidates lose marks by answering a question about ictal, postictal or chronic interictal psychosis as though the three were interchangeable, when what the examiner is testing is precisely their separation. They are distinguished by their timing to the seizure, by whether a lucid interval intervenes, by their duration, and by the state of the record between attacks. Fix which psychosis is being asked about before you write a word about its features.

45.4 The psychoses of epilepsy and forced normalisation

Three psychoses, plus the paradox. Beyond the peri-ictal band sits the chronic, schizophrenia-like psychosis that arises in the interictal baseline, and alongside it **forced normalisation**, the counter-intuitive phenomenon in which behavioural disturbance emerges as seizures are suppressed and the record quietens. The examinable skill is holding these apart from one another and from a primary psychotic illness, and the prompts target exactly the discriminators you are expected to produce.

- Recall the clinical features of the schizophrenia-like psychosis of epilepsy and the seizure history against which it typically develops.
- Recall the historical eponym for forced normalisation and how it relates to the notion of an alternative psychosis.
- Give the three features that mark a psychosis as epileptic rather than as a primary psychotic disorder.
- State the antipsychotic considerations specific to a patient with epilepsy, and where the seizure-threshold ranking of agents is developed.

The last prompt points across a boundary examiners like to probe. The discrimination of epileptic psychosis from primary schizophrenia is owned by its own topic, and the phenomenology of the primary illness belongs to a separate chapter, the Schizophrenia: aetiology, phenomenology, classification and course notes, which is where the comparator should be read rather than reconstructed from memory. This chapter owns the epilepsy-related psychoses and their separation from a primary disorder; it does not own the primary disorder itself.

GOOD TO REMEMBER

When a patient's fits have just come under good control and a psychosis then appears, do not reflexively read it as a new, unrelated illness. The improving seizure control may itself be driving the disturbance, and the presentation belongs on the differential of the alternative-psychosis phenomenon before it is called anything else. A psychosis that arrives as the seizures recede is a prompt to rethink the regimen, not to press on with it unchanged.

45.5 Mood, anxiety and suicide: the disease and the drug

The domain where attribution decides the answer. Depression is among the commonest psychiatric comorbidities of epilepsy and among the most under-recognised, and its phenomenology does not always keep to the textbook shape of a primary depressive episode.

Suicide risk is elevated in epilepsy on its own account, and, separately, a regulatory signal implicates the antiseizure drugs as a class. These two suicidality stories must never be conflated: one is intrinsic to the disease, the other attributed to the medication. The prompts keep them apart.

- Recall the screening instrument designed specifically to detect depression in epilepsy and what makes it suited to that setting.
- Give the magnitude of the elevated suicide risk in epilepsy expressed against the general population, and the factors that raise it further.
- Name the regulator that issued the class warning on antiseizure-drug-attributable suicidality, and how that signal changes prescribing and monitoring.
- State how the phenomenology of depression in epilepsy can depart from a classical major depressive episode.
- Recall the anxiety presentations of epilepsy and the one that is most often mistaken for a primary panic disorder.

Antidepressant choice here follows the same seizure-threshold logic as antipsychotic choice, and the mechanism behind that logic is owned by its own topic rather than repeated across every prescribing decision. When a prompt asks which agents are safest, the answer is an application of that shared principle, not a separate list to memorise in isolation.

45.6 Personality, behaviour and cognition

Where disease and drug are hardest to tell apart. The interictal personality changes of temporal lobe epilepsy, the eponymous behavioural cluster a minority develop, the episodic aggression that is far more often interictal and non-directed than a true ictal act, and the cognitive trajectory of the illness all sit in this domain, and all force the same question: is this the epilepsy, or is it the medication? That attribution is the whole examination point, and it is why the cognitive effects of the disease and the cognitive adverse effects of the drugs are taught as two separate topics that must not be collapsed into one.

- List the components of the eponymous interictal behavioural syndrome of temporal lobe epilepsy, and name it.
- Distinguish the general interictal personality change, of which viscosity is the marker, from that named syndrome.
- Recall the pattern of aggression and episodic dyscontrol in epilepsy and why a directed, purposeful assault is rarely a true ictal phenomenon.
- Separate the cognitive effects attributable to the epilepsy and to repeated seizures from the cognitive and behavioural adverse effects of the antiseizure drugs.
- Recall the psychiatric outcomes, both improvement and the de novo disorders, that can follow epilepsy surgery.

PITFALL

The commonest clinical error in this domain is single-cause thinking: attributing a patient's mental slowing, irritability or low mood entirely to the epilepsy while the antiseizure drug quietly drives it, or the reverse. Before the disease is blamed, the drug and its dose, its recent changes and its known behavioural profile must be interrogated, and before the drug is blamed, the seizure burden and the underlying lesion must be weighed. Attribution here is rarely clean; the discipline is holding both candidates open until one is excluded.

45.7 Functional events, and the chapters next door

The great imitator, and the material this chapter borrows. A psychogenic non-epileptic seizure is the imitator the epilepsy clinic must exclude, and the skill is not a single sign but a weighing of the semiology, the investigations and the psychiatric context. The prompts below cover that ground and then point outward: several specifics a candidate reaches for in this field are owned by other chapters, and it is worth knowing where they live so the borrowed material is read at its source rather than half-remembered here.

- Give the clinical features that most reliably separate a functional non-epileptic event from an epileptic seizure at the bedside.
- Recall the role of **video-electroencephalography** and of the timed serum prolactin, and the limits of each, in confirming the diagnosis.
- State how the diagnosis should be communicated and the stepped psychological and pharmacological management that follows.
- Recall the prognosis of a functional seizure disorder and what changes when it coexists with genuine epilepsy.

The material this chapter leans on but does not own is worth naming so that revision goes to the right place. The general construct of a functional neurological disorder, within which the functional seizure sits, is developed in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes. The mood-stabiliser lens on valproate, carbamazepine and lamotrigine, their bipolar indications and monitoring, belongs to the Mood disorders: pharmacotherapy notes, while this chapter keeps only the antiepileptic lens. The full adverse-effect profile of clozapine, beyond the seizure-threshold angle, is the Schizophrenia: management, antipsychotics and adverse effects notes. Delirium in its own right, with its formal assessment and prevention, is the Stroke, TBI, delirium and focal brain syndromes notes, and the full anatomy of the limbic system is the Functional Neuroanatomy and Neurophysiology notes. Cross-refer to these by name; do not try to carry their numbers in this chapter's memory.

IN INDIA

Several of these topics carry a distinctly Indian answer. Valproate in a woman of childbearing potential is a harder decision where reliable contraception and pre-conception planning are less assured, and the drug remains widely prescribed on grounds of cost and availability. Phenobarbital continues in first-line use in many resource-limited settings for the same reasons, which shapes both the seizure control the psychiatrist inherits and the behavioural and cognitive burden that comes with it. A functional seizure disorder often presents late and heavily stigmatised, sometimes framed by the family in spiritual rather than medical terms, so the way the diagnosis is communicated matters even more than in the settings where the evidence base was written. Video-electroencephalography sits in tertiary centres alone, so much of the peri-ictal placement and the exclusion of a functional event is a clinical judgement made from a careful history rather than from a recording.

45.8 The master map, and walking into the examination

One table to sweep the whole field before you turn the paper. The map below compresses the chapter to a single view: each domain, the discrimination the examiner turns on, and the withheld targets you should be able to produce. Read it as a self-test: if any recall target is blank, the owning topic is where to go, not this cell.

DOMAIN	THE DISCRIMINATION THE EXAMINER TURNS ON	RECALL TARGETS (SUPPLY THE ANSWERS)
Classification and localisation	focal versus generalised onset, and which localisation is psychiatrically loud	the temporal-lobe and frontal-lobe behavioural signatures and their non-epileptic mimics
The peri-ictal axis	place any symptom by its timing to the seizure	the temporal windows and which syndrome occupies each
The psychoses of epilepsy	ictal versus postictal versus chronic interictal, plus forced normalisation	the lucid interval, the typical durations, and the interictal record state for each type
Epileptic versus primary psychosis	what an epileptic psychosis preserves that a primary disorder loses	the three features that mark it as epileptic rather than primary
Mood, anxiety and suicide	the epilepsy-intrinsic story versus the drug-attributed signal	the epilepsy-specific screen, the suicide-risk magnitude, and the regulator warning
Personality, behaviour and cognition	disease versus drug attribution	the interictal change, the eponymous cluster, and the drug cognitive profile
Functional events and the drug interface	functional event versus true seizure, and mood-stabiliser versus antiepileptic lens	the differentiating semiology, the confirmatory investigations, and where the mood-stabiliser evidence lives

A revision map of the chapter: the withheld recall targets are owned and developed by the individual topics and are named here only to be tested, never re-taught.

When the question actually arrives, run the three questions of the master frame in order and let them route the answer: situate the disturbance in time, decide whether the disease, the drug, or an independent disorder is responsible, and name the imitator you have excluded to reach the label. A candidate who reasons this way in the viva, rather than reciting a syndrome from the middle, shows exactly the discrimination the examination is built to reward, and the individual facts fall into place around it because they were learned as answers rather than as a list. The chapter is long, but the skill it teaches is short: timestamp, attribute, exclude.

When	Whose	What else	Then treat
PLACE IT ON THE PERI-ICTAL AXIS	THE DISEASE, THE DRUG, OR INDEPENDENT	THE IMITATOR TO EXCLUDE	SAFETY, AND THE SEIZURE THRESHOLD

References

The canonical sources this chapter is grounded in. Every fact-bearing specific in the notes traces to one of these; where a primary paper is named in the text, it is cited inline.

1. David AS, Fleminger S, Kopelman MD, Lovestone S, Mellers JDC. Lishman s Organic Psychiatry: A Textbook of Neuropsychiatry. 4th ed. Wiley-Blackwell.
2. Boland R, Verduin ML, eds. Kaplan and Sadock s Comprehensive Textbook of Psychiatry. 11th ed. Wolters Kluwer; 2024.
3. Kaufman DM, Geyer HL, Milstein MJ. Kaufman s Clinical Neurology for Psychiatrists. 9th ed. Elsevier.
4. Taylor DM, Barnes TRE, Young AH. The Maudsley Prescribing Guidelines in Psychiatry. 15th ed. Wiley-Blackwell.
5. Standard EEG references (Niedermeyer s Electroencephalography; clinical EEG atlases).
6. Martin A, Bloch MH, Volkmar FR, eds. Lewis s Child and Adolescent Psychiatry. 5th ed. Wolters Kluwer.
7. Brunton LL, et al, eds. Goodman and Gilman s The Pharmacological Basis of Therapeutics. McGraw-Hill.
8. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 5th ed, Text Revision (DSM-5-TR). APA; 2022.
9. World Health Organization. ICD-11 Clinical Descriptions and Diagnostic Requirements (CDDR). WHO.
10. Johnstone EC, et al, eds. Companion to Psychiatric Studies. Churchill Livingstone.

Additional primary sources cited inline: Dulcan's Textbook of Child and Adolescent Psychiatry, Panksepp Affective Neuroscience, RC Jiloha Forensic Psychiatry.

PART II

II

Neurology Foundations for Psychiatrists

The neurology a psychiatrist actually needs: where a lesion puts itself, the headache that is not migraine, and the sleep disorder hiding behind the mood complaint.

01 The bedside examination, what it localises, and the investigations it earns

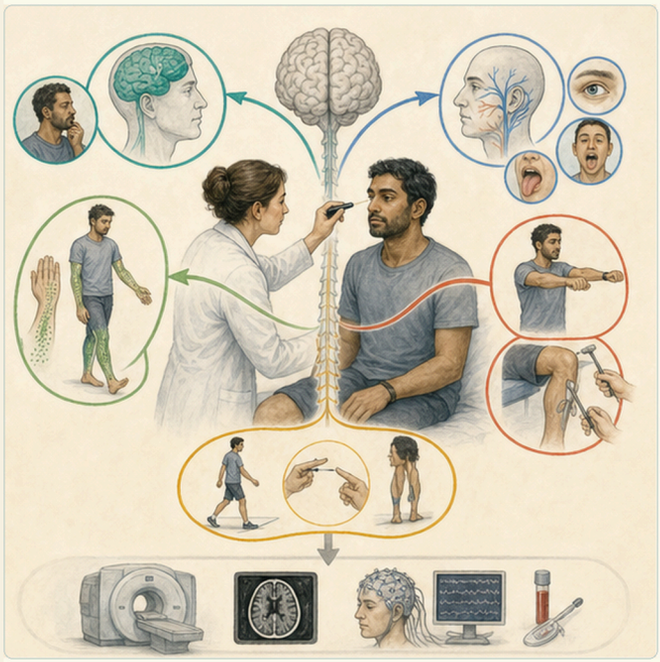
02 Headache and the psychiatric interface

03 Sleep: the architecture and its disorders

04 Consolidate and apply

05 References

VISUAL 2.1



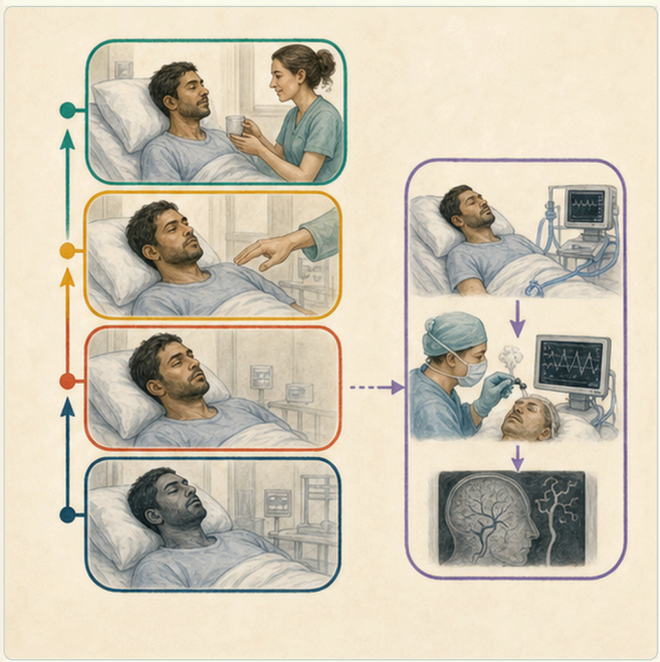
The neurological examination earns localisation

- Higher functions
- Cranial nerves
- Motor and reflexes
- Sensory
- Cerebellar and gait
- Targeted investigations

The examination is organised to localise the lesion first and to justify targeted investigation second.

Source lock: day-31.final.html#sec-t02

VISUAL 2.2



Disorders of consciousness as observable states

- Coma
- Unresponsive wakefulness
- Minimally conscious state
- Emergence
- Brain-death determination

Consciousness states are distinguished by arousal and reproducible purposeful behaviour; brain-death determination is a separate clinical process.

Source lock: day-31.final.html#sec-t07

VISUAL 2.3



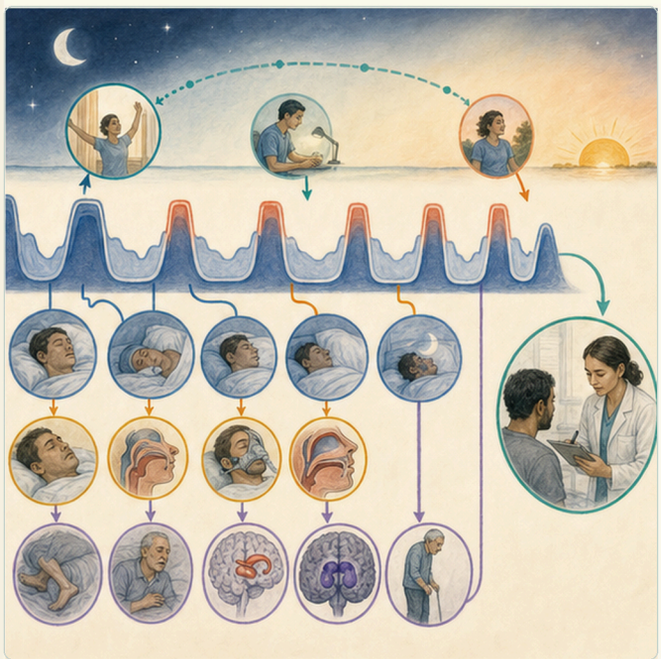
Headache at the psychiatric interface

- Primary headache
- Red flags
- Psychiatric comorbidity
- Medication overuse
- Prevention

Headache assessment holds primary headache, secondary red flags, psychiatric comorbidity, medication overuse and prevention in one formulation.

Source lock: day-31.final.html#sec-t08

VISUAL 2.4



Sleep architecture connects night to diagnosis

- NREM
- REM
- Circadian rhythm
- Breathing
- Movement and neurodegeneration

Sleep complaints become clinically legible when architecture, circadian timing, breathing and movement phenomena are examined together.

Source lock: day-31.final.html#sec-t12

PAPER IV

Neurology foundations for psychiatrists

Three bands . one complete notes document

The bedside examination, what it localises, and the investigations it earns

1 · Neurology for the psychiatrist: what this chapter owns, and what it points to

Orientation is the gatekeeper of the neurological assessment in psychiatry. It establishes whether the patient can take part in a meaningful interview, follow the examination, retain the immediate purpose of the encounter and give information that can safely be interpreted. This is not a decorative opening question. It is the first safeguard against mistaking a disorder of attention, arousal, memory, language, perception or consciousness for a purely psychiatric account of behaviour.

For the psychiatrist, orientation is best understood as a bedside judgement about the patient's current relationship to self, place, time and situation. The answer matters less as a recital of isolated facts than as evidence about how the patient is organising present experience. A person may state a fact correctly yet have reached it by guesswork, rehearsal, cueing from relatives or a fragmentary understanding of the encounter. Conversely, an anxious, frightened, poorly educated or sensory-impaired patient may hesitate without being globally disoriented. The examiner therefore asks, listens, observes and then decides how much weight the remainder of the interview can bear.

1.1 What orientation tests at the bedside

Orientation is the capacity to locate oneself coherently in the immediate world. In clinical work it is not a single faculty. It is the visible product of sufficient wakefulness, sustained attention, registration of current information, access to autobiographical knowledge, language comprehension and the ability to communicate an answer. The patient must also understand the social meaning of the question. A reply to a question about place is only useful if the patient knows that the question concerns present location rather than domicile, birthplace, workplace or an imagined setting.

The practical value of orientation lies in its economy. It gives the examiner an early sample of contact, comprehension and reliability before the history becomes detailed. It also changes the stance of the interview. When orientation is sound, a psychiatrist can generally proceed to the higher-function, affective, psychotic and risk assessment while remaining alert to focal clues. When orientation is doubtful, the task becomes more basic: establish the level and fluctuation of awareness, obtain collateral information, examine for acute medical or neurological contributors and avoid treating every unusual statement as a belief held with normal alertness and cognitive access.

Orientation is related to, but is not synonymous with, **attention**. A person can give a practised answer to routine orientation questions while being unable to hold the thread of conversation. Equally, an attentive person may be uncertain about an unfamiliar environment without having a global disturbance. The difference is clinically important because attention tells the examiner whether information is being taken in now, while orientation tells the examiner whether the patient can place current experience into a usable frame. A brief answer never replaces observation across the interview.

Nor is orientation simply a test of factual knowledge. A patient may know general public facts yet be unable to identify the current setting, or may fail a conventional question because the setting has been changed, renamed or poorly explained. The clinician should seek the level of practical orientation needed for the immediate decision: Can the patient understand who is speaking, where care is occurring, what has happened and what is being proposed? This makes orientation clinically useful in unfamiliar wards, emergency settings, homes, clinics and remote encounters alike. It also prevents needless confrontation over a detail that has little bearing on the patient's present capacity to engage.

RULE **Treat orientation as evidence about the reliability of the encounter, not as a pass-or-fail recital.** Interpret each answer with its route to the answer, the patient's alertness, language, sensory access and the consistency of the surrounding interview.

1.2 The domains and their clinical meaning

Inquiry commonly moves through orientation to person, place, time and situation. These domains should be explored conversationally, using questions suited to the patient's language, culture and immediate context. The aim is not to catch the patient out. It is to determine whether the internal account is coherent, whether uncertainty is acknowledged appropriately and whether an answer remains stable when the question is approached from another angle.

- **Orientation to person** concerns the patient's sense of personal identity and recognition of relevant people. A difficulty here can reflect a disturbance of self-knowledge, memory, recognition, language, psychosis or the meaning of the social interaction. It should prompt careful exploration rather than an immediate assumption about severity.
- **Orientation to place** asks whether the patient can identify the present setting in a way that fits the encounter. Confusion may be revealed not only by a wrong location but by a plausible location that does not explain why the patient is there.
- **Orientation to time** samples the patient's grasp of temporal context. The useful clinical question is whether the patient can situate the present moment sufficiently to make sense of events, medication, admission, sleep and symptom onset, not whether every calendar detail can be recited.
- **Orientation to situation** asks whether the patient understands what is happening and why professional contact is occurring. It connects cognitive orientation to judgement, insight and the immediate practical demands of care.

These domains should not be forced into a false hierarchy. Their pattern may help the clinician describe the problem, but no domain by itself locates a lesion or establishes a diagnosis. The anatomical framework belongs in the Functional Neuroanatomy and Neurophysiology notes, and the detailed neurological examination belongs in the companion examination section of this chapter. At this bedside stage, the psychiatrist's duty is to recognise when the pattern is discordant with the proposed psychiatric formulation and to make that discordance visible in the record.

MNEMONIC

PERSON - PLACE - TIME - SITUATION

Use this as a quiet mental sweep, not a script. After each domain, ask whether the answer is coherent, independently generated and consistent with the patient's alertness and the practical purpose of the assessment.

1.3 How to ask without creating an artefact

Good orientation testing begins before the formal question. Notice whether the patient turns toward the examiner, tracks the topic, responds after a reasonable pause, asks for clarification when necessary and recognises why questions are being asked. Observe whether the patient is hearing adequately, can see environmental cues, has access to a familiar language and is being overwhelmed by pain, breathlessness, fear, intoxication, fatigue or a crowded clinical setting. These conditions can alter performance without proving a disorder of orientation.

Questions should be simple, direct and open enough to show the patient's own frame of reference. Ask for clarification rather than supplying the answer. If an answer is uncertain, revisit it later in a naturally different form. This distinguishes a transient lapse, a misunderstood question and a stable inconsistency. It also keeps the examination humane. A patient who feels tested may become guarded, oppositional or ashamed, especially when already frightened by hospitalisation. Such reactions are data about the interaction, not proof that cognition is intact or impaired.

Collateral information is part of this task, not an afterthought. Relatives, nursing staff and records can clarify the patient's usual baseline, recent change, fluctuations, exposure to substances or medicines, and the circumstances of presentation. Their account should support rather than replace direct examination. The psychiatrist should record the source and distinguish observed behaviour from reported information. When the patient cannot participate reliably, the history becomes a reconstruction, and the limits of that reconstruction must be stated plainly.

TRAP **A correct answer is not automatically an intact function.** Rehearsed responses, environmental prompting and preserved fragments of knowledge can conceal impaired attention or a fluctuating state; judge the answer in the flow of the interview.

The examiner must reduce avoidable disadvantage before interpreting failure. Use the patient's preferred language where feasible, arrange communication support where needed and

make sensory aids available. Consider whether unfamiliarity with formal institutions, literacy demands or cultural assumptions have changed the question's meaning. This is not a lowering of clinical standards. It is how the examination becomes specific to the function it claims to assess rather than to the patient's social comfort with the examiner.

1.4 Separating orientation from the psychiatric mental state

Disorientation can coexist with depression, mania, anxiety, psychosis, dissociation, substance-related states and behavioural disturbance, but it must not be absorbed into any of them without thought. The content of a statement and the conditions under which it is produced are separate clinical questions. A patient may voice an implausible explanation because they hold a delusional belief, because they are confused about their surroundings, because language has failed, because they are attempting to fill a gap in memory, or because they do not understand the question. These possibilities can overlap. Their distinction determines whether the interview is primarily a phenomenological assessment, an urgent medical assessment or both.

Insight is also not interchangeable with orientation. A person may accurately identify place and purpose while rejecting illness, declining treatment or interpreting events through a fixed belief system. Conversely, a person may express apparent acceptance of illness while being unable to describe why they are in the setting or what has just occurred. Psychiatry often rewards a richly elaborated account, but elaboration should not be mistaken for reliability. The more fluent the narrative, the more important it is to check whether it remains anchored to the immediate context.

Dissociation deserves the same discipline. Experiences of unreality, detachment, altered identity or gaps in recall can be clinically compelling, yet they do not remove the need to examine alertness, attention and orientation. The psychiatrist should neither dismiss a dissociative account as feigned confusion nor label a confused patient as dissociative because the history sounds psychologically meaningful. The relevant phenomenology is covered in the Anxiety, OCD and trauma-related disorders notes and the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes; at the neurological bedside, the immediate job is to establish what the patient can currently know, hold and use.

Formal cognitive screens and more extensive cognitive assessment are not substitutes for this bedside reasoning. Their selection, scoring and interpretation belong in the Psychometry, Assessment and Descriptive Psychopathology notes. Here, an abnormal orientation response should lead to a richer account of the patient's functioning, not to a score-driven conclusion divorced from arousal, communication and collateral evidence.

PITFALL

Do not call an implausible answer "psychotic" before establishing that the patient understood the question, could attend to it and can place the encounter in present reality. Content without cognitive context is an unsafe shortcut.

1.5 Recognising when orientation changes the clinical priority

Fluctuation is often more informative than a single failed item. A patient who is coherent in one moment and unable to sustain the same frame of reference later requires repeated observation and a timeline from those who have been present. Variation in alertness, attention, speech, behaviour or orientation should move the clinician away from a one-off mental-state label and toward an account of changing brain function. This does not itself establish cause, but it changes urgency and broadens the differential.

Do not confuse apparent improvement with reliable recovery. A patient may seem clearer after rest, reassurance or environmental simplification, yet still be unable to maintain the new level of engagement when demands increase. In the same way, an apparently impaired response may improve once pain is relieved, a familiar person is present or communication is made accessible. Serial assessment therefore asks a simple but powerful question: is the patient's orientation stable enough for the decision now required? The answer can be qualified. It need not become a claim of normality before the psychiatrist can communicate safely with the wider team.

Where orientation is impaired or cannot be confidently assessed, the psychiatrist should first make the encounter safe and information-rich. Confirm immediate physiological and behavioural safety through the appropriate clinical team; seek collateral; review the temporal relation to illness, injury, substances and treatment; and repeat the examination as the state permits. The detail of the physical neurological examination is taught elsewhere in this chapter. Investigation choices and exam-to-imaging reasoning are likewise separate from the imaging modalities described in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes. The important orientation lesson is that a psychiatric formulation is provisional when its cognitive platform is unstable.

The same caution applies to severe agitation or withdrawal. The observable syndrome may make a conventional interview impossible, but that is a reason to adjust method, not to abandon neurological thinking. Describe whether the patient can be engaged, whether responses are purposeful, whether orientation can be sampled at all and what prevents a reliable assessment. This preserves clinical uncertainty honestly and helps the next clinician detect change rather than inherit a misleading categorical label.

> **GOOD TO REMEMBER**
>
> When orientation is doubtful, document what the patient did, not only what they failed. The route of response, consistency, environmental cues, barriers to testing and collateral baseline often guide the next assessment more than a bare statement that the patient was "disoriented".

1.6 Recording an orientation assessment that another clinician can use

A useful note identifies the domains explored, the patient's responses in plain language, the degree of confidence in those responses and the conditions of testing. It should show whether information was volunteered, prompted or inferred from behaviour. It should also name important limitations: reduced arousal, inattention, language mismatch, hearing or visual

difficulty, distress, inability to cooperate, sedating treatment, or missing collateral. Avoid replacing these observations with a global adjective that conceals uncertainty.

Record the temporal story. Is the presentation described as new, longstanding, episodic, progressive, sudden or variable? Was the patient reportedly functioning differently earlier? Does the patient's account agree with observed behaviour and collateral reports? Such language does not make a diagnosis, but it creates a defensible bridge from the psychiatric interview to medical review. It also supports serial assessment. A later clinician should be able to tell whether the person's ability to orient has changed, or whether the apparent change merely reflects a different question, a different setting or a different level of support.

In handover, communicate the practical implication. State whether the patient can give a reliable account, participate in consent discussions, report symptoms accurately and be left to self-report risk. If uncertainty remains, say what needs reassessment and under what conditions. This is particularly important when deciding how much meaning to assign to mood, psychotic content, refusal, apparent non-cooperation or a request to leave. Orientation is not a bureaucratic field in the mental state examination. It is a declaration of the evidential quality of the rest of it.

BEDSIDE OBSERVATION	WHAT TO CLARIFY	HOW TO RECORD THE IMPLICATION
Confident response	Whether it is independently generated and fits the conversation	State the context supporting reliability
Uncertain response	Question comprehension, sensory access, anxiety and familiarity with the setting	Describe the barrier before inferring impairment
Inconsistent response	Attention, fluctuation, cueing and collateral baseline	Record variation and need for serial review
Unable to engage	Arousal, behaviour, communication and immediate safety	State that orientation cannot be reliably assessed and why

1.7 The neurological lens for psychiatry

Orientation teaches a habit that extends through the whole psychiatric examination: first establish the conditions under which a symptom can be known. The question is not merely "What does the patient say?" It is "What current brain state, communicative capacity and environmental context made that statement possible?" This protects against premature closure in both directions. It prevents the clinician from medicalising every unusual experience, but it also prevents a psychiatric diagnosis from becoming a reason not to notice altered cognition.

That habit should remain proportionate. A patient with a coherent interview, stable orientation and no discordant neurological clues does not need a theatrical neurological workup merely because they have a psychiatric syndrome. Conversely, a polished psychiatric history should not reassure the clinician when it is produced on a platform of impaired attention, changing awareness or unreliable orientation. The subsequent examination, localisation reasoning and imaging decision are covered in their designated sections. Sleep phenomena require the parallel

neurological and psychiatric framing in the Eating and sleep-wake disorders notes. Electroencephalographic questions belong with the Epilepsy and psychiatry notes.

Self	Place	Time	Situation
IDENTITY AND RELEVANT PEOPLE	CURRENT SETTING	PRESENT TEMPORAL CONTEXT	PURPOSE OF THE ENCOUNTER

Orientation is not a memory quiz: it establishes whether the psychiatric interview rests on a reliable account of present reality.

2 · The neurological examination relevant to psychiatry (higher functions, cranial nerves, motor, sensory, reflexes, cerebellar, gait)

The neurological examination is not an ornamental appendix to the psychiatric assessment. It is the bedside method that separates impaired cognition from impaired access to cognition, medication effects from primary motor disease, affective expression from corticobulbar dysfunction, and functional symptoms from deficits that obey neuroanatomy. Marks are earned by showing an orderly examination, describing the sign precisely, and stating what the pattern localises.

The safest routine is a **fixed sequence**: mental status, cranial nerves, motor system, reflexes, sensation, cerebellar system, and gait. Mental status comes first because impaired attention, comprehension, or language can make every later response unreliable. The accompanying figure should be read as a working bedside route, not as a checklist to recite without examining the patient.



Fig 31.1 **The bedside neurological examination as a structured sequence.** The seven stations run in the order given by Kaufman's Clinical Neurology for Psychiatrists (Box 1.1): mental status, cranial nerves, motor system, reflexes, sensation, cerebellar system and gait. The order is itself the teaching: mental status leads because cognitive impairment may preclude accurate assessment of every other station, and gait closes because it needs every level intact and integrated, which is what makes it the single most valuable assessment of noncognitive nervous system function. Each station earns its place in a psychiatric assessment by the misreading it prevents. Attributions per box (Kaufman's Clinical Neurology for Psychiatrists, Hutchison's Clinical Methods, Clinical Methods in Psychiatry (AIIMS)).

MIND	PATTERN	ASYMMETRY	GAIT
VALIDATES	BEATS SEVERITY	LOCALISES	INTEGRATES

2.1 Conduct, sequence, and formulation

A screening examination can usually be completed in **twenty minutes or less**, provided the examiner uses the same route every time and returns for detail where an abnormality appears. Do not abandon the sequence because an obvious deficit is found. A florid tremor does not exclude pyramidal weakness, and a psychiatric history does not make an objective cranial nerve sign less important.

MNEMONIC

Mind, Cranium, Movement, Reflex, Sensation, Coordination, Walking

Use this as a prompt for the sequence: **mind** before instructions, **cranium** for cranial nerves, **movement** for bulk, tone and power, then **reflex**, **sensation**, **coordination**, and **walking**.

The examination should answer what the patient experiences, what the examiner can demonstrate, where the dysfunction lies, and what processes could produce it. “The leg feels dead” is a symptom; a reproducible pyramidal pattern, reflex asymmetry, or inconsistency during synergistic movement is a sign. Move from description to localisation before diagnosis. Imaging follows that reasoning; full pathway anatomy belongs to the Functional Neuroanatomy and Neurophysiology notes.

RULE **Examine in sequence, interpret in patterns, and localise before naming a disease.** A single brisk reflex or uncertain sensory boundary is weak evidence; convergent findings across independent parts of the examination are much stronger.

Before testing, observe the patient entering, sitting, speaking, and reaching. Explain each manoeuvre, compare sides, and retest unexpected findings differently. If arousal is inadequate, higher-function testing cannot be interpreted. If language or hearing is impaired, alter the method rather than labelling the patient uncooperative.

2.2 Higher functions at the bedside

The bedside mental-status component samples attention and cooperation, orientation, language, memory for immediate, recent and remote events, and higher intellectual functions such as calculation and abstraction. It begins during spontaneous conversation. Note whether the patient sustains the task, understands its purpose, initiates a response, monitors errors, and persists. These observations often explain a poor test performance more accurately than the final answer alone.

Bedside language examination samples fluency, comprehension, repetition, and naming. Listen for effort, phrase length, grammatical structure, paraphasic errors, empty fluent output, and awareness of mistakes. Test comprehension with progressively less contextual commands, repetition with material that cannot be guessed from context, and naming with familiar objects and their parts. Naming is vulnerable across dysphasic syndromes. Severe comprehension impairment can invalidate apparent memory, calculation, praxis, and executive testing even when those capacities remain intact. In psychiatric practice this prevents fluent but meaningless speech

being mistaken for formal thought disorder, and non-fluent output being mistaken for mutism, depression, or deliberate refusal.

Apraxia is inability to perform a learned act despite sufficient motor and sensory capacity. Ask the patient to mime tool use, wave, salute, or copy demonstrated hand postures. Confirm that weakness, sensory loss, poor comprehension, and inattention do not explain failure. A multistep command is broader than a pure praxis test because it also recruits language, memory, sequencing, and executive control.

Agnosia is modality-specific failure of recognition despite preserved basic sensation and relevant semantic knowledge. A patient who cannot identify an object by sight may identify it by touch and may still describe its purpose. The comparison between modalities is the point. Similarly, cortical sensory testing asks whether a normally felt stimulus can be integrated and recognised, rather than whether it is detected at all.

Formal screens and neuropsychological batteries may quantify impairment, but administration and scoring belong to the Psychometry, Assessment and Descriptive Psychopathology notes. At this bedside stage, the psychiatrist should state the function tested, control for confounds, describe the error, and say whether it suggests a focal language or praxis disorder, a diffuse cognitive problem, or unreliable access to otherwise preserved function.

2.3 Cranial nerves: a psychiatry-focused sweep

Test smell one nostril at a time with a familiar, innocuous aromatic substance. Irritative substances are unsuitable because nasal trigeminal endings can detect them despite olfactory loss. Smell complaints matter because self-report is unreliable and anatomically impossible performance may reveal invalid responding. On a four-alternative forced-choice task, performance **below twenty-five per cent** is below chance and cannot be explained by genuine anosmia alone.

For the optic nerve, assess acuity, fields by confrontation, and the fundi. Examine each eye independently before comparing them; binocular field testing can miss a chiasmal pattern. In the **pupillary light reflex**, the optic nerve carries the incoming signal and the oculomotor nerve carries the constrictor response. Light in either eye should constrict both pupils. Failure after stimulation from a particular eye, with preserved bilateral responses from the other, indicates an afferent defect. Preserved consensual constriction with failure of a particular pupil to constrict indicates an efferent defect on that side.

Inspect lids, pupils, resting eye position, and conjugacy, then test pursuit while asking about diplopia. Oculomotor impairment combines ptosis, a dilated pupil, and outward deviation. Abducens impairment produces inward deviation without lid or pupil findings. Trochlear injury may produce compensatory head tilt away from the affected side. Dysconjugacy should not be attributed to cortical dementia: preserved conjugacy despite advanced cerebral disease directs abnormal findings toward the brainstem, peripheral nerve course, or neuromuscular transmission.

Test facial sensation in the forehead, malar region, and jaw distribution, preserving the anatomical boundaries. The angle of the jaw is not supplied by the trigeminal territory, and complete facial loss that stops sharply at the hairline is anatomically suspect. In the **corneal reflex**, the ophthalmic trigeminal division is afferent and facial nerves provide the bilateral blink. Absent blinking after stimulation from a particular cornea, with bilateral blinking from the other, points to an afferent lesion; failure of a particular eyelid to close whichever cornea is stimulated points to an ipsilateral facial efferent lesion.

Observe the face at rest and during forehead wrinkling, eye closure, and smiling. A peripheral lesion weakens the ipsilateral upper and lower face; a central corticobulbar lesion predominantly weakens the contralateral lower face because upper facial movement has bilateral cortical input. Hearing can be screened by whispered material presented to each ear separately. Unilateral central lesions do not produce deafness because auditory representation is bilateral; apparent unilateral deafness beside hemiparesis deserves anatomical review.

For bulbar function, listen to spontaneous speech and ask for repeated lingual, labial, and pharyngeal syllables, then inspect palatal elevation during phonation. Lower motor bulbar and corticobulbar syndromes share dysarthria and dysphagia, so these do not make the distinction. Hypoactive gag and jaw responses, respiratory involvement, and absent emotional lability favour bulbar dysfunction; brisk gag and jaw responses with emotional lability and cognitive impairment favour pseudobulbar dysfunction. In **pseudobulbar affect**, crying or laughter is involuntary and dissociated from the underlying mood; true depression may nevertheless coexist. Finally inspect the tongue at rest and on protrusion: a unilateral hypoglossal lesion produces wasting and deviation toward the weak side.

2.4 Motor system: bulk, tone, power, and pattern

Inspect before touching. Compare muscle bulk, posture, spontaneous movement, tremor, and fasciculation. Marked wasting suggests denervation and a lower motor pathway problem; upper motor lesions produce much less wasting, and later. Apparent hollowing in a thin or older person is not enough if strength is preserved. Fasciculations are visible twitches of individual motor units in resting muscle. They gain meaning from distribution, weakness, and wasting, not from their mere presence.

Assess tone with the patient relaxed and by passive movement at different speeds. Spasticity is velocity-dependent: brisk stretch produces a catch and may then yield. Extrapyramidal rigidity resists movement through the range and affects opposing muscle groups; it may feel uniform or intermittently ratcheted by tremor. Test the wrist with passive flexion, extension, and rotation, and use contralateral voluntary movement to reveal subtle rigidity. This is particularly useful when screening a patient receiving dopamine-blocking medication. Active resistance in every direction may instead be paratonia, voluntary guarding, pain, or catatonic negativism, and the quality and context must be described.

Power is tested isometrically by opposing the intended action. Compare sides and proximal with distal groups, stabilise the joint, and prevent substitution. Most groups require separate testing in

each limb. Mild plantar-flexion weakness may be clearer during toe walking and dorsiflexion weakness during heel walking. The **Medical Research Council scale** has **six grades from zero to five**:

- Grade 0 is no contraction and grade 1 is a trace.
- Grade 2 is movement with gravity eliminated and grade 3 is movement against gravity.
- Grade 4 is movement against resistance and grade 5 is normal power.

Record the movement tested, not only a global grade. The scale compresses much clinically important weakness into the resistance grade and works poorly for small distal movements where gravity contributes little. Functional capacity therefore remains essential.

FEATURE	UPPER MOTOR PATHWAY PATTERN	LOWER MOTOR PATHWAY PATTERN
Weakness	Pyramidal distribution	Myotomal, nerve, or distal pattern
Bulk	Relatively preserved early	Marked wasting with denervation
Tone	Spastic	Flaccid
Tendon reflexes	Pathologically brisk	Reduced or absent
Plantar response	Extensor response may be elicited	No extensor response from the lower motor lesion itself

Pattern localises better than absolute severity. Hemiparesis involving lower face, arm, and leg suggests the contralateral cerebral hemisphere or brainstem; paraparesis directs attention to the spinal cord; distal weakness affecting all limbs favours the peripheral nervous system. For subtle pyramidal weakness, ask the patient to extend and supinate the arms with eyes closed. Downward drift with pronation is abnormal. This manoeuvre exposes a coherent motor pattern, whereas an isolated complaint of heaviness does not.

2.5 Reflexes: objective signs and level logic

Tendon reflexes are monosynaptic stretch responses. They test an intact sensory afferent, spinal connection, motor efferent, muscle, and descending modulation. A missing response is therefore not automatically a motor lesion; sensory neuropathy or radicular disease may interrupt the afferent limb. Position the limb comfortably, ensure relaxation, strike the tendon briskly, and watch as well as feel the contraction. Compare sides at matched levels.

REFLEX	PRINCIPAL ROOTS	PRACTICAL TECHNIQUE
Biceps	C5-C6	Place a finger on the tendon and strike the finger
Brachioradialis	C5-C6	Place a finger over the tendon region and strike
Triceps	C6-C7	Support the relaxed arm and strike the tendon
Finger	C8	Elicit with the hand relaxed
Adductor	L2-L3	Observe adductor contraction
Knee	L3-L4	Support the flexed leg and strike the patellar tendon
Ankle	S1	Hold the foot and strike the calcaneal tendon

Normal briskness varies widely, especially with anxiety and incomplete relaxation. Reinforce an apparently absent lower-limb reflex by asking the patient to interlock and pull the fingers at the moment of the strike; reinforce an upper-limb response with contralateral gripping. Do not diagnose neuropathy until reinforcement has been attempted. Conversely, do not diagnose an upper motor lesion from briskness alone. Asymmetry, spread, clonus, tone, weakness pattern, and plantar response supply the context.

The jaw jerk is useful when dysarthria, dysphagia, emotional lability, or widespread spasticity raises a corticobulbar question. Place a finger across the relaxed open chin and tap it lightly. A soft rebound is expected; a depressed response supports lower motor bulbar dysfunction, while a forceful response supports corticobulbar dysfunction. Segmental localisation uses a lost reflex at a given level with pathologically brisk reflexes below it to identify a focal cord level.

To elicit the plantar response, warn the patient, support the foot, and draw a blunt disposable stimulus firmly along the lateral sole from the heel toward the base of the little toe. The minimum extensor response is upward movement of the great toe; fanning may accompany it but is not required. The **Babinski sign** is described as present or absent, not “positive” or “negative.” In an agitated patient with increased tone and brisk reflexes, flexor plantar responses are reassuring. When definite spasticity, pyramidal weakness, pathologically brisk reflexes, and clonus already converge, the plantar response adds less; when the other signs are equivocal, it may decide whether a central lesion is present.

2.6 Sensory examination: tract logic with disciplined scepticism

The sensory examination is the least objective part of the neurological examination because it depends almost entirely on the patient's report. The answer is not to dismiss it. Define the question, demonstrate the stimulus, keep timing and force unpredictable, compare homologous sites, proceed in an anatomical order, and tentatively accept the report while looking for reproducibility and functional consequences.

Test light touch, pain, temperature when indicated, vibration, and joint position. Pain and temperature travel together in the lateral spinothalamic system after crossing near entry to the cord. Position, vibration, and discriminative cortical functions depend on posterior-column input ascending ipsilaterally before crossing in the medulla. Light touch also has anterior spinothalamic representation. This arrangement makes selective preservation of temperature with claimed complete loss of pain anatomically incoherent, while dissociation between pain and position can be anatomically meaningful.

For position sense, close the patient's eyes, hold the sides rather than the top and bottom of a distal phalanx, and move it slightly upward or downward in random order. Side-grasping removes pressure clues. If distal perception is impaired, move proximally and use larger excursions. For vibration, apply a noiselessly activated low-frequency fork to distal bony points and move proximally if sensation is reduced. If reports appear unreliable, randomly apply the fork vibrating or silent while the patient's eyes remain closed. The concealed comparison tests validity without confrontation.

Cortical sensory testing is meaningful only when primary sensation is adequate. For extinction, touch either side alone and then both simultaneously; detection of each alone but omission of a side during bilateral stimulation indicates sensory inattention. For tactile recognition, ask the patient with eyes closed to identify a familiar object that can be named on sight. For graphesthesia, compare recognition of writing on the palm with eyes open and closed. These tests require attention, language, and cooperation.

PITFALL

Do not diagnose a functional sensory deficit merely because the map looks odd. Repeat the examination, check comprehension and attention, test tract-linked modalities, and look for objective motor, reflex, cerebellar, and gait evidence. Sensory signs carry less predictive weight than positive motor and gait signs.

2.7 Cerebellar examination, stance, and gait

Cerebellar hemispheres coordinate ipsilateral limbs, while the vermis coordinates axial structures. Cerebellar lesions cause incoordination rather than primary weakness, pathologically brisk reflexes, or an extensor plantar response. Examine speech, eyes, upper and lower limbs, stance, and gait, while testing proprioception because sensory ataxia can imitate cerebellar incoordination.

For the finger-nose test, place your finger far enough away to require a full reach and ask the patient to alternate between it and the nose. Cerebellar dysmetria produces an irregular trajectory and an action tremor whose amplitude increases near the target. Rapid alternating movement is tested by repeated alternating palm and dorsum contact on the same spot. Uneven force, irregular rhythm, and loss of alternation support ipsilateral incoordination, but this finding is not specific. For the heel-shin test, the supine patient places the lifted heel below the opposite knee and runs it down the shin. Tremulous placement and a wavering descent indicate incoordination.

Romberg's test begins with the feet together and eyes open. Only after confirming that the patient can stand in that condition should the eyes be closed, with the examiner ready to catch the patient. Loss of balance specifically after eye closure indicates impaired proprioceptive input, commonly from posterior-column or peripheral sensory disease, not a cerebellar lesion. A patient unable to stand even with visual input has ataxia, but does not thereby have this sign.

Gait is probably the most valuable noncognitive neurological assessment because it integrates motor pathways, peripheral nerves, coordination, proprioception, balance, attention, and practical confidence. Observe initiation, base, stride, arm swing, turning, symmetry, foot clearance, and response to tandem, heel, and toe walking when safe. A slow usual speed of **0.7 metres per second or less** is associated with increased risks of dementia, stroke, falls, disability, hospitalisation, and death. The number is a risk marker, not a diagnosis.

Pattern recognition then directs the differential. A broad unsteady gait suggests cerebellar ataxia. Difficulty with tandem walking may reveal mild gait ataxia. A festinating pattern suggests an extrapyramidal disorder, including a drug-induced syndrome. Circumduction of a stiff weak leg is hemiparetic. Excessive hip and knee flexion to clear the foot is a steppage pattern. Pelvic-girdle weakness produces waddling. Apparent inability to stand or walk with dramatic near-falls, yet preserved protective balance and no injury, suggests astasia-abasia. Observe gait as the patient enters the interview room.

> **GOOD TO REMEMBER**
>
> A patient encountered in bed can still have the most localising part of the examination omitted. If it is safe, watch the patient stand, start, turn, tandem, and return to the chair. The transition movements often reveal more than straight-line walking.

2.8 Psychiatric discriminators: functional signs, catatonia, and drug effects

The governing principle for a suspected functional deficit is a positive inconsistency with neuroanatomy, not an impression that the patient appears psychologically distressed. Motor and gait signs are more useful than sensory signs. Look for internal conflict between voluntary testing and automatic or synergistic movement: variable give-way effort rather than smooth yielding, a hand that avoids striking the face despite alleged paralysis, normal heel or toe walking despite marked manual ankle weakness, or a distribution that cannot be produced by a single anatomical lesion.

The **Hoover sign** tests involuntary hip extension synergy. Cup the heel of the apparently weak leg while the patient flexes the opposite hip against resistance. Downward pressure from the allegedly weak heel demonstrates preserved extension that was not produced during direct testing. The complementary pattern is absent downward pressure from the unaffected heel when the patient is asked to raise the allegedly weak leg. A related abductor manoeuvre shows reflex abduction of the “weak” leg while the unaffected leg is actively abducted. These are positive motor signs; they should be demonstrated respectfully and interpreted with the whole examination.

Functional sensory patterns include a sharp midline boundary, loss of vibration over only one half of a continuous midline bone, and pain loss with preserved temperature. Genuine proprioceptive loss should impair balance in darkness, stance with eyes closed, and relevant daily actions. Nevertheless, old follow-up series found a later neurological diagnosis in as many as **fifteen per cent** of patients initially labelled with hysteria or conversion, although those cohorts often had rudimentary examinations and investigations. Continue follow-up when the course changes.

Primitive grasping is elicited by placing the index and middle fingers in the palm. Automatic gripping, especially if unilateral, is a frontal release sign; in an inert mute patient it argues against an exclusively depressive explanation. The anatomy belongs to the Functional Neuroanatomy and Neurophysiology notes. Do not confuse this with forced handshaking in catatonia after an instruction not to take the offered hand.

Catatonia must be examined, not merely watched. Observe mutism, staring, stupor, posturing, stereotypy, echo phenomena, excitement, and withdrawal; elicit tone phenomena, negativism, waxy flexibility, automatic obedience, forced grasping, and ambitendency. Waxy flexibility begins with resistance, gradually yields to positioning, and maintains the posture. In mitgehen the limb follows the slightest pressure despite an instruction to resist; mitmachen requires more manipulation. Neurological examination is essential because catatonia can resemble drug effects, akinetic states, delirium, seizures, and advanced cognitive disorders.

TRAP **Do not treat “Gegenhalten” as a universally settled label.** A clinical tradition uses it for active resistance in advanced dementia, another for catatonic negativism in which resistance matches the examiner's force, and another associates variable resistance with frontal release. Describe what the limb did, how resistance changed with force and speed, and the accompanying signs.

In a patient receiving antipsychotic medication, examine spontaneous movement, facial animation, resting tremor, bradykinesia, rigidity, subjective restlessness with observable movement, and involuntary oral, lingual, facial, truncal, or limb movements. The Simpson-Angus Scale, Barnes Akathisia Rating Scale, and Abnormal Involuntary Movement Scale can support serial documentation, but their administration and scoring belong to the Schizophrenia: management, antipsychotics and adverse effects notes. Chorea may initially look like ordinary fidgetiness; tardive dyskinesia often involves the face, lips, jaw, and tongue; asterixis is a brief loss of sustained tone. Describe the movement before attributing it to illness, anxiety, or medication.

Examine with sensitivity. An attempt to “catch” inconsistency can worsen distress and obscure useful signs. Functional and structural disease can coexist. Emotional unconcern is not diagnostic: **la belle indifference** has no credible discriminating value and occurs with neurological disease. Base the conclusion on positive signs, anatomical coherence, longitudinal review, and appropriate investigation, not personality or bedside theatre.

Mental status validates the examination; pattern localises it; gait integrates it; anatomical inconsistency must be demonstrated, never presumed.

Lesion localisation and the imaging decision

Why it matters clinically. A psychiatric formulation can become unsafe when an observed change in language, behaviour, movement, attention, or gait is treated as a psychiatric meaning before it has first been treated as a neurologic sign. **Localisation**, also called topographic diagnosis, asks what part of the nervous system has been affected, using signs and, most often, symptoms. It is not a decorative neurological add-on to the mental-state examination. It is the disciplined step that decides whether a putative psychiatric presentation has a coherent neural address, whether it instead points away from a lesion, and what question an investigation should be asked to answer.

The psychiatrist need not reproduce a neurologist's full examination to use this reasoning. The required competence is to **appreciate neurologic signs and appraise the neurologist's conclusion**. That means separating the observation from its interpretation, resisting a psychiatric default, and recognising when a bedside pattern has more explanatory force than a familiar diagnostic label. The functional anatomy that makes a sign intelligible belongs in the Functional Neuroanatomy and Neurophysiology notes; the present task is to use that map without redrawing it.

3.1 Localisation is a step in diagnostic reasoning

Neurologic diagnosis proceeds through **four** linked operations: recognise impaired function, localise it, construct an aetiological differential, and then use ancillary procedures to decide which proposed cause is present. **Localisation** is therefore neither a substitute for diagnosis nor an inference made after a scan has supplied one. It is the bridge between a finding and a focused differential. Confusing these operations produces a familiar error: treating a radiological abnormality as the diagnosis before asking whether it accounts for the patient's observed deficit.

Recognition comes first because an unrecognised sign cannot be localised. It depends on knowing what normal performance looks like and on examining carefully enough to see a departure from it. Mild chorea may be dismissed as ordinary fidgetiness; slow eye movements may be missed if the examiner notices only whether the eyes complete the movement. Both errors begin before the localising exercise has even started. In psychiatric practice, where restlessness, unusual motor behaviour, reduced engagement, and disordered speech are often interpreted through psychopathology, this preliminary vigilance is particularly important.

Before attempting a locus, ask **three** questions: whether the reported dysfunction is real, whether it is pathological enough to warrant formal work-up, and whether it is neurologic rather than a disorder of the organ mediating that function. These are not sceptical questions directed at the patient. They are tests of the clinical observation. A sensory complaint elicited during examination is part of the examination, not mere history, and may be as objective as an observed wrist drop. Equally, a history should guide attention without hardening into preconception: it suggests whether a central or peripheral problem may be present, while the routine examination still checks all major areas.

SIGN	SITE	CAUSE	TEST
RECOGNISE IMPAIRED FUNCTION	LOCALISE THE PATTERN	FORM THE DIFFERENTIAL	CHOOSE AN ANCILLARY PROCEDURE

RULE **Give a sign its neurologic status before giving it psychiatric meaning.** Unless clear psychiatric manifestations accompany a neurologic symptom or sign, it should be taken at face value while its pattern is assessed.

This is a corrective to a durable diagnostic bias. Some movement disorders, including dystonias, were formerly cast as psychogenic before their organicity was generally recognised. The appropriate stance is neither indiscriminate testing nor reflexive disbelief. It is an accountable formulation in which a sign is first allowed to constrain the possible anatomy.

3.2 Reading the map: address and intersection

Neuroanatomy is the road map for localisation. The map has morphological structures and their functional representations: damage to a structure alters the function it mediates. Applied localisation has **two** basic routes. The **address route** begins with a characteristic constellation that directly identifies a site. The **intersection route** begins with separate signs, traces the pathway implicated by each, and locates the lesion where those pathways meet. The aim is not to recite anatomy but to make every observed finding do localising work.

REASONING ROUTE	QUESTION AT THE BEDSIDE	WHAT MAKES IT PERSUASIVE	CLINICAL CONSEQUENCE
Address	Does the whole pattern name a site?	A syndromic constellation supplies an anatomic address.	Use the pattern to narrow the differential.
Intersection	Which pathways are implicated separately?	The affected pathways converge at a shared locus.	Seek the accompanying sign that makes the location precise.
Temporal check	Were the signs acquired together?	Only contemporaneous findings may be forced into one lesion.	Separate accumulated deficits before localising.

An address-route example is the combination of resting tremor, bradykinesia, and rigidity, which points to injury of the substantia nigra. More often, a single sign only yields a range of possible locations. A third-nerve palsy, by itself, can arise along an extended course. Add contralateral hemiparesis occurring at the same time, however, and the intersection localises the process to the crus cerebri. The lesson is more general than the particular syndrome: one sign opens a corridor; the added, anatomically coherent sign identifies the room.

MNEMONIC

PATTERN - PATHWAY - PLACE - PHASE

Pattern asks whether the signs already provide an address. If not, identify the **pathway** indicated by each finding, locate their shared **place**, and check the **phase** of onset so old and new deficits are not falsely intersected. This is a reasoning scaffold, not a replacement for anatomy.

The temporal check prevents an elegant but false conclusion. The examination can display the accumulated end result of multiple lesions. Signs acquired in widely separated periods should not be made to intersect at a single locus simply because they coexist today. Here the history has a restricted but decisive localising role: it determines whether findings belong to the same event. Otherwise, the history chiefly contributes aetiology, whereas the examination carries the greater localising weight.

3.3 What the examination localises, and what history explains

The examination has greater localising value than the history. By tracking back the pathways mediating impaired functions found at the bedside, a lesion can often be localised even when the history is sparse or unreliable. This is especially useful in liaison work, where collateral may be incomplete, the patient may be distressed or unable to cooperate, and the narrative may be shaped by the very disorder that needs explanation. It does not diminish the history. It assigns it its proper job.

The temporal evolution of an already localised deficit helps define cause. The same left-sided hemiparesis carries different aetiological implications when it develops over minutes, over days, or insidiously over months: cerebrovascular disease or epilepsy, infection or demyelinating disease, and tumour or a degenerative process respectively. The anatomical conclusion remains the same; the causal differential changes. This division of labour stops the clinician from allowing a dramatic tempo to dictate an anatomical site that the examination has not supported.

- **History:** establish onset, evolution, and whether apparently related signs belong to one clinical event.
- **Examination:** identify the impaired functions and construct the anatomical pattern.
- **Formulation:** combine locus and tempo into a restrained differential that includes plausible disease and dangerous alternatives.

The motor examination illustrates the distinction. Severity tells the clinician about functional capacity, but the pattern of weakness supplies more localising information. The level-by-level pattern itself is addressed in the Functional Neuroanatomy and Neurophysiology notes. At this stage, the psychiatrist should read a documented motor pattern as evidence about site, not merely as a measure of disability.

A useful formulation also travels along **two axes**: the affected level, broadly central nervous system, peripheral nervous system, or muscle; and the distribution, focal versus diffuse. Together, site and extent direct the differential. Discrete lesions are the classical terrain of localisation, with cerebrovascular disease the commonest cause and demyelination, infection, trauma, and tumour also often behaving as discrete lesions. System lesions instead affect arrays of neurons serving a functional system. The method remains useful for them, but the symptom pattern may point to an affected pathway rather than the primary cellular target. This is why a localising conclusion should be stated as an inference from signs, not as an overconfident claim that the disease process has been anatomically exhausted.

3.4 Higher functions, posture, and gait demand pattern recognition

Precision generally decreases as one ascends the neuraxis. Lower-level lesions tend to produce stable findings, whereas higher-level lesions may vary even within a single examination. The correct question for a higher function is therefore not simply whether the patient can perform it at some moment, but whether the patient performs it consistently in a normal way. Higher networks can mask a deficit, so sampling must be sufficient to defeat that masking. This is a clinical discipline, not a demand for a scored cognitive screen; formal screens and their interpretation belong in the Psychometry, Assessment and Descriptive Psychopathology notes.

In practice, a patient who manages a task once but fails it unpredictably has not thereby demonstrated intact function. For native speakers, any difficulty repeating a sentence should be regarded as abnormal. Record the performance itself, its consistency, the patient's engagement, and the accompanying neurologic signs. Such documentation helps the next clinician distinguish fluctuating capacity, language impairment, impaired attention, and a failure of testing conditions without pretending that a single bedside answer settles the matter.

Posture and gait need a related but distinct approach because they are assessed chiefly by functional observation, rather than by isometric strength testing or sensory questioning. Their localising value comes from characterising the movement or postural pattern and seeking accompanying neurologic signs. Some patterns are highly stereotyped and strongly localising; cautious gait and central disequilibrium, by contrast, have many potential causes and are harder to localise. The examination technique for gait and the rest of the physical neurological examination are addressed elsewhere in this chapter; here the point is to use the observed pattern as a hypothesis, then test that hypothesis with associated signs.

PITFALL

Do not call a gait or postural complaint non-neurologic merely because muscles and joints can also alter movement. Clinical bias tends to favour a non-neurologic diagnosis when the neural control of posture or gait is actually impaired. Observation must be followed by a search for a coherent accompanying neurologic pattern.

3.5 Formulation includes the limits of localisation

The formulation should answer the **Four Questions of Neurology**: what are the symptoms, what are the signs, where is the lesion, and what is the lesion. In psychiatric work, the equally important negative conclusion is that neurologic disease does not explain the presentation. Neither conclusion is reached by impression alone. The clinician must show how the signs cohere anatomically, or why they fail to do so, and must keep the resulting differential proportionate to the evidence.

Localisation is valuable in most cases, but it is partly an art and is not applicable to every important neurologic illness. Alzheimer disease is an example of a setting in which the classical focal-lesion method is not the right intellectual frame. Similarly, widespread or symmetric changes should not be forced into a discrete lesion formulation. The method is strongest when it clarifies a specific pattern, less decisive where disease is diffuse, degenerative, or network-based. A

good examiner acknowledges this boundary rather than manufacturing a misleading anatomic precision.

The differential begins with diseases most consistent with the signs and locus, includes unlikely but potentially life-threatening conditions, and avoids the intellectual flourish of unlikely but fascinating explanations. This is not anti-curiosity. It is an insistence that the differential stay connected to the patient rather than to the examiner's imagination. A concise written formulation makes that connection visible and gives imaging or other testing a defensible purpose.

TRAP **A single abnormality is not a licence to announce a precise lesion.** One sign often underdetermines the locus. Seek a second anatomically coherent sign, then ask whether the findings appeared in the same clinical period before applying the intersection route.

Conversely, an anatomically impossible deficit points away from a lesion locus. Preserved temperature sensation with loss of pain perception is nonanatomic and should prompt a careful consideration of psychogenic origin. This is the inverse of localisation, not a shortcut to disbelief. Long follow-up of classic hysteria and conversion cohorts found specific neurologic conditions emerging in **up to 15%** of patients. Those cohorts had limited examinations and testing, but the practical warning remains: a psychogenic conclusion must be positively grounded in the pattern, never merely inferred from psychiatric context or diagnostic inconvenience.

3.6 The liaison problem: language that looks psychiatric

The psychiatric presentation may be the sign. Sudden fluent aphasia can account for apparent anger, acute psychosis, consternation, behavioural change, or confrontations around management in liaison settings. Its disordered output may also resemble psychotic speech. A clinician who sees bizarre language and assumes thought disorder can miss the neurologic basis of the presentation, particularly when there is no obvious hemiparesis and attention cannot be captured well enough to examine sensation or visual fields.

The discrimination is clinical and longitudinal. Schizophrenic speech usually develops gradually in the context of long-standing psychiatric illness; language abnormalities can become increasingly similar to aphasia as thought disorder develops. Fluent aphasia usually presents suddenly, may occur with stroke risk factors, and often leaves the patient aware that communication is failing, seeking help, and giving short responses. Repetition of polysyllabic words and complex phrases is an important discriminator because people with schizophrenia can do this unlike most people with fluent aphasia. None of these observations should be isolated into a mechanical rule. Frightened, confused people with sudden aphasia may themselves be agitated or irrational.

QUESTION	FLUENT APHASIA PATTERN	PSYCHOTIC-SPEECH PATTERN	WHY IT MATTERS
Tempo	Usually sudden	Usually gradual	Tempo changes the aetiological frame.
Repetition	Often impaired	May be retained	Test language, not only content.
Patient stance	May recognise communication failure and seek help	Interpret in the broader psychiatric history	Behaviour alone is not diagnostic.
Associated signs	Seek reflex, sensory, and visual-pathway clues	Do not assume their absence from a brief encounter	Associated signs can localise the problem.

The localising endpoint is usually a small discrete structural lesion in the temporoparietal region, often a stroke, although neurodegenerative illnesses can also cause fluent aphasia. The detailed focal lobe syndromes belong in the Stroke, TBI, delirium and focal brain syndromes notes. The immediate psychiatrist's task is simpler and more urgent: recognise that the speech may be language failure, document the pattern, look for accompanying signs, and frame the investigation around that localisation.

GOOD TO REMEMBER

"Gibberish" is a description, not a diagnosis. When language is suddenly abnormal, test repetition and seek a neurologic pattern before attributing the communication failure to psychosis, personality, or non-cooperation.

3.7 From a bedside locus to the imaging question

Ancillary procedures should follow clinical diagnosis if they are to realise their diagnostic potential. This is not an argument against imaging, neurophysiology, or molecular investigation. It is an argument against using them as an unstructured search. **The locus reached on examination names the region that the scan must interrogate**. A patient with a left-sided hemiparesis and a right third-nerve palsy that appeared together, for example, requires careful attention to the midbrain when MRI is obtained. The examination has put a question to the study.

That ordering protects against false-positive attribution. A broad, unlocalised approach to neuroimaging may reveal a striking congenital brain cyst and wrongly make it responsible for a neurologic disorder while the actual disease remains overlooked and untreated. Careful localisation is the guard against this error. It also makes ancillary procedures more thoughtful, reducing patient discomfort and wasted resources. The question is not, "What can a scan find?" but, "Does the study answer the anatomical and aetiological question posed by this patient's signs?"

Some diagnoses cannot be closed at the bedside alone. Stroke and brain tumour require imaging studies, and confirmation of seizures often requires EEG. The modality, its indications,

sequences, and red-flag lens are covered in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes and should not be reconstructed from this section. Here, the decision workflow is sufficient: establish the clinical syndrome, state the likely locus and differential, and request the study that can adjudicate that framed question.

Localisation also determines what kind of diagnostic question is being asked. Aphasia usually implies a discrete dominant cerebral-hemisphere injury and therefore raises a structural question for CT or MRI. Dementia instead raises a neurodegenerative question, with blood tests as well as CT or MRI in its evaluation. The distinction is not academic: it changes the evaluation before it changes the label. When an imaging study shows a different process from the clinical formulation, abnormal scan findings routinely override the clinical impression. This should encourage humility, not passivity: a clear bedside formulation makes a discrepant result intelligible and helps the clinician revisit both the patient and the image.

> Localise from the bedside pattern first; then make every investigation answer the question that localisation has already defined.

4 · Electroencephalography in psychiatry: normal rhythms, common abnormalities and clinical indications

Why EEG still matters to the psychiatrist. Electroencephalography is not a laboratory badge for a psychiatric diagnosis. Its value lies in a disciplined change of question: from "Which psychiatric disorder is this?" to "Could an epileptic, encephalopathic, toxic, or other cerebral process be contributing to this presentation?" That distinction protects patients from both over-investigation and premature psychologising. The normal frequency bands, the morphology of epileptiform discharges, and the distinction between interictal and ictal phenomena are established in *the Epilepsy and psychiatry notes*; the present discussion applies that vocabulary to the psychiatric workup.

EEG is a real-time sample of cortical electrical activity, not a general screen for brain disease. A useful request therefore begins with a clinical problem, a timeline, medication exposure, and the precise alternative diagnosis being tested. In uncomplicated psychiatric illness, recordings are commonly **normal or only nonspecifically abnormal**, with such findings as excess beta or theta activity, isolated sharp waves or spikes, or mild disorganisation. These patterns do not convert a clinical syndrome into a neurological diagnosis.

4.1 Recording language and the meaning of a normal study

Before interpreting an abnormality, the psychiatrist should be able to read the report's spatial language. The **10-20 International System** places electrodes according to measured proportions of head landmarks, so that comparable labels refer to comparable cortical regions despite differences in head size. Electrodes are positioned at **10% or 20%** of the relevant measured distances. This is why serial recordings and reports from different laboratories can be compared meaningfully rather than treated as idiosyncratic maps.

The labels are a clinical shorthand, not a claim that an electrode records only one circumscribed cortical site. In the standard nomenclature, F, T, C, P and O denote frontal, temporal, central, parietal and occipital locations; *z* identifies the midline; odd numbers are left sided and even numbers right sided. Cz is the vertex. The conventional system comprises **21** electrodes, including scalp and aural reference sites. A psychiatrist need not become an electroencephalographer, but should recognise when a report is describing a focal distribution, a generalised background change, or a technically interpretable routine recording.

GOOD TO REMEMBER

Electrode names describe a standardised scalp location. They help communicate distribution; they do not by themselves establish the aetiology of a psychiatric presentation.

A normal EEG is similarly contextual. It can be highly informative when the clinical question is diffuse toxic-metabolic encephalopathy, yet it cannot reliably detect or exclude a structural lesion. When structural pathology is being considered, neuroimaging is the standard investigation; a normal EEG must not be used as a structural rule-out. The relevant imaging approach is covered in *the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes*.

4.2 Indication-driven use in general psychiatry

The governing principle is that a **routine EEG screen is unwarranted** in psychiatric practice. There is no accepted diagnostic EEG indication for psychiatric disorders as defined in the current diagnostic manuals. The test earns its place when it alters the differential towards an organic cause or answers a sharply framed seizure-related question. Asking for it simply because symptoms are severe, unusual in content, or treatment-resistant without further reasoning invites nonspecific findings and false certainty.

RULE **Order EEG to test an organic alternative, not to certify a psychiatric diagnosis.** The clinically useful psychiatric role is differential diagnosis of organic aetiology and investigation of sleep disorders, rather than diagnostic confirmation of a DSM syndrome.

Its yield rises when there is an **atypical presentation, atypical age at symptom onset, or atypical treatment response**. In that setting, the report is sought to identify paroxysmal activity or focal or diffuse slowing that would redirect the assessment. This does not mean every late presentation or difficult-to-treat illness needs a recording. It means the clinician should articulate why the phenomenology, course, or treatment response makes a cerebral contribution plausible and what the result would change.

CLINICAL QUESTION	EEG CONTRIBUTION	INTERPRETIVE BOUNDARY
Uncomplicated psychiatric syndrome	Usually none beyond occasional nonspecific findings	Do not use as a routine screen
Atypical course or response	Seek paroxysmal activity or focal/diffuse slowing	Interpret alongside the syndrome and examination
Acute agitation or unresponsiveness	Detect diffuse slowing or ongoing epileptic activity	Acts on the immediate organic differential
Cognitive decline with selected warning features	Supports a specific encephalopathic or periodic pattern	Not a routine dementia test

A **paroxysmal EEG finding** alone does not diagnose epilepsy. The retrospective clinical history must still support an epileptic nature for the events. In an atypical psychiatric presentation, such a finding should lower confidence in a purely psychiatric formulation and prompt re-examination of the history, collateral account, medication list, and neurological assessment. It is a reason to think again, not a licence to relabel the patient.

TRAP **"Spikes equal epilepsy, therefore the presentation is not psychiatric."** EEG and clinical semiology must be reconciled. An isolated paroxysmal finding changes diagnostic confidence; it is not independently diagnostic.

4.3 Acute disturbance: delirium, toxicity and nonconvulsive seizures

In an acutely agitated, fluctuating, or nonresponsive patient, EEG can be immediately decisive because the key distinction is between a delirious encephalopathic state and ongoing epileptic activity. **Diffuse slowing** supports delirium, while evidence of **nonconvulsive status epilepticus** demands a different urgent pathway. The accompanying figure places these patterns in the broader psychiatric workup. This is an investigation of the acute brain state, not an adjunct to a routine mental-state examination.

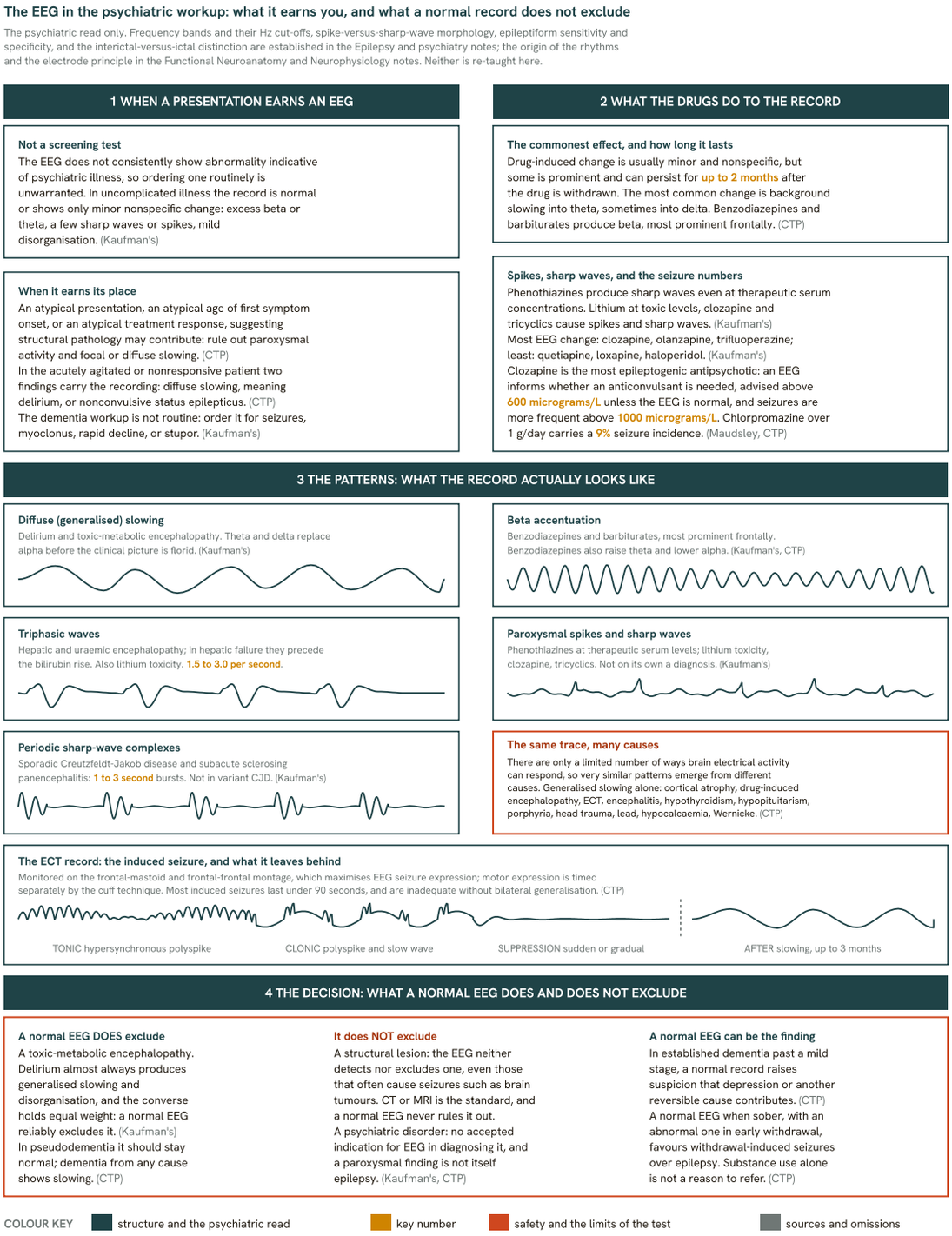


Fig 31.2 The EEG in the psychiatric workup: diffuse slowing, triphasic waves, and the psychotropic and ECT record. The decision is the teaching, not the tracing. The EEG is not a screening test and has no accepted role in diagnosing a psychiatric disorder; it earns its place when the presentation, age of onset or treatment response is atypical, and when an agitated or nonresponsive patient may be delirious or in nonconvulsive status epilepticus. Read the patterns for what they exclude: a normal record reliably excludes a toxic-metabolic encephalopathy, but never a structural lesion and never a psychiatric diagnosis. Triphasic waves are the one shape pointing somewhere specific, and lithium toxicity is their mimic. EEG abnormality in up to 20% of healthy individuals appears nowhere here as fact: its source calls it a belief still needing study. The EEG fundamentals belong to the Epilepsy and psychiatry notes, and the origin of the rhythms and the electrode principle to the Functional Neuroanatomy and Neurophysiology notes. (Kaufman's Clinical Neurology for Psychiatrists; Kaplan and Sadock's CTP; The Maudsley Prescribing Guidelines.)

In toxic-metabolic encephalopathy, theta and delta activity can replace alpha early, sometimes while behavioural or cognitive change is still subtle; the typical record shows generalised slowing and disorganisation. Background slowing is nonspecific because many different processes converge on the same electrical response. It should therefore trigger a targeted clinical search rather than a satisfied conclusion. A normal EEG, however, **reliably excludes toxic-metabolic encephalopathy**, making the test especially useful when delirium is genuinely in the differential.

Triphasic waves are a more suggestive pattern: high-voltage, frontally dominant or diffuse slow waves with a characteristic blunt or rounded spike-like onset, occurring at **1.5 to 3.0 Hz**. They often point toward hepatic or uraemic encephalopathy and, in hepatic failure, may precede the bilirubin rise. The full pattern may also include frontal triphasic activity with loss of organised occipital alpha. Yet the psychiatrist must retain the medication differential: lithium toxicity is a recognised cause of triphasic waves, and metabolic causes remain common.

PITFALL

Do not dismiss diffuse slowing because a psychotropic plasma concentration is within the therapeutic range. Drug-induced encephalopathy can show diffuse slowing despite a therapeutic serum level, so toxicity remains a clinical possibility alongside metabolic and endocrine causes.

Psychotropics complicate this reading. Significant diffuse slowing in an unstable patient receiving psychotropic medication, particularly an older adult, should prompt consideration of medication toxicity as well as alternative causes of encephalopathy. Lithium at intoxicating levels can be associated with diffuse slowing, paroxysmal spikes and triphasic waves, and these EEG abnormalities may remain after the serum concentration has normalised. The EEG is therefore a measure of cerebral effect, not merely a surrogate for the current blood value.

21	1.5–3.0 Hz	LESS THAN 90 s
STANDARD ELECTRODES	TRIPHASIC WAVES	MOST ECT SEIZURES

4.4 Medication effects: read the drug exposure before the tracing

Medication history is part of EEG interpretation, not a footnote. Psychotropic-induced changes are usually minor and nonspecific, but some persist for up to 2 months after withdrawal. The commonest effect is background slowing into theta, sometimes delta. A report should therefore be read with the current regimen, recent discontinuations, dose changes, intoxication risk, and clinical state in view. Generalised slowing is not a diagnosis, and its differential includes drug-induced encephalopathy, ECT and systemic illness.

EXPOSURE	CHARACTERISTIC EEG TENDENCY	CLINICAL READING
Benzodiazepines	Beta enhancement, most prominent frontally; slight theta increase and alpha decrease	Can reduce epileptiform activity
Barbiturates	Frontal beta during use; paroxysmal activity and spikes after abrupt withdrawal	Withdrawal discharges may be behaviourally silent
Phenothiazines	Sharp waves	May occur at therapeutic concentrations
Lithium toxicity	Diffuse slowing, spikes and triphasic waves	Abnormality can outlast normalisation of the serum level
Typical antipsychotics	More alpha, lower frequencies and less beta	Spikes and seizures may occur at high doses
Tricyclic antidepressants	Slow and fast rhythm increase, lower alpha frequency, paroxysmal slow waves and spikes	Seizures may occur at higher doses
SSRIs	Less alpha and more theta	Lower seizure rates than tricyclics

The table is a pattern-recognition aid, not a prescribing hierarchy. Benzodiazepine-related **frontal beta activity** is a classic sedative-hypnotic signature. Barbiturate withdrawal can produce generalised paroxysmal activity and spikes for up to 3 to 4 weeks after the last exposure, sometimes without overt motor seizures. Medication effects can thus mimic disease, expose withdrawal physiology, or coexist with the illness that led to treatment.

The operational consequence is modest but important. An abnormal result should prompt a review of temporal relationships: did the change appear after introduction, dose escalation, toxicity, abrupt discontinuation, or ECT? Does it fit the patient's vigilance, cognition and neurological signs? A plausible drug effect may explain a trace without explaining the whole patient. Conversely, attributing slowing to a familiar prescription can obscure concurrent systemic illness or nonconvulsive seizure activity. The report becomes useful when it is treated as evidence within a formulation rather than as a pharmacological fingerprint printed in isolation.

Clozapine requires particularly careful integration of clinical, EEG and plasma-level information because it is the most epileptogenic antipsychotic and EEG abnormalities are common in treated patients. It can increase delta, theta and beta activity over frontal, central and parietal areas, with paroxysmal slowing and spikes. An EEG may help determine whether anticonvulsant treatment is needed, although the interpretation is complex. At plasma levels above **600 micrograms/L**, anticonvulsant use is recommended unless the EEG is normal; seizures occur more frequently above **1000 micrograms/L**. This is a deliberately scoped clozapine-management decision, not a general threshold for abnormal EEGs.

MNEMONIC

Drugs before discharges

Before treating an EEG discharge as disease, ask what the patient has received, stopped, accumulated, or cleared. The medication list often explains the trace, but it must never replace assessment for concurrent encephalopathy or seizures.

4.5 EEG during and after electroconvulsive therapy

EEG monitoring during ECT documents the induced seizure and helps assess its cerebral expression. The ictal EEG resembles a generalised tonic-clonic seizure and its aftermath. Frontal-mastoid and frontal-frontal montages are used to maximise seizure expression; during the tonic phase, hypersynchronous polyspike complexes are followed by polyspike-and-slow-wave complexes, and the clonic phase is followed by suppression. Motor expression is assessed separately, so an EEG record is not merely a more technical version of observing movement.

Most induced seizures last less than 90 seconds, but duration alone does not establish adequacy. Bilateral generalisation on EEG is the stated adequacy criterion. This distinction matters when a seizure appears brief, or when motor expression is limited by anaesthesia and muscle relaxation: the clinical team needs evidence of a generalised cerebral seizure, not a stopwatch result isolated from the tracing.

After ECT, slow-wave activity can develop over frontal regions or the whole cerebrum and persist for up to 3 months. With unilateral treatment, slowing may remain restricted to the treated side. Postictal suppression and slow-wave activity are more likely in responders than nonresponders. Slow-wave change is not simply an adverse marker: it is associated with both memory impairment and effective treatment of depression. Interpretation should therefore acknowledge a benefit-cost signal rather than treating post-ECT slowing as automatically pathological.

4.6 Cognitive decline and selected disease signatures

EEG is not indicated routinely in dementia assessment. Common dementia-producing illnesses tend to produce slowing or other minor nonspecific abnormalities, which may be difficult to distinguish from age-related change. The recording becomes more useful when seizures, myoclonus, rapidly progressive decline, stupor, suspected nonconvulsive seizures, toxic-metabolic encephalopathy, or a dementia with a specific EEG pattern is under consideration.

In early Alzheimer disease, background activity usually slows to below 8 Hz; later records are slow and often disorganised. Vascular dementia can also cause abnormalities, but EEG cannot reliably differentiate it from Alzheimer disease. Conversely, a normal EEG in a patient thought to have dementia beyond a mild stage should raise suspicion of depression or another reversible contributor. Depression-related cognitive impairment may have a normal or only mildly slowed EEG, but EEG cannot quantify the relative contribution of depression and mild dementia when both are present. The fuller diagnostic framework belongs in *the Dementias and neurocognitive disorders notes*.

The striking exception is the periodic sharp-wave complex. In sporadic Creutzfeldt-Jakob disease and subacute sclerosing panencephalitis, dementia and myoclonus may be accompanied by **periodic sharp-wave complexes**. Classically, fairly regular bursts occur across channels at intervals of 1 to 3 seconds, with low activity between bursts; myoclonic jerks are their clinical counterpart. Variant Creutzfeldt-Jakob disease does not show this characteristic periodic pattern. The clinical context remains decisive, but this is one of the rare psychiatric-neurological differentials in which EEG can provide a near-definitive signature.

4.7 Substance use, ageing and a practical reporting discipline

Drug use alone is not a sufficient indication for EEG referral. With infrequent exceptions, recreational or dependent use does not introduce frank abnormalities into the visually analysed tracing. The question becomes useful when there are seizures, altered consciousness, a concerning withdrawal state, or an alternative neurological diagnosis. In alcohol-related seizures, a normal EEG during sobriety with an abnormal early-withdrawal EEG favours withdrawal-induced seizures rather than epilepsy with comorbid alcohol dependence. The substance-use assessment itself belongs in *the Substance use disorders notes*.

A practical report-reading discipline prevents two opposite errors. First, do not inflate a minor nonspecific change into an explanation for hallucinations, mood symptoms or personality change. Second, do not call a record "nonspecific" and stop when the patient is clinically delirious, fluctuating, or difficult to arouse. The EEG contribution is strongest when its finding is converted into a focused next question: diffuse slowing asks about global cerebral dysfunction; a periodic pattern asks about a narrow diagnostic class; apparent epileptic activity asks whether the witnessed events and collateral history are epileptic. This preserves the hierarchy in which the patient, not the tracing, remains the object of diagnosis.

Older psychiatric patients deserve a lower threshold for organic thinking when seizures are suspected. Older adults may account for as many as one-fourth of new-onset epilepsy cases. This does not make EEG a screening test for every late-life psychiatric presentation. It means that late onset, episodic phenomena, fluctuating awareness, unexplained falls, atypical behaviour, and abrupt cognitive change should be actively tested against an organic differential rather than absorbed into a familiar psychiatric label.

When reviewing or requesting an EEG, document the question in a sentence: "Is there evidence of diffuse encephalopathy, ongoing epileptic activity, or a focal abnormality that would alter the formulation?" Then document the clinical state during recording and relevant medication exposure. If the study is normal, state what it does and does not reduce in the differential. If it is abnormal, distinguish a nonspecific background change from a disease-linked pattern, and return to the patient rather than allowing the report to become the diagnosis.

A well-framed request also makes the eventual report safer to use. Give the electroencephalographer the target symptom, the temporal pattern, recent events, medication and substance exposure, and whether the patient was confused, drowsy or clinically recovered during the recording. On return, reconcile the result with the original question before changing

the psychiatric formulation. That small discipline prevents an incidental tracing feature from acquiring more authority than the history.

- State the clinical phenomenon that prompted the request and the organic alternative under consideration.
- Record the patient's state during the study and every exposure that could alter the background or provoke a withdrawal effect.

EEG in psychiatry is an indication-driven test: use it to detect cerebral processes that change the formulation, never to validate a psychiatric label.

5 · Cerebrospinal fluid examination and its role in the neuropsychiatric workup

CSF examination is a question-driven investigation, not a routine psychiatric screen. Its value lies in sampling the biochemical and cellular environment of the central nervous system when infection, inflammation, bleeding, pressure disturbance, or a selected neurodegenerative process is plausible. The marks are therefore earned less by reciting a laboratory list than by showing sound sequence: decide whether the result can change the differential, establish that puncture is safe, do not postpone time-critical treatment, request the right studies, and interpret the pattern in its clinical context.

Lumbar puncture is the usual route to CSF. It is invasive and uncomfortable, and its commonest complication is post-lumbar puncture headache. That simple fact should discipline requesting behaviour without making the test seem exceptional or mysterious. A well-chosen study may convert a broad neuropsychiatric presentation into a treatable infectious or inflammatory diagnosis; a poorly chosen study exposes the patient to discomfort and risk while producing results that are difficult to interpret.

5.1 Start with the clinical question

The first decision is not "Which CSF tests should I order?" but "What process am I trying to confirm or exclude?" Acute headache with fever and neck stiffness raises a different question from subacute behavioural change, progressive cognitive decline, or papilloedema. CSF is most useful when the differential contains a process that directly alters pressure, cells, protein, glucose, immunoglobulins, microbial material, or disease-associated proteins. It is much less useful as a broad fishing exercise after an unrevealing psychiatric assessment.

A classic bedside trigger is the presence of at least **two of three** features: headache, fever, and nuchal rigidity. In that setting, puncture is directed toward meningitis, subarachnoid haemorrhage, or another inflammatory disorder of the central nervous system. The triad is a prompt to act, not a substitute for clinical judgement. Neuropsychiatric presentations may foreground confusion, altered behaviour, reduced engagement, or cognitive change; the psychiatrist must still look actively for neurological and systemic clues rather than waiting for a fully expressed textbook syndrome.

The **emergency CSF indication** is central nervous system infection, particularly meningitis. Encephalitis also requires urgent analysis and is managed presumptively as herpes simplex encephalitis until CSF polymerase chain reaction is negative. This distinction matters because diagnostic urgency and treatment urgency are coupled but not identical: the sample should be obtained promptly when safe, while treatment must not be held hostage to the procedure.

RULE **Send CSF only when the clinical question can be stated before the needle is inserted.** "Exclude organicity" is not a sufficient request; name the suspected compartmental process and the result that would alter management.

5.2 Safety before puncture, treatment without delay

The decisive hazard is not raised pressure in the abstract. It is a pressure gradient created by a mass lesion or obstructive hydrocephalus. Removing CSF from the spinal compartment can lower pressure below the lesion while intracranial pressure remains unopposed, precipitating transtentorial displacement. The feared outcome is **brain herniation** and death. This is why the pre-procedure neurological examination is not ceremonial paperwork: fundoscopy, arousal, and focal signs determine whether imaging must come first.

Pre-lumbar-puncture imaging is mandatory when papilloedema, any impairment of consciousness, or any focal neurological sign is present. A mass lesion or obstructive hydrocephalus contraindicates puncture. Local sepsis matters as well: a sacral decubitus ulcer contraindicates the procedure because the needle may carry organisms into the spinal canal. These are different mechanisms of harm, and each must be checked rather than compressed into the vague instruction "rule out contraindications."

MNEMONIC

Fundi, Consciousness, Focality, Compartment, Skin

Before puncture, check the **fundi** for papilloedema, **consciousness** for any impairment, the examination for **focality**, imaging for a dangerous intracranial **compartment** lesion when triggered, and the puncture-site **skin** for infection.

Sequence becomes especially important in suspected pyogenic bacterial meningitis. If clinical assessment finds no contraindication, puncture follows expeditiously after assessment and blood cultures. If imaging is required, antibiotics begin after assessment and blood cultures rather than waiting for the scan or the subsequent puncture. CSF obtained after imaging has established safety may remain diagnostically useful even after treatment has begun.

PITFALL

Do not turn "image before puncture" into "image before antibiotics." In suspected bacterial meningitis, treatment must not wait for imaging or lumbar puncture. The safety sequence protects the patient from herniation; it does not justify delaying antimicrobial treatment.

Outside suspected acute bacterial meningitis or subarachnoid haemorrhage, the usual sequence is to postpone puncture until imaging has excluded an intracranial lesion when one is a concern. In those rapidly evolving conditions, diagnosis is time-sensitive, but the bedside safety screen still governs whether puncture can proceed. “Urgent” never means “blind.”

5.3 What the procedure is meant to capture

The needle is inserted caudal to the lower boundary of the spinal cord to avoid cord injury. The procedural objective is not merely to obtain clear fluid. Depending on the question, the study may need a pressure measurement, visual inspection, cell counts and differential, chemical analysis, microbiological studies, or specialised protein and immunological tests. The request form should therefore carry the working diagnosis and the clinically relevant timing, rather than the unhelpful instruction “CSF routine.”

Opening pressure is essential when papilloedema persists despite a good-quality magnetic resonance study that does not show a structural cause. In that context, pressure measurement and CSF analysis are required together: the former pursues idiopathic intracranial hypertension, while the latter helps exclude low-grade chronic meningitis. Raised pressure without a mass lesion is not itself the same contraindication as a dangerous pressure gradient; after appropriate imaging, puncture in idiopathic intracranial hypertension is diagnostic and may occasionally be therapeutic.

The **routine CSF profile** comprises colour, red and white blood cell counts, protein, and glucose. These measurements answer different questions. Appearance may disclose blood or pigment; the cell count establishes whether there is pleocytosis and the differential suggests the inflammatory response; protein reflects disruption and inflammatory burden; glucose must be interpreted with blood glucose. The accompanying figure places these core constituents alongside opening pressure and oligoclonal immunoglobulins, and links them to the decision to send CSF.

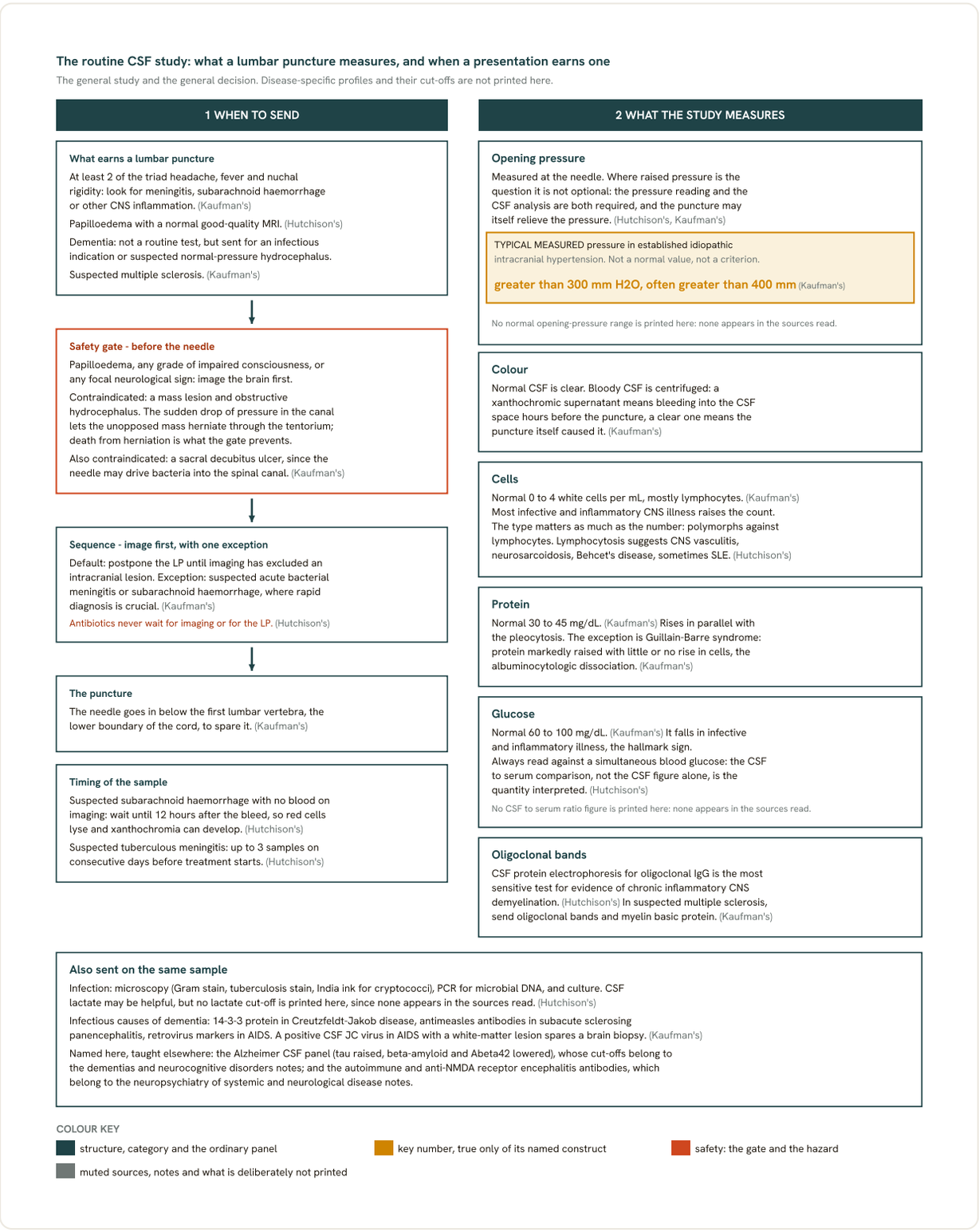


Fig 31.3 The routine CSF study: the constituents measured and the general when-to-send logic. The decision, not the panel, is the teaching: a presentation earns a lumbar puncture, the safety gate (papilloedema, any impaired consciousness, any focal sign) decides whether imaging comes first, and only a mass lesion or obstructive hydrocephalus, not raised pressure itself, forbids the needle. The study then returns pressure, colour, cells, protein, glucose read against a simultaneous blood glucose, and oligoclonal bands. Disease-specific profiles are deliberately absent. Three quantities that a reader may expect are omitted because they were not found in the sources read: the normal opening-pressure range, the CSF to serum glucose ratio, and the CSF lactate cut-off; the only pressure figure shown is the typical measured pressure in established idiopathic intracranial hypertension, which is neither a normal value nor a diagnostic criterion. Attributions per box (Kaufman's Clinical Neurology for Psychiatrists, Hutchison's Clinical Methods).

ASK	CHECK	SEND	PAIR
WHAT DIAGNOSIS COULD CSF CHANGE?	IS PUNCTURE SAFE NOW?	ONLY THE RELEVANT PANEL	RESULT WITH CONTEXT

5.4 Read the routine profile as a pattern

Normal values are the reference frame, not the final diagnosis. Normal CSF is clear. Its white cell count is **0 to 4 WBC/mL**, mostly lymphocytes; protein is **30 to 45 mg/dL**; and glucose is **60 to 100 mg/dL**. Glucose is never interpreted as an isolated CSF number: a simultaneous blood glucose sample is required. The source material establishes the pairing principle but not a numerical CSF-to-blood ratio, so the safe examination answer is to state the comparison without inventing a cut-off.

DOMAIN	NORMAL REFERENCE	INTERPRETIVE QUESTION
Colour	Clear	Is blood or pigment present?
White cells	Within the stated range, mostly lymphocytes	Is there pleocytosis, and which cell type predominates?
Protein	Within the stated concentration range	Does it rise with inflammation, or rise disproportionately to cells?
Glucose	Within the stated concentration range	How does it compare with simultaneous blood glucose?
Red cells	Interpret appearance and processing	Does blood represent an earlier subarachnoid bleed or procedural trauma?
Pressure	Measure when the clinical question requires it	Is pressure itself part of the suspected disorder?

The useful habit is to move through the same axes every time: appearance, pressure when indicated, cells, protein, glucose paired with blood, then targeted microbiology or immunology. A single abnormality rarely carries the whole interpretation. A cell count without the differential is incomplete; low CSF glucose without simultaneous blood glucose is context-poor; raised protein without attention to the cells misses a classic dissociation.

Interpretation is strongest when it separates observation from inference. “Lymphocyte-predominant pleocytosis with raised protein” describes the sample; “therefore this is a particular infection” goes beyond it and must be tested against the syndrome, timing, microscopy, molecular result, and culture. Conversely, a result within the routine reference profile does not answer a question that requires a specialised assay. The panel must match the hypothesis. This distinction prevents overcalling nonspecific abnormalities as well as false reassurance after an incomplete study.

Trend and coherence also matter, even when the page does not supply disease-specific numerical tables. Ask whether cells, protein, and glucose move in a mutually plausible direction, whether procedural contamination explains the appearance, and whether the result fits the neurological examination and imaging. If one element is strikingly discordant, reconsider the working

diagnosis, the sample, and the requested test before creating an elaborate explanation. CSF is evidence within a case formulation, not an autonomous diagnosis.

GOOD TO REMEMBER

Arrange the blood glucose at the same time as the puncture. CSF glucose must be compared with a simultaneous blood glucose concentration; a later or unpaired value weakens interpretation.

5.5 Infection and inflammation: order the panel, then recognise the pattern

When infection is the question, the minimum reasoning set is broader than "cells, protein, sugar." It includes white cell count, whether polymorphonuclear neutrophils or lymphocytes predominate, paired CSF and blood glucose, microscopy for microorganisms, polymerase chain reaction, and appropriate culture. Protein contributes to interpretation, and CSF lactate may be helpful. Microscopy may include Gram stain, staining for tuberculosis, and India ink for cryptococci. A numerical lactate threshold is not supplied here and should not be improvised.

- State the suspected infection so the laboratory can select appropriate microscopy, molecular analysis, and culture.
- Interpret the white cell count together with the differential rather than labelling every cellular response "infection."
- Pair CSF glucose with simultaneous blood glucose and treat protein as part of the pattern.
- Integrate microbiological evidence with the clinical syndrome and treatment timing.

Most infectious or inflammatory central nervous system illnesses produce **pleocytosis**. Protein tends to rise in parallel, while glucose falls to an abnormally low level in the characteristic generic pattern. This directional pattern is more defensible than memorising ungrounded disease-specific tables. It guides the next question, but overlap means it cannot replace culture, microscopy, molecular testing, or clinical judgement.

The classic exception is **albuminocytologic dissociation**: markedly elevated CSF protein with little or no increase in white cells, seen in Guillain-Barré syndrome. The teaching value is conceptual. A high protein result does not automatically imply a cellular inflammatory meningitis; the relationship between protein and cells is itself diagnostic information.

Suspected low-grade tuberculous meningitis may require repeated sampling on consecutive days before treatment to improve the chance of identifying and culturing mycobacteria. The verified source permits a finite repeat strategy, but the exact count is deliberately not printed here. This is less immediately urgent than an acute central nervous system infection, yet it remains an essential investigation when the syndrome warrants it.

5.6 Blood-stained CSF: traumatic tap or subarachnoid bleeding?

Blood introduced by the needle can falsely suggest subarachnoid haemorrhage. The laboratory distinction depends on centrifuging the bloody sample and examining the supernatant. A clear supernatant supports blood introduced during the procedure. A yellow supernatant indicates

xanthochromia, produced by degradation of haem after blood has been present in the CSF space, and therefore supports an earlier true bleed.

Timing is part of the test. When cranial imaging does not show subarachnoid blood but clinical suspicion remains, CSF examination should be delayed until at least **12 hours after the suspected bleed**. This interval allows red cells to lyse and haemoglobin to be metabolised into pigments detectable by spectroscopy or visible as xanthochromia. Sampling too early can make procedural blood and subarachnoid blood impossible to distinguish reliably.

TRAP **"Bloody CSF equals subarachnoid haemorrhage" is wrong.** Ask what the centrifuged supernatant shows and whether sufficient time elapsed for xanthochromia to develop.

This example captures a wider examination principle: pre-analytical details can determine whether a result is meaningful. The same observed abnormality may arise from disease or from the procedure. Interpretation therefore begins before the laboratory report, with the indication, timing, and quality of the sample.

5.7 Pressure and chronic inflammatory studies

Idiopathic intracranial hypertension illustrates why "raised intracranial pressure contraindicates lumbar puncture" is an unsafe oversimplification. After good-quality imaging has excluded a mass lesion, puncture is essential to measure pressure and analyse CSF. Established disease may show markedly raised measured pressure, with low protein, normal glucose, and no cells. The diagnostic threshold belongs to the dedicated idiopathic intracranial hypertension discussion and is not interchangeable with a typical measured pressure. The key distinction here is structural: raised pressure without a mass lesion does not create the same puncture hazard as a compartmental pressure gradient.

For suspected chronic inflammatory demyelination, CSF protein electrophoresis detecting **oligoclonal IgG** is the most sensitive evidence of chronic inflammatory central nervous system demyelination. In suspected multiple sclerosis, CSF is sent for oligoclonal bands and myelin basic protein. A very high cell count or very high protein may be incompatible with the working diagnosis and should trigger revision rather than forced confirmation.

CSF lymphocytosis can also accompany central nervous system vasculitis, neurosarcoidosis, Behçet disease, and sometimes systemic lupus erythematosus. These associations tell the psychiatrist when inflammatory disease should remain in the differential; they do not constitute a stand-alone diagnostic algorithm. The autoimmune and anti-NMDA receptor encephalitis antibody workup, along with the detailed neuroinfection panels, belongs in the Neuropsychiatry of systemic and neurological disease notes.

5.8 The neuropsychiatric workup: selective, not routine

A **dementia workup** does not routinely include lumbar puncture. It becomes appropriate when the presentation suggests an infectious cause, including neurosyphilis, subacute sclerosing panencephalitis, or Creutzfeldt-Jakob disease, or when suspected normal-pressure hydrocephalus makes pressure measurement and CSF withdrawal part of the diagnostic assessment. This is a

high-yield boundary: progressive cognitive symptoms alone do not justify a generic CSF panel, but a specific infectious, disease-directed, or pressure-related hypothesis may.

CLINICAL QUESTION	WHAT CSF CONTRIBUTES	BOUNDARY OF THIS SECTION
Infectious cognitive disorder	Disease-directed proteins, antibodies, retroviral markers, antigens, or polymerase-chain-reaction-detectable DNA	Order to the suspected organism or syndrome
Normal-pressure hydrocephalus	Pressure assessment and withdrawal of CSF	Interpret within the full clinical and imaging assessment
Progressive multifocal leukoencephalopathy in AIDS with a white-matter lesion	**JC virus CSF testing**; a positive result can obviate brain biopsy	A clear example of CSF replacing a more invasive test
Alzheimer disease biomarker question	Increased tau with decreased beta-amyloid and amyloid-beta peptide	The Dementias and neurocognitive disorders notes own the panel and its cut-offs
Autoimmune encephalitis or endemic neuroinfection	Targeted antibody or infection studies	The Neuropsychiatry of systemic and neurological disease notes own the algorithms and titres

Examples of disease-directed testing include protein associated with Creutzfeldt-Jakob disease, antimeasles antibodies in subacute sclerosing panencephalitis, retroviral markers in AIDS, and specific antigens or polymerase-chain-reaction-detectable DNA in other infections. These are not a licence to send every marker in every patient. Each test belongs to a prior clinical hypothesis, and the result must be interpreted within that disease pathway.

The same ownership principle prevents unnecessary duplication. Alzheimer disease CSF biomarkers may be named here by their direction of change, but their numerical interpretation belongs to the Dementias and neurocognitive disorders notes. Autoimmune encephalitis antibodies and detailed neuroinfection findings are indications for targeted CSF work, while their algorithms and titres belong to the Neuropsychiatry of systemic and neurological disease notes.

Before accepting the report, return to the original question. Confirm that the relevant measurement or assay was actually performed, that timing and treatment do not distort its meaning, and that the pattern fits the examination. If not, revise the hypothesis rather than stretching the result to fit it.

Memorise the sequence: define the question, exclude a dangerous pressure gradient or local infection, never delay urgent antimicrobial treatment, obtain pressure when it is part of the question, and interpret cells, protein, glucose, microbiology, and specialised studies as one clinical pattern.

6 · Soft neurological signs and neurological correlates of schizophrenia and developmental disorders

Soft neurological signs are not minor neurology. They are bedside observations that make a psychiatrist ask a more disciplined question: is the finding a focal deficit that requires localisation, a treatment-emergent movement disorder, or a non-localising sign of altered neurodevelopmental or integrative function? **Neurological soft signs** are clinically observable abnormalities that cannot be assigned to impairment of a particular brain region. "Soft" therefore describes their limited localising value, not their clinical importance or the amount of disability a patient experiences.

For psychiatry, the value of this lens is longitudinal and comparative. The signs can be present before drug exposure, may accompany cognitive and symptom dimensions, and can be encountered across psychotic and developmental presentations. They do not diagnose schizophrenia, ADHD, autism, or any other disorder in isolation. Instead, they sharpen formulation, improve the baseline against which later motor adverse effects are judged, and identify occasions when the ordinary psychiatric examination must widen into a careful neurological assessment.

6.1 What makes a sign "soft"?

A **hard neurological sign** has sufficient pattern, asymmetry, or associated deficit to support anatomical localisation. In contrast, soft signs are nonfocal abnormalities in the integration and execution of function. They are commonly observed through tasks involving coordination, sensory integration, developmental reflexes, balance, motor planning, and motor control. This is a functional description rather than a focal localisation.

RULE **"Soft" means non-localising, not benign.** A subtle, bilateral and non-lateralising performance abnormality can contribute to psychiatric formulation; a lateralised or gross abnormality must be read first as a possible focal neurological sign.

The distinction is easiest to understand when the same manoeuvre can be read in different ways. **Dysdiadochokinesia** means impaired rapid alternating movements. In a clearly lateralised presentation with intention tremor and other limb-coordination problems, it has localising significance for cerebellar hemisphere injury. A mild, bilateral irregularity without a localising pattern is instead an observation that may sit within a soft-sign assessment. The manoeuvre has not changed; its clinical meaning has changed with the total neurological pattern.

This is also why soft signs must never become a shortcut around a proper examination. A hard-sign finding should not be collapsed into an NSS score or explained away as "part of schizophrenia." The physical neurological examination and applied localisation are addressed in the Functional Neuroanatomy and Neurophysiology notes and the Stroke, TBI, delirium and focal brain syndromes notes.

PITFALL

Calling an abnormal movement “soft” before checking for asymmetry, weakness, sensory loss, altered reflexes, medication exposure, or a changing level of function is unsafe. Soft-sign language describes a pattern of non-localisation; it is not a permission to stop looking for neurological disease.

6.2 The domains that organise observation

The most useful teaching structure is the DSM-5-TR grouping: impairments in motor coordination, sensory integration, and sequencing of complex movements, with left-right confusion and disinhibition of associated movements included in the clinical picture. This grouping gives the examiner a way to notice patterns without pretending that any isolated task is diagnostic. Motor coordination asks whether an action is steady, timed, and appropriately graded. **Sensory integration** asks whether sensory information is being synthesised into an accurate percept or body schema. Sequencing asks whether the patient can organise a complex motor act in the required order. Associated-movement disinhibition concerns unwanted accompanying activity rather than the intended action alone.

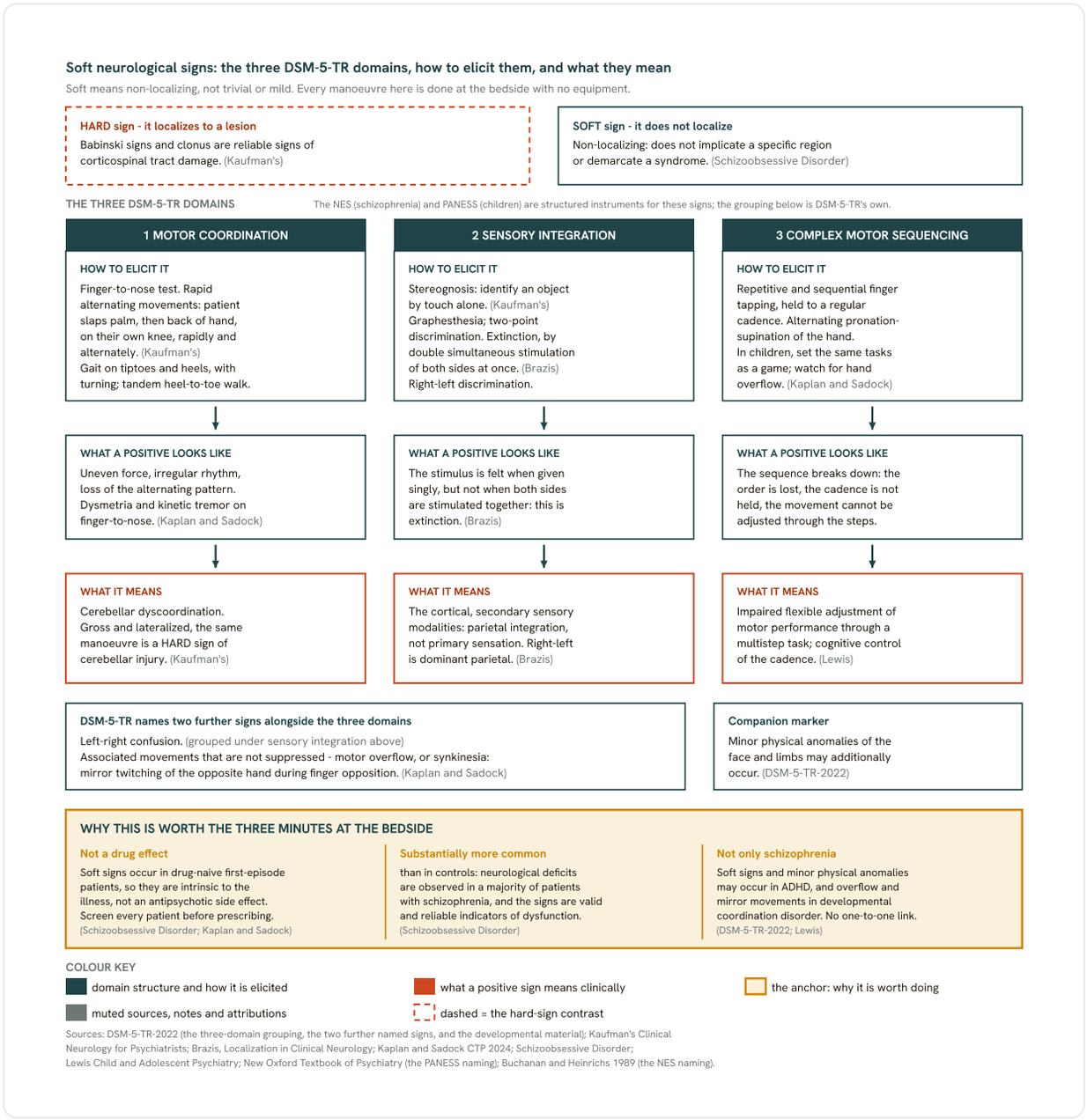


Fig 31.4 Soft neurological signs: the three DSM-5-TR domains, how each is elicited, and what a positive sign means. DSM-5-TR-2022 states that the soft signs common in schizophrenia comprise impairments in motor coordination, sensory integration and motor sequencing of complex movements, together with left-right confusion and associated movements that are not suppressed; minor physical anomalies of the face and limbs may additionally occur. The signs are called soft because they do not localize to a lesion, not because they are trivial or mild: the same manoeuvre that reads as a soft sign when it is subtle and bilateral reads as a hard sign when it is gross and lateralized. They are substantially more common in schizophrenia and in the developmental disorders than in controls, they are present in drug-naive first-episode patients, and every manoeuvre here is done at the bedside with no equipment. Grouping cited to DSM-5-TR-2022; elicitation and interpretation attributed per box (Kaufman's Clinical Neurology for Psychiatrists; Brazis, Localization in Clinical Neurology; Kaplan and Sadock CTP 2024; Schizoobsessive Disorder; Lewis Child and Adolescent Psychiatry).

CLINICAL DOMAIN	WHAT THE EXAMINER IS WATCHING FOR	INTERPRETIVE DISCIPLINE
Motor coordination	Balance, posture, precision and the smooth control of movement	Look for consistency, laterality and associated cerebellar or pyramidal signs before calling it soft.
Sensory integration	Integration of touch, position, body-side information and complex sensory perception	Ensure that primary sensation, comprehension and attention are adequate for the task.
Complex motor sequencing	Ordering, switching and sustaining a multistep motor programme	Separate an execution error from misunderstanding, fear, pain, fatigue, or severe psychopathology.
Associated-movement disinhibition	Unintended accompanying movement or overflow during a voluntary task	Describe what is seen and document baseline status before attributing later change to medication.

The accompanying figure is best used as a map for observation, not as a diagnostic algorithm. Within the motor domain, a rapid alternating-movement task can reveal irregular rhythm, uneven force, and failure to maintain alternation. Within the sensory domain, **stereognosis** is recognition of an object's form by touch. It is a cortical sensory modality, as are graphesthesia and related integrative tasks, and should not be mistaken for a basic test of light touch. Right-left confusion similarly belongs to an integrative task domain and should be interpreted alongside the rest of the neurological examination.

Extinction offers a useful contrast. On double simultaneous stimulation, the affected side is perceived when stimulated alone but not when both sides are stimulated together. This is a focal attentional-sensory phenomenon, not a generic soft sign to be casually scored. The lesson is practical: an NSS battery can direct attention to integrative function, but it cannot replace the clinician's duty to decide whether a result localises.

MNEMONIC

CIS

Use **C**oordination, **I**ntegration and **S**equencing as the bedside spine. Then look separately for associated-movement disinhibition. The mnemonic organises observation; it does not convert the domains into diagnostic criteria.

6.3 Primitive reflexes and motor overflow

Primitive reflexes require particular care because their presence can be discussed under soft-sign traditions, yet the finding may carry a harder neurological implication in the appropriate context. **Frontal release reflexes** include snout, suck, rooting, jaw jerk, palmomental and grasp responses, most of which are normally present in infancy. Their appearance in an adult is abnormal. Several such reflexes, interpreted with the history and the remainder of the examination, can suggest

congenital cerebral injury, frontal lobe damage, or neurodegenerative illness. They should never be treated as a diagnostic signature of a functional psychiatric syndrome.

For the psychiatrist, their importance is that pathological reflexes can support an organic basis for personality change and correlate to some degree with cognitive impairment. The relevant frontal-system anatomy belongs in the Functional Neuroanatomy and Neurophysiology notes; here, the examination lesson is to record the sign, its distribution, and its neurological context rather than using it as decorative neuropsychiatric jargon.

Synkinesia, or motor overflow, denotes unintentional movement accompanying a voluntary action. Mirror movements, contralateral finger twitching during a finger-opposition task, and facial grimacing during hand movements illustrate the phenomenon. In children, overflow can be read as a developmental immaturity; in adults, the same observation needs to be set against medication history, neurological examination, and course. “Disinhibition of associated movements” is a descriptive observation, not a licence to infer a particular lesion.

6.4 Schizophrenia: correlates, course and meaning

NSS are common in schizophrenia, but they are not specific to it. Their significance lies in convergence with a developmental reading of illness, not in diagnostic exclusivity. They have been observed in **dopamine-antagonist medication-naive first-episode psychosis** and in most people with chronic schizophrenia. This makes a purely iatrogenic explanation inadequate, while still leaving room for medication to add new extrapyramidal phenomena later in the course.

Evidence also links NSS with cognitive deficits and with negative and disorganised symptoms. A further association has been reported with cognitive and motivation-expression deficits and with positive psychopathology resistant to initial antipsychotic treatment. These are correlates, not a severity scale that determines treatment choice. The clinician should therefore avoid using a soft-sign burden to make a prognostic declaration to a patient or family. It is better used to enrich the baseline description, direct collateral developmental history, and make monitoring for treatment-emergent movement change more defensible.

There is a trait argument and a state argument, and neither should be caricatured. Soft signs have been identified in people at high risk, in monozygotic co-twins discordant for schizophrenia, and in first-degree relatives, supporting their potential as **trait markers**. Prospective developmental evidence is also striking: children who later developed schizophrenia had **more than one soft neurological sign** and poorer motor-skill performance at several childhood assessments. Yet some motor and cortical signs may improve as psychopathology improves, whereas harder signs can appear more static and trait-like. The careful conclusion is that the construct contains both state-sensitive and trait-like elements.

This pattern fits the broader association of neurological signs with cognitive dysfunction, developmental anomaly, and diffuse brain pathology. It should be named, then bounded: the full neurodevelopmental account of schizophrenia belongs in the Schizophrenia: aetiology, phenomenology, classification and course notes. Reported neural-circuit and structural correlations are mechanistic correlates, not a bedside imaging indication.

CLINICAL QUESTION	WHAT NSS CAN CONTRIBUTE	WHAT NSS CANNOT ESTABLISH
Was movement present before treatment?	A documented baseline for later comparison	That a later movement is necessarily unrelated to medication
Is there a neurodevelopmental signal?	A prompt to obtain developmental and family history	A diagnosis of schizophrenia or aetiological proof
Is cognition relevant to current disability?	A reason to assess cognition carefully and functionally	A substitute for formal cognitive assessment
Could this be focal disease?	A cue to inspect for localising features	A reason to ignore a focal or progressive deficit

The cognitive screen and its formal interpretation are covered in the Psychometry, Assessment and Descriptive Psychopathology notes. The acute adverse-effect framework belongs in the Schizophrenia: management, antipsychotics and adverse effects notes. The present task is narrower and clinically potent: identify what was present before prescribing, describe it precisely, and refuse false certainty about causation.

NONLOCALISING	CIS	BASELINE	INTRINSIC
DEFINING FEATURE	CLINICAL SPINE	BEFORE PRESCRIBING	NOT SIMPLY DRUG EFFECT

6.5 Structured assessment and the baseline motor record

The **Neurological Evaluation Scale** is a structured instrument for assessing neurological signs in schizophrenia. It contains **26 items**, organised into motor coordination, sensory integration, sequencing of complex motor acts, and a residual "others" grouping. Each item is rated on an ordinal **0 to 2** scale using standardised instructions, with higher scores indicating greater disruption. Its psychometric literature should be read with attention to the instrument's stated scope and the licence boundary.

The NES is useful for standardising observation and for research-quality description. It is not a neurological consultation in a booklet, and this chapter does not reproduce its protected items, wording, or scoring anchors. In ordinary practice, a structured screen should be embedded within the general motor examination: gait, station and balance, abnormal movement at rest and with arms outstretched, coordination, tone, and strength. Finger-to-nose and rapid alternating movements are standard cerebellar manoeuvres; finger-to-nose also samples proximal arm movement, kinetic tremor, and dysmetria.

GOOD TO REMEMBER

Record spontaneous movements and soft signs before the first psychiatric drug exposure whenever feasible. Abnormal movements can precede treatment and have a broad differential, including congenital, developmental, neurodegenerative and acquired cerebral conditions, as well as normal ageing. A later adverse-effect judgement is only as good as the baseline record.

Baseline signs may also be relevant when monitoring for treatment-emergent extrapyramidal adverse effects. This does not make NSS a deterministic prediction tool, but it is a strong practical

reason to document the motor examination rather than retrospectively reconstruct it after an adverse effect occurs. Careful appraisal of movement belongs to general psychiatric practice because emotional, cognitive and motor disorders involve interrelated basal-ganglia and frontal-subcortical circuits.

6.6 Developmental interpretation in children

In developmental psychiatry, a soft sign is often best understood as delay in a normally acquired achievement: integration of motility, smooth and accurate motor sequencing, or auditory-visual integration. This language avoids prematurely labelling a child with a neurological abnormality when the observed phenomenon may represent immaturity. Its value depends on developmental context, functional impact, and whether the observation persists beyond what is expected for the child.

Associations with psychopathology have been demonstrated when **at least 3 soft neurological signs** are present in children older than **7 years**. The age qualifier matters: earlier in development, many signs may represent delayed achievements rather than persisting dysfunction. This is a threshold for an association, not a diagnostic criterion and not an invitation to count minor behaviours without a developmental examination.

Assessment can be made collaborative and game-like. Useful observations include ball throwing, walking on a line, heel-to-toe walking, overflow of the hands while walking on the outside of the feet, repetitive or sequential finger tapping, and alternating pronation-supination. The child should understand the task and have an opportunity to practise. A poor performance can arise from anxiety, inattention, oppositionality, pain, unfamiliarity, language difficulty or reduced comprehension, so the examiner must distinguish a stable motor pattern from situational non-cooperation.

- Observe quality, not merely completion: rhythm, bilateral symmetry, sequencing and overflow often provide more information than success or failure.
- Compare across tasks: a single awkward action is weak evidence, whereas a convergent pattern across coordination, integration and sequencing is more meaningful.
- Link the finding to daily function: handwriting, play, self-care, classroom demands and social confidence determine its practical significance.

In developmental coordination disorder, mirror movements and choreiform movements of unsupported limbs are described as neurodevelopmental immaturities or soft signs rather than neurological abnormalities, and their diagnostic role remains unclear. Developmental coordination disorder occurs in **5 to 8 per cent** of children aged **5 to 11 years**. The crucial clinical distinction is that overflow may support a developmental formulation but does not independently make the diagnosis.

6.7 ADHD, communication disorders and broader developmental correlates

ADHD has no diagnostic physical sign, though subtle motor delays, NSS and minor physical anomalies may occur. **Minor physical anomalies** described in this context include

hypertelorism, a highly arched palate and low-set ears. Marked co-occurring clumsiness or motor delay should prompt separate consideration of developmental coordination disorder rather than being absorbed into ADHD. Soft signs or minor anomalies may modestly increase confidence in an ADHD diagnosis, but there is no one-to-one relation.

The proposed reading of ADHD soft signs concerns cognitive control of motor inhibition and excitation during repetitive actions, flexible adjustment in multistep tasks, and integration of proprioceptive input and body-position sense in gait and balance. A reported association with reduced bilateral frontal cerebral blood-flow measures does not make functional imaging part of routine ADHD workup. The finding is a correlate, not a test to order.

Similar observations have been described in speech sound disorder and language disorder, alongside subtle motor-skill deficits and mixed cerebral dominance. These observations argue for a broad developmental review where function is impaired, not for the false claim that an NSS profile can identify a single psychiatric diagnosis.

TRAP **Do not turn a developmental correlate into a diagnostic sign.** Clumsiness, overflow, minor anomalies and soft signs can strengthen the decision to examine development more fully, but they do not replace syndrome-level history, impairment assessment or differential diagnosis.

6.8 A practical psychiatric approach

LOCUS is the routine outpatient, emergency or inpatient assessment where movement and neurological history should be captured before treatment alters the picture. FOCUS is the current motor phenotype, its onset and course, associated cognition or behaviour, and any localising or progressive features. MODUS is careful observation, a focused neurological examination, structured documentation when useful, collateral developmental history, and escalation for neurological evaluation when the pattern is focal, progressive, or otherwise concerning.

For a person presenting with schizophrenia, an examination focused on the extrapyramidal system should be part of routine assessment. This is not an optional neuropsychiatry flourish. It protects against misattributing a pre-existing movement abnormality to treatment, provides a defensible baseline, and may identify an alternative neurological explanation for apparent psychiatric change. In a child, the same principle means observing movement in an age-appropriate context and making function, not a checklist total, the centre of interpretation.

A soft neurological sign is a non-localising clue to integrative or developmental dysfunction; document it, contextualise it, and never let it obscure a focal neurological deficit or a medication-related movement disorder.

7 · Disorders of consciousness, coma, the vegetative and minimally conscious states and brain death

The marks lie at the boundaries. Separate arousal from awareness, coma from wakeful unresponsiveness, purposeful behaviour from reflex movement, and severe impairment from

death. A wrong label may conceal reversible neurological disease, deny a conscious person communication, or confuse a family during critical care.

Use an anatomical and behavioural approach: test arousal, seek reproducible evidence of awareness, examine the brainstem rostrocaudally, and describe observations before naming a syndrome. Higher-function testing belongs to the arousable patient. The descriptive ladder is covered in the Psychometry, Assessment and Descriptive Psychopathology notes, while delirium belongs to the Stroke, TBI, delirium and focal brain syndromes notes.

7.1 The architecture of consciousness

Consciousness depends on interaction between the ascending reticular activating system and the cerebral hemispheres. This gives the clinician separable axes: arousal, supported by the activating system, and awareness, expressed through organized hemispheric function. Alertness is necessary for ordinary awareness but does not guarantee cognition, emotional capacity, or meaningful engagement. Conversely, a patient may retain an intact mind while motor output is almost completely abolished.

The ascending reticular activating system occupies the paramedian tegmentum of the posterior pons and midbrain and extends toward the posterior hypothalamus and thalamic reticular formation. Diffuse thalamocortical projections regulate cortical activity. Ocular motor structures lie within this system, explaining why eye and pupil findings localise brainstem dysfunction. Sedatives may depress the same synaptic network that structural disease can damage.

RULE **Coma requires failure of the activating system or failure of both cerebral hemispheres.** A unilateral hemispheric lesion is therefore an exceptional and usually transient explanation unless it produces secondary brainstem compromise.

Clinical neurology describes a descending sequence of **alert, lethargic, stuporous, and comatose**. A lethargic patient appears asleep but can be brought temporarily to an awake state by sufficient stimulation, remaining inattentive, disoriented, and cognitively impaired when roused. A stuporous patient remains unarousable and gives only rudimentary motor or verbal responses. In coma, the eyes remain closed, limb movement is reflexive, verbal response is absent or minimal, and there is no meaningful environmental interaction. Coma is usually transitional, lasting from hours to weeks before recovery, death, or evolution into another disorder of consciousness.

7.2 Bedside examination of coma

Build a pattern from pupils, spontaneous and reflex eye movements, motor symmetry, posturing, and respiration while assessing reversible toxic and metabolic causes. The light reflex resists metabolic dysfunction, so unilateral abnormality strongly suggests a structural midbrain or oculomotor lesion. Drugs, hypothermia, and acute anoxia can invalidate this shortcut.

- **Pupils:** metabolic or bilateral diencephalic dysfunction usually leaves small, reactive pupils. Tectal or pretectal midbrain dysfunction produces midsized or slightly enlarged pupils without a light response but with a ciliospinal response. Tegmental midbrain dysfunction

produces midsized, often unequal pupils lacking both responses. Pontine dysfunction causes small pupils, while pontine haemorrhage may produce pinpoint pupils that still constrict under magnification.

- **Herniation:** lateral transtentorial compression of the oculomotor nerve produces an ipsilateral, widely dilated pupil with a sluggish or absent light response, the Hutchinson pupil. If hemiparesis appears on the same side as the dilated pupil, the Kernohan notch phenomenon is a possible false-localising explanation.
- **Eye movements:** as coma deepens, roving movements disappear before the oculocephalic response, and the caloric response is lost last. Pupillary reactivity may persist after eye movements are no longer elicitable in metabolic coma.
- **Respiration:** Cheyne-Stokes respiration is associated particularly with bilateral thalamic dysfunction but may occur with descending-pathway lesions extending to the upper pons. Apneustic breathing points toward the lower pontine lateral tegmentum, cluster breathing toward low pontine or high medullary dysfunction, and ataxic breathing toward dorsomedial medullary damage and impending respiratory failure. Metabolic disturbance can imitate these patterns.

Oculovestibular testing supplies a stronger stimulus than the oculocephalic manoeuvre. After confirming an intact tympanic membrane and elevating the head by 30 degrees, 50 mL of ice water is instilled over 30 seconds. This is a specialist bedside procedure, not a casual test, because technique and ear pathology determine whether an absent response is interpretable.

MNEMONIC

Pupils, Ocular, Corneal, Gag, Motor

The brainstem-reflex sweep in coma follows **P**upillary light response, **O**cular responses, **C**orneal reflexes, **G**ag and cough, and **M**otor response. Read the findings as a linked rostrocaudal pattern, never as isolated tricks.

7.3 Motor response and posturing

Decorticate posturing consists of shoulder and arm adduction, elbow flexion, and wrist pronation and flexion, with extension of the leg. With a unilateral hemispheric lesion it is contralateral. **Decerebrate posturing** consists of upper-limb extension and pronation with forceful plantar flexion, and may follow severe metabolic injury or upper brainstem dysfunction. More caudal progression may produce arm extension with weak leg flexion in pontine tegmental damage and total flaccidity with medullary involvement.

TRAP **Posturing does not prove a structural lesion.** Toxic-metabolic coma commonly produces both flexor and extensor patterns. Structural patterns are often asymmetric, but the posture name alone must not be converted into an anatomical diagnosis.

Motor symmetry has greater discriminating value. Marked asymmetry argues against purely metabolic encephalopathy. Tremor, asterixis, myoclonus, and seizures favour toxic-metabolic

dysfunction, although metabolic and structural disease may coexist. Asterixis may disappear as coma deepens. Document the stimulus, best response on each side, spontaneous movement, and serial change.

7.4 Structured assessment of responsiveness

The **Glasgow Coma Scale** records eye opening, verbal response, and best motor response. It is a compact description of observable neurological function, not a diagnosis and not a substitute for the brainstem examination. Report the components as well as the total because identical totals can represent clinically different response patterns.

COMPONENT	RESPONSE	SCORE
Eye opening	Never	1
	To pain	2
	To verbal stimuli	3
	Spontaneously	4
Best verbal response	None	1
	Incomprehensible sounds	2
	Inappropriate words	3
	Disoriented and converses	4
	Oriented and converses	5
Best motor response	None	1
	Extension, decerebrate rigidity	2
	Flexion, decorticate rigidity	3
	Flexion withdrawal	4
	Patient localizes pain	5
	Patient obeys	6

The total ranges from **3 to 15**, with lower totals indicating poorer neurological function. In the traumatic brain injury severity bands used by Kaufman, 3-8 denotes severe injury or coma, 9-12 moderate injury, and 13-15 mild injury. These are trauma-severity bands, not a universal taxonomy of consciousness, and they must not be merged with alternative bands that classify a different construct.

E	V	M	E + V + M
EYE OPENING	VERBAL RESPONSE	MOTOR RESPONSE	REPORT COMPONENTS

The scale correlates closely with outcome after major head trauma but poorly after minor trauma. On the first day after traumatic brain injury, **90%** of patients at the minimum score have a fatal outcome, and most survivors do not regain consciousness. Yet that score cannot determine death.

Cerebral hypoxia and intubation limit interpretation, and the scale does not belong in a mental status examination for dementia or circumscribed cognitive deficit: it grades responsiveness, not cognition.

PITFALL

Writing only a total hides clinically important information and may create false precision when the verbal component is untestable. Record the observed component pattern and the reason for any untestable response.

A low Glasgow Coma Scale score describes impaired responsiveness; it never, by itself, declares death.

Teasdale G, Jennett B. Assessment of coma and impaired consciousness: a practical scale. Lancet 1974;2(7872):81-84. The scale reproduced here is acknowledged as copyright of Glasgow University and is cleared only for the free, non-commercial resident PDF. This clearance does not extend to related prognostic charts, imputation tools, or commercial channels.

The accompanying figure consolidates the bedside distinctions described here.

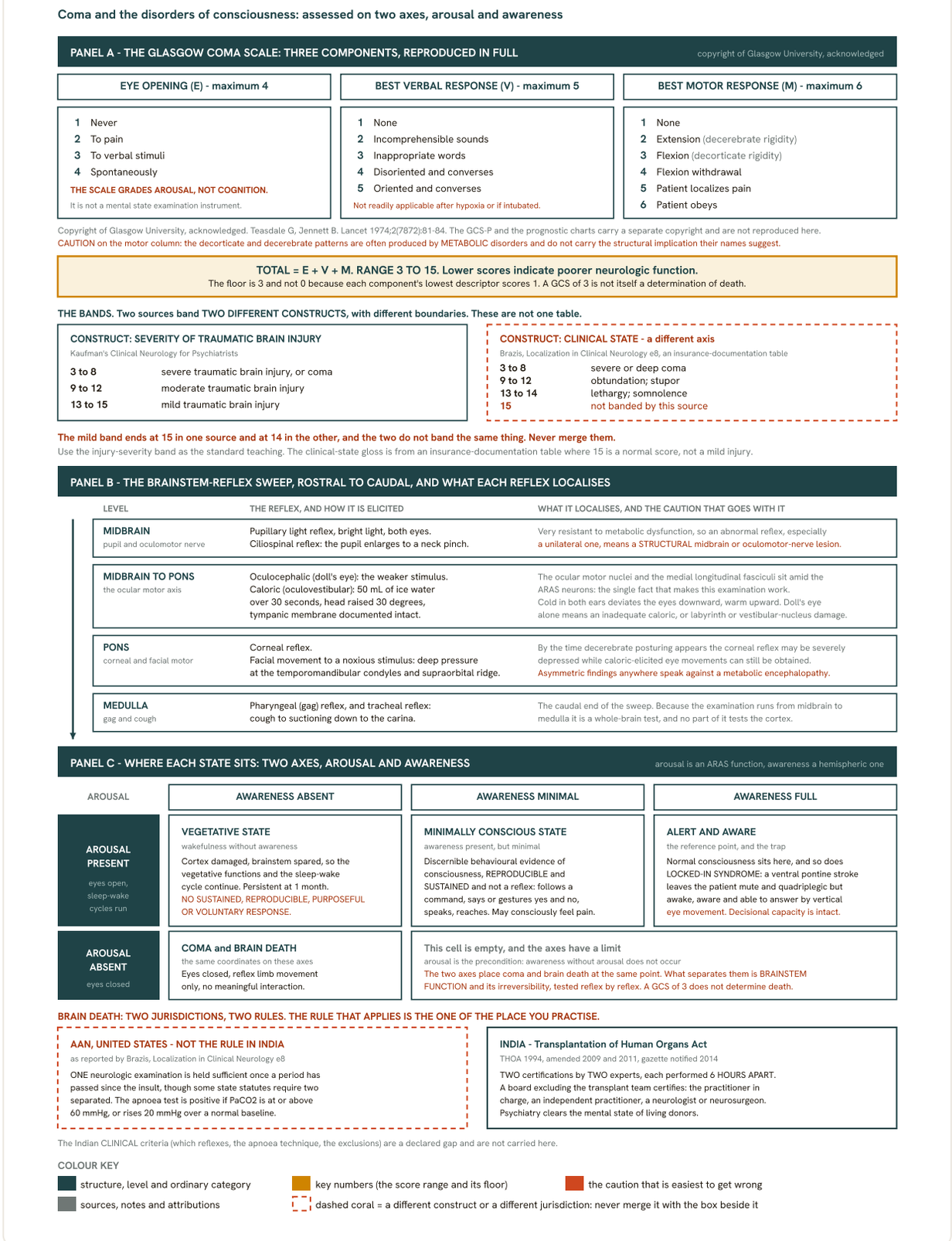


Fig 31.5 Coma and the disorders of consciousness: the Glasgow Coma Scale, the brainstem-reflex sweep, and where each state sits. Consciousness is assessed on two axes, arousal and awareness: coma means either the ARAS is down or both hemispheres are. The Glasgow Coma Scale is the copyright of Glasgow University, acknowledged on the plate, where the licence conditions sit. Brazis prefers 'unresponsive wakefulness syndrome', on the ground that responses are all the examiner can evaluate. (Kaufman's Clinical Neurology for Psychiatrists; Brazis, Localization in Clinical Neurology e8; RC Jiloha, Forensic Psychiatry: An Indian Perspective.)

7.5 Vegetative and minimally conscious states

Vegetative state is wakefulness without awareness: widespread cortical or immediately subcortical damage coexists with relative brainstem preservation. The eyes open, sleep-wake cycling continues, and vital vegetative functions persist, but there is no evidence of awareness of self or environment, language comprehension or expression, or sustained, reproducible, purposeful or voluntary behaviour. Cranial and spinal reflexes vary, and incontinence is typical.

The word persistent is temporal rather than synonymous with permanent. It applies when the vegetative state remains for **at least 1 month** after acute traumatic or nontraumatic injury, or for the same duration in degenerative, metabolic, or developmental disease. Prognosis depends strongly on cause and duration. Kaufman gives categorical irrecoverability thresholds of 1 year after traumatic injury and 3 months after neurodegenerative illness, whereas Brazis frames recovery after comparable durations as unlikely or rare rather than impossible. The conflict should be stated, not flattened into certainty.

Minimally conscious state retains reproducible, sustained, non-reflex behavioural evidence of consciousness. This may be command-following, a verbal or gestural yes-no response even if inaccurate, intelligible speech, appropriate smiling or crying, an answer by speech or gesture, or purposeful reaching and manipulation. Such patients may consciously perceive pain and generally have a more favourable outcome than patients in a vegetative state. Organized cortical activity may be evident on EEG or functional imaging in the minimally conscious state when meaningful activity is absent in the vegetative state.

STATE	AROUSAL	AWARENESS AND BEHAVIOUR	KEY SEPARATION
Coma	Absent	No meaningful interaction	Eyes remain closed
Vegetative state	Present	No sustained, reproducible purposeful response	Wakefulness without awareness
Minimally conscious state	Present but impaired	Reproducible, sustained, non-reflex evidence	Behaviour proves at least intermittent awareness
Locked-in syndrome	Present	Awareness and decisional capacity intact	Output failure, not impaired consciousness
Death by neurological criteria	Absent	Irreversible cessation of all functions of the entire brain	A formal jurisdiction-bound determination

The diagnosis remains epistemically difficult because motor output is the examiner's access to awareness, yet motor response is precisely what severe injury may abolish. Some patients meeting behavioural criteria for chronic vegetative state have shown command-following or awareness on functional neuroimaging. For this reason, unresponsive wakefulness syndrome or persistent unresponsiveness may be preferable language. Imaging may challenge the bedside inference, but structural damage alone does not settle awareness.

7.6 Locked-in syndrome and akinetic mutism

Locked-in syndrome is the inverse of vegetative state. The patient is mute and quadriplegic, yet awake, aware, cognitively intact, and capable of decisions. Eye or eyelid movement permits communication. A typical ventral pontomedullary lesion interrupts bulbar and corticospinal output while sparing the dorsal activating system, hemispheres, and ocular pathways. The EEG is relatively normal.

> **GOOD TO REMEMBER**
>
> In an apparently unresponsive patient with open eyes, ask for vertical eye movement or eyelid movement to command. Correct responses permit a communication code and a formal assessment of cognition and capacity. Horizontal gaze may be impaired by the pontine lesion.

Severe peripheral weakness can create the same de-efferented appearance. Myasthenia gravis, amyotrophic lateral sclerosis, and Guillain-Barre syndrome may abolish cranial and limb movement; a complete locked-in state can even lose pupillary responses and be mistaken for anoxic brain damage. A normal or mildly slow EEG is a crucial clue that cerebral function persists. The ethical consequence is immediate: a conscious patient may express treatment preferences once a reliable communication method is established.

Akinetic mutism describes a seemingly awake patient who is silent and motionless, with eyes that may track movement but little evidence that attention or interest can be engaged. Descending motor tract signs are limited, while frontal release signs may occur. Bilateral anterior cingulate or frontal lesions, diencephalomesencephalic reticular dysfunction, and lesions of the globus pallidus or hypothalamus are recognised substrates. Autonomic arousal with tachycardia and sweating during discomfort may resemble catatonia and does not discriminate. Frontal release or corticospinal signs and slow-wave EEG abnormalities favour akinetic mutism; a normal or desynchronised low-voltage fast EEG favours catatonia.

7.7 Functional, ictal and autoimmune mimics

Do not diagnose function by theatricality. In functional or dissociative unresponsiveness, the eyelids may be held forcibly shut against opening, or the patient may stare with quick blinks. Pupils are normally positioned and reactive, tone and reflexes are normal, and the oculocephalic response may be random or absent. The decisive discriminator is caloric stimulation: classic vestibular nystagmus with a preserved quick phase requires functioning frontal eye fields and therefore demonstrates cortical activity. True coma lacks that quick phase.

Roving eye movements of light coma cannot be produced voluntarily and therefore argue against feigning. Forced downward gaze has been reported in feigned coma but is not specific because sedative-induced coma may show it during caloric testing. Recurrent functional unresponsiveness is often misdiagnosed as epilepsy or migraine, replacing formulation with repeated disease-labelled treatment.

Nonconvulsive status epilepticus may resemble metabolic encephalopathy without clear focal signs. Passively elevate the lids and look for small-amplitude, mainly vertical eye movements,

then inspect for subtle intermittent focal or multifocal rhythmic movement. Suspicion warrants prompt EEG; the broader seizure assessment belongs to the Epilepsy and psychiatry notes.

Anti-NMDA receptor encephalitis is a critical psychiatric trap. A psychiatric presentation may be followed by choreic or other movement disorder, respiratory insufficiency, and coma. Akinetic-mutism-like or catatonia-like states may alternate with agitation, while resistance to eye opening and absent response to pain can misleadingly appear dissociative. The combination of psychiatric symptoms and apparently functional examination findings must therefore increase, not reduce, concern for this treatable autoimmune disorder. Its full investigation belongs to the Neuropsychiatry of systemic and neurological disease notes.

7.8 Death by neurological criteria

Death by neurological criteria is clearer clinical language than brain death because it avoids implying an intermediate state followed by a later, more real death when ventilation is withdrawn. The diagnosis requires irreversible cessation of all functions of the entire brain. It is categorically different from coma, vegetative state, minimally conscious state, and locked-in syndrome.

The following clinical framework is the American Academy of Neurology position for the United States, not the Indian clinical standard. Before examination, clinicians establish an irreversible proximate cause, exclude central nervous system depressant drugs and neuromuscular blockade, exclude severe electrolyte, acid-base, or endocrine disturbance, ensure core temperature is above 36 degrees Celsius, and ensure systolic blood pressure is at least 100 mmHg.

The neurological assessment requires coma, absence of brainstem reflexes, and apnoea. The reflex examination demonstrates absent bilateral pupillary light responses, absent oculocephalic and oculovestibular responses, absent corneal responses, absent facial movement to noxious stimulation, and absent gag and cough. Spinally mediated movements do not refute the diagnosis when every brainstem-mediated response is absent.

Under the same United States framework, the apnoea test evaluates respiratory drive using a carbon dioxide challenge after physiological prerequisites are met and oxygenation is maintained. It supports the diagnosis when respiratory movement remains absent and arterial carbon dioxide reaches at least **60 mmHg**, or rises by 20 mmHg above a normal baseline. This is an intensive-care procedure with explicit preparation and abort conditions, not an examination-room manoeuvre.

Ancillary testing is used when clinical assessment cannot be completed or interpreted. Hypothermia, drug overdose, and metabolic derangement can mimic electrocerebral silence; silence alone does not prove irreversibility, while persisting EEG activity refutes the diagnosis. Fixed pupils after acute neurosurgical injury also do not prove irreversibility: among 40 patients, 25% recovered function, although none recovered after more than 6 hours of fixed pupils.

Spinal automatisms may persist after total irreversible brain destruction. These include withdrawal, stretch reflexes, toe or abdominal movements, respiratory-like movements, and the

Lazarus sign. Such automatisms occur in up to 40% of heart-beating cadavers, usually within the first 24 hours. Their apparent purposefulness can distress families and junior staff; the decisive point is that no facial, gag, cough, or other brainstem-mediated response is present.

7.9 Indian certification and psychiatric liaison

IN INDIA

The Transplantation of Human Organs Act, 1994, amended in 2009 and 2011 and gazette notified in 2014, recognises brain death as a definite form of death. Certification requires two certifications by two experts, performed 6 hours apart, and certifiers must not belong to the transplant team. These are certification rules. The allowed evidence does not supply Indian clinical testing criteria, so the United States criteria above must not be substituted for local statutory, state, and institutional requirements.

LOCUS: care occurs in the emergency department and intensive-care unit, with later work at the bedside family meeting and within the transplant pathway. **FOCUS:** acutely, protect against a false diagnosis by establishing irreversibility and excluding confounders; subsequently, provide an unambiguous explanation of death, prepare relatives for spinal movements, and separate the death determination from the donation decision. **MODUS:** the work is clinical, procedural, statutory, communicative, ethical, and social. The governing frames are the American position only where its jurisdiction applies and the Indian statutory framework.

For the psychiatrist, capacity problems sit on opposite sides of this boundary. A locked-in patient needs a communication channel before capacity is judged. In transplantation practice, psychiatry clearance is required for the mental state of living donors. In brain death, the primary team raises organ donation and the next of kin may authorise it. Psychiatry identifies impaired understanding, coercion, conflict, or psychopathology and keeps death communication separate from donation counselling.

Families often interpret open eyes, reflex orienting, posturing, or spinal automatisms as awareness or suffering. Clear language should acknowledge what they have seen, state which responses are reflexive, explain which findings would demonstrate awareness, and avoid implying that withdrawal of ventilation causes death. That precision is both neurologically correct and psychologically protective.

Headache and the psychiatric interface

8 · Migraine: psychiatric comorbidity (depression, anxiety, bipolar disorder) and shared mechanisms

Migraine is a neurological disorder with a psychiatric interface, not a personality diagnosis. For the psychiatrist, the important task is to recognise a genuine migraine syndrome, identify comorbid mood and anxiety symptoms without collapsing them into the headache, and decide whether a temporal relationship is evidence of causation or simply coexistence. The distinction

matters because symptoms may be present during an attack, within an aura, between attacks, or as an independent psychiatric disorder. Each setting calls for a different formulation.

The International Headache Society categorisation places migraine among primary headaches. That framing is useful in psychiatric practice: the diagnosis rests on the characteristic clinical syndrome, whereas examination and laboratory findings are generally normal. It does not mean that the suffering is minor. Recurrent pain, sensory sensitivity, anticipatory fear, disrupted functioning and repeated health-care contact can all shape mood and behaviour. Conversely, depression, anxiety and bipolar-spectrum symptoms can alter help-seeking, adherence, sleep, substance use and the interpretation of bodily symptoms. The consultation must therefore hold both sides of the interface in view.

8.1 Establish the migraine phenotype before attributing psychiatric meaning

A psychiatric formulation starts with phenomenology. The **ICHD-3 migraine without aura criteria** require a recurrent attack pattern, a headache lasting **4-72 hours** when untreated or unsuccessfully treated, characteristic pain features, associated nausea or vomiting or light and sound sensitivity, and exclusion of a better explanation. This is not an invitation to diagnose every severe headache as migraine. It is a reminder that the syndrome has a recognisable structure, and that the history should test that structure before depression or anxiety is made the explanatory centre of the case.

The distinction is particularly important when patients describe neurologic symptoms as “panic,” “dissociation,” or “hallucinations.” In **migraine with aura**, the neurologic symptoms are fully reversible and may be visual, sensory or dysphasic, without motor weakness in the typical-aura description. A symptom may develop gradually and each aura symptom has a bounded duration of **5-60 minutes**. The headache may begin during the aura or follow it. This temporal sequence is often more diagnostically useful than the emotional language used to describe it.

Ask for a chronological account in ordinary language: what was noticed first, how it changed, whether a positive phenomenon appeared alongside a loss of function, what followed, and whether the same sequence has recurred. This protects the patient from an erroneous psychiatric label and protects the psychiatrist from missing a neurological syndrome. It also avoids a false binary in which a patient is assumed to have either migraine or emotional distress. An attack can be neurologically typical and psychologically frightening at the same time.

The history is also where the psychiatrist decides what needs parallel assessment. A stereotyped recurrent sequence makes a migraine formulation more coherent, but it does not abolish the need to examine a new or changed presentation in its clinical context. Conversely, a normal neurological examination between familiar attacks should not be used to imply that the pain, disability or comorbid emotional symptoms are unreal. The psychiatric task is interpretive: identify the syndrome, the timing of its effects and the patient’s response to them, rather than forcing all symptoms into a single category.

RULE **Diagnose the headache syndrome and the psychiatric syndrome on their own evidence before deciding how they are related.** Temporal proximity alone can support a hypothesis, but it cannot settle causation.

8.2 Depression: a reciprocal association, not a simple reaction to pain

Migraine-depression comorbidity is clinically substantial and appears reciprocal. Major depression increases subsequent migraine risk, while migraine is associated with an increase in major-depression risk of **up to 4-fold**. The practical implication is that neither diagnosis should automatically be treated as secondary to the other. Pain can worsen mood, but the observed association is not exhausted by understandable demoralisation in a person with recurrent pain.

~40%	~60%	up to 4-fold
OF YOUNG ADULTS WITH MAJOR DEPRESSION AND/OR ANXIETY HAVE MIGRAINE	OF PEOPLE WITH MIGRAINE HAVE A LIFETIME ANXIETY OR MOOD DISORDER HISTORY	INCREASE IN MAJOR-DEPRESSION RISK ASSOCIATED WITH MIGRAINE

These figures are not prevalence estimates for an individual clinic. Their value is to make comorbidity an active part of the routine review. Ask about low mood, loss of interest, guilt, hopelessness, anxiety, panic and self-harm separately from the pain history. Then ask how each condition affects the other: whether attacks precede withdrawal, whether anticipatory worry constricts activity between attacks, whether depression reduces self-care, and whether illness behaviour changes during periods of headache freedom. A longitudinal account is more informative than a single cross-sectional label.

There is an important nuance for examination answers and clinical reasoning. One account describes the depressive association as relatively specific to migraine rather than other severe headache disorders, while other material in the corpus contests that specificity. The reliable conclusion is the reciprocal association itself, not an overconfident claim that depression identifies migraine. A patient can have depression and another headache disorder; conversely, a patient with migraine may have normal mood between attacks. Comorbidity increases clinical vigilance but does not replace neurological diagnosis.

GOOD TO REMEMBER

When a patient with migraine reports “depression,” establish whether this denotes persistent syndrome-level mood change, attack-related dysphoria, avoidance driven by fear of the next attack, medication effects, or more than one of these. The management target becomes clearer only after that separation.

8.3 Anxiety, panic and the bipolar spectrum

The psychiatric association extends beyond depression. Migraineurs are more likely to have **generalised anxiety disorder, panic disorder and bipolar disorder**. This should prompt a proper psychiatric assessment, not a shortcut from headache to diagnosis. Anxiety may be an independent disorder, a response to an unpredictable and disabling illness, an affective component of an aura, or an acute reaction to a frightening neurological symptom. Panic

symptoms may similarly be reported around attacks without establishing that every episodic autonomic complaint is panic disorder.

The bipolar link needs especially disciplined language. Studies described in the source associate migraine specifically with phobias and with **hypomania/bipolar-spectrum conditions**, whereas the depression association may be found across headache disorders more generally. The evidence supplied here does not license a claim about a particular bipolar subtype. Do not convert "spectrum" into a more precise diagnosis than the history supports. Assess past periods of elevated or irritable mood, altered activity and their relation to headache independently, using the framework in the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes.

A useful formulation separates predisposing, precipitating and perpetuating influences without mistaking that structure for aetiological proof. Recurrent severe pain may increase vigilance to bodily sensations and reduce confidence in planning. Anxiety may then amplify avoidance, while low mood may shrink activity and social contact. These are clinically meaningful feedback loops even when no single symptom can be declared the original cause. The formulation should identify modifiable maintaining factors while retaining the neurological diagnosis as a real disorder in its own right.

For the same reason, avoid the outdated **"migraine personality"** formulation. Migraine is not confined to people of a particular social class or to those characterised as rigid, perfectionistic and competitive. Such a label can make the clinician mistake selection effects, coping style or current distress for a causal personality pattern. It also shifts attention away from the patient's actual syndrome. Personality formulation may still be clinically useful when it is based on the individual assessment, but it must not be smuggled in as an explanation for migraine.

TRAP **Do not diagnose bipolar disorder from migraine plus irritability alone.** Irritability can be part of an aura or an attack-state experience; a bipolar-spectrum diagnosis requires its own longitudinal evidence.

8.4 Aura and attack-state symptoms can resemble psychiatric phenomena

Migraine can transiently generate symptoms that fall naturally into psychiatric language. The psychiatric content of migraine aura includes anxiety, depression and irritability, alongside aphasia and altered perception of size, shape or time. During an attack, dysphoria and inattentiveness may look like a depressive or confusional state. Some patients withdraw; others appear unusually active. After recovery, an altered affective state may also be reported. The point is not to psychiatricise these symptoms, but to locate them accurately in the attack sequence.

Visual experience is a common source of confusion. **Visual migraine aura** may include a scotoma, flashing zigzag lines, coloured crescents, tubular vision or distortion of objects. A particularly useful discriminating feature is the **simultaneous positive and negative visual phenomena**: a scintillating experience may occur with an opaque or missing area. This pattern helps distinguish migraine aura from occipital seizure and from a psychiatric hallucination, but it

should be interpreted with the full time course and neurological context rather than used as a solitary test.

CLINICAL QUESTION	MIGRAINE-ORIENTED ANSWER	PSYCHIATRIC IMPLICATION
How does the symptom begin?	Ask whether it emerges gradually and evolves.	A gradual evolving symptom should prompt an aura history before a primary psychiatric inference.
What is experienced?	Look for positive phenomena alongside a deficit or obscuration.	Mixed positive and negative visual experience favours a migraine formulation in the stated differential.
What else is present?	Clarify sensory, language, perceptual and affective changes.	Fear, dysphoria or irritability may be attack-linked rather than independent syndromes.
What follows?	Establish the relationship to headache and recovery.	Repeated stereotyped sequencing is stronger evidence than a symptom label alone.

This approach is a diagnostic safeguard rather than an excuse to dismiss psychiatric symptoms. A patient may have an aura and a panic disorder, or migraine-related dysphoria and major depression. The relevant question is whether the symptom is confined to the migraine sequence, persists outside it, or has both forms. The Epilepsy and psychiatry notes should be used for seizure assessment; the Anxiety, OCD and trauma-related disorders notes cover diagnosis of the anxiety disorders themselves.

MNEMONIC

Sequence

For a possible aura, reconstruct the **sequence**: onset, evolution, paired positive and negative phenomena, headache relationship and recovery. The mnemonic is a history-taking spine, not a diagnostic criterion by itself.

8.5 Shared mechanisms: useful bridge, necessary caution

Mechanistic explanations can help the psychiatrist understand why a neurological pain disorder and psychiatric syndromes travel together, but they must not be represented as settled fact where the evidence only supports a proposal. In the model described by Kaufman, **spreading neuronal depression** begins with altered cortical neuronal metabolism and changes in neuronal activity that spread from posterior to anterior cortex. It is proposed to account for the aura and to activate the trigeminal system, followed by release of vasoactive neuropeptides and painful meningeal processes. This model displaced the older simple vascular account and places cortex, meninges and brainstem within the same clinical picture.



Fig 31.6 Migraine and psychiatric comorbidity: an observed comorbidity, and a bridge that is only proposed. Knowing which half is which is the discriminator, and the plate carries that on its face rather than in this caption: the solid teal half is observed, every dashed coral box and arrow is proposed. Three specifics are absent because they do not appear in the sources read, each marked where it belongs: any single bipolar subtype, any sensitisation mechanism, and any proportion of the comorbidity attributable to the bridge. (Kaufman's Clinical Neurology for Psychiatrists; Kaplan and Sadock's Comprehensive Textbook of Psychiatry 2024.)

The accompanying figure is best read as a conceptual bridge: a cortical event is linked to aura, trigeminal activation and pain, while shared biological systems may help explain why mood and anxiety disorders are frequent in the same patients. It is not a diagram of a proven unitary cause. In particular, it should not be used to infer that a patient's depression or anxiety is merely a migraine symptom.

The **serotonergic limb** adds a clinically familiar connection. Platelet serotonin concentration falls at migraine onset; serotonin-containing neurons are concentrated in the dorsal raphe nucleus; and triptans act primarily as 5HT-1B and 5HT-1D receptor agonists at trigeminal nerve endings, likely limiting the release of vasoactive neuropeptides. These observations make serotonin biologically plausible in migraine. They do not establish one shared abnormality as the cause of all comorbidity.

Related theories propose serotonin as a shared substrate for the association of migraine with epilepsy, major depression and anxiety. The word "propose" matters. This is a hypothesis that connects clinical observations and treatment pharmacology; it is not proof that serotonin explains the relationship in every patient. A good answer therefore gives the bridge, preserves the hedge, and returns to clinical formulation: migraine burden, psychiatric syndrome, temporal pattern, vulnerability and treatment exposure.

8.6 Risk assessment is part of migraine review

Comorbidity changes safety work. People with migraine have increased lifetime suicidal ideation and suicide attempts compared with the general population. In those with concomitant migraine and fibromyalgia, suicidal ideation is reported at **~60%** and suicide attempts at **15-20%**. The source also notes that the excess risk is not wholly explained by depression. This means that a clinician should not use the absence of an obvious major depressive syndrome as reassurance that suicide risk has been dealt with.

Ask directly about suicidal thoughts, intent, planning, past attempts, access to means and current supports whenever the clinical presentation indicates risk. Then integrate migraine into the formulation: severity and unpredictability of attacks, pain-related desperation, sleep disruption, functional losses, comorbid anxiety or mood symptoms, other pain syndromes and substance use. The headache history can clarify precipitants and burden, but it should never substitute for a full psychiatric risk assessment. Risk management follows the severity and immediacy of the person's presentation rather than the diagnostic label alone.

- Separate risk that intensifies during pain from risk that persists between attacks.
- Document the patient's account of functional losses and current supports.
- Reassess after changes in headache burden, psychiatric state or treatment exposure.

PITFALL

Do not reduce suicidal thoughts in a person with migraine to "understandable distress" and stop there. The association with suicidality requires direct assessment, including when depression is not the only apparent clinical problem.

8.7 Prescribing and diagnostic attribution at the interface

Medication choice should be made for the whole clinical syndrome, rather than by treating pain and psychiatric symptoms as unrelated lists. Tricyclic antidepressants are effective when migraine coexists with depression and are described as more effective for migraine than SSRIs or SNRIs. This is a comparative statement about migraine benefit, not a reason to ignore psychiatric indication, tolerability, previous response or the broader psychopharmacology plan. The Mood disorders: pharmacotherapy notes provide the general psychiatric pharmacology; migraine prophylaxis regimens are addressed separately in this chapter.

There is also a well-known interaction question. Concurrent use of an SSRI or SNRI with a triptan or dihydroergotamine carries a low but often-cited risk of **serotonin syndrome**. The clinically mature response is neither reflexive avoidance nor casual dismissal. Reconcile all medicines, identify the purpose of each serotonergic agent, explain relevant symptoms to the patient, and coordinate with the clinician treating the headache. Headache itself can also be an adverse effect of psychotropic medication, so a new headache after a psychiatric prescription deserves a temporal medication review rather than an automatic attribution to anxiety.

Prescribing at this interface is collaborative. The psychiatrist should make explicit which symptoms the psychiatric treatment is expected to target and which headache outcomes require review by the clinician managing migraine. This prevents one clinician from silently assuming the other has monitored the interaction burden, the patient's understanding or the effect of treatment on functioning. It also creates room to address psychological work: education about the distinction between attack symptoms and psychiatric symptoms, support for paced return to valued activity, and work with avoidance or catastrophic interpretations where these are present.

Diagnostic coding requires the same restraint. DSM-5 describes depression triggered by severe headaches as **Depressive Disorder due to Another Medical Condition**, but high migraine-depression comorbidity can make that causal claim difficult to defend. Use a "due to" formulation only when the evidence for causation is persuasive. Where reports of migraine are fabricated for opioid or another tangible benefit, the relevant alternatives named in the source are Factitious Disorder Imposed on Self and Malingering. These are serious determinations, not conclusions to draw from inconsistency or from a clinician's frustration with recurrent attendance.

> In migraine, psychiatric comorbidity is common and clinically consequential, but the diagnosis of a mood, anxiety or bipolar-spectrum disorder - and any causal attribution between it and headache - must rest on its own longitudinal evidence.

9 · Medication-overuse headache, tension-type headache and the psychiatric overlap

The psychiatric consultation can either interrupt or perpetuate a headache cycle. A patient may present with anxiety about a progressive head pain, low mood secondary to disability, insomnia, repeated requests for sedatives or analgesics, or apparent treatment resistance. The psychiatrist's task is not to annex the neurological diagnosis. It is to recognise when repeated symptomatic treatment is part of the disorder, distinguish a primary tension-type pattern from

transformed headache, and organise prescribing across settings so that one clinician does not unknowingly undo another's plan.

Chronic daily headache is a useful clinical syndrome rather than a shortcut to a single cause. It describes headache lasting 4 hours or longer on **at least 15 days each month** for at least 3 months. The central practical question is then retrospective: was this an episodic migraine or tension-type headache that gradually transformed, a newly persistent daily illness, a post-traumatic or psychiatric presentation, or a syndrome sustained by medicines? That history changes the plan more than the current pain adjective alone.

MOH	TTH	CBT
REPEATED RELIEF CAN SUSTAIN PAIN	COMMON PRIMARY HEADACHE	A TREATMENT PARTNER, NOT AN AFTERTHOUGHT

9.1 Medication overuse: a diagnostic habit of mind

Medication-overuse headache is best understood as a self-reinforcing clinical loop. It commonly develops on a pre-existing migraine substrate: acute medication is taken, rebound pain follows, and the rebound is interpreted as a reason for further self-administered medication. The patient is not necessarily seeking intoxication or behaving irrationally. They are pursuing immediate relief in a system in which immediate relief has become part of the longer-term problem. That formulation allows a firm discussion without moralising.

Several routes lead to chronic daily headache. Episodic migraine or tension-type headache may lose their episodic pattern; a new daily persistent presentation may begin in someone with little previous headache; post-traumatic and psychiatric contexts may contribute; and medication overuse can pave the route regardless of the original phenotype. In adults, prolonged exposure to headache medicines is the dominant risk factor. In children and adolescents, chronic daily headache usually occurs without such a history. This contrast prevents the psychiatrist from applying an adult explanation mechanically to a younger patient.

The medication history must include prescribed drugs, over-the-counter products, combination preparations, medicines borrowed from family, and tablets taken for sleep, distress or neck discomfort rather than explicitly labelled as headache treatment. The commonly implicated groups are **aspirin-butalbital-caffeine compounds**, triptans, non-steroidal anti-inflammatory drugs, benzodiazepines and opioids. This is psychiatry-facing neurology: butalbital-containing preparations and benzodiazepines may be introduced through psychotropic prescribing, while opioid exposure is often uncovered only when the history is asked in a non-accusatory way.

RULE **In a patient with increasingly frequent headache, every apparently helpful acute medicine is part of the diagnostic history.** Count the product, the indication the patient gives for it, the prescriber, and the sequence of relief and recurrence before adding another symptomatic agent.

Risk is not uniform across drug classes. Triptan or opioid use on **more than 10 days per month** is reported as a risk threshold for chronic daily headache, with development in less than 2 years. This is a risk statement, not a formal diagnostic criterion. By contrast, frequent non-opioid

analgesic use may require almost 5 years before chronic daily headache emerges. Barbiturate-containing products are particularly concerning: use on **at least 5 days per month** is associated with rebound headache and progression of headache disorders. Acute antimigraine medicines can also lead to chronic daily headache with use as little as 2 to 3 times a week.

PITFALL

Do not diagnose medication-overuse headache from a single threshold alone. The available frequency statements describe risk, not the formal per-class diagnostic criteria. Equally, do not dismiss a combination product because the patient calls it a "regular painkiller"; the sedative and caffeine components can obscure what is actually being overused.

9.2 Why the phenotype can deceive

Transformed chronic daily headache may look remarkably like tension-type headache at the consultation. Patients often describe generalised, waxing and waning, dull, pressing and non-pulsatile discomfort of mild-to-moderate intensity. Yet the discomfort can be incapacitating even when the pain is not dramatic. During transformation, the original headache may shed its distinctive qualities and associated aura-like or autonomic features. A cross-sectional history therefore risks labelling the current pain as uncomplicated tension-type headache and missing the antecedent migraine pattern and medication cycle.

Ask for the earliest memorable attacks, not only the current month: what did the first disabling headaches feel like, did activity worsen them, were there associated symptoms, and when did tablets become routine? Also ask what changed when the headache became almost daily: work attendance, family responses, sleep, food intake, and the patient's confidence in coping. These questions locate headache in its behavioural economy. They do not replace a neurological assessment, and the migraine disorder and its psychiatric comorbidity are addressed in the companion migraine discussion.

Psychiatric symptoms are neither incidental nor sufficient explanation. Chronic daily headache is associated with depression and anxiety, functional disability, stressful life events, disability, head trauma, obesity and socioeconomic adversity. In adolescents, depression and anxiety are the prominent risk factors. Depression is reported with chronic daily headache, migraine and tension-type headache, but not with trigeminal neuralgia or other secondary headache syndromes. A psychiatric diagnosis should therefore prompt a fuller headache formulation, not conclude it.

A useful formulation separates the pain disorder, the medicine-rebound loop, emotional distress and disability, and the care system that supplies medicines. Once separated, these processes can be addressed together without claiming that distress "caused" the pain. This is particularly important when a patient has been told that a bilateral pressing pain proves psychological tension. It does not.

9.3 Tension-type headache: recognise the pattern without psychologising it

Tension-type headache is the most common primary headache. It is often described as intermittent, bilateral frontal or cervical dull pain, with a pressing or squeezing quality. An episode may last 30 minutes to 7 days. The key clinical contrast with migraine is not simply severity. The patient typically has pain without the migrainous accompaniment of nausea, vomiting, photophobia, phonophobia, hyperacusis or autonomic disturbance.

"Tension" is historically sticky but clinically misleading. The disorder is not understood as the result of either muscle contraction or psychological tension. Neck discomfort may be a consequence of altered head movement in response to pain rather than proof that muscular tension caused it. This matters in psychiatry because a patient with stress and a headache can be given an etiological story that is both plausible and wrong. Stress may be clinically relevant, but relevance is not the same as a simple causal explanation.

MNEMONIC

PRESS

Pressing or tightening pain, **R**outine activity does not aggravate it, **E**mpty of prominent migrainous accompaniments, **S**ymmetric or bilateral pattern, **S**ustains usual activity. Use this as a bedside memory aid, then confirm the formal criteria and medication history.

Behaviour is a high-yield discriminator. People with tension-type headache usually tolerate the pain and continue daily activity; headache that is exacerbated by activity is more likely to be migraine. The disorders may nevertheless coexist, either together or in alternating periods. Many neurologists place them at opposite ends of a hypothesised spectrum arising from a shared but unknown physiological disturbance. That is a useful conceptual possibility, not an established mechanism and not permission to call every mild migraine "tension."

CLINICAL AXIS	TENSION-TYPE PATTERN	MIGRAINE-FACING CONTRAST
Location	Bilateral or symmetric pressure	May be hemicranial
Pain quality	Dull, pressing or tightening, non-pulsatile	Throbbing quality is more typical
Activity	Usually continues ordinary activity	Activity-related exacerbation favours migraine
Associated symptoms	Absence of prominent gastrointestinal and sensory concomitants	Nausea and sensory symptoms support migraine
Course	May coexist with migraine	Do not force an either-or history

9.4 Formal tension-type classifications

The **ICHD-3 frequent episodic tension-type headache** classification requires at least 10 episodes occurring on 1 or more but fewer than 15 days per month for at least 3 months. Episodes last from 30 minutes to 7 days. They have at least two of bilateral location, pressing or

tightening non-pulsatile quality, mild or moderate intensity, and no aggravation by routine activity. There is no nausea or vomiting, and no more than one of photophobia or phonophobia; another disorder must not better account for the headache.

ICHD-3 chronic tension-type headache occurs on **at least 15 days per month** for more than 3 months. The headache lasts for hours and may be continuous. The same core pain-character features apply. The chronic classification permits no more than one of photophobia, phonophobia or mild nausea, but excludes moderate or severe nausea and vomiting. Frequency alone cannot distinguish chronic tension-type headache from chronic migraine or chronic daily headache. Pain character, associated symptoms, prior phenotype and medicine use must do the discriminating work.

TRAP **A bilateral, pressing daily headache is not automatically chronic tension-type headache.** A transformed migraine with medication overuse can lose its original migrainous features and resemble the tension-type pattern. Ask backwards before labelling forwards.

The classification is a discipline of exclusion as well as description. It prevents the clinician from substituting a psychosocial impression for the actual symptom pattern. It also stops a false dichotomy: a person can have both migraine and tension-type headache, and an acute medicine used for either may contribute to chronic daily headache. A reliable formulation therefore names the current phenotype, the historical phenotype and the medication exposure separately.

9.5 Acute treatment and the prevention of iatrogenic chronicity

For tension-type headache occurring less than twice a week, acute treatment at onset with over-the-counter analgesics may suffice. Aspirin, aspirin-caffeine products, acetaminophen and non-steroidal anti-inflammatory drugs are named options. The apparently ordinary nature of this advice is precisely why it needs a ceiling in the patient's understanding: daily use often leads to chronic daily headache. The clinician should frame acute treatment as a rescue tool, not a standing response to every discomfort.

ACUTE OPTION	EVIDENCE STATEMENT WITHIN THIS SOURCE	MEDICATION-OVERUSE RELEVANCE
Ibuprofen 800 mg	First choice; lowest reported gastrointestinal bleeding or perforation risk among the compared options	Still requires a bounded acute-use plan
Naproxen sodium 825 mg	Next choice; higher gastrointestinal bleeding risk	Do not substitute repeated use for review
Aspirin or paracetamol 500 to 1000 mg	Comparable effectiveness in the cited comparison	Ask about all combined preparations
Caffeine 130 or 200 mg	Augments simple analgesics and ibuprofen in controlled trials	Combination products can conceal exposure

LOCUS is usually outpatient care, but its safety depends on a shared plan among all prescribers. FOCUS in the acute phase is to clarify what is being taken and stop automatic escalation; in the

short and middle term it is to restore a non-rebound pattern and treat disability, depression or anxiety; in the longer term it is to prevent recurrence of fragmented prescribing. MODUS includes medication review, psychologically informed education, behavioural treatment, and liaison with neurology and primary care. Relevant headache guidance should be consulted for an individual regimen, particularly when comorbidity or withdrawal risk is present.

GOOD TO REMEMBER

The most useful prescription may be a single reconciled medicine list. It should record every acute product, who supplied it, the symptom for which it is used, and the agreed clinician responsible for changing it. Fragmented prescribing is a mechanism of medication overuse, not merely an administrative inconvenience.

9.6 Withdrawal, containment and collaborative care

When chronic daily headache is attributed to medication overuse, the culprit medicine must be eliminated, but this is not a casual instruction to "just stop." Abrupt cessation can produce withdrawal symptoms as troublesome as opioid withdrawal. Rebound headaches may continue after detoxification, and relapse of medication use is common. The psychiatrist should provide containment and avoid reintroducing the same cycle simply to settle the immediate crisis.

LOCUS may need to shift from outpatient care to medically supervised withdrawal. Comorbid depression and a history of excessive use of any medicine are reasons neurologists often consider hospitalisation; withdrawal from over-the-counter preparations, opioids or excessive conventional antimigraine medicines may also require it. FOCUS is safety and containment in the acute phase, sustained discontinuation through the immediate rebound period, then relapse prevention and treatment of psychiatric comorbidity. MODUS is coordinated medical supervision, clear prescribing boundaries, psychological support, and a plan for the underlying headache disorder rather than a replacement dependence on another symptomatic medicine.

All of the patient's physicians should work together, actively seeking psychiatric disorder that may be obvious or initially hidden. This is not an instruction to psychologise the headache. Depression, anxiety, hopelessness about pain and medicine-related behaviours can make withdrawal harder, while uncontrolled pain and disability can intensify psychiatric symptoms. Chronic daily headache generally resists treatment; botulinum toxin has a role and tricyclic antidepressants, valproate and topiramate may help. Regimen selection belongs with the treating headache clinician. The psychotropic and anticonvulsant cross-use in migraine is covered separately in the migraine prophylaxis discussion.

9.7 Prevention in chronic tension-type headache

For chronic tension-type headache, preventative treatment may include amitriptyline or topiramate. When a tricyclic is selected for this indication, the cited approach is to start amitriptyline or clomipramine at 10 to 25 mg before bed and increase gradually. The reported average required amitriptyline dose for chronic tension-type headache is **50 to 75 mg per day**. These are tension-type headache prophylactic figures, not antidepressant dosing and not migraine-prophylaxis dosing.

The same source reports that most authorities recommend stopping treatment after 6 months regardless of efficacy, with a reduction of 20% to 25% every 2 to 3 days as a way to avoid rebound headache. LOCUS is outpatient care when risk is contained. FOCUS is prevention rather than repeated rescue medication; MODUS combines an individually prescribed preventive medicine, symptom monitoring, behavioural work and reconciliation of acute drugs. The general pharmacology of antidepressants and anticonvulsants belongs in the Mood disorders: pharmacotherapy notes and the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes.

Selective serotonin reuptake inhibitors have not been convincingly shown to prevent tension-type headache. This is a useful corrective to the tempting but simplistic idea that any antidepressant should help a disorder historically named for psychological tension. A tricyclic's benefit here should be discussed as a pain-preventive strategy, while depression and anxiety are assessed and treated on their own merits.

9.8 Behavioural treatment and the psychiatric contribution

Behavioural treatment is not a consolation prize for patients whose pain persists. Relaxation and biofeedback, alone or combined, have been tested in tension-type headache. Cognitive-behavioural stress-management approaches are effective, particularly for people with high stress when combined with relaxation or biofeedback. Improvement may appear more slowly than with pharmacological treatment, but can be sustained for several years without monthly therapist contact. Stress-management techniques combined with a tricyclic antidepressant are superior to either approach alone.

For children and adolescents with chronic headache, relaxation and cognitive-behavioural therapy reduce frequency and severity. In adults, insight-oriented psychotherapy and psychoanalysis do not alleviate the headache itself, although they may still provide insight, reduce anxiety, treat depression and offer other benefits. This boundary is clinically humane: do not promise headache relief from a therapy that is not shown to deliver it, and do not deny the therapy's other legitimate goals.

- Validate pain as pain, even when stress worsens coping or disability.
- Make acute-medication use visible before attempting to change it.
- Formulate depression and anxiety as comorbidity that can amplify suffering and complicate withdrawal.
- Use behavioural strategies to expand coping and reduce reliance on repeated rescue medication.

> A daily bilateral pressing headache should trigger a retrospective migraine history and a complete medicine count before it is called chronic tension-type headache.

10 · Psychotropics in migraine prophylaxis and antidepressant or anticonvulsant cross-use

The examination issue is not simply which psychotropic can prevent migraine. The higher-value task is to keep indication, dose and therapeutic purpose aligned when the same medicine crosses neurology and psychiatry. Amitriptyline may be chosen for migraine in a person who is depressed, and topiramate may be familiar from anticonvulsant practice, but neither familiarity licenses transfer of a dose from one indication to another. This is prescribing by indication, not prescribing by drug name.

The psychiatrist therefore needs a compact prophylaxis framework: identify the preventive indication, select a suitable agent after discussing benefit and risk, write the regimen with its migraine scope, and review safety in the context of mood symptoms, self-harm risk, pregnancy potential and concurrent prescribing. The disorder, its psychiatric comorbidity and its shared mechanisms are considered elsewhere in this chapter. General psychopharmacology belongs in the Mood disorders: pharmacotherapy notes, while the general beta-blocker and anxiolytic profile of propranolol belongs in the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes.

RULE **A cross-use drug never carries a context-free dose.** Write the indication in the same sentence as the dose, because a familiar drug name can conceal a materially different therapeutic range and purpose.

10.1 Read the prescription as an indication-dose pair

A psychotropic used in migraine prophylaxis sits at an interface between specialties. The prescription may look familiar to a psychiatrist, yet the clinical question has changed: the target is prevention of migraine attacks rather than treatment of a mood syndrome or seizure disorder. This changes how the dose is interpreted, how response is judged and which safety discussion has priority. A correct answer therefore begins with the indication and only then names the agent and regimen.

NICE migraine-prevention options are propranolol, topiramate or amitriptyline after a full discussion of the benefits, risks and suitability of each. This is a choice among reasonable options, not a command to use every option in a fixed sequence. The discussion should incorporate the patient's previous treatment experience, concurrent medicines, likely acceptability and risks that become especially important in psychiatric practice.

Cross-use can be clinically useful because one prescriber may recognize more than one relevant domain. It can also create anchoring: seeing amitriptyline and assuming an antidepressant regimen, or seeing topiramate and assuming an anticonvulsant regimen. The disciplined response is to ask, “What is this medicine being used to prevent or treat here?” before reading any number.

MNEMONIC

IDI: Indication, Dose, Interpretation

Indication tells you which regimen is valid; **D**ose must be written with that scope; **I**nterpretation asks whether the observed benefit belongs to migraine prevention rather than to another property of the medicine.

In migraine prophylaxis, never import the psychiatric or anticonvulsant dose merely because the drug is shared across specialties.

10.2 Propranolol: the reference preventive and the overdose problem

Propranolol is the most commonly used migraine prophylactic in *Essentials of Medical Pharmacology*. For *migraine prophylaxis*, it is started at **40 mg twice daily** and may be increased up to **160 mg twice daily**. Those figures belong to migraine prevention. They do not describe its anxiolytic use, and this fragment does not reproduce its general beta-blocker pharmacology.

The reason propranolol provides a useful reference point is that it gives the candidate a benchmark against which the cross-use agents can be understood. Its response figures can be set beside the attack-reduction evidence for topiramate, as in the strip below. The comparison is about migraine outcomes, not about which drug is more familiar to the psychiatrist. Response assessment should therefore remain tied to the preventive target rather than to nonspecific impressions of feeling better or worse.

UP TO 70%	4 WEEKS	50% REDUCTION
PROPRANOLOL: FREQUENCY AND SEVERITY	PROPRANOLOL: EFFECT GENERALLY SEEN	TOPIRAMATE: HALF OF PATIENTS ACROSS TWO RANDOMISED TRIALS

The central psychiatric complication is not a subtle pharmacological distinction. In a person with depression and migraine, **propranolol overdose risk** may increase the danger of self-harm because toxicity and deterioration after overdose can be rapid. NICE advises caution and harm-minimising prescribing in precisely this comorbid group. A preventive choice that is reasonable neurologically may therefore be unsuitable when access to medication, current suicidal intent, impulsivity or the feasibility of safe dispensing changes the risk calculation.

GOOD TO REMEMBER

Before prescribing propranolol for migraine to a depressed patient, make overdose safety part of drug selection rather than an afterthought. The practical response may include choosing a safer option, limiting the amount accessible at one time and involving supports where clinically appropriate. These are risk-management principles, not substitutes for a full suicide-risk assessment.

10.3 Amitriptyline: migraine benefit is not antidepressant treatment

For *migraine prophylaxis*, amitriptyline is prescribed at **25 to 50 mg at bedtime**. The scope is essential: this is not an antidepressant-dose statement. General pharmacology and psychiatric

dosing should be read in the Mood disorders: pharmacotherapy notes. Importing a mood-disorder regimen into a migraine answer is not an ambitious answer; it is a category error.

The decisive concept is that the **antimigraine effect of amitriptyline** is independent of its antidepressant property. A patient does not need to improve from depression for the preventive migraine effect to be clinically meaningful, and a migraine response does not demonstrate that depression has been adequately treated. The two outcomes require separate assessment even when one medicine appears on both problem lists.

Amitriptyline can still be attractive when migraine and depression coexist because the class suits that clinical combination. “Suits” does not mean that one migraine regimen automatically treats both conditions. It means the prescriber may consider the overall fit while keeping indications, outcome measures and dose decisions separate. It is effective in many patients but causes **more adverse effects than propranolol**, so apparent convenience must be weighed against tolerability.

TRAP **“Amitriptyline prevents migraine because it treats the associated depression.”** The grounded discriminator is the opposite: its antimigraine action is independent of its antidepressant property. Comorbidity may influence selection, but it does not explain the preventive effect.

This distinction also prevents false conclusions during follow-up. If headache burden improves while depressive symptoms persist, the migraine regimen may be working and the mood disorder may still require its own treatment plan. If mood improves while migraine does not, the shared drug name should not obscure failure of the preventive target. One medicine can therefore generate two clinically separate response narratives.

10.4 Anticonvulsant cross-use: sequence by indication, not familiarity

Valproic acid, gabapentin and topiramate illustrate anticonvulsant cross-use. In migraine, the cited pharmacology source places anticonvulsants below beta blockers in efficacy and uses them particularly when other drugs have not worked or propranolol is unsuitable. That **lower efficacy than beta blockers** is a sequencing statement for migraine prevention; it is not a judgment about their value in seizure treatment.

For *migraine prophylaxis*, valproic acid is used at 400 to 1200 mg per day. For the same indication, gabapentin has some preventive effect at 300 to 1200 mg per day. These doses should not be expanded into general pharmacology, monitoring or psychiatric prescribing rules here. Valproate's broader use belongs in the Mood disorders: pharmacotherapy notes, and anticonvulsant treatment more generally belongs in the Epilepsy and psychiatry notes.

Topiramate makes the dose-scope problem especially visible. For *migraine prophylaxis*, **topiramate titration** begins at **25 mg once daily** and is gradually increased to **50 mg once or twice daily**. By contrast, for the *anticonvulsant indication*, the dose may be increased up to 100 to 200 mg twice daily. The latter can be four times the migraine-prevention target and must never be printed as though it were the migraine regimen.

Evidence of preventive benefit should also be read in migraine terms. The trial result in the evidence strip communicates the kind of outcome being pursued: fewer attacks, not seizure control and not a psychiatric effect. It should not be converted into a claim about anticonvulsant efficacy or psychiatric benefit. Topiramate's broader pharmacology, including its relevance to weight, cognition and raised intracranial pressure, is considered in the idiopathic intracranial hypertension section of this chapter.

PITFALL

A medication reconciliation that records only "topiramate" without the indication invites a dosing error. Record migraine prophylaxis explicitly, verify why any concurrent prescriber is using it, and reconcile the intended target before altering the regimen.

10.5 Flunarizine and the Indian-practice divergence

Guideline lists are shaped by jurisdiction and should not erase familiar local practice. NICE names propranolol, topiramate and amitriptyline for consideration, but it does not name flunarizine. Indian pharmacology teaching and practice nevertheless include **flunarizine for migraine prophylaxis** as a standard option. The correct synthesis is to present the UK recommendation accurately and then identify the Indian option separately, without pretending that both documents contain the same list.

IN INDIA

For *migraine prophylaxis*, flunarizine is used at 10 to 20 mg once daily in adults and 5 mg once daily in children. Its routine place in Indian practice is a genuine jurisdictional difference from NICE, not an omission to be silently "corrected" in either direction.

This comparison teaches a broader method for postgraduate answers. State the named guideline's recommendation as that guideline's recommendation. Then state the locally relevant alternative and its source. A candidate loses precision by merging the two into an imaginary universal ladder. A clinician loses more: a locally available and familiar option may be missed, or a foreign recommendation may be applied without considering access and context.

The same discipline applies to children. A paediatric dose grounded for flunarizine does not authorise extrapolation of adult regimens for other medicines. In June 2025, the use of **topiramate and amitriptyline for migraine prevention in children and young people was off-label** under the cited NICE guidance. Off-label status calls for explicit prescribing responsibility and explanation; it is not synonymous with ineffectiveness or prohibition.

10.6 Pregnancy potential and self-harm risk are selection variables

Preventive prescribing is not complete when the drug and dose are written correctly. Psychiatric practice changes the salience of two safety questions: could the available medicine be used in self-harm, and could the regimen expose a pregnancy? These questions belong at the point of selection because discovering them after dispensing may leave the patient with a theoretically suitable but practically unsafe option.

For topiramate, the restriction is categorical in the cited guidance. It **must not be used for migraine prophylaxis during pregnancy**. In women of childbearing potential, it must not be used unless the conditions of the **Pregnancy Prevention Programme** are fulfilled, including highly effective contraception. This is especially important when the psychiatrist encounters topiramate on a medication list and might otherwise regard it as merely a familiar cross-specialty drug.

For propranolol, the psychiatric safety lens is different. The concern is access to a medicine capable of serious toxicity in overdose in a population that may have depression and self-harm risk. The response is individualised risk minimisation rather than automatic exclusion of every depressed patient. Ask about current and past self-harm, intent, impulsivity, available quantities and who controls medication access, then incorporate that information into the preventive choice.

- **Before selection:** establish the migraine-prevention target, current psychiatric state, pregnancy potential and overdose context.
- **At prescribing:** document the indication beside the regimen and explain why this option fits the individual risk profile.
- **At review:** assess migraine outcome separately from mood, seizure or nonspecific symptom change.
- **Across prescribers:** reconcile who owns titration, monitoring and renewal so that a dose from another indication is not imported inadvertently.

10.7 Comparative prescribing map

The doses have been taught at their primary clinical locations above. The table instead compares the reason each agent appears in a psychiatry-facing migraine discussion, the boundary that prevents indication transfer, and the application most likely to alter a prescribing decision. It is not a complete pharmacology table and should not be used to infer unlisted contraindications, adverse effects or monitoring schedules.

AGENT	PLACE IN MIGRAINE PREVENTION	CROSS-USE BOUNDARY	PSYCHIATRIC-INTERFACE APPLICATION
Propranolol	Most commonly used preventive in the cited pharmacology source	Its migraine regimen must not be confused with anxiolytic use	Overdose toxicity and rapid deterioration matter when depression or self-harm risk is present
Amitriptyline	Effective in many patients, but with more adverse effects than propranolol	Its preventive effect is independent of antidepressant action	Assess migraine response and depressive symptoms as separate outcomes
Flunarizine	Standard Indian option absent from the NICE list	This is a jurisdictional difference, not antidepressant or anticonvulsant cross-use	State the Indian option separately from UK guidance
Valproic acid	Anticonvulsant option within a class whose migraine efficacy is lower than beta blockers	Its mood-disorder and anticonvulsant uses do not define the migraine regimen	Keep general pharmacology in its owning chapters
Gabapentin	Has some migraine-prophylactic effect	Familiarity from anticonvulsant practice does not make indications interchangeable	Judge benefit against the migraine target
Topiramate	Preventive option with attack-reduction evidence	The migraine target must remain distinct from the higher anticonvulsant dose	Apply pregnancy and Pregnancy Prevention Programme restrictions
Erenumab	Migraine-specific preventive rather than psychotropic cross-use	It illustrates a different type of preventive decision	Indian availability and cost require local verification

Erenumab is included for completeness because it represents a migraine-specific preventive rather than psychiatric or anticonvulsant cross-use. It is a fully human monoclonal antibody that blocks the **CGRP receptor**. For *migraine prophylaxis*, it is given as 70 mg subcutaneously once a month and significantly reduced monthly migraine days in people with 4 to 14 migraine days per month. Shared drugs require unusually careful dose scoping; this migraine-specific agent instead raises practical questions about local access. Indian availability and affordability are not established by the allowed evidence and should be checked rather than assumed.

10.8 Management: LOCUS, FOCUS and MODUS

Management begins in the outpatient LOCUS. Migraine prophylaxis is usually a planned, longitudinal decision, while emergency or inpatient care becomes relevant when psychiatric risk, overdose or another acute safety issue changes the setting required. The guideline anchor for the choice is NICE CG150, which recommends considering propranolol, topiramate or amitriptyline

after discussing benefits, risks and suitability. Indian practice must additionally retain flunarizine as a locally standard option rather than treating the NICE list as globally exhaustive.

FOCUS. The immediate focus is safe selection and an unambiguous indication-dose prescription. The short-term focus is tolerability, adherence and whether the preventive plan is being followed as intended. The continuing focus is reduction in migraine burden while separately tracking psychiatric symptoms, pregnancy-related safety and self-harm risk. The purpose of separation is not bureaucratic: it prevents a response in one domain from being mistaken for adequate treatment in another.

MODUS. Drug treatment is the owned mode in this fragment. Psychological and social care remain important when stress, mood symptoms, adherence, family support or self-harm risk affect the treatment plan, but this section does not duplicate psychotherapy or full migraine management. Procedural options are not specified in the allowed evidence. Collaborative care means the psychiatrist, physician or neurologist agrees on the indication, chosen regimen, titration responsibility and review target.

The prescribing sequence is simple. Write “for migraine prophylaxis” beside every shared-drug regimen; choose among appropriate preventives only after discussing individual benefit, risk and suitability; screen specifically for propranolol access in a self-harm context and for topiramate restrictions related to pregnancy potential; do not treat comorbidity as proof that one regimen is adequately treating both conditions; and cross-refer general psychopharmacology rather than importing its doses into the migraine plan.

The final examination answer can be concise without being thin: state the preventive choices, give only indication-scoped regimens, identify amitriptyline's independent antimigraine effect, contrast the migraine and anticonvulsant topiramate doses, name flunarizine's Indian place, and close with the propranolol overdose and topiramate pregnancy safeguards. That sequence demonstrates clinical reasoning rather than a memorised list.

11 · Idiopathic intracranial hypertension and psychotropic associations (topiramate, weight)

The marks lie at the interface between headache, threatened vision, obesity and psychopharmacology. **Idiopathic intracranial hypertension** is the preferred name for raised intracranial pressure without an identified cause. The older labels, pseudotumor cerebri and benign intracranial hypertension, are historically recognisable but clinically misleading: there is no tumour, yet the disorder is not benign because untreated optic-disc swelling can culminate in irreversible loss of vision. For the psychiatrist, the practical task is not to manage every headache as neurological disease. It is to recognise the pattern that warrants examination of the optic discs, protect vision while the diagnosis is established, and review medication and weight without making a premature causal attribution.

This topic also corrects an attractive but unsafe shortcut. Obesity is strongly associated with the disorder, and topiramate can suppress appetite and reduce weight. That does not establish

topiramate as treatment for idiopathic intracranial hypertension. The verified treatment statement names acetazolamide as first-line. The standard topiramate reference does not recommend combining topiramate with acetazolamide. The useful lesson is therefore a prescribing tension, not a therapeutic equivalence.

RULE **In headache with visual symptoms, examine or arrange examination of the optic discs before debating psychotropic causation.** A normal-looking general neurological examination does not neutralise papilloedema or its threat to sight.

11.1 Clinical phenotype and why "benign" is wrong

The typical clinical setting is a young woman with obesity, sometimes with menstrual irregularity. Female sex and obesity carry the most robust association. This demographic pattern should raise suspicion, but it must not become a stereotype: the diagnosis rests on the clinical syndrome, examination, imaging, cerebrospinal-fluid pressure and exclusion of another cause. Conversely, psychiatric comorbidity or psychotropic exposure must not be allowed to make a neurological complaint seem functional by default.

The characteristic symptom cluster is a dull, generalised headache, pulsatile tinnitus and transient episodes in which vision briefly darkens. These visual obscurations are especially important because patients may describe them vaguely as "blackouts", "giddiness" or anxiety. Careful history distinguishes a transient visual phenomenon from loss of consciousness. Nausea or vomiting may join the syndrome. Persistent evolution matters more than a single dramatic adjective: a headache that persists and accumulates visual or gastrointestinal features deserves a different response from a familiar episodic migraine-like pattern.

CLINICAL DOMAIN	GROUNDED FINDING	PSYCHIATRIC IMPLICATION
Optic discs	**Papilloedema** is the cardinal sign	Fundoscopy cannot be replaced by a reassuring mental-state or general examination
Symptoms	**Dull generalised headache, pulsatile tinnitus and transient visual obscurations**	Ask what "blackout" means before classifying it as panic, dissociation or syncope
Neurological examination	It may otherwise remain normal despite florid optic-disc swelling	Normal power, reflexes, coordination and gait do not close the case
Visual consequence	An enlarged blind spot may progress to optic atrophy and blindness if untreated	The referral endpoint is preservation of sight, not merely relief of headache

Raised pressure may stretch the **abducens nerve and produce unilateral or bilateral inward deviation of the eye**. This is a classic false-localising sign: it is compatible with the syndrome and does not, by itself, prove a focal pontine lesion. Its presence may generate diplopia and makes the eye examination still more important. The broader framework for raised intracranial pressure and hydrocephalus belongs in the Stroke, TBI, delirium and focal brain syndromes notes.

PITFALL

Do not let “neurological examination otherwise normal” become “no neurological disease”. The decisive abnormality may be on optic-disc examination, while the rest of the bedside examination remains unexpectedly unremarkable.

11.2 Diagnosis is a structured exclusion

The **Modified Dandy criteria** organise the diagnosis around **six** elements: symptoms or signs of raised pressure; no localising sign apart from abducens palsy; an awake and alert patient; imaging without an explanatory lesion or thrombosis; raised lumbar-puncture opening pressure with normal cerebrospinal-fluid composition; and no alternative explanation. The diagnostic opening-pressure threshold is greater than **25 cm H2O**. This is a criterion, not a licence to diagnose from pressure alone. The clinical context, imaging and fluid composition remain part of the same diagnostic statement.

MRI with MR venography is used to exclude an intracranial mass and interrogate venous drainage. Imaging may show compressed, slit-like ventricles, while venography may show slowing or occlusion of intracranial venous drainage. These findings do not convert every case into idiopathic disease. “Idiopathic” is a residual label: when a venous sinus thrombosis or another cause explains the pressure, the case is reclassified as secondary intracranial hypertension. Detailed modality selection and sequence interpretation are covered in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes.

The lumbar puncture contributes pressure measurement and analysis of the fluid. In idiopathic intracranial hypertension the glucose remains normal and cells are absent; protein may be low. Thus the defining abnormality is pressure in a fluid that is otherwise essentially normal. A cellular, biochemical or other unexpected abnormality should prompt reconsideration rather than being forced into the idiopathic label. Cerebrospinal-fluid technique and the general neuropsychiatric work-up are addressed elsewhere in this chapter.

MNEMONIC

PIVOT: Persistent headache, Inspect discs, Venous imaging, Opening pressure, Threat to sight

Persistent evolving headache with visual symptoms; **I**nspect the optic discs; use **V**enous imaging while excluding another cause; interpret the **O**pening pressure within the full diagnostic criteria; remember the **T**hreat to sight.

11.3 Management: sight is the endpoint

LOCUS. A stable patient without rapidly progressive visual concern may enter care through outpatient neurology or ophthalmology, but new visual loss, significant optic-disc swelling or clinical deterioration requires urgent specialist assessment. The psychiatrist’s locus is recognition, medication reconciliation, communication and continuity of mood treatment. Definitive

diagnostic and vision-preserving decisions belong with clinicians who can examine the discs, assess visual function, arrange imaging and perform lumbar puncture.

FOCUS. The acute focus is to confirm raised pressure, exclude an identifiable cause and protect sight. The short-term focus is control of the pressure syndrome and objective surveillance of visual risk. The continuing focus is to prevent optic atrophy, review relevant comorbidity and medication exposure, and avoid destabilising psychiatric illness through reflex prescribing changes. Headache relief matters, but it is not the sole or safest measure of success.

MODUS. The grounded pharmacological approach uses carbonic-anhydrase inhibitors and diuretics, with **acetazolamide as first-line treatment**. Repeated lumbar puncture may be used by neurological teams to measure pressure and drain cerebrospinal fluid. Refractory disease may require a cerebrospinal-fluid shunt or optic-nerve-sheath fenestration; the procedural purpose is prevention or slowing of visual loss. Weight and medication review are relevant supportive actions, but they do not substitute for pressure-directed and sight-directed care.

- Refer for a clinician who can assess papilloedema and complete the diagnostic lumbar-puncture evaluation.
- Review all medicines and comorbidities before assigning causality to a psychotropic.
- Coordinate any mood-stabiliser change with the patient and the neurological team rather than acting on association alone.

GOOD TO REMEMBER

A patient may report less headache while vision remains at risk. Keep the management target anchored to the optic nerves and visual function, especially when procedural escalation is being considered.

11.4 Lithium: association is not simple causation

Lithium is among the medication exposures associated with idiopathic intracranial hypertension, alongside tetracycline-related antibiotics, hormonal contraceptives and vitamin A. Association, however, is not proof that lithium caused an individual case. This distinction is crucial because people receiving lithium may also have obesity, hypertension, chronic kidney disease or another relevant exposure. Some published cases also involved another implicated medicine, and reported lithium concentrations could remain therapeutic.

0.03 to 4.7 per 100,000 COUNTRY-SPECIFIC ADULT INCIDENCE	**almost exactly 9:1** FEMALE:MALE RATIO IN A SWEDISH DATABASE PERIOD	**7.70 → 4.75** LITHIUM ODDS RATIO: GENERAL TO OBESE CONTROLS	**17** LITHIUM-ASSOCIATED CASES IN A REVIEW

The control-group comparison is the teaching point. Against obese controls rather than general-population controls, the odds ratio falls from 7.70 to 4.75, which is a reduction of roughly two fifths of the apparent lithium signal. That pattern shows how strongly obesity can confound an observed drug association. Residual confounding by hypertension and chronic kidney disease

further limits causal certainty. The small published case count, co-prescribed implicated drugs and therapeutic lithium concentrations all argue against inflating a rare association into an expected toxicity.

Routine screening for idiopathic intracranial hypertension is not required simply because lithium is prescribed. The clinically useful posture is selective suspicion. Ask specifically about a persistent headache that is new or evolving, rather than assuming every episodic migraine-like headache represents raised pressure. Escalation is warranted when persistence is joined by nausea, vomiting or transient visual change. Referral is needed for papilloedema assessment and lumbar puncture when the syndrome raises concern.

TRAP **Do not make lithium discontinuation the first response to a diagnosis of idiopathic intracranial hypertension.** First examine competing medicines, obesity and relevant comorbidity, then discuss causality and viable mood-stabiliser alternatives with the patient and neurologist.

11.5 Weight is the shared clinical hinge

The overlap between psychiatry and idiopathic intracranial hypertension is partly created by weight. Obesity is among the strongest associated factors for the neurological disorder. A weight-gain problem arising during psychotropic treatment can potentially strengthen that background risk, while topiramate may suppress appetite and produce weight loss. The metabolic mechanisms and management of psychotropic-associated weight gain belong in the Endocrine and metabolic psychiatry notes; the neurological task here is to recognise why weight trajectory changes the risk conversation.

Topiramate-associated appetite suppression and weight loss can be desirable in a patient who is overweight or obese, but problematic in someone who is underweight. In epilepsy studies, the weight effect was more evident at higher baseline weight and was greater in females than in males of similar baseline weight. That is strikingly aligned with the population in whom idiopathic intracranial hypertension is most often encountered. It makes topiramate clinically interesting, but does not turn a shared demographic pattern into evidence of disease treatment.

Controlled studies also support a weight effect in psychiatric populations, including reduction of adiposity in women receiving olanzapine. This can inform a broader metabolic plan, but it must remain properly scoped. A medicine selected to address weight or another established indication is not thereby a pressure-lowering treatment. No topiramate regimen for idiopathic intracranial hypertension is established by the source base used here, and migraine-prophylaxis dosing belongs to the dedicated headache pharmacotherapy discussion.

RULE **Weight benefit and disease-specific efficacy are different constructs.** Topiramate may help weight in the same demographic that is vulnerable to idiopathic intracranial hypertension. That overlap cannot be used to claim that it treats the neurological disorder.

11.6 Why topiramate is not acetazolamide

Topiramate has several pharmacodynamic actions: state-dependent sodium-channel blockade, positive modulation of GABA-A receptors, and antagonism at kainate and AMPA receptors, together with **weak inhibition of the CA-II and CA-IV carbonic-anhydrase isoenzymes**. The relation between these biochemical actions and each clinical effect is not fully established. The word "weak" is load-bearing. It prevents a category error in which any carbonic-anhydrase inhibition is treated as equivalent to acetazolamide.

The verified consequences attached to topiramate's carbonic-anhydrase effect are not a cerebrospinal-fluid mechanism. They include reduced urinary citrate, increased urinary pH and a contribution to nephrolithiasis; the same action may contribute to paraesthesia. About **1.5%** of exposed adults develop nephrolithiasis, and adequate fluid intake may reduce risk. Carbonic-anhydrase inhibition also underlies concern about hyperchloraemic non-anion-gap metabolic acidosis. Baseline and periodic serum bicarbonate measurement is recommended, with dose reduction, discontinuation or alkali considered when acidosis is detected.

Acetazolamide shares clinically recognisable adverse effects such as paraesthesias and kidney stones, and may also cause gastrointestinal upset, hypokalaemia, rash and, rarely, aplastic anaemia. This overlap helps explain why stacking carbonic-anhydrase inhibitors is not a neutral act. In a patient whose neurological treatment already includes acetazolamide, adding topiramate purely because weight loss seems attractive creates avoidable interaction risk.

PITFALL

Combining topiramate with acetazolamide is not recommended because the combination can increase the risks of metabolic acidosis, nephrolithiasis and heat-related problems. Do not convert "each inhibits carbonic anhydrase" into "combine them for greater benefit".

11.7 Topiramate adverse effects can imitate the problem

The **topiramate cognitive and neuropsychiatric adverse-effect signature** includes confusion, psychomotor slowing, impaired concentration or attention, memory difficulty, speech or language problems, depression or other mood problems, sedation and fatigue. Patients often experience this as slowed thinking or difficulty finding words rather than volunteering the label "cognitive adverse effect". These costs matter in postgraduate trainees, professionals, students and anyone whose recovery depends on cognitive performance. They must be weighed against any weight benefit.

Topiramate is also associated with increased risk of suicidal ideation or behaviour across epilepsy and psychiatric populations. Assessment should therefore include change in mood, hopelessness and suicidality, not only weight and headache. Paraesthesia and renal stones may arise from the same carbonic-anhydrase pharmacology discussed above. A new somatic complaint after starting the medicine should be characterised rather than automatically attributed to anxiety or to the intracranial-pressure disorder.

The most important visual discriminator is drug-induced ocular disease. Topiramate is rarely associated with **acute myopia and secondary angle-closure glaucoma**, presenting with acute reduction in visual acuity or ocular pain, usually within **one month** of initiation and reversing with discontinuation. Idiopathic intracranial hypertension, by contrast, threatens vision through papilloedema, visual-field change and eventual optic atrophy. A patient taking topiramate can therefore develop visual symptoms through a mechanism that is not idiopathic intracranial hypertension.

- Transient visual obscurations with persistent headache direct attention toward papilloedema and raised pressure.
- Acute ocular pain or abrupt loss of acuity after starting topiramate raises concern for its ocular adverse reaction.
- Either presentation warrants prompt eye assessment; neither should be dismissed as a nonspecific medication complaint.

11.8 The lithium, acetazolamide and topiramate junction

A lithium-treated patient who develops idiopathic intracranial hypertension sits at a difficult prescribing junction. Lithium is an associated exposure, although the evidence is tenuous; acetazolamide is first-line neurological treatment; and topiramate may be considered elsewhere in the psychiatric plan because of weight. Each decision changes the interpretation or monitoring of the others. The clean approach is to separate diagnosis, causality, neurological treatment and metabolic strategy rather than trying to solve all of them with a single drug switch.

The acetazolamide interaction evidence comes from oedema management rather than an idiopathic-intracranial-hypertension treatment study: acetazolamide lowers lithium concentrations by **31%**. The practical inference is that a lithium-treated patient started on acetazolamide requires serum-level monitoring and dose adjustment guided by measured concentrations. Topiramate, particularly at high dose, may also affect serum lithium concentrations; its prescribing information recommends monitoring lithium when the medicines are co-administered in that setting. The clinical importance of this interaction remains incompletely characterised, so certainty should not be overstated.

A disciplined review separates the questions that are often collapsed. First, confirm that the raised-pressure diagnosis is secure and that an identifiable secondary cause has been excluded. Next, ask which medicines and comorbidities plausibly contribute, and grade the strength of evidence for each rather than treating every association equally. Finally, establish what the neurological team needs to protect vision and what drug-level or biochemical monitoring follows from that treatment. This sequence preserves diagnostic clarity while allowing urgent care to proceed.

The psychiatrist then protects continuity of effective mood treatment while remaining willing to change it when the individual causal assessment supports doing so. Obesity, hypertension, chronic kidney disease, other implicated medicines and viable alternatives all belong in the shared decision. The patient should hear a balanced explanation: lithium has been associated

with the disorder, but the association is uncommon and confounded; stopping it reflexively is not the recommended first move; and vision-preserving neurological care must proceed without waiting for perfect causal certainty.

Memorise the tension: acetazolamide is first-line for idiopathic intracranial hypertension; topiramate is only a weak carbonic-anhydrase inhibitor. It is not established here as treatment for the disorder and should not be combined with acetazolamide.

Sleep: the architecture and its disorders

Sleep neurophysiology, breathing, circadian rhythm and investigations

Why these marks belong in neurology. Sleep is not diagnosed from stillness alone. It is a changing physiological state expressed through cerebral activity, eye movements, skeletal-muscle tone, ventilation, oxygenation and observable behaviour. The neurological task is to identify the system that controls state, recognise when breathing or timing is disturbing that state, and choose a recording that can make the suspected disturbance measurable. This creates a continuous clinical argument: circuit, clock and breathing determine what happens during sleep; the investigation converts each of those processes into a signal that can be examined.

This section deliberately provides that neurological frame rather than duplicating a sleep-medicine chapter. Detailed sleep architecture, the hypnogram, formal staging, the two-process model, sleep-disorder criteria and treatment algorithms belong in the Eating and sleep-wake disorders notes. Molecular circadian physiology and melatonin dynamics belong in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes. Here, the emphasis is on the landmarks a psychiatrist should recognise and the reasoning that links a clinical question to a physiological channel.

STATE	BREATH	CLOCK	TRACE
AROUSAL CIRCUIT	VENTILATORY INTEGRITY	CIRCADIAN ALIGNMENT	PHYSIOLOGICAL CORRELATE

12.1 State control: the neurological map

The useful starting point is not a list of sleep stages but a map of competing state-control systems. The **transmitter pattern links dopamine, serotonin, histamine and acetylcholine with wakefulness; reduced dopamine and serotonin with increased GABA activity characterises both NREM and REM sleep, while acetylcholine remains active in wakefulness and REM rather than NREM**. This is a state association, not a licence to infer sleep from the concentration of any single transmitter. It tells the candidate why sleep and wakefulness are network phenomena and why a drug acting at one transmitter may alter vigilance without reproducing normal sleep physiology.

Hypocretin, also called orexin, maintains wakefulness rather than initiating it. Loss of its activity is therefore best understood as failure to sustain the waking state, producing episodic loss

of wakefulness, rather than as a simple excess of a sleep-generating signal. That distinction matters because it prevents a common conceptual reversal: a wake-stabilising system can be clinically decisive even when it is not the system that first turns wakefulness on. The detailed orexin framing, hypersomnolence syndromes and their management are covered in the Eating and sleep-wake disorders notes.

The named anatomical landmarks provide the circuit vocabulary. **Wake-side nodes include the tuberomammillary nucleus, locus coeruleus, raphe nuclei, pedunculopontine and laterodorsal tegmental nuclei, lateral hypothalamus and ventral tegmental area; the ventrolateral preoptic area is the sleep-side node**. For the psychiatrist, their value is organisational: distributed brainstem, hypothalamic and forebrain-linked systems regulate a global behavioural state. The complete arousal-system anatomy, flip-flop teaching, sleep architecture and hypnogram should be revised from the Eating and sleep-wake disorders notes rather than reconstructed here.

RULE **Think in physiological systems before diagnostic labels.** Ask whether the dominant disturbance concerns state maintenance, ventilation, circadian alignment or the expression of state in measurable channels.

12.2 From circuits to observable physiology

A patient can appear asleep while the underlying physiology is fragmented, mis-timed or behaviourally dissociated. Conversely, a patient may report little or no sleep despite a recording that demonstrates an organised sleep pattern. The examiner therefore needs two levels of description. The clinical level contains the history of sleep opportunity, timing, continuity, daytime consequence, witnessed behaviour and breathing. The physiological level asks what the brain, eyes, muscles, cardiorespiratory system and body were doing at the same time. Neither level replaces the other.

This distinction explains why normal sleep architecture is foundational even when it is not re-taught in this chapter. Architecture supplies the reference pattern against which abnormal breathing, loss of expected muscle atonia, unusual movements, behaviours and state transitions become legible. It also prevents a category error: a symptom such as fatigue, poor concentration or nocturnal fear is not itself a physiological state. It may arise from disrupted sleep, circadian misalignment, impaired ventilation, a nocturnal event, a psychiatric disorder, medication effects or more than one process acting together. The sleep study can answer selected physiological questions, but it does not replace the formulation.

MNEMONIC

STATE - BREATH - CLOCK - TRACE - CONTEXT

Build the neurological account in that order: identify the state-control question, check whether ventilation disrupts sleep, decide whether timing is aligned, select the trace that can capture the event, and interpret it in clinical context.

The sequence is particularly useful in psychiatry because the presenting complaint often sits downstream from the disturbance. Irritability, cognitive inefficiency, mood symptoms or unusual nocturnal behaviour may bring the patient to a psychiatrist, while the decisive physiological abnormality lies in ventilation, timing or state-linked motor control. The correct response is neither to psychologise the complaint nor to treat the trace as the whole patient. It is to connect symptom, timing, witness account and physiological hypothesis.

12.3 Sleep-related breathing: one mechanism, several diagnoses

The shared neurological burden. **Obstructive sleep apnoea, central sleep apnoea and sleep-related hypoventilation share impaired ventilation during sleep, commonly accompanied by intermittent or sustained hypoxaemia and sleep disruption**. Their immediate mechanisms are not interchangeable, but the shared consequence is crucial for psychiatric reasoning. Repeated disruption of sleep continuity and impaired oxygenation can alter daytime alertness and cognitive or emotional functioning. A psychiatric presentation may therefore be downstream from a physiological sleep disorder even when the patient does not volunteer a sleep complaint.

Breathing history should remain mechanistic. Ask whether the account suggests obstruction, loss of respiratory drive, inadequate ventilation or a mixed picture, and whether the suspected disturbance is episodic or sustained. Witnessed nocturnal events, daytime consequences, medical context and medication exposure help form the pre-test hypothesis. The formal diagnostic indices, screening instrument, severity bands, positive-airway-pressure treatment and psychotropic-choice rules belong in the Eating and sleep-wake disorders notes and should not be inferred from symptoms alone.

The association with psychiatric illness is clinically important but causally untidy. In a sleep-apnoea cohort, **mood disorders, anxiety and posttraumatic stress disorder, psychosis and dementia were significantly more frequent than in the non-sleep-apnoea comparison group**. The direction remains contested. Sleep disruption and hypoxaemia may contribute to psychiatric symptoms, while psychiatric illness or its associated circumstances may predispose to apnoea. Comorbidity therefore supports active case-finding; it does not prove that either disorder caused the other.

> **PITFALL**
>
> Do not convert improvement-resistant mood or cognitive symptoms directly into a sleep-apnoea diagnosis. The correct move is to recognise the possibility, obtain the relevant sleep and breathing history, examine the medical context, and test the physiological hypothesis when indicated.

For an examination answer, keep the layers separate. State the **mechanism** as impaired ventilation with disturbed sleep and impaired oxygenation. Describe the **clinical expression** as nocturnal breathing disturbance with daytime neurobehavioural consequences. Then identify **confirmation** as a recording chosen to demonstrate the suspected respiratory disturbance during sleep.

12.4 Circadian timing: sleep ability versus sleep placement

Sleep generation and sleep timing are related but different questions. A person may possess the physiological capacity to sleep yet attempt it at a phase that is misaligned with internal timing or the external schedule. The **suprachiasmatic nucleus of the hypothalamus acts as the internal pacemaker for the sleep-wake cycle and also coordinates circadian hormonal and metabolic rhythms**. This gives circadian disturbance its broad clinical reach: the issue is not merely clock time on the bedside table, but alignment among internal physiology, environmental cues and required behaviour.

The need for entrainment becomes intuitive when external time cues are removed. Under such conditions, the internal cycle tends to lengthen to **24.5 to 25 hours**, rather than remaining locked to the environmental day. The discrepancy may seem small within one cycle, but without repeated resetting the internal phase drifts away from social time. External cues are therefore active synchronisers, not decorative accompaniments to an otherwise perfectly fixed clock.

Light is the central cue to understand. **Darkness promotes pineal melatonin synthesis and release so concentrations rise at night, whereas natural and artificial light suppress synthesis and release so concentrations fall during daylight**. The clinically important inference is that exposure and timing matter together. Light is not a generic sedative or stimulant; it is information supplied to a timing system. Exact phase-response rules, melatonin markers, circadian disorder subtypes, timed-light treatment and shift-work management are taught in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes and the Eating and sleep-wake disorders notes.

TRAP **Do not describe every complaint of sleeping late as inability to sleep.** First distinguish failure of sleep generation from sleep occurring at an internally preferred but externally inconvenient phase.

12.5 Polysomnography as a set of physiological questions

Polysomnography is best learnt as simultaneous measurement, not as a mysterious machine that prints a diagnosis. A **full polysomnogram records cerebral activity through EEG channels, right and left ocular movements, chin and other muscle activity through EMG, oxygen saturation and other vital signs, together with video of movement, vocalisation and behaviour**. Each channel answers a different question, while simultaneity allows the reader to decide whether changes belong to the same event.

PHYSIOLOGICAL QUESTION	RECORDING DOMAIN	WHAT THE PSYCHIATRIST GAINS
What state is the cerebrum expressing?	EEG	A cerebral correlate against which other signals can be timed
Are characteristic eye movements present?	Right and left ocular channels	Ocular activity linked to the simultaneous cerebral and muscle pattern
Is muscle tone present, reduced or abnormally retained?	Chin, limb or other EMG	A measurable motor correlate rather than a witness description alone
Is oxygenation or other vital physiology disturbed during sleep?	Oxygen saturation and other vital-sign recordings	Temporal linkage between impaired oxygenation, physiological consequence and sleep disruption
What did the patient visibly do?	Time-linked video	Correlation of movement, vocalisation or behaviour with the physiological trace

The table should be read horizontally and vertically. Horizontally, each row moves from a clinical question to a signal and then to its interpretive use. Vertically, the channels create converging evidence about one period of recorded behaviour. A movement on video means something different when accompanied by expected muscle physiology than when it appears during a state in which that physiology should be absent. Likewise, an oxygen change is more informative when its relation to breathing and sleep disruption is visible.

IN INDIA

Access to comprehensive polysomnography may be uneven and cost can determine what is feasible. Referral should therefore state the physiological question clearly and include the relevant witness account, medication context and differential diagnosis. A costly recording without a defined question is less useful than a targeted study whose channels can adjudicate competing explanations.

12.6 Channel concordance: how the trace becomes neurophysiology

The diagnostic value lies in relationships among channels. **EEG separates sleep states, ocular channels show the eye movements, and EMG carries the muscle-atonia signal that distinguishes REM from NREM**. This makes a physiological absence, such as failure of expected REM atonia, recordable rather than merely descriptive. Video then shows the behaviour occurring at that moment. The study can therefore link a witnessed movement to cerebral state and muscle physiology instead of asking the clinician to choose between history and trace.

Interpretation requires concordance. A technically plausible signal should fit the neighbouring channels, the recorded video and the clinical question. Apparent movement may be irrelevant if it lacks the expected temporal relationship; an apparently normal channel may be unhelpful if the event was not captured; and an abnormal trace may be incidental to the presenting complaint. The report is strongest when it states what event occurred, which channels changed

together, which physiological state was present and whether that combination answers the referral question.

This is also why montage detail and formal staging cannot be replaced by a short list of electrodes in a psychiatry answer. Instrumentation determines what can be observed, but interpretation depends on validated recording practice and specialist scoring. Those details, along with sleep-stage scoring and the formal indices derived from the study, belong in the Eating and sleep-wake disorders notes. The raw electrophysiological principle of EEG is covered in the Epilepsy and psychiatry notes. Here, the transferable skill is to match each clinical uncertainty to the channel that could resolve it.

GOOD TO REMEMBER

When referring for a sleep study, write a testable question: suspected respiratory events, abnormal behaviour linked to REM physiology, repetitive limb movement, parasomnia or a possible sleep-only seizure. “Poor sleep” alone does not tell the laboratory what physiological event must be captured.

12.7 What polysomnography can confirm

The indication follows from the mechanism under suspicion. **Polysomnography can confirm sleep apnoea, REM sleep behaviour disorder, periodic limb movements and parasomnias; it is preliminary in narcolepsy assessment, for which a multiple sleep latency test can become diagnostic in the appropriate clinical setting**. This is not a list of interchangeable referrals. Each condition is supported by a different relation among cerebral state, muscle activity, ocular movement, respiratory physiology or recorded behaviour.

CLINICAL SUSPICION	PHYSIOLOGICAL TARGET	ROLE OF THE STUDY
Sleep-related breathing disorder	Ventilatory disturbance, oxygenation and associated sleep disruption	Confirms that the respiratory event occurs during recorded sleep and shows its physiological consequence
REM sleep behaviour disorder	REM state, muscle atonia and time-linked behaviour	Converts reported enactment into a state-linked motor finding
Periodic limb movements	Repetitive limb activity in relation to sleep	Measures the movement rather than relying on bed-partner description alone
Parasomnia	Behaviour in relation to physiological state	Helps classify the event and examine competing explanations
Suspected narcolepsy	Overnight context before daytime sleep-propensity testing	Provides the preliminary study; the subsequent test is interpreted with the clinical syndrome

The practical principle is to seek an event that the available channels can capture. If the question is breathing, the study must connect ventilation to oxygenation and sleep disruption. If the question is dream enactment, the study must connect behaviour to REM state and muscle tone. If

the question is a parasomnia or repetitive movement, video and motor channels must be interpreted against cerebral state. If the question is narcolepsy, overnight recording prepares the ground for the appropriate daytime investigation rather than replacing it.

Formal diagnostic thresholds, respiratory indices, movement indices, daytime test thresholds and actigraphy protocols are intentionally not reproduced here. They are covered in the Eating and sleep-wake disorders notes. In an answer focused on neurological foundations, marks come from stating what each test measures, why the measurement fits the hypothesis and how it changes the differential diagnosis.

12.8 Limited and negative uses: when a normal trace helps

Polysomnography has a limited role in routine insomnia evaluation, but a demonstrated normal sleep pattern may assist indirectly with nocturnal panic attacks, paradoxical insomnia, factitious sleep-wake presentations and seizures occurring exclusively during sleep; it may also reveal an underlying disorder in sleep-maintenance insomnia. This is the psychiatrist's distinctive use of the investigation. A normal or organised trace is not “nothing”; it can weaken a proposed physiological explanation and redirect attention to mismatch between experienced sleep, observed behaviour and recorded state.

The word indirectly matters. A normal sleep pattern does not by itself diagnose panic, intentional symptom production or misperception of sleep. It changes the evidential balance when integrated with history and observed behaviour. Similarly, a recording that captures a stereotyped nocturnal event with an epileptic correlate can reclassify an apparent sleep disorder, but seizure diagnosis and EEG interpretation require the framework in the Epilepsy and psychiatry notes. A negative recording is also bounded by what was sampled: failure to capture the habitual event is not equivalent to proving that the event never occurs.

Before requesting a study for insomnia, ask:

- Is there a specific alternative disorder that the recording could demonstrate?
- Is the complaint primarily about sleep timing, sleep opportunity, perceived sleep or an observable nocturnal event?
- Would a normal trace meaningfully narrow the differential diagnosis?

If none of these questions identifies a measurable target, more instrumentation may add data without adding understanding. Careful history remains the gatekeeper.

12.9 Integrated clinical and examination method

The section can be consolidated into a single reasoning sequence. Begin with the complaint but translate it into physiology. Decide whether wake maintenance, sleep generation, ventilation, circadian placement or an abnormal state-linked behaviour is the leading problem. Use collateral history when the event occurs outside the patient's awareness. Then select the signal capable of testing the hypothesis and state what finding would support it. Finally, return the result to the clinical context rather than allowing the laboratory label to replace formulation.

A compact answer can follow this order:

1. **Name the system and disturbance:** connect arousal and sleep-side nodes, wake-maintaining orexin, the circadian pacemaker or ventilatory control to unstable wakefulness, disrupted sleep, impaired oxygenation, circadian misalignment or abnormal motor expression.
2. **Choose the measurement:** cerebral, ocular, muscle, respiratory, vital-sign or video channel, interpreted simultaneously.
3. **Define the boundary:** explain what the study can confirm, what it only supports and what a normal recording can or cannot exclude.

This approach also keeps cross-references disciplined. Architecture, formal sleep-disorder classification, diagnostic indices, circadian subtypes and treatment belong in the Eating and sleep-wake disorders notes. Molecular clock physiology and detailed melatonin markers belong in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes. Seizure electrophysiology belongs in the Epilepsy and psychiatry notes. The neurological foundation retained here is the bridge from system to signal.

Memorise the chain: state-control circuit, breathing and circadian timing generate the clinical hypothesis; simultaneous physiological channels make that hypothesis testable.

13 · Insomnia disorder: assessment, hyperarousal model and pharmacological and CBT-I management

Insomnia is not adequately understood as simple sleep loss. For the psychiatrist, its most useful neurological formulation is a disorder in which the capacity for sleep is repeatedly opposed by persistent arousal. This framing makes sense of a familiar clinical mismatch: the person reports exhausting nights, yet may present by day as tense, alert, mentally over-engaged, or worried rather than simply somnolent. The **daytime extension of hyperarousal** is therefore part of the syndrome's clinical logic, not merely a consequence of lying awake.

This section deliberately takes the neurobiological lens. Formal diagnostic assessment, insomnia severity instruments, behavioural treatment and the pharmacological treatment ladder belong in *the Eating and sleep-wake disorders notes*. They should be used to establish the disorder and plan treatment; the present account explains why the reported experience is biologically coherent. The model is neither a licence to dismiss distress as "anxiety" nor a claim that every person with insomnia has identical laboratory findings. It is a way of linking subjective wakefulness, physiological activation and the difficulty of disengaging from wake-promoting processes.

13.1 The clinical problem the model is trying to explain

Patients often describe a night in which tiredness and wakefulness coexist. They may want sleep intensely, monitor its absence, and experience the bed as a setting in which thought and bodily vigilance become more prominent. A purely deficit-based account would expect inadequate sleep to produce an uncomplicated increase in objective daytime sleepiness. The hyperarousal model instead proposes that the same processes that sustain wakefulness can remain active across the night and spill into waking life.

The relevant distinction is between fatigue and sleep propensity. Fatigue can be experienced as depletion, poor concentration, low motivation or a sense of having been worn down. Sleep propensity concerns the tendency to fall asleep under standardised laboratory conditions. In insomnia, the latter may be reduced, and patients **may not show increased objective daytime sleepiness** compared with people without sleep disorders. This does not invalidate the complaint. It asks the clinician to hear two true things at once: restorative sleep has been disrupted, and the arousal system may still be preventing the expected drift towards sleep.

RULE **Do not equate a complaint of poor sleep with a measured tendency to fall asleep.** The mismatch itself can be clinically informative when it is interpreted as possible persistent arousal rather than as proof that the patient is exaggerating.

This formulation is especially useful in psychiatric practice because the consultation can otherwise become falsely binary. Either the patient is said to have a "real" sleep problem, or the sleep problem is reduced to a secondary emotional symptom. Hyperarousal permits a more precise account: cognitive, autonomic and cortical activation can be present together, while their relative contribution differs across patients and across phases of illness. The clinician can then assess sleep without prematurely assigning a single cause.

GOOD TO REMEMBER

A patient who is exhausted but not obviously sleepy has not thereby failed to demonstrate insomnia. Ask how alertness, worry, bodily activation and effort to sleep change from evening into the next day; the pattern is often more informative than the adjective "tired".

13.2 Cortical arousal in the sleeping brain

The most direct support for the model comes from sleep electroencephalography. Compared with good sleepers, people with insomnia have **greater high-frequency EEG power** both around sleep onset and during non-rapid-eye-movement sleep. The finding is consistent with increased cortical arousal persisting when the person is meant to be asleep. It matters because sleep is not defined only by stillness, closed eyes or elapsed time in bed. Its quality depends on the brain's capacity to reduce wake-like processing.

More specifically, the electrophysiological signature described in insomnia includes globally increased fast beta activity in the **16 to 32 Hz** range and more centrally increased alpha activity in the **8 to 12 Hz** range during non-rapid-eye-movement sleep. The point for a psychiatrist is not to turn these values into a bedside test. They are research-level markers that explain why a person can appear to have slept yet experience sleep as light, unrefreshing or vulnerable to intrusion by thought and environmental signals.

Fast-frequency EEG components during sleep are interpreted as an electrophysiological indicator of arousal. This is a convergent observation rather than a diagnostic signature to be sought in every patient. The model does not require the clinician to infer a particular tracing from a history. Its value is conceptual: insomnia can involve wake-like cortical activity within a state that conventionally looks like sleep.

LEVEL OF OBSERVATION	WHAT THE HYPERAROUSAL FORMULATION CONTRIBUTES	CLINICAL READING
Subjective night	Wakefulness can feel effortful, vigilant and mentally active.	Explore the quality of being awake, not only the duration of wakefulness.
Sleep EEG	**Wake-like cortical activity** can persist at sleep onset and in non-rapid-eye-movement sleep.	Sleep may contain an arousal component.
Daytime state	**Daytime hyperarousal** may co-exist with a poor night.	Do not use apparent activation to dismiss impairment.
Laboratory physiology	**Bodily activation** can be described across autonomic, endocrine and metabolic domains.	Interpret findings as support for a systems model, not as a single confirmatory test.

TRAP **Do not present high-frequency EEG activity as though it were a clinical diagnostic criterion.** It is evidence for a model of arousal, while insomnia remains a clinical disorder requiring the assessment framework in the sleep-wake chapter.

13.3 Autonomic, endocrine and metabolic activation

The cortical account is strengthened by evidence beyond the EEG. Laboratory measures in insomnia have shown **generalised HPA-axis activation** and increased arousal, although the findings are not consistent across studies. That qualifier must remain in the clinician's account. The model is supported by a pattern of observations, not by a uniform biomarker profile that every patient must express.

Reported correlates include raised whole-body metabolic rate, altered heart-rate variability, and altered norepinephrine and cytokine secretion. Cortisol and adrenocorticotrophic hormone have been found to be raised particularly in the **evening and early sleep period**. The timing is clinically intelligible: a system that should be winding down may remain physiologically mobilised as the patient approaches sleep. It also cautions against an overly psychological account of arousal. Thoughts about sleep matter, but the model is not confined to thoughts.

These observations should not be used as a work-up checklist. In ordinary psychiatric practice, they do not license indiscriminate endocrine, autonomic or inflammatory testing for insomnia. Their significance is explanatory. They show that the clinical experience can involve distributed activation across brain, endocrine and bodily systems. This is why the patient may describe the night as both mentally and physically "switched on", even when there is no external demand requiring alertness.

Cortical	Autonomic	Endocrine	Metabolic
STATE REGULATION	BODILY STATE	PHYSIOLOGY	ENERGY USE

The word "hyperarousal" is therefore best read as a systems-level shorthand. It refers to an interaction between cortical activation, bodily activation and attentional engagement with sleep. It does not imply that all insomnia is caused by stress, that the patient is voluntarily maintaining

wakefulness, or that a normal laboratory measure excludes the condition. The measured domains offer mutually reinforcing perspectives, while their inconsistency reminds us to avoid turning a model into an overconfident explanation of an individual case.

There is a further clinical advantage to this systems account. It helps explain why interventions aimed at only one surface feature may feel incomplete to a patient. Someone may reduce time awake in bed but still describe sleep as effortful; another may feel less worried about sleep but remain physically keyed up at night. Such observations do not prove a discrete biological subtype. They simply remind the clinician that cortical, cognitive and bodily components can travel together without being identical. A formulation should therefore be revised by the patient's longitudinal report rather than defended as a fixed explanatory label.

For the same reason, physiological language needs to be used with restraint in the consultation. Explaining that insomnia can involve activation across several systems can reduce shame and make the complaint easier to discuss. Explaining it as a measured endocrine abnormality in a particular patient, when no such measurement exists, is false precision. The senior clinical move is to use biology to deepen empathy and sharpen questions, not to convert a useful model into a laboratory diagnosis.

13.4 Failure of deactivation rather than absence of sleep capacity

Neuroimaging has encouraged a particularly useful reframing. The proposed problem is an **inability to switch off arousal-related circuits** rather than an inability to switch on sleep-related circuits. This is framed as suggestive evidence rather than settled fact, but it is clinically generative. It shifts the question from "Why cannot this brain make sleep?" to "What is keeping wake-promoting activity from relinquishing control?"

That distinction avoids a common therapeutic misunderstanding. If the difficulty is conceived only as an absence of sleep, treatment can become a race to impose sleep from outside. If it is conceived as persistent activation, the psychiatric task becomes broader: reduce the conditions that reinforce alertness, address maladaptive attention to sleep, and use treatment plans that are proportionate to the clinical setting. The detailed behavioural and pharmacological approaches are deliberately considered in *the Eating and sleep-wake disorders notes*; this neurological formulation explains their target without reproducing their protocols.

The word "switch" should not be understood as a simple on-off button. It is a clinical metaphor for state regulation. Arousal ordinarily supports attention, threat detection, thought and action. In insomnia, those functions may be insufficiently downregulated when sleep requires disengagement. The resulting experience is often paradoxical: the person can feel unable to stop trying to sleep precisely because the effort itself maintains a state of monitoring and readiness.

MNEMONIC

OFF

Observe the mismatch; **F**ind what maintains wakefulness; **F**ormulate the pattern from night into the waking hours. "OFF" keeps the clinical focus on disengagement rather than on forcing sleep.

13.5 A whole-day formulation

The model becomes most clinically useful when it is extended beyond the night. Some patients with insomnia are **hyperaroused and anxious in the daytime** and, despite poor sleep, do not necessarily feel sleepy. This is not a universal presentation and should not be used as a required feature. It is an explanatory pattern that helps the psychiatrist understand why a patient may be distressed, irritable, cognitively overactive and physiologically tense while also reporting a severe sleep complaint.

In the interview, this supports a timeline rather than a single question about "hours slept". Ask about the transition into evening, the onset of monitoring or worry, the bodily sense of activation in bed, the quality of nocturnal wakefulness, and the patient's state after rising. The aim is not to convert the consultation into a laboratory experiment. It is to establish whether the story describes a state that fails to disengage across contexts.

This approach also improves differential clinical reasoning without trespassing into other sleep-disorder material. Marked involuntary sleep episodes, unusual nocturnal behaviours, breathing-related symptoms, circadian misalignment, medication effects and mood-state changes may each demand their own assessment path. Name the possibilities, but do not force them into the hyperarousal model. Their diagnostic and management frameworks are set out in *the Eating and sleep-wake disorders notes*, and mood syndromes are addressed in *the Mood disorders: classification, depressive and bipolar syndromes, neurobiology notes*.

> **PITFALL**
>
> Do not call all daytime activation "evidence of anxiety". A poor sleeper who remains alert may be showing the very paradox that the hyperarousal model predicts. Equally, do not use that model to bypass assessment for a mood episode, substance effect, or another sleep-wake disorder.

13.6 How the model should shape assessment language

The assessment contribution of this model is qualitative. It teaches the psychiatrist to distinguish sleep opportunity from sleep capacity, tiredness from sleepiness, and a subjective sense of poor sleep from the physiological processes that may sustain it. It also invites attention to the patient's relationship with wakefulness: is the person frightened by it, struggling against it, monitoring it, or interpreting it as evidence of impending failure? These are clinically actionable formulations, even though the treatment methods themselves are owned elsewhere.

Use tentative language. "The history may suggest persistent arousal" is better than "the patient has excessive cortisol" or "the EEG is hyperaroused." The former is a formulation; the latter makes an unmeasured biological claim. This distinction protects the patient from false precision and protects the clinician from treating a group-level research finding as though it were an individual laboratory result.

The model is also a guard against moralising. Hyperarousal is not laziness, weak will or a simple failure of sleep hygiene. It describes a state in which brain and body may continue to privilege alertness. That formulation validates the patient's suffering while preserving clinical curiosity

about psychiatric comorbidity, substances, physical illness and other sleep conditions. It is more disciplined than assuming a single cause and more humane than treating insomnia as a complaint that should resolve through exhortation.

At the end of a psychiatric interview, the hyperarousal formulation should help organise the record, not replace diagnostic work. Useful language identifies what has been reported, what remains to be clarified, and which alternative explanations need their own pathway. It prevents two opposite errors: giving a biological explanation before the sleep history has been taken, and withholding a biological explanation until an investigation proves it. In most cases the formulation remains provisional and is tested against the course of symptoms and response to appropriately chosen treatment.

- Document the patient's account of mental and bodily activation separately from the complaint of inadequate sleep.
- Record daytime fatigue and daytime sleepiness as related but non-interchangeable experiences.
- State competing psychiatric, medical, substance-related and sleep-wake explanations explicitly rather than allowing "hyperarousal" to become a catch-all conclusion.

This way of writing the formulation also improves communication with the patient. It acknowledges that the symptom is real, makes uncertainty visible, and avoids making a speculative neurobiological explanation sound like a verdict. It leaves room for the person's own account of stress, mood, routines, bodily discomfort and the meanings attached to a poor night. Those details are not distractions from neuroscience. They are the clinical contexts in which persistent arousal is recognised and addressed.

13.7 Limits of the model and the bridge to treatment

Hyperarousal is an organising model, not a complete final mechanism. It remains under investigation whether the principal driver is **cortical hyperactivity or excess wake-stabilising orexin**. That uncertainty is important. It prevents a false impression that a single neurotransmitter account has solved insomnia, and it creates a principled bridge to the neurobiology of wake-stabilising systems considered with hypersomnolence disorders.

A practical formulation therefore has several layers. At the experiential level, the patient feels unable to disengage from wakefulness. At the cortical level, fast-frequency activity during sleep supports the possibility of residual arousal. At the bodily level, autonomic, endocrine and metabolic observations suggest that activation may not be confined to conscious thought. At the systems level, imaging suggests deficient downregulation of arousal-related circuitry. None of these layers should be over-read in an individual patient, but together they make the syndrome more intelligible.

Management should follow a full sleep-wake assessment and the appropriate treatment framework, not an isolated biomarker or an appealing neurobiological story. The psychiatrist's neurological contribution is to recognise persistent arousal as a plausible target, to avoid

mistaking fatigue for sleepiness, and to communicate that insomnia can be biologically real even when it does not look like ordinary sleep deprivation. For diagnostic assessment, cognitive behavioural treatment for insomnia and pharmacological management, see *the Eating and sleep-wake disorders notes*.

> Insomnia is best understood as a possible failure to downregulate arousal across the night and, in some patients, into the day.

14 · Narcolepsy and hypersomnolence disorders: hypocretin, cataplexy and management

The clinical question is not simply whether a patient sleeps too much. Narcolepsy is a disorder of unstable boundaries between wakefulness and sleep, and the orexin system gives psychiatrists a neurological way to understand that instability. This perspective prevents an unhelpful split between "neurological" symptoms and "psychiatric" experiences. A person may describe frightening imagery, immobility, irresistible sleepiness, emotional consequences, or apparent changes in behaviour. The task is to establish when the experience occurs, what state transition surrounds it, and whether it belongs to a coherent sleep-wake syndrome.

The marks in this area lie in linking a state-boundary symptom to its physiology without claiming more diagnostic certainty than the history supports. Detailed diagnostic criteria, formal sleep testing, disease classification and the treatment ladder belong in *the Eating and sleep-wake disorders notes*. The neurological foundation here is the wake-stabilising system, the logic of rapid eye movement sleep intrusion, and the psychiatric interview consequences of taking sleep timing seriously.

14.1 The orexin system: one system, two names

Orexin and hypocretin are two names for the same peptide neurotransmitter system. The terminology reflects a historical naming collision: one line of work emphasised the system's hypothalamic location, whereas the other drew on appetite in the name. Thus hypocretin **1** and **2** correspond to orexin A and B. The practical point is simple: a guideline, laboratory discussion, or medicine name may use either vocabulary. They should not be read as rival transmitters or as separate diagnostic entities.

The system should be conceived as a stabiliser rather than as a generic switch that merely starts wakefulness. During waking, **tonic firing of orexin neurons** maintains arousal. That is a useful correction to a crude "awake versus asleep" model. Wakefulness is an active, sustained state that must hold attention, muscle tone, perception and behaviour together while competing sleep processes remain possible. When its stabilising influence is lost, the clinical picture can include porous transitions between states rather than a simple excess of ordinary tiredness.

This system also matters beyond sleep. Orexins are implicated in **feeding and reward**, alongside other behaviours. That broader reach explains both the appetite-derived term and the psychiatric interest in the system. It does not license a reductionist reading of eating, motivation or reward

symptoms as proof of an orexin disorder. Rather, it reminds the clinician that arousal is embedded in behavioural regulation and that sleep histories deserve a place in a psychiatric formulation.

A useful formulation therefore has two levels. At the biological level, orexin helps maintain a coherent waking state. At the clinical level, the person may experience the failure of that stability as sudden sleepiness, a mismatch between bodily state and intended activity, or events that appear at the boundary between sleep and waking. This translation prevents a common conversational mistake: asking only whether the person is tired. It is more informative to ask how alertness behaves across the day, what situations expose the difficulty, and whether sleep-related experiences intrude into the waking account.

Wake	Feeding	Reward	Language
OREXIN STABILISES AROUSAL	A RECOGNISED WIDER FUNCTION	A RECOGNISED WIDER FUNCTION	OREXIN AND HYPOCRETIN ARE INTERCHANGEABLE

RULE **Read orexin as the system that steadies waking.** This makes the symptoms of narcolepsy intelligible as instability at a state boundary, not as a failure of will or a purely subjective complaint.

14.2 Hypothalamic source and the logic of cerebrospinal fluid measurement

The relevant cells are **lateral hypothalamic neurons**. They produce hypocretin and release some of it into cerebrospinal fluid. This anatomical fact explains why **cerebrospinal fluid hypocretin measurement** is biologically meaningful: the assay is not an indirect psychological marker but samples a peptide that reaches that compartment.

For a psychiatrist, the important distinction is between the rationale for a test and the test's place in a diagnostic pathway. A result is interpreted with clinical phenotype and specialist sleep assessment; it does not replace a careful history. The numerical laboratory threshold, the associated genetic marker, and the formal testing framework are deliberately not reproduced here. They are taught in the diagnostic section of *the Eating and sleep-wake disorders notes*. Keeping this boundary clear avoids the common error of treating a biomarker as a detached screening answer.

The hypothalamic location is also conceptually useful. It joins sleep-wake regulation to a region familiar to psychiatrists through motivation, appetite and endocrine discussion. Yet localisation should not become a shortcut to diagnosis. The clinician's job remains syndromic: identify persistent sleepiness, reconstruct state-linked events, and decide whether the whole account warrants formal sleep evaluation. A test can refine a well-framed question; it cannot repair a history that has not distinguished sleepiness from fatigue, low drive, medication-related sedation, or a fluctuating medical state.

That distinction has direct consequences for the referral letter. A useful referral does not merely state that a patient is “sleepy”. It describes the duration and pattern in ordinary language, the timing of episodes, sleep-related imagery or paralysis if present, the medications and substances

that may alter alertness, and the functional situations in which reduced alertness matters. It also records the psychiatric context without allowing it to become a catch-all explanation. This is not administrative detail. It lets the receiving clinician see why a sleep-wake mechanism is being considered and what alternative explanations still need attention.

GOOD TO REMEMBER

A cerebrospinal fluid assay has a direct physiological rationale because hypocretin produced in the lateral hypothalamus is secreted into cerebrospinal fluid. The result belongs within specialist diagnostic reasoning, not in isolation.

14.3 Narcolepsy as a hypocretin-deficit model

The discovery of a **selective loss of hypocretin-producing neurons** changed the explanatory centre of narcolepsy with cataplexy. The syndrome is no longer best understood as an undifferentiated complaint of daytime sleepiness. The model connects a discrete arousal system with the instability of wakefulness and with sleep phenomena appearing in an inappropriate state. It also gives a coherent account of why symptoms that might initially seem unrelated can be elicited in the same interview.

The mechanism of neuronal loss **may be autoimmune**. The hedge matters. “May” identifies a plausible pathophysiological account without turning a developing inference into an established individual cause. In an examination answer, this is the appropriate level of certainty. In clinical work, it protects against the equally unhelpful habits of presenting an aetiological theory as a confirmed diagnosis or dismissing the biological model because the patient also has understandable psychological distress.

Hypersomnolence is especially easy to flatten into an everyday word. The interview should instead ask what the patient means by sleepiness. Is there a pull towards sleep, an inability to remain alert, a loss of energy, slowed initiation, or sedation after a substance or medicine? These descriptions can overlap in ordinary language, but they direct different lines of enquiry. A temporal map of sleep, waking, experiences at the edge of sleep, medication exposure and functional consequences often provides more neurological information than a broad question about “tiredness”.

Psychiatric comorbidity or emotional reactions do not invalidate this enquiry. Fragmented function, fear of symptoms, shame about apparent sleepiness, and interpersonal misunderstanding may all shape the presentation. The neurologically disciplined move is to preserve phenomenology before assigning explanation. If the report is state-bound and internally consistent, it deserves sleep-wake formulation even when the consultation began in a psychiatric clinic.

This approach is particularly valuable when the narrative sounds inconsistent at first hearing. A patient may be able to describe an episode in striking detail and nevertheless be unable to say why it occurred. That is not evidence of fabrication. State transitions are brief and subjective, and recall can be shaped by fear, embarrassment and the language available to the patient. The

interviewer's role is to reconstruct the sequence patiently: the preceding state, the event itself, the recovery, and the surrounding sleep pattern. A coherent temporal account carries more weight than an initially dramatic label.

PITFALL

Do not use a plausible psychological explanation to close the sleep history. A patient can have distress, altered mood or medication exposure and still require a careful account of whether experiences cluster around transitions between wakefulness and sleep.

14.4 REM intrusion: the unifying neurological frame

REM sleep phenomena intruding into wakefulness provide the unifying physiological frame for cataplexy, sleep paralysis and sleep-related hallucinations in narcolepsy. This is a mechanism, not a licence to diagnose narcolepsy from any one experience. It explains why a wakeful person may report a bodily state resembling REM-related atonia or imagery resembling dream experience, while still retaining enough awareness to describe the event.

Cataplexy is best held here as a state-boundary phenomenon within the syndrome. Its detailed phenomenology, diagnostic role, and treatment are covered in *the Eating and sleep-wake disorders notes*. The essential neurological insight is that it is not best conceptualised as ordinary weakness, a voluntary behaviour, or a global loss of consciousness. The same frame makes sleep paralysis intelligible as the motor component of REM physiology persisting briefly into waking.

This formulation gives psychiatrists a disciplined route through seemingly dramatic accounts. First ask whether the episode sits at a sleep boundary. Then ask what consciousness, movement, imagery and recall were like during it. Finally, seek the wider sleep-wake context rather than deciding from an isolated symptom. The sequence is useful because it resists two opposite errors: medicalising every unusual nocturnal experience and treating a state-linked biological event as evidence of primary psychosis.

MNEMONIC

State, Sequence, Syndrome

State: locate the event near sleep or waking. **Sequence**: separate movement, imagery and awareness. **Syndrome**: decide only after the wider sleep-wake account has been taken. This is a history structure, not a diagnostic criterion.

TRAP **Calling every paralysed or hallucinatory episode narcolepsy.** These events can be compelling, but their presence alone does not establish the syndrome.

14.5 State-boundary hallucinations and the psychiatric interview

Sleep-related hallucinations typically occur at sleep onset or on awakening. The labels **hypnagogic and hypnopompic** are worth using because they force the clinician to document timing rather than merely record “hallucinations”. Their characteristic quality can be vivid and image-like; the experience may be immobile, frightening, and last for minutes. It commonly

recedes when a light is turned on. Those details point to a state transition and are often more discriminating than the emotional intensity of the account.

The interview must neither minimise nor overinterpret the experience. A frightened patient may understandably describe it in the language of an external presence or threat. The clinician can validate the fear while asking precise questions: Were the eyes open? Was the event linked to falling asleep or waking? Could the person move or speak? Did orientation return with environmental change? Was there a comparable experience in the fully awake daytime state? The purpose is phenomenological clarification, not adversarial cross-examination.

Collateral information can be useful when episodes disrupt shared sleep, work or family routines, but it should supplement rather than replace the patient's account. It may clarify observed behaviour, recovery, or the practical effect of sleepiness. It cannot by itself determine the internal experience of imagery, paralysis or awareness. The same caution applies to labels supplied by family members. A description such as “fainting”, “fits”, “possession”, or “behavioural problem” is a starting point for inquiry, not a conclusion. The psychiatric interview remains a careful exercise in translating lay descriptions into state, sequence and context.

It is reasonable to infer that state-bound timing, vivid visual quality, preserved recall, immobility and resolution with light should widen the differential beyond a primary psychotic process. That inference is not a claim that narcolepsy is commonly mistaken for any particular psychiatric disorder, nor does it make the distinction automatic. Psychiatric formulation remains necessary, especially when symptoms are not consistently tied to the sleep-wake boundary or when other psychopathology requires its own assessment.

HISTORY AXIS	STATE-BOUNDARY SLEEP EXPERIENCE	PSYCHIATRIC ASSESSMENT IMPLICATION
Timing	Sleep onset or awakening	Document the transition, not only the content
Perceptual form	Vivid, image-like and immobile	Clarify form before assigning meaning
Course	Can persist for minutes	Ask for sequence and recall
Environment	May disappear with light	Ask what changed the episode
Emotion	Can be frightening	Validate distress without letting fear determine the diagnosis

14.6 Sleep paralysis is informative but not diagnostic

Sleep paralysis is a brief intrusion of normal REM atonia into wakefulness. This gives it a neurological explanation without making it pathognomonic. Sleep paralysis and hypnagogic or hypnopompic hallucinations are **nonspecific symptoms**: they may occur alone or alongside other sleep disorders. The central skill is therefore calibration. An isolated report warrants careful exploration, but it cannot carry the whole diagnosis.

The distinction between "compatible with" and "diagnostic of" is a high-yield habit across neurology and psychiatry. Patients understandably seek a single label for an alarming event; clinicians are tempted to provide one, particularly when a symptom resembles a familiar textbook syndrome. Better practice is to identify the experience accurately, assess its frequency and context without inventing a threshold, and then decide whether the broader account supports referral for specialist evaluation.

When questioning, make room for ordinary and culturally shaped language. "Could not move" may refer to true bodily immobility, fear, dissociation, exhaustion, or a metaphor for helplessness. "Saw something" may denote imagery at a state boundary, an illusion, a dream remembered on waking, or an experience requiring a different psychiatric formulation. Clarification protects against both invalidation and premature diagnostic closure. It also helps the patient feel that the clinician is taking an apparently strange experience seriously without assuming its cause.

- Anchor the account to the transition into sleep or return to waking.
- Separate immobility, imagery, awareness and memory of the event.
- Ask whether the event occurs in isolation or within a broader sleep-wake complaint.
- Record functional and safety consequences in the patient's own routine.

14.7 Management: psychiatric contribution within a sleep-specialist pathway

LOCUS: management usually begins in outpatient collaboration, where psychiatric and sleep histories can be integrated without forcing the patient to choose between services. Urgent assessment is appropriate when sleepiness or its consequences create immediate risk, but the durable work is longitudinal: establish a reliable description of the sleep-wake problem, review concurrent mental symptoms and medicines, and ensure that the patient reaches the diagnostic pathway able to test the working formulation. Formal sleep investigations, diagnostic thresholds and syndrome-specific treatment belong in *the Eating and sleep-wake disorders notes*.

FOCUS: in the near term, the focus is accurate symptom mapping and reduction of preventable harm from unrecognised sleepiness or destabilising symptoms. Over time, focus expands to the practical burden: stigma, work or study disruption, anxiety about episodes, family misunderstanding and the effects of a chronic explanatory label. Psychiatric review can distinguish secondary demoralisation from a separate mood, anxiety, substance-related or psychotic syndrome, while avoiding the false choice that only one domain can be "real".

MODUS: the psychiatrist contributes explanatory psychoeducation, a structured temporal history, medication review, support for adherence to specialist assessment, and treatment of independently established psychiatric conditions. Drug selection for narcolepsy and pharmacological suppression of REM-related symptoms require the disorder-specific guidance and specialist context given in *the Eating and sleep-wake disorders notes*. Psychological work is not a substitute for the neurological assessment, but it can help a patient recover agency, communicate symptoms accurately, and manage the social consequences of an episodic disorder.

Shared formulation is often the most useful intervention at the psychiatric interface. Write down which experiences are clearly state-bound, which are less clear, and which mental symptoms need parallel assessment. This gives the patient a way to report change without having to defend a single explanation. It also makes later review more coherent: a newly recognised sleep disorder may clarify part of the presentation, while a coexisting psychiatric condition may still need independent treatment. Neither discovery cancels the other.

Explain the model in plain clinical language: the problem can involve the brain's regulation of wakefulness and the boundary between waking and REM sleep. Such an explanation often reduces moralised interpretations such as laziness, weak motivation or deliberate behaviour. It should remain provisional until the diagnostic process is complete. The goal is not to promise certainty early; it is to offer a credible account of why careful assessment is worthwhile.

> The must-memorise principle: state-boundary symptoms are clues to a sleep-wake formulation, not stand-alone proof of narcolepsy.

Sleep, movement and neurodegeneration

Why it matters clinically. Abnormal movement around sleep is not merely a sleep complaint. It may expose failure of a brainstem motor-control system, signal an adverse effect or withdrawal state created by prescribing, or provide a window into neurological disease. The psychiatrist therefore has to identify the state in which movement occurs, reconstruct the medication timeline, and decide how much neurological meaning the phenomenon can legitimately carry.

This section follows that reasoning from rapid eye movement sleep behaviour disorder to restless legs syndrome. It does not repeat formal sleep-disorder criteria, polysomnographic indices, or disorder-specific treatment algorithms, which belong in the Eating and sleep-wake disorders notes. Instead, it develops the neurological lens: how normal REM immobility is generated and lost, why a secondary drug phenomenon must not be given the prognostic meaning of an apparently spontaneous disorder, how iron and dopamine may converge in restless legs syndrome, and why timing is more discriminating than the mere presence of an urge to move.

15.1 The REM atonia circuit

Normal REM sleep combines an activated dreaming brain with profound suppression of skeletal muscle activity. **REM atonia** is generated by perilocus ceruleus or subcoeruleus systems that produce functional quadriparesis, atonia and areflexia in limb and trunk muscles. This state-dependent immobility is protective because it prevents dream content from being translated into purposeful movement. The essential idea is not that motor programmes disappear during REM sleep. They are actively prevented from reaching the final common motor pathway.

The circuit has **two** major inhibitory routes. A glutamatergic projection from the **subcoeruleus area** reaches the medullary reticular formation, which then supplies GABAergic and glycinergic inhibition to motoneurons. A parallel glutamatergic projection recruits GABAergic interneurons that also inhibit motoneurons. Reduced noradrenergic and serotonergic drive contributes

additional disfacilitation. Thus, REM muscle silence reflects both active inhibition and withdrawal of excitatory support rather than a single descending switch.

RULE **Dream enactment is a failure of state-dependent motor inhibition, not evidence that dreaming has simply become more intense.** The neurological question is where REM motor gating failed; the psychiatric question is whether disease, medication or withdrawal altered that gating.

This systems view also explains why the phrase **REM sleep without atonia** is mechanistically informative. It names a dissociation between the REM state and the motor inhibition normally coupled to it. The concept is more precise than treating every nocturnal movement as equivalent, because different movements arise before sleep, during non-REM transitions, during REM sleep, or independently of sleep altogether.

15.2 What lesion evidence can and cannot localise

Experimental lesion work supports the circuit model. Small lesions in the pontine reticular formation lateral to the locus coeruleus, or in the medial medulla, can abolish REM atonia by removing inhibitory input to spinal motoneurons. This is powerful causal evidence that the motor block depends on a distributed pontomedullary pathway. It also warns against imagining a purely cortical explanation for complex behaviour during REM sleep.

Animal and human localisation do not carry equal certainty. Bilateral perilocus coeruleus lesions can produce a presumed REM sleep behaviour disorder phenotype in animals. In humans, however, there is only a suggestion of associations with diffuse hemispheric lesions, bilateral thalamic abnormalities or brainstem lesions. The clean experimental locus therefore becomes a broader and less certain clinical network. A patient with the phenotype cannot be reverse-localised to a tiny pontine lesion from behaviour alone.

The practical formulation should preserve levels of inference. Loss of atonia shows failure of a physiological function; it does not by itself prove a focal structural lesion. A compatible structural disorder may disrupt the network, a diffuse neurodegenerative process may impair it, and a medicine may alter state control without producing a visible lesion. Conflating those levels turns a useful circuit model into false localisation.

GOOD TO REMEMBER

When the history suggests dream enactment, document the sleep-state relationship and the exposure timeline separately. The same outward behaviour can reflect circuit disease, a medication effect or rebound after withdrawal, and those formulations do not carry the same neurological implication.

15.3 The prescribing paradox in REM sleep behaviour disorder

Psychiatrists meet a paradox: medicines that suppress REM sleep can nevertheless provoke the symptoms and signs of REM sleep behaviour disorder. One proposed explanation is that the medicine allows persistent muscle activity during the REM sleep that remains, thereby uncoupling REM generation from its motor inhibition. This mechanism is explicitly provisional,

but the clinical association is sufficiently important to make medication review part of neurological interpretation. **Drug-precipitated REM sleep behaviour disorder** is therefore a state-control problem produced in the context of exposure, not automatically evidence of an emerging primary neurodegenerative process.

Tricyclic antidepressants, monoamine oxidase inhibitors and selective serotonin reuptake inhibitors are implicated classes. The operational exposure list is wider: biperiden, caffeine, venlafaxine, selegiline and serotonin agonists may provoke or worsen the syndrome, alongside the implicated antidepressant classes. The list matters because the less obvious agents can be missed when the review is reduced to a generic question about antidepressants.

Withdrawal supplies the mirror-image mechanism. Episodes may appear during withdrawal from alcohol, meprobamate, pentazocine or nitrazepam. **REM rebound** after withdrawal may precipitate attacks, so an apparently contradictory history of symptoms emerging after a drug was stopped can still be pharmacologically coherent. Current exposure and recent cessation must be reconstructed as parts of the same REM-regulation history.

- Establish whether the behaviour was observed during sleep rather than inferred from a vivid dream report.
- Record current REM-suppressing or otherwise implicated substances, including agents outside routine antidepressant review.
- Ask about recent withdrawal as carefully as current use, because rebound can reverse the apparent temporal logic.
- Keep a secondary exposure formulation separate from the later question of neurological disease association.

> **PITFALL**
> Do not label every medication-associated episode as idiopathic REM sleep behaviour disorder and then attach a neurodegenerative prognosis to it. First decide whether the same phenomenology is better formulated as secondary to a medicine or withdrawal.

15.4 Nosology and the neurodegeneration window

The distinction between phenomenology and diagnosis is crucial. **DSM-5** diagnostic framing excludes episodes attributable to medication, illicit substances or medical conditions. This makes it likely that drug-precipitated episodes would not be accepted as the primary disorder because they are secondary, although that conclusion should remain cautious. A patient can therefore show the same REM motor disinhibition while receiving a different diagnostic formulation and a different prognostic interpretation.

At the other end of the reasoning pathway, REM sleep behaviour disorder can be associated with neurological disease. Polysomnographic documentation is ordinarily required in the diagnostic framing, with an exception when a **synucleinopathy** is already established. That exception is worth naming because it exposes the close clinical bridge between REM atonia failure and

neurodegeneration, but the detailed diagnostic rule belongs in the Eating and sleep-wake disorders notes.

The reported neurological associations include Parkinson disease, dementia, progressive supranuclear palsy, Shy-Drager syndrome and narcolepsy. This list is a window, not a conversion calculator. The associations justify neurological attention but do not assign an individual conversion probability. Medication-precipitated or withdrawal-emergent behaviour, by contrast, requires its secondary cause to remain visible in the formulation. The diseases themselves belong in the Dementias and neurocognitive disorders notes and the Neuropsychiatry of systemic and neurological disease notes.

QUESTION	WHAT IT TESTS	INTERPRETIVE LIMIT
Was REM motor inhibition lost?	State-dependent circuit failure	Does not localise a focal lesion by itself
Was an implicated medicine present?	A secondary exposure formulation	Does not justify primary-disorder prognosis
Did episodes follow withdrawal?	A rebound mechanism	Stopping a substance does not exclude causation
Is neurological disease already established?	The neurodegeneration bridge	Does not replace disease-specific assessment

15.5 Restless legs syndrome as a waking sensory-motor state

Restless legs syndrome is best understood as a sensory-motor state at the boundary of sleep rather than as movement occurring in sleep. Its **four** linked elements are uncomfortable paresthesias before sleep, an irresistible drive to move the legs, relief when the legs are moved, and return of the sensations when movement stops. The sequence matters more than any single symptom. Paresthesia generates an urge, movement temporarily resolves it, and immobility allows it to recur.

The urge arises primarily during rest, inactivity or bedtime, and the movements are not the movements of actual sleep. Patients may rise and walk, move their feet while seated, or make bicycling movements in bed to obtain relief. These actions can look like pacing or motor restlessness, but their meaning lies in the context and sensory consequence. Movement is purposeful self-relief, not merely an observed excess of activity. The accompanying figure sets these observations against their mimics; they need not be turned into another checklist here.

MNEMONIC

REST

Rest brings it on; **E**asing follows movement; **S**ensory discomfort drives the movement; **T**he discomfort returns when movement stops. The mnemonic preserves the linked sequence rather than reducing the syndrome to restless movement.

This is also why polysomnography is not the instrument that establishes the clinical syndrome itself. The relevant urge and relief occur while the patient is awake. Sleep investigation may answer a different question about movements occurring after sleep begins, but it cannot substitute for a careful sensory and temporal history.

15.6 Distinguishing restless legs syndrome from akathisia

The trap exists because the subjective architecture overlaps. People with restless legs syndrome, tics and **akathisia** may experience psychological discomfort when resisting an urge and relief after complying. An urge-relief sequence therefore does not discriminate by itself. Likewise, walking or repetitive leg movement can be shared outward behaviour. Diagnosis depends on the temporal geography of the complaint: where, when and under what state conditions the urge appears.

The circadian and rest-linked pattern is the strongest discriminator; drug-related akathisia and hypnic jerks lack the striking circadian pattern and periodicity of the restless-legs complex. The accompanying figure brings the discriminating axes and the worked medication case together without treating any isolated feature as decisive. Its deeper lesson is that exposure changes prior probability, while phenomenology determines what the observed movement actually represents.

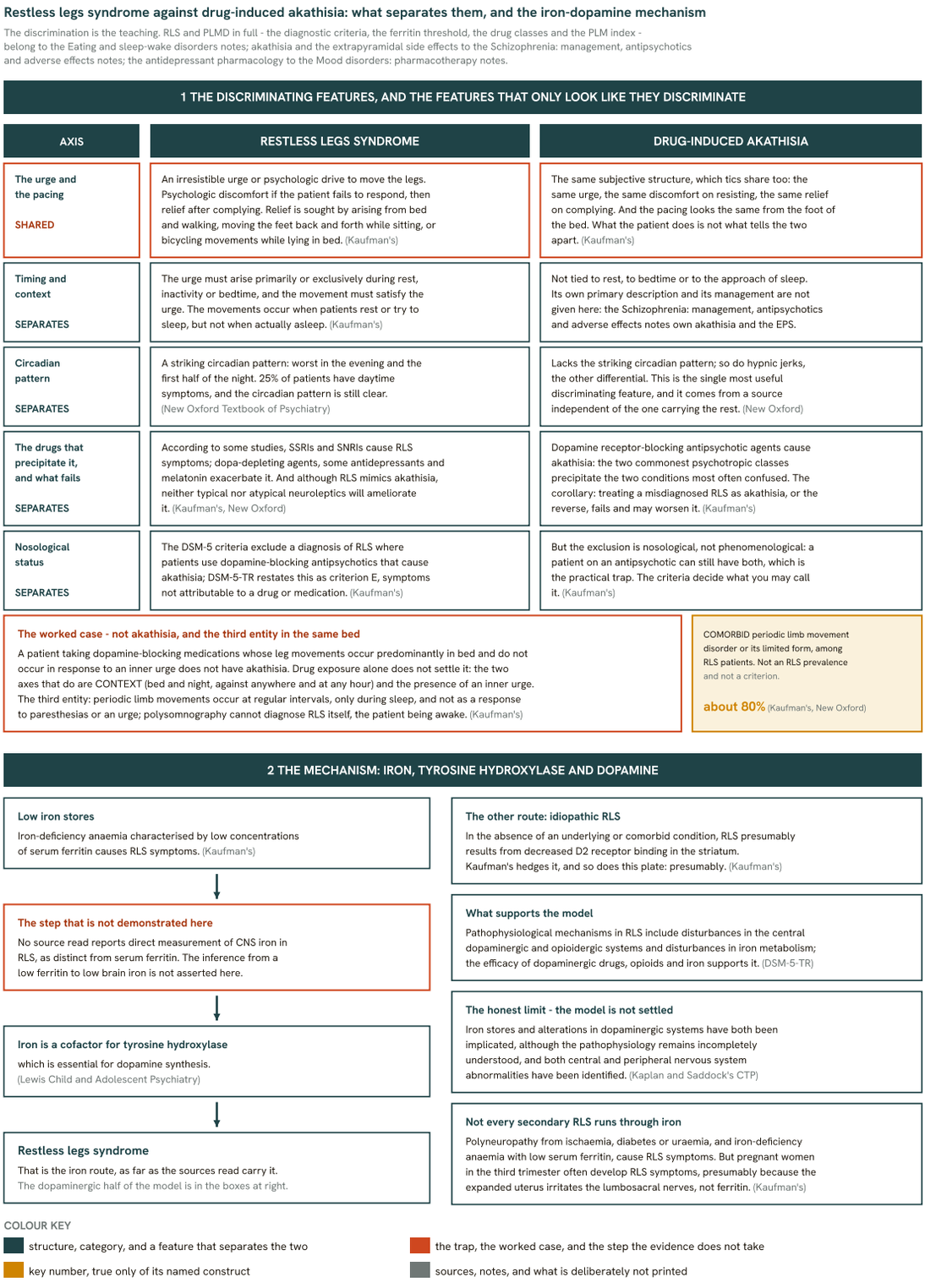


Fig 31.7 **Restless legs syndrome against drug-induced akathisia.** The urge and the pacing are shared, so neither discriminates; the context, the circadian pattern akathisia lacks, the precipitating drug class and the failure of neuroleptics to ameliorate RLS do. Akathisia appears only by contrast, cross-referred above. The iron chain is drawn with its hole visible: no source read measures CNS iron in RLS, so the ferritin-to-brain-iron step is named, not asserted, and the idiopathic D2 route is kept separate. Sources: Kaufman's Clinical Neurology for Psychiatrists, New Oxford Textbook of Psychiatry, Kaplan and Saddock's CTP, DSM-5-TR, Lewis Child and Adolescent Psychiatry.

TRAP **Do not diagnose akathisia from antipsychotic exposure plus leg movement alone.** Ask whether there is generalized inner restlessness, then map symptoms against rest, bedtime, circadian pattern and relief. Exposure is context, not the diagnosis.

Nosology creates an additional layer. DSM-5 excludes restless legs syndrome when symptoms are attributable to a dopamine-blocking antipsychotic causing akathisia. This is a rule about diagnostic attribution, not proof that a treated patient cannot have both syndromes. The clinician must first describe the phenomena accurately and then decide which diagnosis each phenomenon supports. The detailed examination and treatment of extrapyramidal syndromes belong in the Schizophrenia: management, antipsychotics and adverse effects notes.

The figure also prevents a separate collapse between restless legs syndrome and **periodic limb movements**. The useful clinical move is to keep the patient's waking sensory account distinct from a bed partner's observation of movement during sleep. Coexistence does not make the phenomena interchangeable, and one label should not be allowed to erase the other history.

15.7 Iron, dopamine and the limits of a unifying mechanism

The iron-dopamine account is useful because it connects biology, secondary causes and pharmacological observations, but it must remain a model rather than a settled single lesion. Iron is a cofactor for **tyrosine hydroxylase**, which is essential for dopamine synthesis. Reduced iron availability can therefore provide a biologically coherent route to impaired dopaminergic function. In apparently idiopathic disease, decreased striatal **D2** receptor binding has been proposed, although this remains presumptive.

Convergent treatment response adds plausibility without proving a complete causal chain. Evidence implicates disturbances in dopaminergic and opioidergic systems as well as iron metabolism, supported by the efficacy of interventions acting on those systems. Yet iron stores and dopaminergic alterations do not explain every case, and both central and peripheral nervous system abnormalities have been identified. The mechanism should therefore guide questions and investigations while preserving uncertainty.

EVIDENCE STREAM	SUPPORTED INFERENCE	WHAT MUST NOT BE OVERCLAIMED
Iron biology	Iron supports an enzyme essential to dopamine synthesis	Not every case is caused by peripheral iron deficiency
Striatal finding	Reduced dopamine-receptor binding is a proposed idiopathic mechanism	The hypothesis is not a universal lesion
Pharmacological convergence	Dopaminergic, opioidergic and iron-related responses support involvement of these systems	Response does not establish a complete causal pathway
System distribution	Central and peripheral abnormalities have both been identified	Pathophysiology remains incompletely understood

Clinically, the model prevents a false dichotomy between a brain disorder and a systemic disorder. Peripheral neuropathic or metabolic disease can generate the symptom complex, while central state-dependent and dopaminergic mechanisms shape its expression. The sensible formulation can therefore contain a syndrome description, a possible secondary contributor and an explicitly hedged mechanism at the same time.

15.8 Secondary conditions, psychotropics and the depression loop

Secondary associations broaden the neurological work-up. Polyneuropathy related to ischaemia, diabetes or uraemia, and iron-deficiency anaemia with low serum ferritin, can cause restless legs symptoms. Pregnancy is a useful warning against forcing every secondary case through iron: during the **third trimester**, symptoms have been attributed, presumptively, to irritation of adjacent lumbosacral nerves by the expanded uterus rather than to low ferritin. The principle is to search for the relevant biological context without assuming a universal mechanism.

The psychiatric vicious circle. Serotonergic antidepressants may induce or aggravate restless legs syndrome, yet the syndrome itself may predispose to depression, and effective treatment of the restless legs symptoms can reduce depressive symptoms. A clinician may therefore intensify antidepressant treatment for low mood that is partly sustained by an unrecognized sleep-related sensory-motor disorder, worsening the contributor that needs attention. The review must ask whether the mood syndrome, the leg symptoms and the prescription are interacting rather than assuming a simple sequence in which depression came first and every later complaint is merely an adverse effect. General psychotropic pharmacology belongs in the Mood disorders: pharmacotherapy notes.

Medical comorbidities include iron deficiency, end-stage renal disease, neuropathy, fibromyalgia, diabetes, Parkinson disease and multiple sclerosis. These are associations, not proof of causation. In particular, this neurodegenerative comorbidity must not be rewritten as a prodromal relationship. That inference would require separate longitudinal evidence and would confuse coexistence with prediction.

15.9 Epidemiology and an integrated ward formulation

At least 5%	At least 50%
POPULATION PREVALENCE	FAMILY HISTORY

Restless legs syndrome occurs across ages, and familial cases tend to present younger. The high frequency of family history supports asking directly about similar night-time sensory-motor symptoms in relatives rather than relying on a volunteered diagnostic label. The accompanying figure explains why a bed partner's report and the patient's own account may seem to describe different phenomena: each observer has access to a different state and a different part of the history.

An integrated ward formulation should separate observation from inference. “Legs moving” is an observation, not a diagnosis. The history must then supply sensory quality, state, context, subjective drive and the effect of movement. The clinician's job is to resist collapsing those layers

into whichever diagnosis is most strongly suggested by the medication chart. This approach also permits coexistence: a medication-related movement syndrome does not make every later nocturnal movement part of that syndrome.

The same discipline applies to REM phenomena. Describe what was observed, establish its relationship to REM sleep through the appropriate sleep-disorder pathway, identify current or withdrawn precipitants, and only then consider the neurological disease window. RBD and RLS both reward state-based reasoning: movement has different meaning during REM sleep, before sleep while resting, and during sleep without subjective urge.

> Memorise this: in REM dream enactment, separate circuit failure from exposure and withdrawal; in leg restlessness, timing and state outrank the medication label.

Consolidate and apply

16 · Worked vignettes

Why vignettes matter clinically. Neurological assessment in psychiatry is not a ritual performed after the mental-state examination. It is a method for deciding whether an apparently psychiatric presentation can safely remain within a psychiatric formulation, whether it needs parallel medical enquiry, and how urgently the setting of care must change. The useful question is not, "What neurological disease does this patient have?" It is, "What feature of this presentation cannot be adequately explained until I examine, localise, investigate, or escalate?"

These vignettes rehearse the neurological lens rather than reproduce component teaching. Examination, localisation, imaging, electrophysiology, cerebrospinal-fluid work-up, headache syndromes, disorders of consciousness, and sleep disorders each have their own accounts elsewhere in this chapter. Read a vignette as a rehearsal of **problem representation**: compress the presentation into a meaningful sentence, identify the discordant feature, and choose the next defensible action. The diagnosis may remain open. That is often safer than premature certainty.

PRESENTATION	EXAMINATION	TRAJECTORY	CONTEXT
WHAT THE PATIENT REPORTS	WHAT THE CLINICIAN CAN VERIFY	WHAT HAS CHANGED OVER TIME	WHAT MAKES THE PATTERN PLAUSIBLE

16.1 New behavioural change with a discordant neurological finding

A patient is brought with irritability, disinhibition, reduced self-care, and a story from relatives that "the personality has changed." The interview can easily become organised around diagnosis, risk, and medication. It should also become organised around onset, tempo, functional decline, and the reliability of the account. Ask what was present before the behavioural change, what changed first, whether the decline was continuous or fluctuating, and whether the patient can give a coherent account of the change. The collateral history is not merely corroborative; it may be the only route to detecting loss of insight or a deterioration that the patient cannot describe.

The neurological lens begins when the bedside examination yields a finding that does not sit comfortably inside a purely affective, psychotic, or personality-based explanation. A deficit in a higher function, an asymmetry, a change in gait, an unusual movement, or altered arousal changes the task. Do not convert the finding into a named syndrome by pattern-recognition alone. Instead, construct a **sign-symptom mismatch**: the psychiatric symptom is real, but the objective sign demands a parallel neurological account. The point of this formulation is not to make every behavioural disturbance organic. It is to avoid allowing a familiar psychiatric label to silence a discordant observation.

The immediate response has distinct parts.

- Repeat and document the finding in plain examination language.
- Establish whether the finding is new, persistent, progressive, fluctuating, or observed only under one condition.
- Seek collateral about baseline function, safety, medication exposure, substance exposure, recent illness, and observed neurological change.
- Decide whether the setting permits observation and planned work-up, or whether the pattern requires urgent medical assessment.

Localisation is a reasoning discipline, not a parlour trick. The anatomical account and the formal localisation ladder belong in *the Functional Neuroanatomy and Neurophysiology notes*. The psychiatry-facing task is narrower: a reproducible focal or lateralised finding should make the clinician pause before attributing the entire presentation to a primary psychiatric disorder. If imaging becomes relevant, choose it through the examination-to-imaging bridge and consult *the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes* for modality and indication detail.

RULE **An objective neurological finding changes the formulation before it changes the diagnosis.** First establish what the finding is, whether it is reproducible, and what urgency it creates. Diagnostic naming can follow better evidence.

Notice the difference between respecting psychiatric phenomenology and being trapped by it. Disinhibition may be experienced by relatives as “mania”; impaired self-care may be read as “negative symptoms”; apathy may be labelled depression. Such descriptions remain clinically important, but they do not settle causation. The safest formulation holds both levels: the patient has a psychiatric presentation requiring care now, and the presentation contains a neurological feature requiring explanation.

16.2 A fluctuating confusional presentation on the psychiatric ward

A patient admitted for agitation becomes intermittently inattentive, disorientated, frightened, and difficult to engage. At lucid moments the patient appears more coherent, leading staff to debate whether the presentation is behavioural, psychotic, medication related, or deliberate. The mistake is to treat fluctuation as evidence against illness. Fluctuation is itself a finding. It directs

attention to arousal, attention, temporal course, exposure history, systemic illness, and the possibility that more than one process is occurring.

Begin with a **state examination**, not a long cognitive battery. Can the patient sustain attention long enough to understand a question? Does the level of contact change across the interview? Is the patient drowsy, over-aroused, internally preoccupied, frightened, or unable to maintain a coherent line of thought? Re-examination is essential because a single polished interview note can falsely imply stability. The nurse's contemporaneous observations, relatives' description of the baseline, and the sequence of medication administration may reveal the course more clearly than a single clinician encounter.

At this point, separate what belongs to descriptive psychopathology from what belongs to neurological assessment. The phenomenology of clouding, stupor, and related states is developed in *the Psychometry, Assessment and Descriptive Psychopathology notes*. Delirium and focal brain syndromes are developed in *the Stroke, TBI, delirium and focal brain syndromes notes*. This chapter's lens is to recognise that altered attention or arousal changes the reliability of every psychiatric inference made after it. A denial of suicidal intent, an apparently bizarre belief, or apparent non-cooperation cannot be interpreted in the usual way when contact itself is unstable.

The practical sequence is therefore one of containment and clarification. Protect the patient from preventable harm, obtain physiological assessment through the medical team, review recent changes and exposures, and use serial bedside observations to determine the direction of travel. If there is concern for seizure-related or encephalopathic activity, an electroencephalographic question may be appropriate. The technical foundations of that investigation belong in *the Epilepsy and psychiatry notes*; the psychiatric-work-up indications are covered in the chapter's electrophysiology section. A normal-looking interval at the bedside is not permission to discard the preceding pattern.

GOOD TO REMEMBER

When attention and arousal are unstable, the first clinical task is to establish the patient's current state and safety. A polished diagnostic formulation based on an unreliable encounter is less useful than a brief, accurate account of fluctuation and a clear escalation plan.

This vignette also tests professional communication. The handover should state what staff saw, what the patient could and could not do during assessment, what changed, and what needs repeating. It should not hide uncertainty under a label such as “behavioural.” That word explains nothing. It can become a form of diagnostic closure when the patient most needs medical and psychiatric teams to observe together.

16.3 Headache, visual complaint, and a psychiatric medication history

A patient in follow-up for a mood or psychotic disorder describes headache, nausea, visual disturbance, reduced concentration, and worry that medication is “causing brain damage.” The psychiatrist has two responsibilities at once. The first is not to dismiss symptoms because the patient has anxiety, affective illness, or a history of somatic preoccupation. The second is not to

turn every headache into an emergency neurological diagnosis. Good reasoning preserves both responsibilities by asking which features are new, what their trajectory is, whether the patient is functionally impaired, and what the focused examination contributes.

The symptom account should be translated into a **syndrome frame** before medication changes are made. Is headache the dominant complaint, or part of a broader alteration in vision, cognition, alertness, or motor function? Is the visual complaint vague discomfort, an intermittent perceptual symptom, or a sustained loss of function? Has the patient already altered medication without telling the team? Has a rescue medicine become part of the problem? These questions distinguish a medication discussion from an indiscriminate medication reaction.

Do not use the psychiatric diagnosis as a substitute for examination. A focused neurological assessment, including attention to visual function and relevant eye findings, may expose a reason to alter the urgency and pathway of care. The technical examination belongs in the chapter's neurological-examination section. The disorder-specific accounts of migraine, medication-overuse headache, and idiopathic intracranial hypertension belong in their respective sections. The medication choices used for migraine prophylaxis are not interchangeable with psychiatric prescribing: consult *the Mood disorders: pharmacotherapy notes* for general psychopharmacology and the chapter's headache sections for indication-specific decisions.

A useful interview structure is:

- Clarify the symptom narrative without accepting the patient's causal attribution uncritically.
- Map medication exposure, adherence, recent changes, and self-directed use of non-prescribed remedies.
- Examine for the neurological information that can change urgency.
- State explicitly whether the current plan is symptomatic support, planned investigation, or urgent referral.

TRAP **"It is probably anxiety" is not a neurological conclusion.** Anxiety may amplify symptom distress and coexist with a primary headache disorder or another medical process. It cannot replace a history, examination, and safety decision.

The same discipline protects against the opposite error: treating a distressing headache report as proof that the psychiatric medicine must be stopped immediately. Abrupt, poorly reasoned medication changes can worsen the psychiatric illness while failing to address the headache. Explain the uncertainty honestly, decide what must be assessed now, and coordinate with the relevant medical service. The patient hears something much more useful than reassurance: their symptom is being taken seriously without a false promise of instant attribution.

16.4 The sleep complaint that is not merely insomnia

A patient with depression reports poor sleep, daytime fatigue, irritability, impaired concentration, and reduced motivation. The tempting formulation is circular: depression causes poor sleep, poor sleep worsens depression, therefore treatment should proceed as usual. That formulation may be partly true, but it is incomplete until the sleep complaint is unpacked. "I do

not sleep" can mean difficulty initiating sleep, disrupted sleep, insufficient opportunity, irregular timing, involuntary sleep episodes, disturbed breathing, abnormal movements, frightening nocturnal events, or non-restorative sleep. Each history points the assessment in a different direction.

The key term is **sleep-wake phenotype**: the characteristic pattern of sleep, wakefulness, timing, behaviour, and daytime consequence. Elicit it in the patient's ordinary language before translating it into a diagnostic category. Ask the patient and, where possible, the bed partner what happens across the whole sleep period, not only at bedtime. Clarify work schedule, opportunity for sleep, stimulant and sedative use, changes after medication, and safety-sensitive daytime consequences. A patient who says "insomnia" may be using a familiar word for a problem that has a different clinical structure.

This is a place where psychiatrists should resist both overconfidence and unnecessary testing. The sleep-disorder assessment, behavioural treatment, medication choices, and formal sleep investigations are set out in *the Eating and sleep-wake disorders notes*. The neurology chapters of sleep explain the relevant physiology and investigation lens. In routine psychiatric care, the contribution is to recognise when the account has exceeded the boundaries of a straightforward insomnia formulation and to make a clear referral question rather than a vague request to "rule out sleep disorder."

The referral question should specify the observed problem and its consequence: unexplained sleepiness, suspected breathing-related sleep disturbance, abnormal nocturnal behaviour, prominent limb symptoms, unusual dream enactment, or concern about circadian misalignment. This protects the patient from having a sleep service receive only the psychiatric label. It also protects the psychiatrist from changing sedating or activating medicines before understanding whether the change is likely to clarify or further obscure the presentation.

> **PITFALL**
>
> Do not use the word "insomnia" as shorthand for every complaint of poor sleep. A complaint is the opening of the history, not its conclusion. Treating the label instead of the phenotype can conceal a condition whose daytime risk is the most important part of the story.

The psychiatric formulation can then become more precise. Sleep may be a maintaining factor, an adverse-effect concern, a marker of relapse, a co-occurring disorder, or a signal of another neurological process. These are different causal positions and lead to different questions. The value of the sleep lens is not that it produces a fashionable additional diagnosis. It prevents an undifferentiated symptom from governing treatment.

16.5 Apparent non-organic symptoms and the obligation to examine

A patient presents with weakness, altered sensation, a tremor, gait difficulty, episodes of unresponsiveness, or a dramatic movement that appears inconsistent during conversation. The consultation may contain obvious psychosocial stress, dissociation, prior psychiatric treatment, and a strong request for certainty. None of those features removes the obligation to conduct a

respectful neurological assessment. The correct aim is not to catch the patient out. It is to determine whether the observed pattern is internally coherent, whether it maps to a plausible neurological process, and whether there are positive clinical grounds for the formulation being considered.

Use **positive inconsistency** carefully. It means that the examination identifies a reproducible feature whose behaviour under appropriate testing is not compatible with the expected pattern of a structural deficit. It does not mean that a symptom is unusual, emotionally charged, fluctuating, or difficult to explain. Nor does it mean that normal investigations prove a functional presentation. A normal test can be valuable, but it answers only the question it was capable of answering.

The detailed neurological examination is owned by the chapter's bedside-examination section. The clinical account of dissociation and conversion belongs in *the Dissociative, conversion and anxiolytic/hypnotic pharmacology notes*. This vignette teaches the interface: psychiatric formulation gains credibility when it is based on positive examination evidence, coherent history, transparent explanation, and continued readiness to revise. It loses credibility when it is merely the residual category after the clinician becomes impatient.

RULE **Functional symptoms require positive clinical reasoning, not a diagnosis of exclusion made in frustration.** The examination should be collaborative, adequately documented, and repeated if the clinical picture changes.

Communication is part of the neurological intervention. Explain what has been found without implying fabrication or triviality. Distinguish between “we have not yet found a dangerous explanation” and “there is nothing wrong.” The latter is usually false and clinically corrosive. A clear formulation can validate genuine symptoms, preserve trust, and leave room for appropriate neurological review if the course changes.

Medication review also belongs in the formulation, but not as an automatic answer. Some medicines can alter movement, alertness, sleep, or subjective cognition. The assessment must first identify the phenotype of the complaint, establish temporal relations, and examine for objective change. For adverse-effect syndromes and their pharmacology, see *the Schizophrenia: management, antipsychotics and adverse effects notes*. This prevents a common drift in practice: calling a movement “functional” because it is unfamiliar, or calling it a medication adverse effect because a medicine is present.

16.6 Building an integrated neuropsychiatric plan

The final vignette is not a disease presentation. It is the common real-world situation in which a patient has an established psychiatric disorder, a new physical or neurological complaint, an uncertain medication history, and several teams who each see only part of the picture. The psychiatrist's task is to build a **parallel formulation**. One strand addresses current psychiatric symptoms, risk, capacity, engagement, and continuity of treatment. The other addresses the neurological question, its urgency, and the information required to resolve it. Neither strand should cancel the other.

In the outpatient LOCUS, the plan needs a named owner for each action, a time-bound review arrangement expressed in ordinary clinical scheduling, and clear return precautions. In an inpatient LOCUS, serial observation and nursing evidence become central, while the team must guard against diagnostic diffusion in which everybody assumes somebody else has examined the patient. In an emergency LOCUS, stabilisation and escalation come before elegant phenomenology. The same formulation can travel across settings, but the available evidence and acceptable delay differ.

A concise integrated note records the following information.

DOMAIN	RECORD	CLINICAL USE
Psychiatric presentation	Syndrome and immediate risks without overclaiming cause	Preserves psychiatric care
Neurological concern	Symptoms and signs rather than a speculative label	Makes the question visible to collaborators
Examination	Observations that alter the pathway	Explains the urgency decision
Next step	Investigation or specialist question and what would change management	Prevents unfocused referral
Communication	Plan for patient, family, nursing staff, and collaborating services	Creates continuity across settings

This structure prevents the false choice between “organic” and “psychiatric.” Psychiatric illness can coexist with neurological illness. Medication effects can coexist with relapse. A neurological complaint can be genuine even when anxiety increases attention to it. The formulation should therefore be revisable. New collateral, a repeated examination, changing arousal, or an investigation result can appropriately move the balance of probability. Revisability is a strength because it documents what the clinician knew at the time and what evidence would make them act differently.

MNEMONIC

PAUSE

Before closing a neurological question in psychiatric practice, **P**icture the trajectory, **A**ssess the state and examination, **U**ncover exposures and collateral, **S**tate the safety and setting decision, and **E**scalate with a focused question. PAUSE is not a diagnostic algorithm. It is a safeguard against premature closure.

The cross-references are part of the plan, not an admission of weakness. Use *the Functional Neuroanatomy and Neurophysiology notes* for anatomy, *the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes* for imaging and foundational psychopharmacology, *the Psychometry, Assessment and Descriptive Psychopathology notes* for formal assessment, and *the Eating and sleep-wake disorders notes* for sleep-disorder management.

The psychiatrist retains responsibility for integration: making sure the neurological question is visible, the psychiatric treatment remains safe, and the patient is not passed between services without a coherent explanation.

> When a psychiatric presentation contains a neurological discordance, pause, examine, describe the uncertainty, and make the next action answer a focused clinical question.

17 · Consolidation and quick review

The neurological lens is a discipline of attention, not a separate specialty consultation performed in miniature. In psychiatric practice, its purpose is to decide whether the presenting disturbance can safely be understood as primarily psychiatric, whether an accompanying neurological process may be shaping it, and what immediate action follows from that distinction. The task is not to convert every consultation into an exhaustive neurological examination. It is to recognise when the mental state, bodily signs, time course, and functional change do not form a coherent account unless the brain and body are brought back into the formulation.

Consolidation therefore means learning to move from scattered observations to a defensible clinical position. A behavioural change may be real without being diagnostically sufficient. A normal-appearing conversation may coexist with a concerning change in arousal, movement, vision, sensation, continence, coordination, or headache pattern. Conversely, a dramatic symptom report does not by itself establish structural disease. The psychiatrist's work is to observe carefully, state uncertainty accurately, and organise the next step so that neither psychiatric care nor medical safety is abandoned.

17.1 Build a formulation before ordering a test

A **neurological lens** asks a different first question from a checklist: what kind of problem is this patient having now? Begin by describing the clinical state in ordinary, observable terms. Is the dominant disturbance in mood, thought, perception, behaviour, cognition, arousal, movement, or bodily function? Which parts are witnessed, which are reported, and which are inferred from collateral history? This prevents the reflex of treating an investigation as a substitute for formulation.

The useful product is a **syndrome formulation**: a concise account linking the mental-state change to its onset, progression, associated bodily symptoms, treatment exposure, background vulnerability, and consequences for safety. It should be able to accommodate competing explanations. A patient can have a primary psychiatric disorder and a concurrent neurological illness; the choice is not invariably one or the other. Equally, an unusual psychiatric presentation need not be made neurological merely because it is difficult to classify. Formulation keeps the question open while making the next observation purposeful.

Ask what would be surprising if the working explanation were correct. If a presumed affective episode is accompanied by new objective focality, fluctuating arousal, an unfamiliar neurological complaint, or a striking departure from the person's usual illness pattern, the formulation needs

revision. If a presumed neurological disorder is being considered solely because symptoms are intense, the evidential basis may still be weak. The essential habit is to make the discrepancy explicit rather than silently absorbing it into a psychiatric label.

RULE **Investigations answer a formulated question; they do not create one.** State the clinical concern, the competing explanation, and the decision that a result could change before requesting the test.

17.2 Establish the state and the trajectory

The most transferable neurological skill in psychiatry is attention to state. A **state-dependent assessment** recognises that a mental examination is valid only to the extent that the patient can sustain the level of arousal, attention, communication, and cooperation required for it. Confident interpretation of thought content is hazardous when the underlying state is unstable. In that setting, the task shifts from refining psychopathology to describing the disturbance, protecting the patient, and clarifying its cause.

Trajectory gives the state meaning. Obtain an account of what the patient was like before the current change, how the change became apparent, whether it has been stable or variable, and what else changed at the same time. Collateral history is often the bridge between a convincing bedside interview and a misleading formulation. It may reveal a loss of previously routine skills, altered sleep-wake behaviour, a change in gait, episodes of unresponsiveness, new falls, altered intake, or a treatment-related temporal relationship that the patient cannot report reliably.

Do not let the word “chronic” end the enquiry. A longstanding psychiatric diagnosis does not explain every later change, and a longstanding neurological symptom does not make a new deterioration benign. The clinically important question is whether the present pattern is continuous with the patient's established baseline. When it is not, record the difference in concrete language. This keeps the handover useful and makes later reassessment possible.

- Describe the current level of engagement before interpreting elaborate mental content.
- Seek collateral evidence of functional change rather than relying only on diagnostic history.
- Record the sequence of symptoms and treatment exposures in the patient's own clinical timeline.
- Reassess the formulation when the course changes rather than defending the original label.

GOOD TO REMEMBER

A careful description of the change from baseline is often more clinically valuable than a premature conclusion about its cause. It allows the next clinician to see whether the patient is improving, fluctuating, or evolving in a new direction.

17.3 Give positive and negative findings their proper weight

A **positive neurological finding** is an observed abnormality that can be described, repeated, and placed in context. It gains value when it is internally consistent, corroborated by another source of information, or changes the working formulation. A **negative neurological finding** is not a

declaration that neurology has been excluded. It is a statement about what was looked for, under what conditions, and what was not found at that time. This distinction is vital in psychiatric settings, where agitation, withdrawal, fear, pain, medication effects, and limited cooperation can constrain the examination.

Use the examination already taught in this chapter as a targeted extension of the mental-state assessment. Observe spontaneous movement before asking the patient to perform tasks. Notice facial animation, speech effort, posture, balance during ordinary activity, and the way a patient enters or leaves the room. Then examine the relevant domains deliberately, explaining what could and could not be assessed. The full techniques belong to the physical neurological examination section; the consolidating skill is deciding which finding is meaningful for this patient.

Negative findings matter because they limit overinterpretation. They can support a plan to monitor rather than escalate, or they can identify that the concern remains unexplained despite an unrevealing bedside screen. Neither use is passive. The note should distinguish “not present” from “not assessed” and “not assessable.” That small discipline protects against the false reassurance created by a templated normal examination.

TRAP **Calling an examination normal when it was incomplete.** In psychiatric practice, non-cooperation or altered arousal is itself clinically relevant; document the limitation and decide whether observation, collateral information, or a different setting is needed.

17.4 Localise cautiously and think in patterns

Localisation is a reasoning tool, not a performance of anatomical recall. It asks whether a cluster of findings points towards a coherent level or system, whether the pattern is diffuse rather than focal, or whether the findings do not yet support either conclusion. Its value lies in directing attention and communication. It should never be used to make a lesion diagnosis from a single sign, especially when the sign may be influenced by anxiety, medication, effort, pain, or the examination context.

Think in patterns across domains. A change in behaviour becomes more concerning when it travels with a change in consciousness, cognition, motor function, sensory experience, autonomic function, or cranial symptoms. By contrast, a symptom that remains isolated, stable, and compatible with the established clinical picture may call for follow-up rather than an urgent neurological narrative. Pattern recognition must be paired with the humility to say that the pattern is incomplete. The detailed anatomical framework belongs in the Functional Neuroanatomy and Neurophysiology notes; the psychiatrist needs enough applied reasoning to notice when the phenotype does not fit.

Be especially careful with apparent inconsistency. Inconsistency can arise from fluctuating disease, fatigue, communication difficulty, distress, changing attention, pain, medication effects, or an examination technique that has not controlled the task. It may be diagnostically informative, but it is not a shortcut to a conclusion. Repeat the observation, compare it with function outside the formal examination, and describe rather than editorialise.

CLINICAL QUESTION	PSYCHIATRIC FORMULATION REMAINS PLAUSIBLE WHEN	NEUROLOGICAL CONCERN STRENGTHENS WHEN	USEFUL NEXT MOVE
Change in mental state	The presentation remains coherent with history and observed state	The state or course is discordant with the working account	Reconstruct baseline and trajectory
Behavioural disturbance	Observed behaviour and collateral information converge	Behaviour coexists with unexplained bodily or cognitive change	Target the bedside examination
Abnormal movement	The phenomenon is described before it is labelled	It is new, progressive, or accompanied by other signs	Review treatment exposure and examine in context
Reported sensory symptom	The symptom can be followed without premature inference	There is an associated objective or functional pattern	Clarify distribution, consistency, and impact
Uncertain findings	Limitations are recorded honestly	Uncertainty itself affects safety or disposition	Seek senior, medical, or neurological input

17.5 Resist diagnostic overshadowing without abandoning psychiatry

Diagnostic overshadowing occurs when a known psychiatric diagnosis causes clinicians to interpret a new physical or neurological problem as merely another expression of that diagnosis. It is not prevented by indiscriminate testing. It is prevented by returning to the patient's present phenotype, comparing it with baseline, and asking whether the explanation has become too convenient. The converse error is also real: treating all distress, bodily vigilance, or atypical expression as evidence of neurological disease can disrupt appropriate psychiatric care.

The balanced approach is dual-track reasoning. Continue to address immediate psychiatric risks, distress, communication needs, and treatment adherence while pursuing a proportionate medical or neurological assessment. This avoids the harmful pause in care that follows a false choice between "psychiatric" and "organic." It also makes the consultation more respectful. Patients and families should not have to prove that physical symptoms deserve attention because a psychiatric diagnosis already exists.

Medication review is part of this lens. Ask whether the observed change predates treatment, follows an exposure, changes with adherence, or has been previously documented. Do not infer causality from temporal proximity alone. Compare the suspected adverse effect with the whole clinical picture and discuss uncertainty clearly. The detailed adverse-effect framework is covered in the Schizophrenia: management, antipsychotics and adverse effects notes and the Mood disorders: pharmacotherapy notes.

PITFALL

Writing "behavioural" as though it were an explanation. Behaviour is a description of what the patient is doing. A formulation still has to account for the state, trajectory, context, associated signs, and immediate risks.

17.6 Match the investigation to the question

Investigations become clinically useful when their limitations are anticipated. Neuroimaging, electrophysiology, and cerebrospinal-fluid examination answer different questions and occupy different places in the work-up. The psychiatrist should be able to state the concern that prompted the request, identify what a positive or negative result would mean for the current plan, and recognise when an apparently reassuring result does not settle the clinical question. Technical details of imaging belong in the Neurochemistry, Neuroendocrinology, Neuroimaging and Basic Psychopharmacology notes; the psych-work-up role of electrophysiology belongs in the Epilepsy and psychiatry notes; cerebrospinal-fluid pathways require the Neuropsychiatry of systemic and neurological disease notes where relevant.

Before requesting an investigation, clarify the clinical decision at stake. Are you seeking evidence to explain a change in consciousness, a seizure-like episode, a focal complaint, a progressive cognitive change, an inflammatory or infectious process, or an otherwise unexplained neuropsychiatric state? The wording should make the question visible to the receiving team. "Rule out organicity" is not a useful request because it names neither a syndrome nor an action.

After a result returns, bring it back to the bedside. A test result is not self-interpreting, and an incidental finding is not automatically causal. Reconcile it with the state examination, temporal pattern, and prior probability established by the history. If the result does not fit, do not silently select the more convenient story. Clarify the discrepancy with the appropriate specialist and update the written formulation.

- Name the symptom cluster or observed change that generated the request.
- State what decision a result could alter: disposition, monitoring, treatment, or referral.
- Record limitations in timing, state, and examination quality.
- Interpret the result alongside the patient, not instead of the patient.

17.7 Apply the lens across common psychiatric interfaces

The same reasoning method applies whether the presenting interface is headache, sleep, altered consciousness, movement, cognition, or a possible seizure-related event. The content differs, but the sequence does not: establish the state, recover the trajectory, examine for discriminating signs, identify the safety question, and select the appropriate pathway. This chapter supplies the neurological frame; it does not replace the disorder-specific teaching.

For a patient with headache and psychiatric symptoms, ask whether the headache pattern, associated neurological complaints, visual experience, and treatment context require a broader account than stress or somatisation. The disorder-specific discussion belongs in the headache sections of this chapter. For sleep complaints, do not reduce daytime behaviour or cognitive

symptoms to insomnia without considering the sleep-wake context; the disease-level assessment and management are in the Eating and sleep-wake disorders notes. For altered consciousness, distinguish descriptive psychopathology from the neurological problem of arousal and responsiveness, and use the Stroke, TBI, delirium and focal brain syndromes notes for delirium in full.

Movement complaints demand particular care because language is imprecise. Patients may use the same words for inner restlessness, pain, tremor, weakness, cramps, or an urge to move. Begin with the patient's experience and observed behaviour, then review treatment exposure and perform the relevant examination. Do not collapse these possibilities into a single psychiatric label. The distinction between restless legs phenomena and akathisia is addressed in the sleep and movement material, while adverse-effect teaching belongs in the Schizophrenia: management, antipsychotics and adverse effects notes.

STATE	TIME	SIGNS	SAFETY
WHAT IS HAPPENING NOW?	HOW DID IT EVOLVE?	WHAT IS OBSERVED?	WHAT ACTION CANNOT WAIT?

17.8 Communicate uncertainty as an active clinical plan

Diagnostic humility is not vagueness. It is the ability to state what is known, what remains uncertain, what has been examined, and what will trigger a change in plan. This is particularly important when the patient is transferred between emergency, medical, neurological, and psychiatric settings. A useful note permits another clinician to reconstruct the reasoning without guessing which facts were observed and which were assumed.

A good handover names the current psychiatric risks and the concurrent neurological concern in parallel. It specifies the baseline, recent trajectory, pertinent positive and negative findings, limitations of the assessment, investigations requested or reviewed, and the reason for escalation or observation. It also tells the receiving team what deterioration would matter. This is **safety netting**: not an afterthought, but a shared plan for detecting change before it is normalised.

Uncertainty should alter the plan, not merely soften the language. It may justify closer observation, a repeat examination when the patient is more assessable, direct discussion with a medical colleague, or a clearer threshold for transfer. The appropriate response depends on the setting and the immediate risk, but the reasoning can be communicated consistently: the available evidence does not yet account adequately for the presentation, so the team is actively preserving safety while seeking clarification. That is a clinically strong position, not a failure of diagnostic confidence.

Clinical confidence should rise only when the account becomes more coherent. When it does not, seek help early and preserve the evidence that prompted concern. The psychiatrist is not expected to provide the final answer to every neurological problem. The psychiatrist is expected to notice when the current answer does not adequately explain the patient and to act in a way that keeps the patient safe while expertise is brought in.

MNEMONIC

SCOPE

State: define the patient's current level of function and arousal. **C**ourse: reconstruct the change from baseline. **O**bservations: separate observed signs from reported symptoms and untested assumptions. **P**attern: ask whether the findings cohere with the working formulation. **E**scalation: state the safety question and the next responsible service or setting.

When the presentation does not cohere, return to state, course, signs, pattern, and safety before deciding that it is "just psychiatric."

References

The canonical sources this chapter is grounded in. Every fact-bearing specific in the notes traces to one of these; where a primary paper is named in the text, it is cited inline. The list is ordered by how much of the chapter rests on each.

1. Kaufman DM, Geyer HL, Milstein MJ. Kaufman's Clinical Neurology for Psychiatrists. 9th ed. Elsevier.
2. Boland R, Verduin ML, eds. Kaplan and Sadock's Comprehensive Textbook of Psychiatry. 11th ed. Wolters Kluwer; 2024.
3. Brazis PW, Masdeu JC, Biller J. Localization in Clinical Neurology. 8th ed. Wolters Kluwer.
4. Glynn M, Drake WM, eds. Hutchison's Clinical Methods. Elsevier.
5. Tripathi KD. Essentials of Medical Pharmacology. 8th ed. Jaypee Brothers.
6. Clinical Methods in Psychiatry. 1st ed. All India Institute of Medical Sciences; 2024.
7. Nelson JC, Meyer JM, Schatzberg AF, et al. The Lithium Handbook. Cambridge University Press.
8. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 5th ed, Text Revision (DSM-5-TR). APA; 2022.
9. Geddes JR, Andreasen NC, Goodwin GM, eds. New Oxford Textbook of Psychiatry. 3rd ed. Oxford University Press.
10. Tasman A, Riba MB, Alarcon RD, et al, eds. Tasman's Psychiatry. 5th ed. Springer.
11. Taylor DM, Barnes TRE, Young AH. The Maudsley Prescribing Guidelines in Psychiatry. 15th ed. Wiley-Blackwell.
12. Stahl SM. Stahl's Essential Psychopharmacology: Neuroscientific Basis and Practical Applications. 5th ed. Cambridge University Press.
13. Cowen P, Harrison P, Burns T. Shorter Oxford Textbook of Psychiatry. Oxford University Press.
14. Martin A, Bloch MH, Volkmar FR, eds. Lewis's Child and Adolescent Psychiatry. 5th ed. Wolters Kluwer.
15. Casey P, Kelly B. Fish's Clinical Psychopathology. 4th ed. Cambridge University Press.
16. Brunton LL, Knollmann BC, eds. Goodman and Gilman's The Pharmacological Basis of Therapeutics. McGraw-Hill.
17. National Institute for Health and Care Excellence. Headaches in over 12s: diagnosis and management. NICE clinical guideline CG150.
18. Teasdale G, Jennett B. Assessment of coma and impaired consciousness: a practical scale. Lancet 1974;2(7872):81-84. The Glasgow Coma Scale is the copyright of Glasgow University and is reproduced here with that acknowledgement.
19. Buchanan RW, Heinrichs DW. The Neurological Evaluation Scale (NES): a structured instrument for the assessment of neurological signs in schizophrenia. Psychiatry Res 1989;27(3):335-350. Cited as an instrument; no item content is reproduced.
20. Transplantation of Human Organs and Tissues Act and Rules, Government of India, as amended.

Additional sources cited inline: Cicchetti D, ed. Developmental Psychopathology; Jiloha RC. Forensic Psychiatry: An Indian Perspective; Schizoobsessive Disorder (Cambridge University Press). Guideline positions are attributed in the text to the body

that issues them, and where a guideline position and Indian practice diverge, both are named and neither is presented as the other.

Index

Every entry jumps to its page. Numbers are the page of first mention.

#

10-20 International System 306

A

Absence seizures 116
acceptance of the diagnosis 237
acetazolamide as first-line treatment 361
Active recall 276
acute myopia and secondary angle-closure glaucoma 364
address route 301
afterdischarge 56
Agnosia 291
akathisia 299
Akinetic mutism 336
albuminocytologic dissociation 317
alert, lethargic, stuporous, and comatose 330
Alternative psychosis 99
Anti-NMDA receptor encephalitis 127
antimigraine effect of amitriptyline 354
Apraxia 293
aspirin-butalbital-caffeine compounds 346
attention 43
attribution 14
autonomic arousal 26

B

Babinski sign 296
Bedside language examination 292
behavioural teratogenicity 266
Behavioural treatment 350
bidirectional relationship 12
Bodily activation 373
brain herniation 315
burden of normality 202

C

carbamazepine 131
cerebrospinal fluid hypocretin measurement 379
CGRP receptor 357
chlorpromazine-equivalent 130
Chronic daily headache 346
Clozapine 128
clozapine 128
Clozapine seizure incidence 138
comorbid epilepsy 233
comorbidity 21
confounding 45
confusional state 74
Consciousness 23
corneal reflex 291
cortical hyperactivity or excess wake-stabilising orexin 377

D

daytime extension of hyperarousal 372
Daytime hyperarousal 374
de novo postoperative depression 199
de novo psychiatric disorders 198
de novo psychosis 200
Death by neurological criteria 335
Decerebrate posturing 331
Decorticate posturing 331
dementia workup 309
Depressive Disorder due to Another Medical Condition 345
Diagnostic humility 404
Diagnostic overshadowing 402
Diffuse slowing 49
Dissociation 19
dissociative seizure 205
dissociative seizures 205
Drug-precipitated REM sleep behaviour disorder 386
Drug-resistant epilepsy 43
DSM-5 62
duration of illness 236
Dysdiadochokinesia 322

E

emergency CSF indication 315
enzyme-inducing AEDs 252
epilepsy patients 93
Epilepsy-to-psychosis latency 114
epileptic dementia 186
epileptiform discharge 47
epileptogenesis 59
episodic dyscontrol syndrome 181
ethosuximide 115
evening and early sleep period 374

Extinction 297

F

Fast-frequency EEG components 373
feeding and reward 378
fetal valproate syndrome 266
first-generation antipsychotics 128
fixed sequence 211
Fluctuation 284
flunarizine for migraine prophylaxis 355
Focal aware seizure 17
Focal impaired-awareness seizure 22
focal impaired-awareness seizures 22
Focal onset seizures 18
forced normalisation 66
Forced normalisation (Landolt phenomenon) 114
Forced thinking 74
Four Questions of Neurology 303
framework 12
frontal beta activity 311
frontal lobe epilepsy 13
Frontal release reflexes 325
functional neurological disorder 204
functional outcome 238

G

Gastaut-Geschwind syndrome 174
generalised HPA-axis activation 374
Generalised tonic-clonic seizures 91
Generalized onset seizures 18
Glasgow Coma Scale 332
greater high-frequency EEG power 373

H

hard neurological sign 322
highest-yield discriminators 212
Hoover sign 298
hyperaroused and anxious in the daytime 376
hypergraphia 174
hypnagogic and hypnopompic 381
hypomania/bipolar-spectrum conditions 341

I

ICHD-3 chronic tension-type headache 349
ICHD-3 frequent episodic tension-type headache 348
ICHD-3 migraine without aura criteria 339
ictal 12
ictal affective phenomena 67
ictal depression 67
ictal fear 17
ictal psychic phenomena 17
ictal psychosis 32
ictal symptoms 66
Ictal violence 181
ictus 62
Idiopathic intracranial hypertension 316
inability to switch off arousal-related circuits 375
Incidence 12
Insight 285
interface 11
interictal 12
interictal dysphoric disorder 62
interictal period 62
Interictal personality change 15
interictal violence 181
interleaving 276
intersection route 301

J

JC virus CSF testing 321

K

kindling 13

L

la belle indifference 299
lamotrigine 131
lateral hypothalamic neurons 379
Lifetime prevalence 41
Localisation 11
localisation-related epilepsy 188
Locked-in syndrome 334
lower efficacy than beta blockers 354
lucid interval 62
Lumbar puncture 314

M

may be autoimmune 380
may not show increased objective daytime sleepiness 373
Medical Research Council scale 295
medically unexplained presentations 224
Medication-overuse headache 345
migraine with aura 339
Migraine-depression comorbidity 340
Minimally conscious state 329
Minor physical anomalies 324
Modified Dandy criteria 360
mood disorder due to epilepsy 98
more adverse effects than propranolol 354
MRI with MR venography 360

must not be used for migraine prophylaxis during pregnancy 356

N

negative neurological finding 400
neural tube defect 265
Neurological Evaluation Scale 327
neurological lens 289
Neurological soft signs 322
NICE migraine-prevention options 352
non-confrontational stance 228
Nonconvulsive status epilepticus 74
nonspecific symptoms 382
normal or only nonspecifically abnormal 306

O

oligoclonal IgG 317
Opening pressure 316
Orexin 365
Orientation 13
Orientation to person 285
Orientation to place 285
Orientation to situation 285
Orientation to time 85

P

Papilloedema 314
parallel formulation 397
paroxysmal EEG finding 308
patients with epilepsy 116
peri-ictal axis 13
peri-ictal category 63
peri-ictal depression 100
peri-ictal period 62
peri-ictal psychosis 65
peri-ictal temporal axis 12
periodic limb movements 370
periodic sharp-wave complexes 309
phenobarbital 67
pleocytosis 316
Point prevalence 41
positive inconsistency 298
positive neurological finding 400
positive symptoms 110
posterior dominant rhythm 49
postictal anxiety 97
postictal confusion 13
postictal delirium 85
postictal depression 67
postictal mania 97
postictal psychosis 62
postictal recovery 86
Pre-lumbar-puncture imaging 315
Pregnancy Prevention Programme 267
preictal prodrome 13
problem representation 392
propranolol overdose risk 353
pseudobulbar affect 294
psychogenic non-epileptic seizure 37
psychogenic non-epileptic seizures 37
pupillary light reflex 293

R

reliably excludes toxic-metabolic encephalopathy 310
REM atonia 369
REM rebound 386
REM sleep phenomena intruding into wakefulness 381
REM sleep without atonia 385
Remission 41
Restless legs syndrome 384
reverse causation 45
Romberg's test 298
routine CSF profile 316
routine EEG screen is unwarranted 307

S

safety netting 404
second-generation antipsychotics 128
seizure phobia 151
seizure threshold 44
selective loss of hypocretin-producing neurons 380
semiology 13
Sensory integration 322
serotonergic limb 343
serotonin syndrome 343
sharp wave 48
sign-symptom mismatch 393
simultaneous positive and negative visual phenomena 341
Sleep paralysis 381
Sleep-related hallucinations 381
sleep-wake phenotype 396
spike 30
spina bifida 265
spreading neuronal depression 342
state examination 108
state-dependent assessment 400
status epilepticus 17
stereognosis 324
subcoeruleus area 384

syndrome formulation 399
syndrome frame 395
Synkinesia, or motor overflow 326
synucleinopathy 386

T

temporal lobe epilepsy 13
Tension-type headache 345
tonic firing of orexin neurons 378
topiramate 165
topiramate titration 354
Topiramate-associated appetite suppression and weight loss 362
trait markers 326
Transformed chronic daily headache 347
Triphasic waves 309
tyrosine hydroxylase 389

U

unprovoked seizures 41

V

valproate 165
Vegetative state 291
video-EEG telemetry 53
video-electroencephalography 21
vigabatrin 195
viscosity 168
Visual migraine aura 341

W

Wake-like cortical activity 373

X

xanthochromia 317

Z

zonisamide 165

#

“migraine personality” 341

About the authors

Dr. Wilfred D'souza

MD Psychiatry (Resident) · Certified Gestalt Therapist · Weave, Centre for Integrative Psychiatry

Works in integrative psychiatry with Schema Therapy as the primary lens, alongside CBT, DBT, motivational interviewing and emotion-focused therapy, and is a certified Gestalt therapist. His clinical interests run through neurodivergence, ADHD, bipolar disorder, personality disorders, addiction and developmental psychiatry: work grounded in lived experience of attention difficulties long labelled as laziness, which made psychiatry personal before it became professional. He has been developing the Toroidal Model of Psychosis and Affect (TMoP), his computational model of mind, and TMoP thinking shaped the Claude Code AI system he builds with: clinical software, teaching tools and much of this series' production pipeline among its work, bringing psychiatry into the AI era in his own way. Lead author of the Weave Sprint series.

Dr. Niharika Reddy

Consultant Psychiatrist · Weave, Centre for Integrative Psychiatry

Consultant Psychiatrist at Weave, Mumbai, seeing people online through weave.clinic. Her work centres on relationships and family dynamics, family therapy and women's mental health, with a particular focus on neurodivergence in women and on adolescent mental health. Practises integrative, person-centred psychiatry. Co-author of this volume.

THE WEAVE LIBRARY

You revised from one. The whole shelf is open.

Field guides and references written in practice, for the people who do the work and the people living through it. Most are free to read and download.



field guides · references · for clinicians and families · free to read

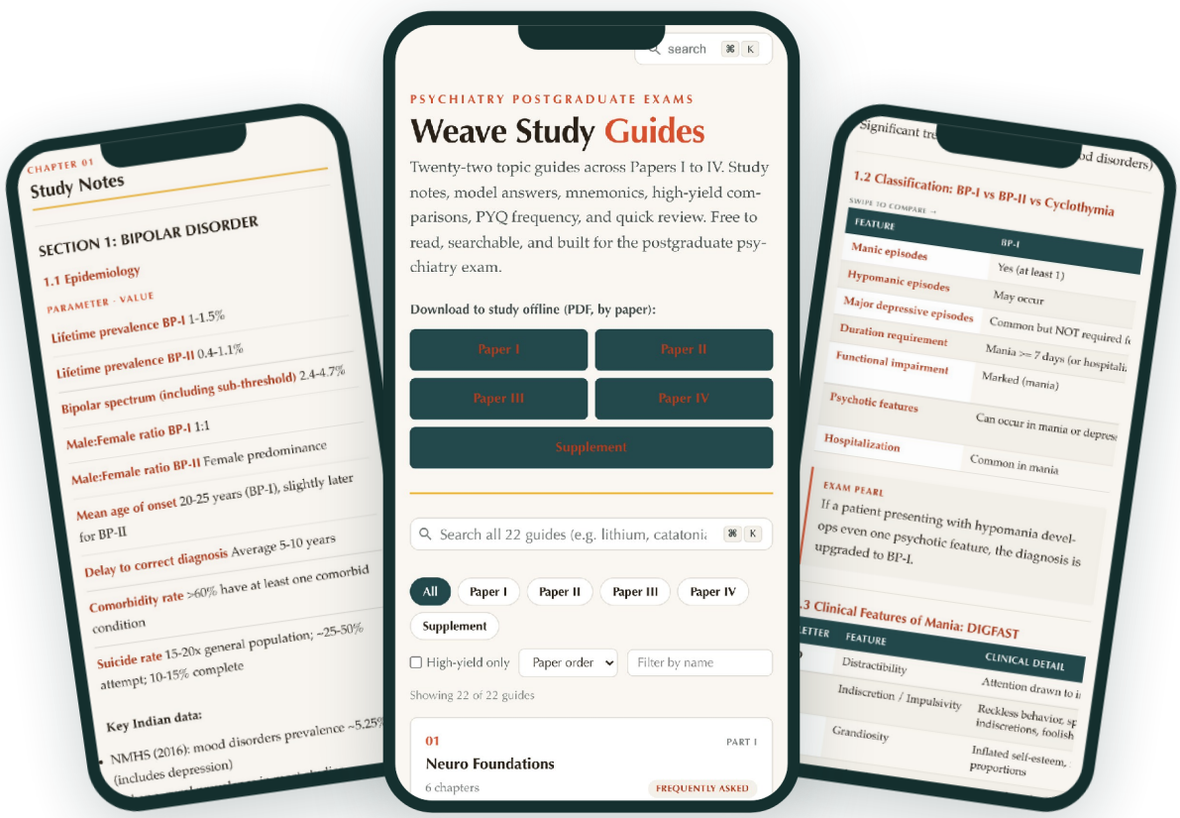
weave.clinic/library

TAP OR SCAN TO OPEN

WEAVE STUDY GUIDES

When the night is short, read the guide.

Twenty-two topic guides across all four papers. Study notes, model answers, mnemonics, high-yield comparisons and previous-question frequency. Free, searchable, built for this exam.



22 guides · all four papers · fully searchable · free to read

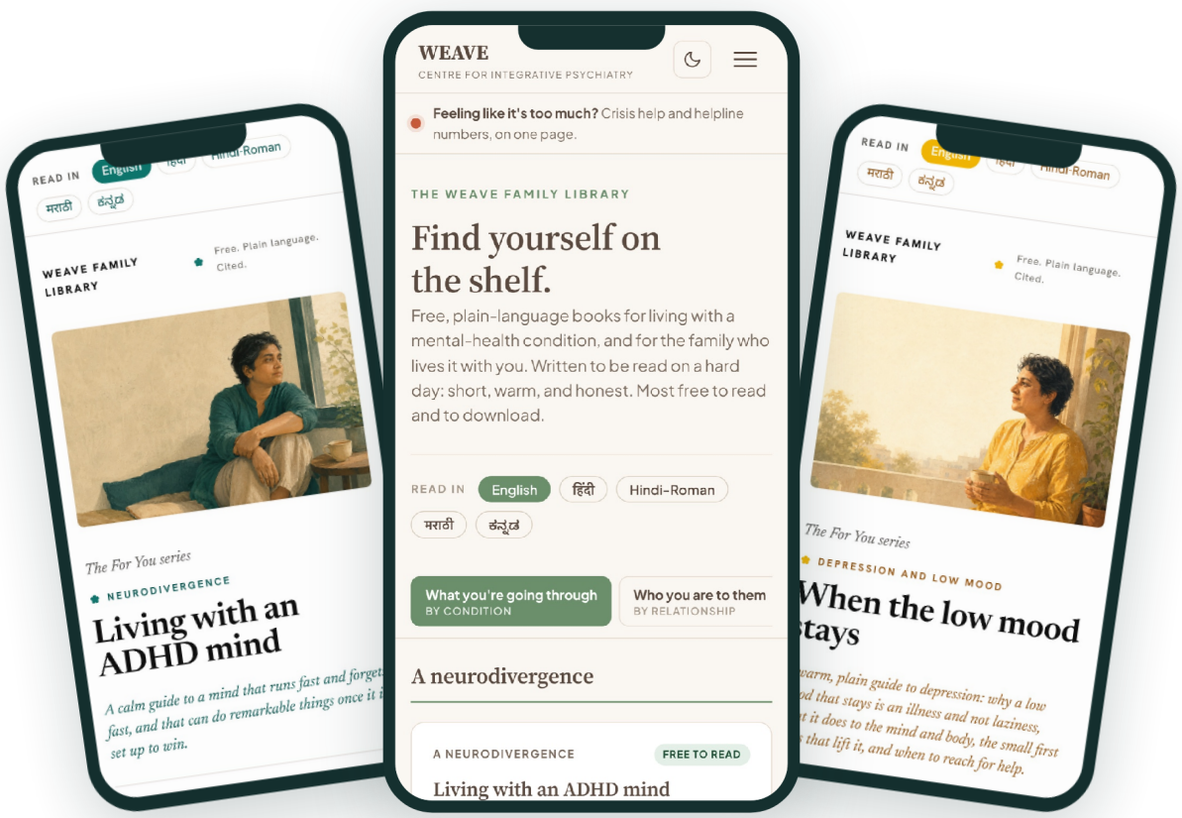
weave.clinic/study

TAP OR SCAN TO OPEN

THE WEAVE FAMILY LIBRARY

Send the family home with more than a prescription.

Free, plain-language books for living with a mental-health condition, written for patients and the people who love them. Find yours by condition, by relationship, or by situation.



plain language · patients and families · five languages · free to read

weave.clinic/family

TAP OR SCAN TO OPEN

A PAPER A DAY

The whole written exam, in one day's reach.

Every paper of the MD psychiatry written exam in one free book, with a colour memory code and clickable navigation, for the last week, the last night, the last hour before the hall.



four papers · one volume · colour-coded · free to read

weave.clinic/revision

TAP OR SCAN TO OPEN

STUDY TOGETHER

You do not have to revise alone.

These notes started as messages in a room full of residents helping each other through. That room is still open. Bring your doubts, your corrections, and the questions the textbooks do not answer.

Join the study group

https://chat.whatsapp.com/H7TsLGK2wFl4bSe2r0Zfzn?mode=gi_t

PRINTING AND DISTRIBUTION

Notes Next Door (NND), Delhi is the official printing and distribution partner for Weave Sprint. The soft-copy PDFs and the complete print-ready set are always available at weave.clinic/sprint, should you prefer to print the notes yourself. Weave Sprint is open source and free, and as part of Weave's commitment to residents it will stay that way, forever. We do, however, strongly recommend a printed set from Notes Next Door: in our experience, they have been the most reliable printing partner we have worked with.

WEAVE SPRINT · PAPER IV · VOLUME ONE

Volume One of the fourth paper: epilepsy and psychiatry, where the two disciplines have argued longest over the same patient, with the peri-ictal syndromes, the psychoses of epilepsy, the non-epileptic attack and prescribing across both illnesses at once; then the clinical neurology a psychiatrist is expected to carry. Two complete sets of notes, written to be understood the first time and recalled every time after, with a colour memory code, a clickable contents and a full index.

Built to understand. Kept to revise.

All the very best for your exam.

WEAVE

weave.clinic · wilfred@weave.clinic · niharika@weave.clinic