WEAVE SPRINT . PAPER IV

Neurology foundations

The neurology a psychiatrist actually needs at the bedside: the examination as an ordered sequence and what each station localises, electroencephalography and cerebrospinal fluid read for what they buy a psychiatric workup, the soft signs that do not localise and still matter, consciousness from coma to brain death with the Indian certification framework set beside the American clinical rule, headache with every prophylactic dose scoped to its indication, and sleep from its architecture to the movement disorders a psychiatrist must tell apart from the drugs they prescribed.

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WEAVE . CENTRE FOR INTEGRATIVE PSYCHIATRY

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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**
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> 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**
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> 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.

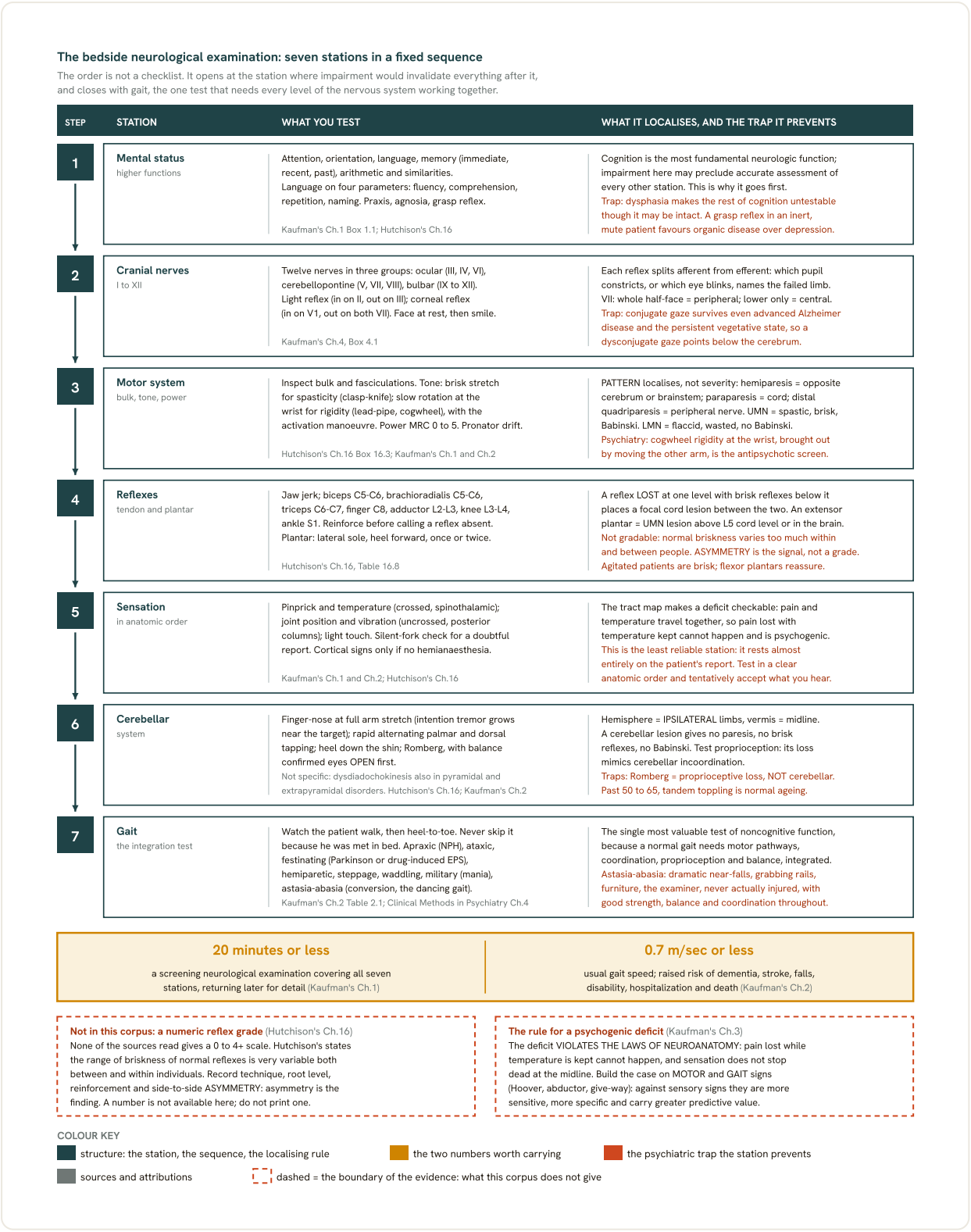

Fig 31.1 **The bedside neurological examination as a structured sequence.** The seven stations run in the order given by Kaufman's Clinical Neurology for Psychiatrists (Box 1.1): mental status, cranial nerves, motor system, reflexes, sensation, cerebellar system and gait. The order is itself the teaching: 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**
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> 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.

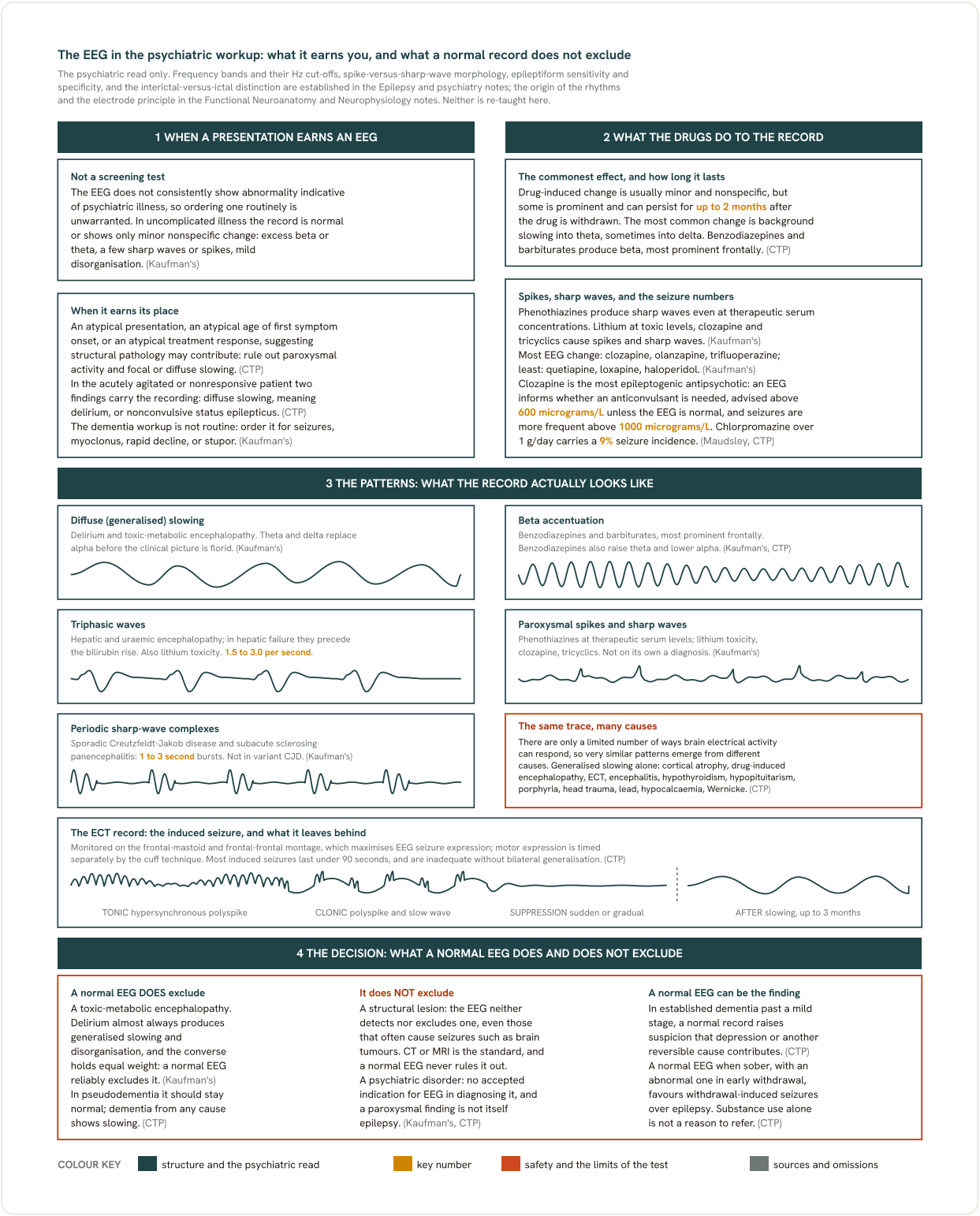

Fig 31.2 The EEG in the psychiatric workup: diffuse slowing, triphasic waves, and the psychotropic and ECT record. The decision is the teaching, not the tracing. The EEG is not a screening test and has no accepted role in diagnosing a psychiatric disorder; it earns its place when the presentation, age 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.

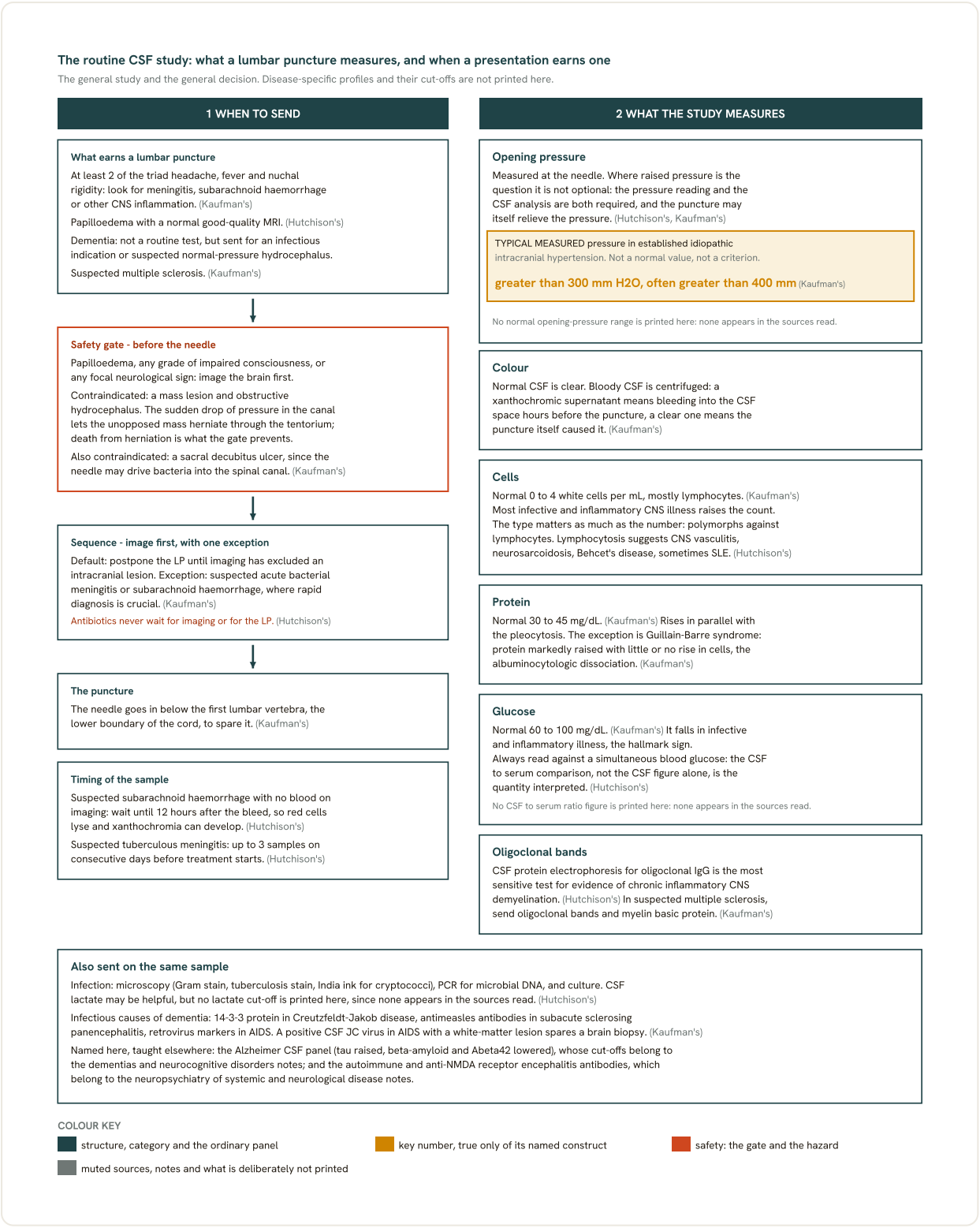

Fig 31.3 The routine CSF study: the constituents measured and the general when-to-send logic. The decision, not the panel, is the teaching: a presentation earns a lumbar puncture, the safety gate (papilloedema, any impaired consciousness, any focal sign) decides whether imaging comes first, and only 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.

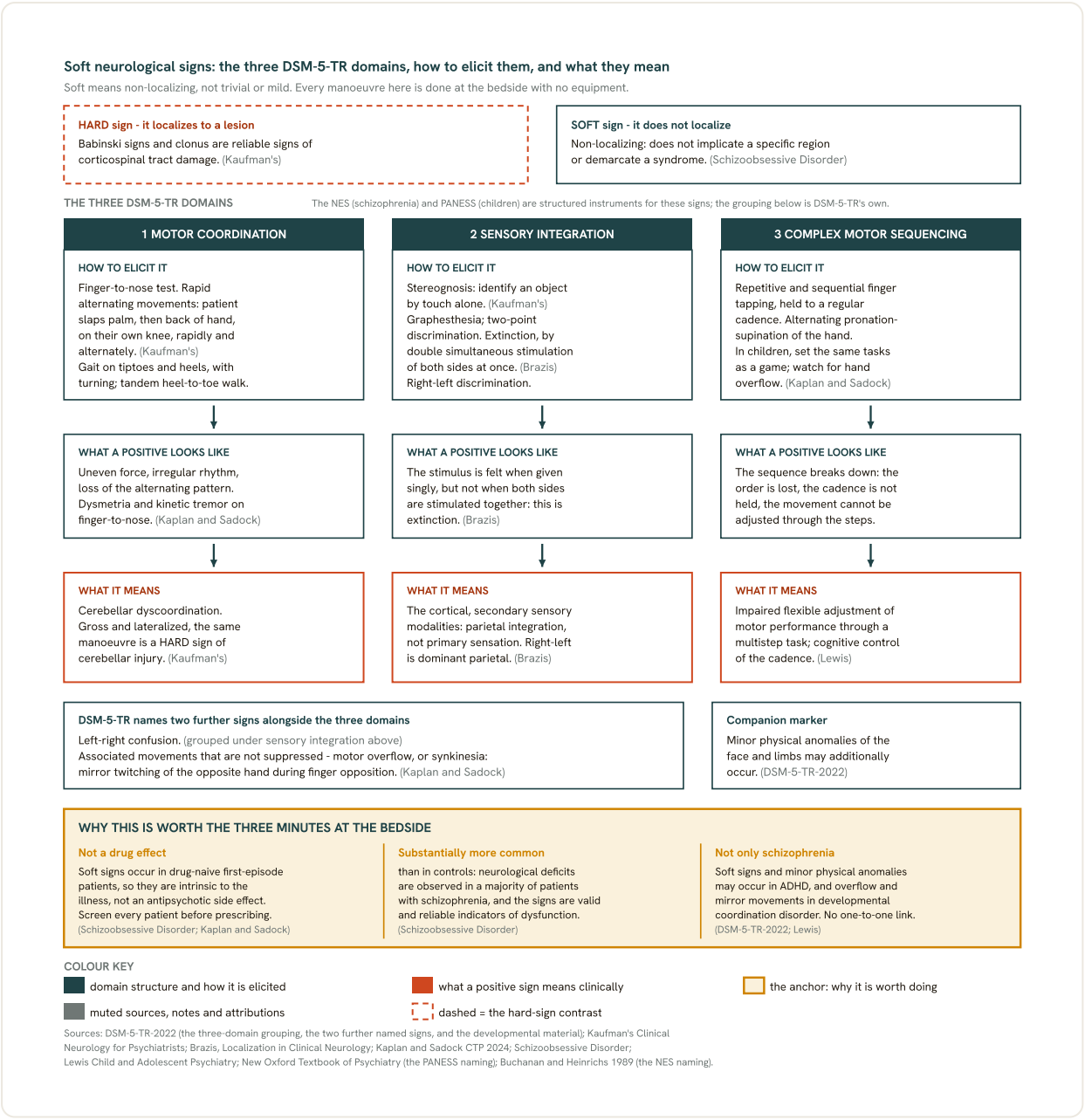

Fig 31.4 Soft neurological signs: the three DSM-5-TR domains, how each is elicited, and what a positive sign means. DSM-5-TR-2022 states that the soft signs common in schizophrenia comprise impairments in motor coordination, sensory integration and motor sequencing of complex movements, together with 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.

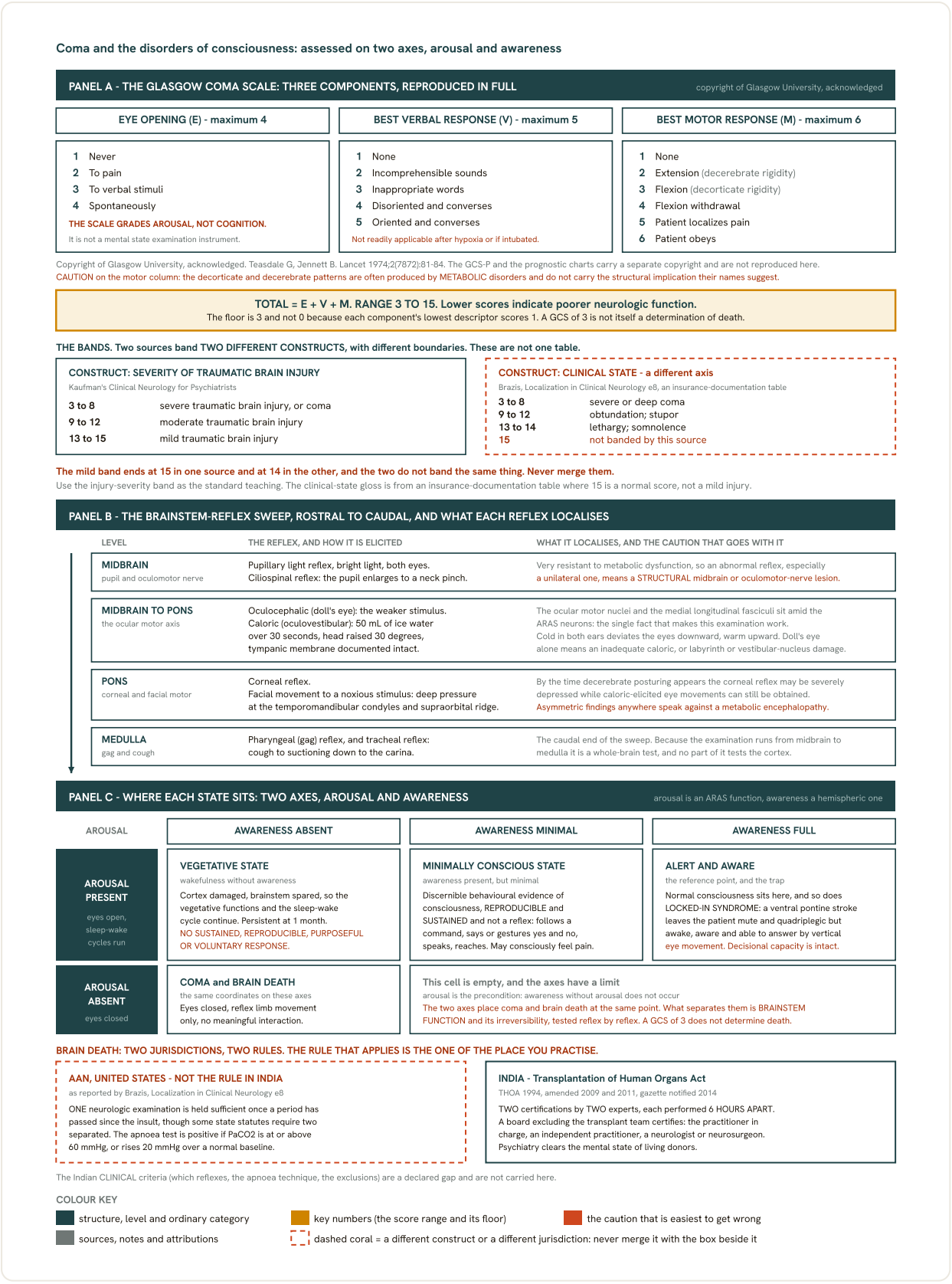

Fig 31.5 Coma and the disorders of consciousness: the Glasgow Coma Scale, the brainstem-reflex sweep, and where each state sits. Consciousness is assessed on two axes, arousal and awareness: coma means either the ARAS is down or both hemispheres are. The Glasgow Coma Scale is the copyright of Glasgow 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.

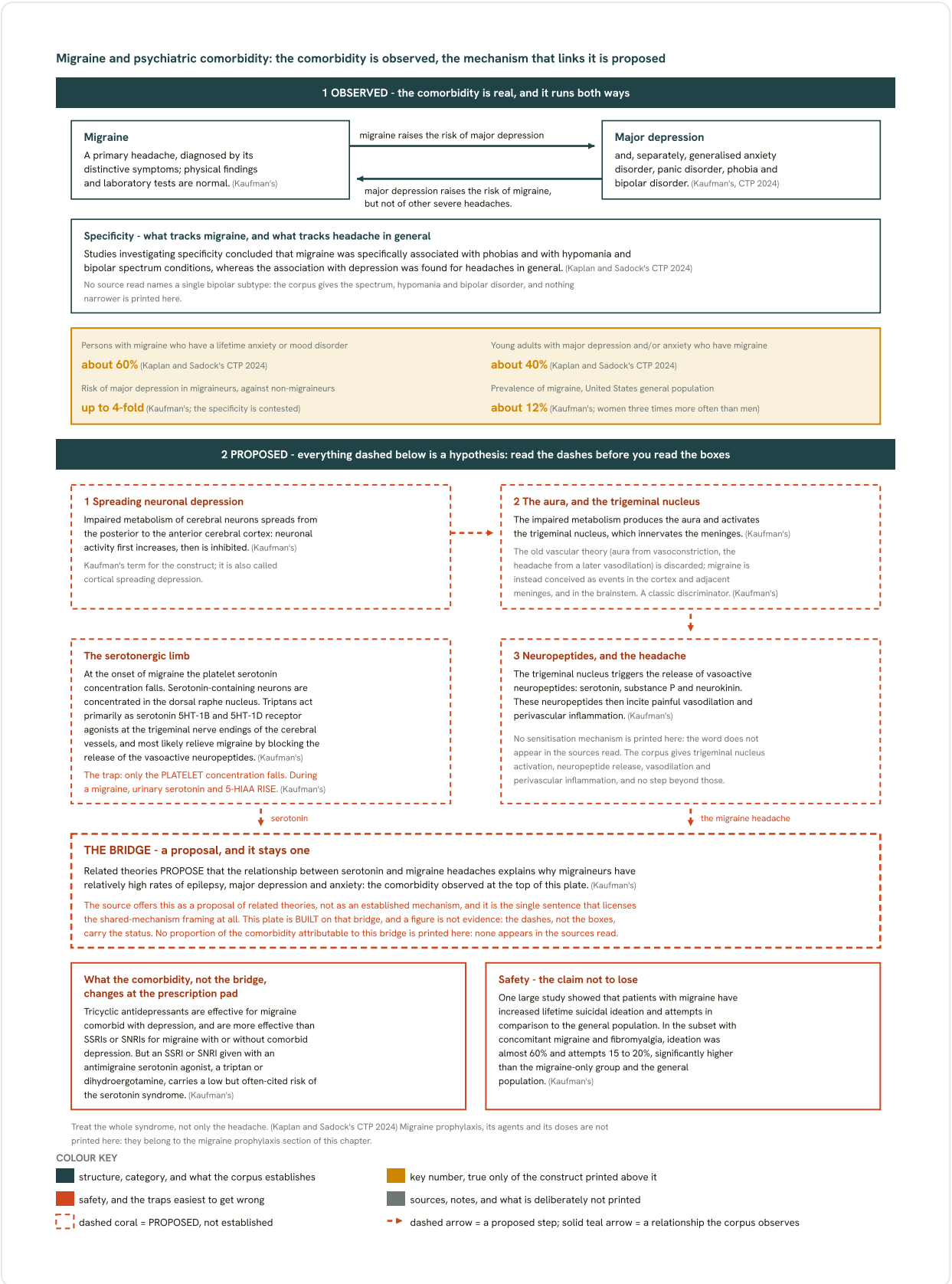

Fig 31.6 Migraine and psychiatric comorbidity: an observed comorbidity, and a bridge that is only proposed. Knowing which half is which is the discriminator, and the plate carries that on its face rather than in this caption: the solid teal half is observed, every dashed coral box and arrow is proposed. 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	**almost exactly 9:1**	**7.70 → 4.75**	**17**
COUNTRY-SPECIFIC ADULT INCIDENCE	FEMALE:MALE RATIO IN A SWEDISH DATABASE PERIOD	LITHIUM ODDS RATIO: GENERAL TO OBESE CONTROLS	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.

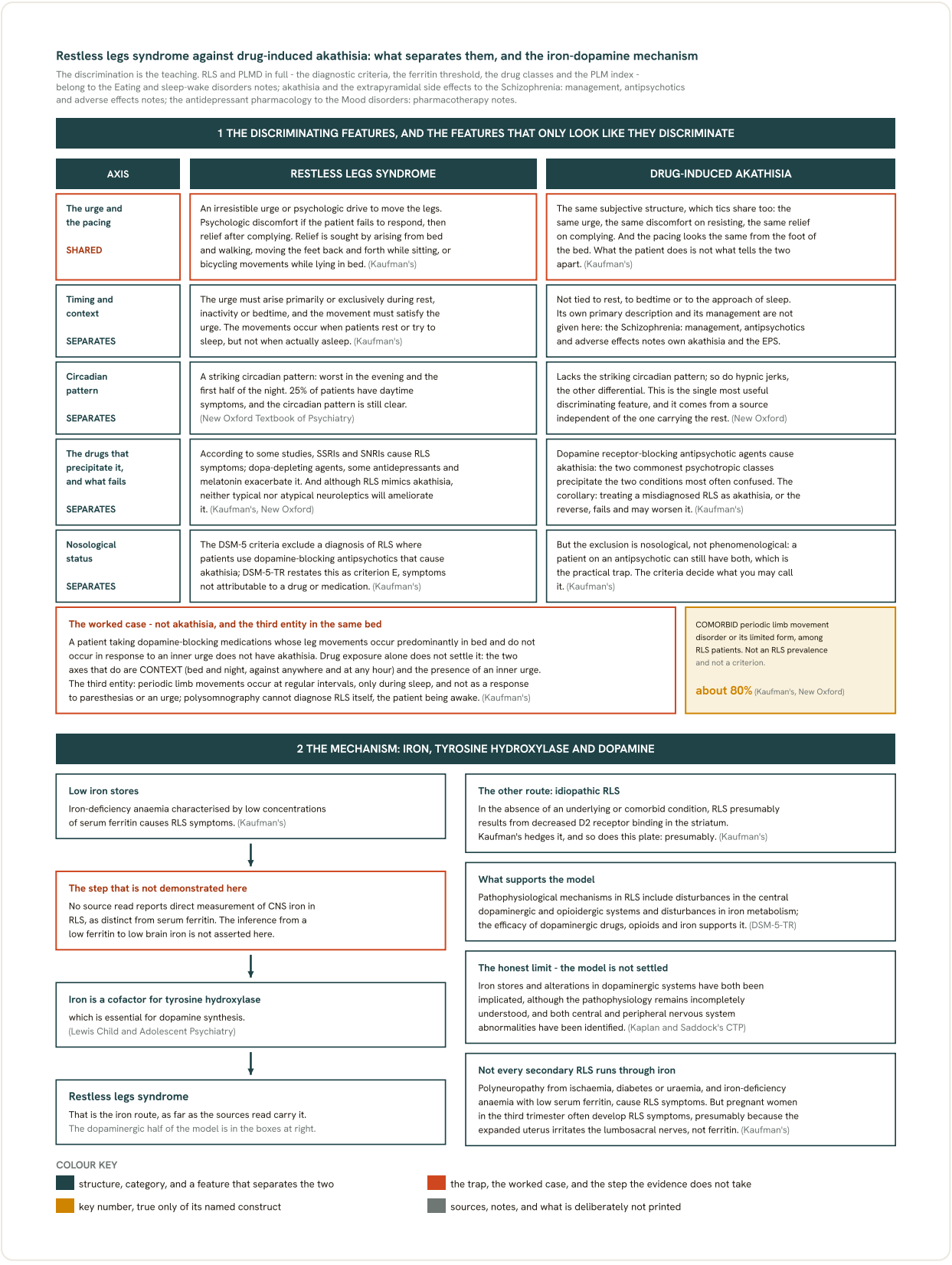

Fig 31.7 Restless legs syndrome against drug-induced akathisia. The urge and the pacing are shared, so neither discriminates; the context, the circadian pattern akathisia lacks, the precipitating drug class and the failure of neuroleptics to ameliorate RLS do. Akathisia appears only by contrast, cross-referred 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."

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