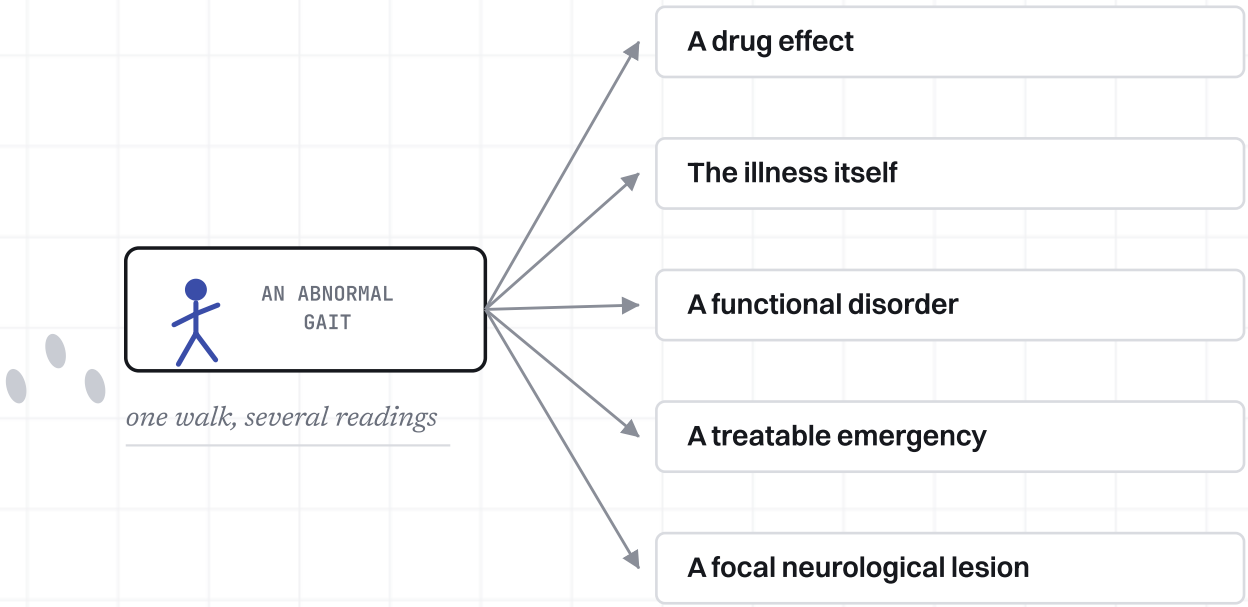


The Way They Walk In.

A gait is the first sign a psychiatrist reads, often before a word is spoken. ***It is shown, not described, and the same abnormal walk has several readings. The skill is classifying it, telling drug-induced from functional from the illness from a focal lesion, and knowing what to do.***

01 The walk as a sign

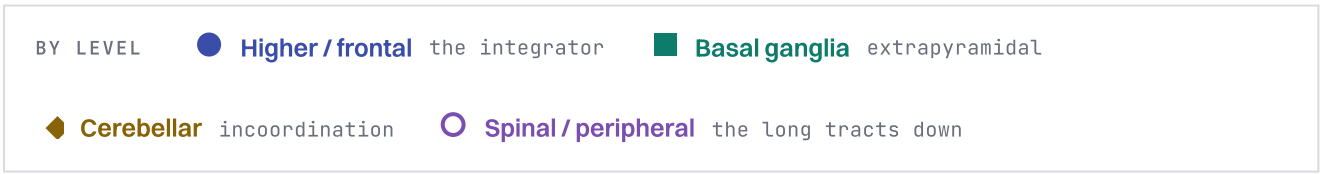
DEFINITIONAL

A gait is read before it is described. The patient crosses the room, and by the time they sit down the experienced clinician has already formed an impression that no question will return. This is an advantage and a trap. An abnormal walk is one of the highest-yield physical signs in medicine, but it is also one of the most over-read and most casually dismissed, because the same abnormal walk has several readings and the differences between them are not obvious from the doorway. The whole skill is telling them apart.

The readings compete. A slow, stiff, shuffling walk in a patient on an antipsychotic may be **a drug effect**, the commonest movement complication a psychiatrist causes. It may be **the illness itself**, the leaden gait of psychomotor retardation in depression, or the stilted, halting gait of catatonia. It may be **a functional disorder**, genuine and involuntary, recognised by its own positive signs. It may be **a treatable emergency** hiding in plain sight, Wernicke encephalopathy, lithium toxicity, normal-pressure hydrocephalus. Or it may be **a focal neurological lesion** that localises to a level of the nervous system and needs a neurologist. Each of these is managed differently, and the first one chosen is too often the last one considered.

Two facts make this a psychiatrist's problem rather than a neurologist's. The first is exposure: the drugs we prescribe cause gait disorders, the illnesses we treat produce them, and the patients we see carry an unusual load of alcohol, falls and polypharmacy. The second is prevalence. Gait disorders are common and rise steeply with age, from roughly one person in ten in their sixties to more than six in ten of those over eighty, and the commonest neurological contributors, sensory ataxia, parkinsonism and the frontal gait disorders, are exactly the ones that sit at the seam between psychiatry and neurology.[6] Gait is no longer regarded as an inevitable casualty of ageing to be shrugged at; it is a localisable sign to be worked.[3]

THE CONCEPT CODE USED THROUGHOUT: BY LEVEL OF LESION



Each colour is also a shape, so the code survives a greyscale photocopy. The four levels run from the top of the nervous system to the periphery. A fifth category sits deliberately off this axis: the functional gait and the psychiatric-state gaits do not localise to a lesion at all, and recognising that is half of what this issue teaches. The figure overleaf is the whole map on one page.

02 How to classify a gait

BY PHENOMENOLOGY, AND BY LEVEL

There are two useful ways to classify a gait, and a clinician uses both at once. The first is **phenomenological**: name the pattern by what it looks like, parkinsonian, ataxic, spastic, steppage, waddling, cautious, functional. The second is **anatomical**: ask what level of the nervous system the pattern points to. The two are tied together, because the dominant phenomenology and its underlying pathophysiology are tightly connected, and what you finally see at the bedside is the disease plus whatever the patient has done to compensate for it.[4] Read the pattern, then read the company it keeps: the accompanying signs, sensory, extrapyramidal, cerebellar, cortical, point to the level, and in older patients the cause is frequently more than one level at once.[5]

The hierarchy. The organising idea behind the anatomical reading is a hierarchy. In the classic scheme of Nutt, Marsden and Thompson, gait control is layered: a lowest level (the peripheral apparatus and the sensory channels that feed it), a middle level (the long motor tracts, the cerebellum and the basal ganglia that shape the stepping), and a highest level (the frontal and subcortical systems that integrate balance, initiation and the decision to walk).[1] The highest level is the one most relevant to psychiatry and the most poorly localising. Damage there does not produce a clean weakness or a clean ataxia; it produces cautious, hesitant, small-stepped and freezing gaits that look odd and defy a single tract, and it was precisely this group that the original work named the higher-level gait disorders.[1,2] Locomotion is itself a higher-order motor behaviour that leans on mental processes, which is why it is so readily disturbed by mood, attention and fear, not only by lesions.[2]

Why phenomenology leads at the bedside. Several classifications coexist, anatomical, aetiological, pathological and phenomenological, and none is complete on its own.[7] For the unusual or non-localising pattern, the most pragmatic route is to deconstruct what you see into its elements, name the phenomenology, and let the accompanying signs localise it, rather than reaching first for a diagnosis.[7] The map overleaf sets the named gaits against the level each points to, with the off-axis category for the gaits that point nowhere on the neuraxis.

THE ORDER TO READ A GAIT IN

1. **Name the pattern.** Parkinsonian, ataxic, spastic, steppage, waddling, cautious, functional. The phenomenology is the entry point and the one thing you can record before you have a diagnosis.
2. **Find the level.** Read the company the gait keeps. Rigidity and reduced arm swing point to the basal ganglia; a wide base and past-pointing to the cerebellum; a sensory level or foot-drop to the periphery; start hesitation and small steps with preserved leg strength to the frontal lobe.[5]
3. **Ask whether it localises at all.** If the pattern is internally inconsistent, distractible, or a slowed walk that tracks mood, it may sit off the neuraxis entirely. That recognition is the move that separates a psychiatrist's reading from a checklist.

FIGURE 1 **The localisation map: each level, and the gaits that point to it**

WHERE THE LESION IS, AND WHAT IT MAKES THE WALK DO — LOCALISATION MAP

LEVEL	STRUCTURE	THE GAITS THAT POINT HERE
HIGHER-LEVEL / FRONTAL	Frontal cortex and its subcortical connections; the integrator of walking	Cautious gait · frontal / apraxic gait (marche a petits pas) · the magnetic gait of NPH · the gait of dementia · gait-ignition failure
BASAL GANGLIA / EXTRAPYRAMIDAL	Nigrostriatal dopaminergic pathways	Parkinsonian / festinant gait (Parkinson's and drug-induced) · hyperkinetic / dyskinetic gait (tardive, chorea)
CEREBELLAR	Cerebellum, especially the anterior vermis, and its connections	Wide-based, unsteady ataxic gait (chronic alcohol, Wernicke, lithium toxicity, acute intoxication)
SPINAL + PERIPHERAL	Corticospinal tracts, dorsal columns, nerve, muscle, vestibular apparatus	Spastic / hemiplegic / scissoring · sensory (stamping) ataxia · high-stepping (foot-drop) · waddling (myopathic) · vestibular · antalgic
OFF THE AXIS: THESE DO NOT LOCALISE TO A LESION		
NO LEVEL NON-LOCALISING	Not a structural lesion. Diagnosed by a positive pattern, not by a level	Functional (psychogenic) gait · the psychomotor-retardation gait of depression · the catatonic gait

Read the pattern, then read the company it keeps: the accompanying signs localise the level.

The gaits a psychiatrist meets most often are the two at the bottom, the ones with no level at all.

HOW TO READ THIS Read down by level of the nervous system, structure on the left, the gaits it produces on the right. The two rows below the dashed line are off the axis: the non-localising gaits, the ones a psychiatrist meets most. The level is a starting point, not a verdict; many older patients have a gait built from more than one level, and the higher-level disorders in particular blur into one another.[1,5]

WHAT THE MAP BUYS YOU

- **It narrows the differential before you touch the patient.** A wide-based stagger and a small-stepped shuffle are different problems pointing to different levels, and the pattern rules half the list in or out from across the room.
- **It separates the localising from the non-localising.** The four levels are organic and refer outward, to a neurologist or to a drug change. The off-axis gaits are the psychiatrist's own territory: they are treated, not referred, once they are correctly named.
- **It keeps the treatable emergencies visible.** Three of the cerebellar entries, Wernicke, lithium toxicity and acute intoxication, and the magnetic gait of NPH at the top, are reversible if caught and disabling if missed. The map keeps them on the same page as the benign ones, so they are never filed under inattention.

03 The localisation crosswalk

EVERY NAMED GAIT, BY LEVEL

This is the reference table for the named gaits. The first four blocks are organic and localise to a level of the nervous system; the last block sits off the axis and does not. Read a block to learn the level; read a row to fix one gait. The glyph carries the level so the page survives a photocopy.

TABLE 1 **Named gaits, by level of the nervous system**

Gait	WHERE IT LOCALISES	CLASSIC CAUSE	WHAT IT MEANS IN PSYCHIATRY
● Frontal / apraxic	Frontal lobes, subcortical white matter	Small-vessel disease, NPH, frontal tumour	The gait of vascular dementia; small steps and start hesitation that mimic depressive slowing
● NPH magnetic	Ventricular enlargement, periventricular tracts	Idiopathic normal-pressure hydrocephalus	A surgically reversible mimic of dementia; refer for shunt assessment, do not file as decline
● Cautious	Adaptive; no focal lesion	Any perceived instability, fear of falling	Anxiety and fear of falling made visible; the commonest higher-level gait in mild dementia
■ Parkinsonian / festinant	Nigrostriatal dopamine	Parkinson's disease; antipsychotics and other dopamine blockers	The movement disorder a psychiatrist most often causes
■ Hyperkinetic / dyskinetic	Striatal output; D2 supersensitivity	Tardive dyskinesia and dystonia; chorea; Huntington's	A late complication of long-term antipsychotic exposure
◆ Cerebellar ataxic	Cerebellum, especially the anterior vermis	Chronic alcohol, Wernicke, lithium toxicity, intoxication, MS	The alcohol-and-lithium gait; treat as an emergency until Wernicke and toxicity are excluded
○ Spastic / hemiplegic	Corticospinal (upper motor neuron) tracts	Stroke, multiple sclerosis, cord lesion, cerebral palsy	Post-stroke; the stiff leg circumducts and the arm is held flexed
○ Sensory ataxic (stamping)	Dorsal columns (B12) or large-fibre sensory nerve	B12 deficiency, alcohol and diabetic neuropathy	Nutritional and alcohol neuropathy; markedly worse in the dark, Romberg positive
○ Steppage (foot-drop)	Common peroneal nerve, L5 root, polyneuropathy	Nerve palsy, polyneuropathy	Uncommon but cleanly localising; the foot slaps the floor
○ Waddling (myopathic)	Proximal pelvic-girdle muscle	Myopathy, hip disease	Rare in psychiatry; consider with long-term steroids
○ Vestibular	Labyrinth, vestibular nerve	Vestibular neuritis, Meniere's disease	Overlaps the anxious, visually dependent gait of persistent dizziness
○ Antalgic (painful)	Bone, joint, soft tissue	Arthritis, injury	A confound, not a neurological gait; exclude pain before reading anything else
◌ Functional (psychogenic)	No lesion; a positive pattern	Functional neurological disorder	Genuine and involuntary; diagnosed by positive signs, never by exclusion
◌ Depression (psychomotor)	No lesion; a state-dependent slowing	Major depressive episode	Slow, stooped, reduced arm swing; state-dependent and reversible with recovery
◌ Catatonic	No lesion; a psychomotor syndrome	Catatonia (mood, psychotic or medical)	Stilted and halting with posturing or negativism; responds to lorazepam and ECT

The peripheral and antalgic gaits are uncommon presentations in a psychiatric clinic, but they localise cleanly and are worth recognising so they are not mistaken for something central. The off-axis block is where most of a psychiatrist's work lives.

04 The differentiation grid

THE TABLE TO REMEMBER

This is the centerpiece. Each row is a gait the psychiatrist actually meets; the columns move from what it looks like, to where it comes from and why, to the one bedside clue that pulls it apart from its neighbours. The sections that follow expand each row and carry the citations.

TABLE 2 **The gaits the psychiatrist meets, and what tells them apart**

Gait	WHAT IT LOOKS LIKE	WHERE IT IS SEEN / THE MECHANISM	THE BEDSIDE CLUE
■ **Drug-induced parkinsonian** BASAL GANGLIA	Bradykinesia, rigidity, reduced arm swing, shuffling, festination; usually symmetric	Antipsychotics and other dopamine blockers; nigrostriatal blockade; 20 to 35% of users	**Onset within weeks** of starting or raising the drug; symmetric; eases on dose reduction
■ **Tardive / dyskinetic** BASAL GANGLIA	Choreoathetoid or dystonic intrusions into stance and stride; posture distorted	Months to years of dopamine-blocker exposure; D2 supersensitivity	**Late and hyperkinetic**, not hypokinetic; worsens on withdrawing the drug
◌ **Depression** NON-LOCALISING	Slow, heavy, stooped, reduced arm swing and stride; a leaden walk	A state, not a lesion; psychomotor retardation	**Tracks mood** and reverses with recovery; no rigidity or cogwheeling
◌ **Catatonic** NON-LOCALISING	Stilted, halting, freezing; posturing, negativism, gegenhalten, mannerisms	Catatonia in mood, psychotic or medical illness	**Other catatonic signs** present; a lorazepam challenge eases it
◌ **Functional** NON-LOCALISING	Variable, effortful; walking-on-ice, dragging or buckling, astasia-abasia	Functional neurological disorder; genuine and involuntary	**Inconsistency and distractibility**; positive signs; uneconomic slowness
● **Cautious / fear of falling** HIGHER-LEVEL	Widened base, short slow steps, reaching for support, en-bloc turns	An adaptive response to perceived instability; anxiety, fear of falling	**Improves with a light touch** or a companion's arm; out of proportion to any deficit
◆ **Cerebellar (alcohol)** CEREBELLAR	Wide-based, unsteady, irregular, titubation; cannot tandem walk	Anterior-vermis degeneration from chronic alcohol and poor nutrition	**Wide base, past-pointing, dysarthria**; not much worse in the dark
◆ **Wernicke** CEREBELLAR / EMERGENCY	Ataxic, wide-based gait, often the most prominent or only sign	Thiamine deficiency; a treatable emergency	**Two of four:** diet, eye signs, gait, confusion; treat on suspicion before glucose
◆ **Lithium toxicity** CEREBELLAR / RED FLAG	Ataxic gait with coarse tremor, dysarthria, nystagmus	Lithium cerebellar neurotoxicity; can occur at therapeutic levels	**New ataxia on lithium is toxicity** until proven otherwise; may persist
◆ **Acute intoxication** CEREBELLAR	Staggering, wide-based, lurching, unsteady	Alcohol or benzodiazepine; reversible cerebellar and vestibular depression	**Acute, fluctuates with the level**; the history and the breath; clears with the drug
● **NPH (magnetic)** HIGHER-LEVEL	Broad-based, short shuffling steps, feet stuck to the floor, start hesitation	Ventriculomegaly; a surgically reversible cause	**Gait dominates the triad**; improves after removing CSF at a tap
● **Frontal / dementia** HIGHER-LEVEL	Small steps (marche a petits pas), wide base, upright, early-preserved arm swing	Cerebral small-vessel disease; frontal-subcortical disconnection	**Strength and coordination preserved** on the couch; predicts vascular dementia

The first column is what catches the eye; the last is what settles the question. The thread through the table is that onset, symmetry, the company the gait keeps, and whether it tracks state are what separate the look-alikes.

05 The gaits the drugs cause

The first gait to learn is the one we cause. Drug-induced parkinsonism is the second commonest cause of parkinsonism after Parkinson's disease, was first described with antipsychotics, and is produced by any dopamine blocker.[9] It is common: prevalence estimates vary with the agent, the dose and how it is sought, running as high as 20 to 35% of antipsychotic users.[10] And the gait is not incidental to it. When the World Health Organization pharmacovigilance database was mined for drug-induced parkinsonism, the two most frequently co-reported terms were tremor and **gait disturbance**, so an unsteady or shuffling walk is one of the leading ways the syndrome announces itself.[12]

What it looks like, and who gets it. Bradykinesia and rigidity with a rhythmic tremor appear within hours to weeks of starting or raising the drug, from blockade of dopamine in the nigrostriatal pathway.[10] The gait is the parkinsonian one, reduced arm swing, short shuffling steps, a stooped posture and festination, but it is more often symmetric than the asymmetric gait of idiopathic disease. Risk rises with older age, female sex, the dose and duration, the agent, cognitive impairment and any pre-existing extrapyramidal signs.[11] The reporting risk is highest for sulpiride and haloperidol, then risperidone, aripiprazole, paliperidone, metoclopramide, olanzapine, quetiapine and clozapine, and is greater in men and in those over 75.[12] It is not only antipsychotics: valproate can cause a reversible parkinsonism, often with cognitive impairment, emerging after years of therapy at ordinary serum levels.[15]

The trap, and the fix. Drug-induced parkinsonism can be clinically indistinguishable from idiopathic Parkinson's disease, which is why a documented exposure matters and why a definitive split sometimes needs dopamine-transporter imaging.[13] The drug may also be unmasking degenerative disease that was already on its way, and although the parkinsonism usually reverses on withdrawal it can take months to do so; hyposmia and DAT imaging help separate pure drug effect from unmasked degeneration.[11,13] The same vulnerability now reaches children: paediatric drug-induced parkinsonism rose roughly tenfold over a decade alongside rising atypical-antipsychotic use, with risperidone and aripiprazole carrying the highest risk.[14] The management move is to switch to a lower-propensity agent rather than escalate, and to remember that anticholinergics relieve this parkinsonism but worsen any co-existing tardive dyskinesia; the full extrapyramidal map is the subject of Aporia 09.[10]

READING THE DRUG-INDUCED GAIT

- **Symmetry and timing give it away.** A symmetric, reduced-arm-swing shuffle that arrived with the drug or its last increase is drug-induced until proven otherwise; review the antipsychotic before reaching for anything else.
- **Asymmetry or persistence changes the question.** If the gait is asymmetric, carries a rest tremor that outlasts the drug, or fails to settle after withdrawal, think idiopathic Parkinson's disease and image rather than simply re-challenge.

05 / cont. The tardive gait

LATE, AND HYPERKINETIC

The second drug-induced gait is the opposite of the first: late instead of early, hyperkinetic instead of slow, and worsened rather than helped by the anticholinergic that rescues acute parkinsonism. The tardive syndromes are not a single movement but a family, dyskinesia, dystonia, akathisia, tremor, myoclonus, and a recognised **tardive gait**, each with its own pathophysiology and treatment, which is why they should never be treated as one uniform pattern.[18,19]

Tardive dystonia distorts the walk. Unlike the classic orofacial dyskinesia, tardive dystonia produces sustained abnormal axial and limb posturing that twists standing posture and gait, and it is more painful, more disabling and more often persistent than classic tardive dyskinesia.[16] It is not protected by a short exposure: in the largest series it arose across all ages and both sexes, appeared in a fifth of cases within the first year with no minimum safe latency, was usually persistent, and responded best to dopamine-depleting agents and, in some, to anticholinergics, though full remission was rare.[17]

Tardive dyskinesia intrudes on stride. Pooled prevalence is about a quarter of treated patients, higher with first-generation than second-generation agents, so a prescriber of dopamine blockers will meet it at scale.[20] Its choreoathetoid movements intrude into stance and stride, and the contemporary pharmacological lever is selective VMAT2 inhibition: valbenazine significantly reduced tardive dyskinesia severity against placebo in a phase-3 trial, and the same mechanism reduces the chorea of Huntington's disease, confirming VMAT2 inhibition as the shared tool for choreic hyperkinesias.[21,22]

THE DISCRIMINATOR

Two drug-induced gaits, opposite in every way

Drug-induced parkinsonism is **early** (hours to weeks), **hypokinetic** (slow, stiff, reduced arm swing), and **eased** by an anticholinergic. The tardive gait is **late** (months to years), **hyperkinetic** (choreic or dystonic), and **worsened** by an anticholinergic. The single most useful question is when the gait began relative to the drug, and whether the movement is too little or too much. The full account of both, with the onset timeline and the management forks, is Aporia 09.

QUICK CHECK

A patient on long-term haloperidol develops an unsteady, writhing walk; trihexyphenidyl is added and the walk worsens. Early drug-induced parkinsonism, or the tardive gait?

A Tardive: it is late and hyperkinetic, and worsened, not eased, by the anticholinergic, the mirror image of acute drug-induced parkinsonism.[18,19]

06 The cerebellar gaits

ALCOHOL, INTOXICATION, WERNICKE, LITHIUM

The alcohol and intoxication gaits. Chronic alcohol and its poor nutrition degenerate the anterior superior vermis, giving a wide-based truncal gait ataxia with relative arm-sparing and no nystagmus, the cerebellar face of the same thiamine-deficiency spectrum as Wernicke; the instability has an objective signature, excess anterior-posterior sway and a 5 to 7 Hz truncal tremor, and it does not always reverse with abstinence.[23,26] Acute alcohol gives the familiar staggering, lurching walk that clears as the drug clears, and so do benzodiazepines, which independently raise falls in older people (odds ratio around 1.4, higher for long-acting agents), so an unsteady gait on a sedative is a prompt to review the load before reaching for a primary neurological cause.[29,36]

Wernicke encephalopathy. The classic triad of confusion, eye signs and gait ataxia is the exception, not the rule, and waiting for it is how the diagnosis is missed: in a necropsy series only a fifth of confirmed cases had been recognised in life.[24] Gait ataxia is often the most prominent of the three, and the unsteady walk may be the only sign in front of you. The discipline is to use the operational (Caine) criteria, which require only two of four, dietary deficiency, oculomotor signs, cerebellar or gait dysfunction, and an altered mental state or memory impairment, and to treat on suspicion.[25] Thiamine is given parenterally and before any carbohydrate, in high dose (the EFNS guideline gives 200 mg three times daily, and many protocols use more for established disease), and its safety margin is wide enough that empirical treatment carries little downside.[27] It is not only an alcohol problem: Wernicke also follows bariatric surgery, prolonged vomiting and psychiatric food refusal, settings a psychiatrist meets in eating disorders and catatonia, where it most often presents as an altered mental state rather than the full triad.[28]

Lithium toxicity. Lithium has a narrow therapeutic window, and its neurological toxicity is a cerebellar one: gait ataxia, a coarse tremor, dysarthria and nystagmus.[30] The dangerous fact is that this neurotoxicity can occur even when the serum concentration is within the therapeutic range, so a new ataxia or dysarthria is a clinical red flag regardless of the level.[31] Worse, it can persist. The Syndrome of Irreversible Lithium-Effectuated Neurotoxicity is defined as neurological sequelae lasting at least two months after lithium is stopped, and persistent cerebellar dysfunction, the ataxic, unsteady gait, is its single most commonly reported sequela; in the largest review of 117 cases, cerebellar dysfunction persisted in 77%, often with concurrent antipsychotic use.[32,33] Vulnerability is not uniform: any prior neurological illness markedly raises the risk, and in that setting toxicity does not correlate with the serum level at all, so a therapeutic number offers false reassurance.[34] Severe poisoning warrants extracorporeal removal: dialysis is recommended where kidney function is impaired and the level exceeds 4.0 mEq/L, or with a decreased conscious level, seizures or life-threatening dysrhythmias whatever the number.[35]

TWO RULES

The cerebellar gait that must not wait

01 **An unsteady, wide-based gait in anyone who uses alcohol is Wernicke until excluded.** Treat with parenteral thiamine before glucose, on two of the four Caine criteria, without waiting for confusion or eye signs.

02 **A new ataxia, slurred speech or unsteady walk in anyone on lithium is toxicity until excluded.** The serum level can be normal and still be toxic; check it, but do not let a therapeutic number reassure you, and act on the gait.

The magnetic gait of NPH. The most important higher-level gait to recognise is the one of normal-pressure hydrocephalus, because it is one of the few surgically reversible causes of gait failure in older adults and it is routinely mistaken for primary dementia.[37] Gait disturbance is the cardinal and most reliably reversible component of the Hakim triad of gait, cognition and continence: shunt surgery most consistently improves mobility, while cognition and continence respond less predictably.[38] The walk is broad-based with short, shuffling, magnetic steps, the feet seeming glued to the floor, with start hesitation and turning all-of-a-piece.

The walk is the test, and the target. Removing cerebrospinal fluid both confirms the diagnosis and predicts the surgery: a positive gait response to a high-volume tap or to prolonged lumbar drainage carries a high positive predictive value for shunt benefit, and in the multicentre SINPHONI study the tap test predicted shunt response with useful accuracy, improving when combined with the opening pressure.[39,40] The clinical consequence is simple and frequently missed. A broad-based, magnetic, small-stepped gait in an older patient with cognitive slowing and urinary urgency is referred for neurosurgical shunt assessment, not filed as untreatable decline.[37]

The frontal gait of small-vessel disease. More broadly, the higher-level or frontal gait disorder is a breakdown in the supraspinal organisation of balance and locomotion, a wide base, short shuffling steps, start hesitation or freezing, en-bloc turns, and preserved leg strength and coordination, and it subdivides into an ignition failure that responds to external cues and an equilibrium failure that does not.[8] Its commonest driver is cerebral small-vessel disease: the burden of periventricular white-matter change is the strongest imaging correlate of gait impairment, whereas amyloid is not.[41] This carries a prognosis. In community-dwelling adults over 75 without dementia, a neurological gait abnormality nearly doubled the overall risk of dementia but specifically predicted non-Alzheimer, vascular, dementia, with the frontal gait the single strongest predictor, and did not predict Alzheimer's disease at all.[42] The small-stepped frontal gait is both a present sign of vascular cognitive impairment and a marker of what is coming.

~2× **A neurological gait abnormality nearly doubled the risk of dementia** in adults over 75, and specifically predicted the vascular kind, not Alzheimer's, with the frontal gait the strongest single predictor.[42]

QUICK CHECK

An older adult walks with a broad-based, magnetic, small-stepped gait, the feet seeming glued to the floor, with cognitive slowing and urinary urgency. What is the one cause you must not file as untreatable decline?

A Normal-pressure hydrocephalus. Gait is the cardinal and most reliably reversible part of the triad, so this is referred for a tap test and shunt assessment, not recorded as irreversible dementia.[37,38]

The cautious gait

The cautious gait is the body's stereotyped answer to feeling unsafe on its feet: a widened base, slow velocity, shortened steps, and turning all-of-a-piece, reaching for the wall and the furniture. It is a higher-level adaptive response, not the sign of a focal lesion.[1] It is the commonest higher-level gait disorder in mild Alzheimer's disease, present in around 40% of patients against 18% of controls, and it can be reproduced in anyone: experimentally raising the threat of a fall heightens anxiety and stiffens the gait, the more so in older adults.[43,72]

Fear of falling drives it, and it feeds itself. The cautious gait is frequently driven by fear of falling, which behaves like a condition in its own right. It is associated with previous falls and independently predicts both future falls and future fear, and it can develop in people who have never fallen, with mild cognitive impairment raising the risk; roughly a quarter of older adults develop it over a year of follow-up.[73,74] The loop is self-reinforcing, fear narrows the gait, the narrowed gait confirms the fear, and the psychiatrist is well placed to break it: balance training and Tai Chi produce a small-to-moderate reduction in fear of falling without increasing the rate of falls.[75]

The anxious, visually dependent gait. A specific pattern sits at the seam of psychiatry and neuro-otology. Persistent postural-perceptual dizziness is a chronic functional vestibular disorder, defined by three months or more of dizziness and unsteadiness that worsen on standing, with self-motion, and in busy visual environments; it is typically set off by an acute organic vestibular event in a person with an anxious temperament, and is explicitly neither a structural disease nor merely a psychiatric one.[76,78] When such patients walk, they show a slow, cautious gait with the body stiffened, the gaze pulled down to the ground and scanning sideways for support, and the severity of these changes tracks their fear of falling.[77] It improves with vestibular rehabilitation, cognitive behavioural therapy and SSRIs, and not with vestibular sedatives.[78]

AT THE BEDSIDE

An older woman, no fall in two years, walks with a widened base and short steps, a hand trailing the wall, and steadies the moment a companion offers an arm. The deficit is out of proportion to any weakness: this is the cautious gait of fear of falling,[73] not a focal lesion.[1] Treat the fear, and balance training or Tai Chi reduces it without raising the fall rate.[75]

The cautious gait is an intelligent adaptation, not a defect; it becomes the problem only when the fear outruns the real risk. Naming the fear, and treating it, is often the treatment for the walk.

The gait of the illness

Some gaits are not caused by a drug or a lesion at all; they are the illness, made visible in how the body moves. Psychomotor disturbance is a core, objectively measurable feature of depression with high discriminative validity, and it may be the single symptom domain that best separates depressive subtypes, predicts antidepressant response, and links depression to basal-ganglia and thalamo-cortical circuitry.[45] Psychomotor retardation is the defining feature of the melancholic subtype, mechanistically tied to dopaminergic dysfunction, which is why a depressed gait can look so much like a parkinsonian one.[46]

What the depressed walk looks like. Objective three-dimensional gait analysis gives the depressive walk a measurable signature: reduced velocity, reduced arm swing, reduced vertical head movement, more lateral sway of the upper body, and a more slumped posture.[44] Some of these are not simply by-products of moving slowly: the neck and thoracic flexion, the head-down stoop, are independent of walking speed.[47] The walk is heavy and leaden, the steps short, the arms still, the gaze and the head down.

It tracks mood, and that is the discriminator. The decisive feature for telling this gait apart is that it follows the affect rather than localising to a structure. Inducing a sad mood with music in never-depressed people reproduces the same slowed, reduced-arm-swing, slumped walk, which shows the pattern is a state marker that lifts as the mood recovers.[44] Gait can even act as an objective, contactless marker of the illness: recorded gait features have classified depression against healthy controls with high sensitivity and specificity.[48] But it is not universal: in young adults, gait parameters showed no significant association with affect or depressive severity, so the depressed gait is most reliable where the retardation is genuinely visible, and is read as one sign among many, never as a test.[49]

THE SIGNAL

The depressed gait, and what it is not

It is slow, heavy and stooped, with reduced arm swing and a short stride, and it tracks the mood. It is **not** rigid or cogwheeled, which is what separates it from the drug-induced parkinsonian gait it most resembles, and it does **not** localise, which separates it from a lesion. The test that settles it is time: the depressive gait lifts with recovery, where a drug effect needs the drug changed and a lesion stays put.

QUICK CHECK

A depressed inpatient walks slowly, stooped, with the arms still. What single bedside finding separates this from the drug-induced parkinsonian gait it mimics?

A Tone: the depressed gait carries no rigidity or cogwheeling and it tracks the mood, reversing with recovery; the drug-induced gait is rigid and needs the drug changed.[44,46]

The other gait that is the illness itself belongs to catatonia, a distinct and recognisable psychomotor syndrome whose motor signs can be reliably rated with the Bush-Francis Catatonia Rating Scale, the preferred instrument for the purpose.[50,52] Its disturbance of stance and gait is one part of a broader picture that runs from immobility and stupor at one pole to agitation at the other, with posturing, negativism, gegenhalten (an oppositional resistance to movement), mannerisms, stereotypies and ambitendency.[50] When such a patient walks at all, the gait is stilted, halting and odd, with freezing and held postures rather than a clean weakness or a clean ataxia. It does not localise, because it is not a lesion; it is a state.

Common, and missed. Catatonia is found in 5 to 18% of psychiatric inpatients and around 3.3% of medical inpatients, yet it is frequently unrecognised and carries a risk of life-threatening complications.[58] Using the same instrument, it was identified in 13.5% of new admissions in India against 9.6% in Wales, with the classic retarded signs of posturing, catalepsy, staring and stupor more frequent in the Indian sample, and it occurred across many diagnoses, not only schizophrenia.[56] A US national analysis found about 60% of catatonia hospitalisations had a primary psychiatric and 40% a primary neurological or medical diagnosis, which is why the syndrome cannot be assumed to be psychiatric and must be actively looked for.[57]

Why the gait is worth recognising: it is treatable. Benzodiazepines are first-line and electroconvulsive therapy is for refractory and malignant cases.[54] In the foundational protocol, 76% of patients responded to lorazepam, the four who did not responded promptly to ECT, and a positive response to an initial parenteral lorazepam challenge predicted the final response; a 2 mg challenge is a reasonable confirmatory dose.[51,53] The urgency comes from the malignant end of the spectrum: malignant catatonia adds autonomic instability, an abnormal temperature, blood pressure, heart rate and respiratory rate, is life-threatening, and is easily mistaken for, and overlaps with, neuroleptic malignant syndrome, which is why an antipsychotic is the wrong reflex for catatonic immobility.[55]

A stilted, posturing or freezing gait is a prompt to examine for the other catatonic signs and, where they are present, to consider a lorazepam challenge, not to escalate an antipsychotic for the slowness.

13.5 vs 9.6% **Catatonia in new admissions, India against Wales,** on the same Bush-Francis instrument.[56] It runs at 5 to 18% of psychiatric inpatients and is routinely missed, yet it is treatable, which is why a stilted, posturing gait is worth recognising.[58]

09 The functional gait

The functional gait is the one most often handled badly, and it is the one a psychiatrist is best placed to handle well. A functional gait disorder is genuine, involuntary and disabling, and it is a positive diagnosis. Functional neurological disorder, including functional gait, is diagnosed by recognisable positive patterns of genuinely experienced signs that vary within and between tasks over time, not by the absence of organic disease.[59] It is part of a broader neuropsychiatric syndrome, in which the constant motor presentations, weakness, fixed posturing and gait disorder, cluster together; it is not a performance, and the older language of hysteria has no place in it.[67] The diagnosis should rest on clear positive evidence from the examination or the nature of the events, not on a normal scan.[66]

The signs that rule it in. The bedside tests with the most evidence for functional limb weakness and gait are Hoover's sign, the hip-abductor sign, drift without pronation, the dragging monoplegic gait and give-way weakness, and the hallmark uniting them is internal inconsistency produced by abnormally focused attention.[62] Hoover's sign captures this exactly: hip extension that is weak on direct command returns to normal strength involuntarily when the opposite hip is flexed against resistance.[65] For stance and gait, the classic six features, momentary fluctuation with suggestion, an excessive slowness incompatible with disease, a psychogenic Romberg that improves with distraction, uneconomic energy-wasting postures, a walking-on-ice gait with fixed ankles, and sudden knee buckling that usually does not end in a fall, identified the disorder in 97% of a video series.[60] Positive signs such as Hoover's and tremor entrainment are rule-in findings that satisfy the diagnostic criteria, and the clinical history alone should not be used to make the diagnosis.[64]

TABLE 3 **The positive signs of a functional gait, and how to elicit them**

Sign	How to elicit it	What a positive result shows
Hoover's sign	Test hip extension on command (weak), then again as the patient flexes the opposite hip against resistance	Extension power returns involuntarily, so the weakness is functional, not organic[65]
Dragging monoplegic gait	Watch how the affected leg is moved forward	It is dragged behind as a single unit, hip rotated, rather than circumducted as a spastic leg is[62]
Excessive slowness	Compare the speed of walking to the apparent deficit	A slowness out of all proportion to any weakness, effortful and energy-wasting rather than economical[60]
Walking on ice	Observe stance and stride	Small, stiff-ankled, cautious steps with the arms abducted, yet with balance reactions preserved[60]
Distractibility	Re-examine the gait or a tremor during a competing cognitive or motor task	The abnormal movement lessens, changes or entrains when attention is drawn away[63]
Astasia-abasia	Ask the patient to stand and walk, then test leg power lying on the couch	Cannot stand or walk, yet leg strength is full when tested supine; near-falls are caught[60]
Give-way weakness	Test power with steady, sustained resistance	Strength yields in ratchety, inconsistent jerks rather than as a smooth, fixed weakness[62]

No sign stands alone; the diagnosis is a pattern of positive findings, recorded and repeatable. The figure overleaf sets the functional pattern against the organic one it is most often confused with.

FIGURE 2 **Functional against organic: the signs that separate them**

TELLING FUNCTIONAL FROM ORGANIC AT THE BEDSIDE — RULE IN, NOT OUT

THE SIGN	ORGANIC	FUNCTIONAL
Hoover's sign	Hip extension stays weak whatever else you do	Extension returns when the other hip flexes to resist
The weak leg	Circumducts (spastic), or is high-stepped	Dragged behind as one unit, not circumducted
Consistency	Fixed and reproducible on every test	Inconsistent; eases or changes with distraction
Slowness	In proportion to the weakness you find	Excessive, uneconomic; a walking-on-ice gait
On the couch	Weak lying down as well as standing	Full power lying down, yet cannot stand or walk

The signs are specific, not sensitive: one positive sign rules functional in; **their absence does not rule it out, and the functional and the organic can coexist.**

HOW TO READ THIS Each row is one sign; read across, organic on the left and functional on the right. One positive functional finding rules functional in; its absence does not rule it out, and the two can coexist.

The honest limits. These signs are specific but not sensitive. Across controlled studies their specificity was high (92 to 100%) but their sensitivity was variable and often low (8 to 100%), the evidence is of modest quality, and no single sign should be used in isolation.[61,63] When raters classified functional gait videos against published frameworks, inter-rater agreement was consistently low and no framework captured every case, so the diagnosis is best anchored to discrete, elicitable signs and recorded video rather than a global impression.[68] And functional and organic disorders coexist, so a positive sign rules the functional disorder in without ruling a lesion out.[63]

HOLD THIS

The functional gait is real

A functional gait disorder is genuine, involuntary and disabling, not feigned. It is diagnosed by the positive signs above, never by a normal scan, and the word that fits it is functional, not hysterical, not put on, not supratentorial. Naming it clearly and without judgement, as a real and treatable disorder of how the nervous system runs the movement, is itself part of the treatment. The management is a confident positive diagnosis, a clear explanation the patient can understand, and specialist physiotherapy, which improves motor symptoms and quality of life, though a large recent trial did not meet its primary functional outcome, so the evidence is real but still maturing.[69,70]

Most of the work is done by watching, in a fixed sequence. Watch the natural walk first, for its speed, base, stride length, arm swing, symmetry and the way the patient turns. Then ask for tandem walking, then test Romberg, and where balance is in doubt, the pull test. The findings are not fed into a guess at the cause but into a practical categorisation by clinical pattern and the company the gait keeps, which is the most reliable route through an unfamiliar walk.[3]

The manoeuvres that decide it. A handful of bedside tests carry most of the discriminating weight. The pull, or retropulsion, test is best done as a single unexpected shoulder pull with the patient forewarned; taking more than two steps backwards, or having to be caught, is abnormal, and the test is reliably rated between examiners.[71] Romberg separates the two ataxias: a cerebellar gait is only modestly worse with the eyes shut, whereas a sensory, dorsal-column ataxia is markedly worse without vision. Tandem walking exposes a mild cerebellar or vestibular deficit the ordinary walk hides. And the single hardest call, drug-induced parkinsonism against idiopathic Parkinson's disease, can be clinically indistinguishable, which is why it turns on a documented drug exposure and, when it must be settled, on dopamine-transporter imaging.[13]

What the gait is worth keeping. An abnormal gait in an older patient is not only a present-tense sign. A neurological gait abnormality nearly doubles the risk of future dementia, and the frontal pattern in particular predicts the vascular kind, so the gait you record today is also a prognosis worth following.[42] The figure overleaf puts the whole sort into one order: the emergency first, the positive functional signs next, then the organic levels, and a focal localising pattern referred on.

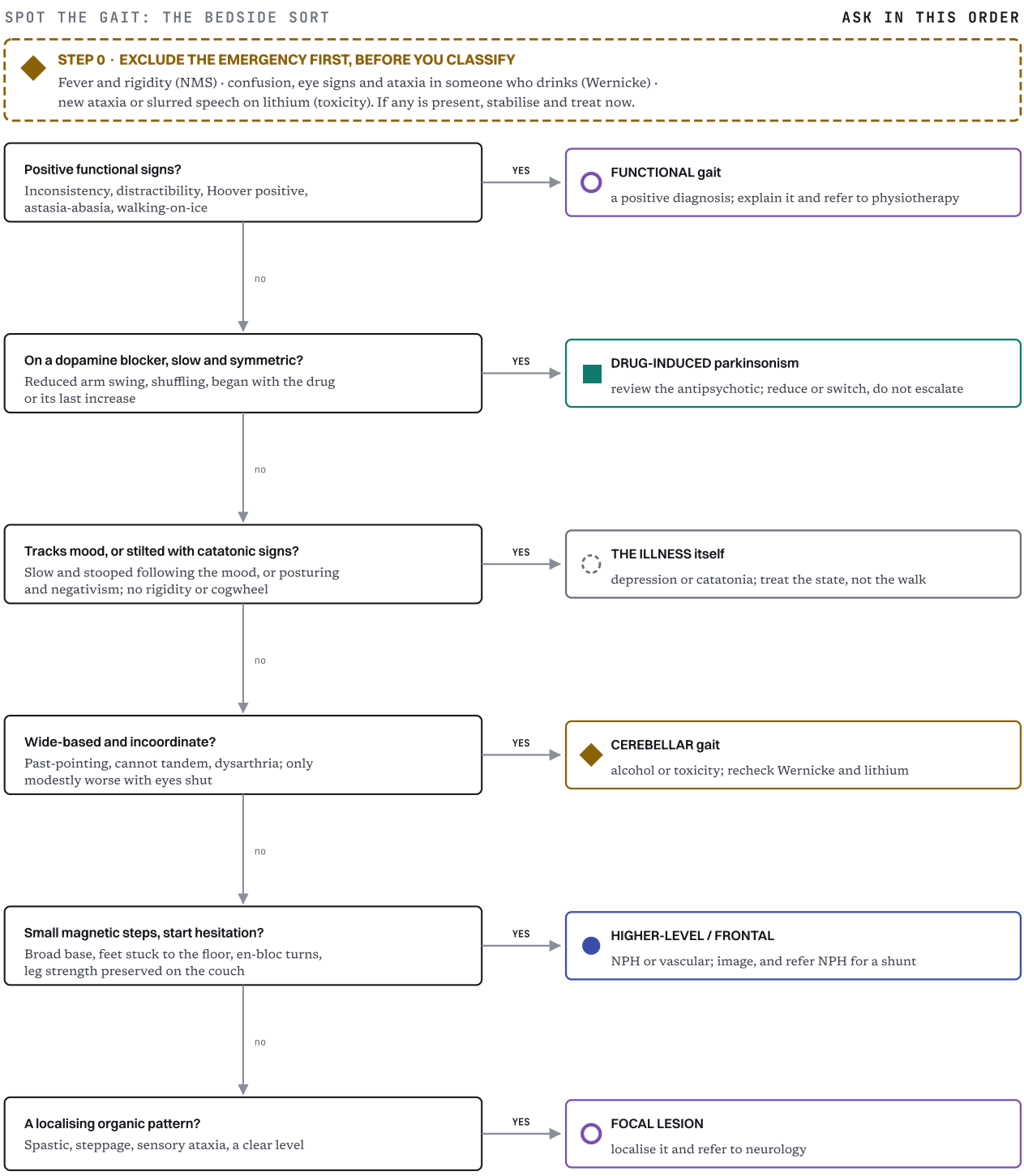
THE EXAM, IN ORDER

- **Watch the natural walk.** Speed, base, stride, arm swing, symmetry and the turn, read before any test.
- **Tandem walk, then Romberg, then the pull test.** Tandem exposes a mild cerebellar or vestibular deficit; Romberg separates a cerebellar from a sensory ataxia; the pull test grades retropulsion.[71]

AT THE BEDSIDE

A man on lithium and an antipsychotic walks wide-based and slurs his words; on Romberg he is only mildly worse with the eyes shut, which points cerebellar rather than sensory. On lithium, a new ataxia is toxicity until excluded, so check the level but act on the gait.[71,13]

FIGURE 3 **Spot the gait: the bedside sort, in safety order**



HOW TO READ THIS Read top to bottom. The dashed box at the top is the emergency gate, checked before anything is classified; then each question in turn, and the first yes names the gait and stops the descent.

The order is a safety order: the emergency first, the functional signs before the organic levels so a real functional disorder is named and not chased, and a focal localising pattern referred. The first yes that fits stops the descent, and any gait that resists the sort is reassessed, not forced into a box.

11 What the walk points to

A gait is a dense diagnostic signal. It localises, naming the level of the nervous system involved. It dates, placing the onset against a drug or an episode. It grades, in the depth of the slowing or the width of the base. And it forecasts: an abnormal gait in an older person nearly doubles the risk of future dementia and points specifically to the vascular kind.[42] Few signs a clinician can gather in the few seconds it takes a patient to cross a room carry as much, and the discipline this issue asks for is simply to spend those seconds on purpose.

The reason to look is sharper than that. A handful of the causes of an abnormal gait are treatable or dangerous, and they are precisely the ones a psychiatrist is positioned to catch and a busy clinic is positioned to miss. Wernicke encephalopathy is reversed by thiamine and is mostly never diagnosed in life. Lithium toxicity is in our own prescription, and its serum level can reassure while the patient deteriorates. Normal-pressure hydrocephalus is surgically reversible and is routinely mistaken for untreatable dementia. The drug-induced gait is corrected by changing a drug we chose. None of these needs a neurologist to suspect. Each needs only the habit of asking, before the gait is filed under anything else, whether it is one of the ones that cannot wait.

DON'T MISS

The gaits that are reversible, or dangerous, or both

01 **Wernicke encephalopathy.** A wide-based, unsteady gait in anyone who uses alcohol. Give parenteral thiamine before glucose, on two of the four Caine criteria, without waiting for confusion or eye signs.

02 **Lithium toxicity.** New ataxia, slurred speech or an unsteady walk in anyone on lithium. Check the level, but treat the gait: the concentration can be in range and still be toxic, and the deficit can persist.

03 **Neuroleptic malignant syndrome.** Fever and rigidity with an immobile, stiff state that overlaps catatonia. Stop the antipsychotic and treat the emergency; do not read the rigidity as a movement side effect to be medicated.

04 **Normal-pressure hydrocephalus.** A broad-based, magnetic, small-stepped gait with cognitive slowing and urinary urgency. Refer for shunt assessment rather than filing it as decline.

05 **The drug-induced gait.** A symmetric, reduced-arm-swing shuffle that began with the drug or its last increase. Review the antipsychotic before anything else.

12 Managing it

Management follows one rule, and it is the same as the diagnostic order: treat the cause, not the gait. Exclude and treat the emergency first; then manage the walk by what it actually is. Adjust the drug for a drug-induced gait, treat the state for a depressed or a catatonic one, take the positive-diagnosis-and-physiotherapy route for a functional one, and refer the focal lesion. The walk is the readout; the cause is the target, and chasing the readout, escalating a sedative for an unsteady walk, medicating an immobility that is catatonia, is how harm is done.

Falls prevention runs underneath all of it, and here the evidence is clear and unusually flattering to the psychiatrist. Exercise reduces the rate of falls by about 23% and the number of people who fall by about 15%, with balance and functional training the active ingredient and Tai Chi a proven option.[79] A structured multifactorial assessment that includes a medication review reduces falls.[80] And the single largest effect of any falls-prevention trial came not from a physiotherapist but from a prescriber: weaning psychotropic users off their drugs cut falls by two-thirds, which makes deprescribing the highest-leverage falls intervention a psychiatrist personally controls.[81]

In Indian practice the weighting shifts. The burden of alcohol-use disorder is large and under-served, with treatment gaps for substance-use disorders reported near 89% in survey data and a strong association between alcohol-use disorder and suicidality, so the alcohol-related gaits, intoxication, cerebellar degeneration and Wernicke, are both commoner and more often missed.[82,83] Catatonia is at least as frequent in Indian admissions as in Western ones.[56] Trihexyphenidyl is the near-universal answer to drug-induced extrapyramidal effects, and limited access to gait laboratories and neurology means the bedside examination carries even more of the diagnostic load.

TABLE 4 **By cause: the first move, and who to involve**

Cause	The first move	Who to involve
Wernicke	Parenteral thiamine before glucose, on two of the four Caine criteria	Medicine; neurology if not improving
Lithium toxicity	Hold lithium and check the level; supportive care; dialysis if severe	Nephrology / toxicology
Drug-induced parkinsonism	Reduce or switch the antipsychotic; short-term anticholinergic	Neurology if asymmetric or persistent
Tardive gait	Switch the agent where possible; a VMAT2 inhibitor; avoid anticholinergics	Movement-disorder neurology
Catatonia	Lorazepam challenge, then a benzodiazepine; ECT if refractory or malignant	ECT service; medicine if malignant
Depression	Treat the depressive episode; the gait follows the mood	None; reverses with recovery
Functional gait	A positive diagnosis, a clear explanation, specialist physiotherapy	Functional-disorder service, physiotherapy
Cautious / fear of falling	Treat the anxiety; balance training or Tai Chi	Physiotherapy, falls service
NPH	Refer on the gait response to a CSF tap	Neurosurgery
Frontal / vascular gait	Vascular risk reduction; physiotherapy	Neurology, falls service

FIGURE 4 **The management pathway: act in this order, prevent falls throughout**

HOW TO READ THIS Read top to bottom as a priority order; each numbered box is one action. The dashed band at the foot is the falls-prevention layer that runs under every step, not a final one.

The numbered steps are an order of priority, not a queue: the emergency is always first and falls prevention is always present. A patient may need more than one step, and the right answer to a mixed gait is to work the steps that apply, in order.

13 Ways to get it wrong

Stopping at the first reading. The commonest error with a psychiatric patient's gait is to attribute it to the obvious cause, the antipsychotic, the depression, the drink, and to stop there. That reflex hides Wernicke behind drunkenness, lithium toxicity behind a little unsteadiness, normal-pressure hydrocephalus behind getting old, and neuroleptic malignant syndrome behind an extrapyramidal side effect. The obvious cause is often the real one, but it is never the first thing to confirm. The corrective is the safety order: exclude the treatable emergency before you accept the easy explanation.

Calling it functional too soon, or with contempt. The opposite error is to reach for functional, or worse for put on, for any gait that looks odd or refuses to fit. This fails twice. It can miss a real organic disorder, because functional and organic disorders coexist and a positive functional sign does not exclude a lesion.[63] And it mistreats a real functional disorder, which is genuine, involuntary and diagnosed by positive signs, not by the absence of a lesion or by a clinician's irritation. The corrective is to rule a functional gait in on its own signs, never to reach for it as a verdict of exclusion.

Treating the gait instead of the cause. The third error is to act on the readout. To escalate a sedative for an unsteady walk and worsen the falls. To add an anticholinergic to a tardive gait and worsen the movement. To medicate a catatonic immobility with an antipsychotic and risk malignant catatonia. To investigate a functional gait without end while withholding the explanation that is itself the treatment. Each treats the visible sign and leaves the cause untouched. The corrective is the management rule, unchanged from the first page: treat the cause, not the walk.

QUICK CHECK

A positive Hoover sign is found. Does it exclude a stroke?

A No: functional and organic disorders coexist, so a positive functional sign rules a functional gait in without ruling a lesion out.[63]

HOLD THIS

The one lesson, if you keep nothing else

An abnormal gait in a psychiatric patient is a sign with several readings, and the danger is in stopping at the first. Exclude the treatable emergency, rule a functional disorder in on its positive signs rather than out by exclusion, and treat the cause, not the walk. Read carefully, the gait is one of the most useful signs in psychiatry, it localises, it dates, it grades, it forecasts. Read lazily, it is one of the most dangerous, because the things it hides are the things that could have been fixed.

14 Before they walk out

A gait is read before a word is spoken, and almost all of the skill is in not stopping there: classifying the walk, telling the look-alikes apart, knowing what each points to, and acting on the cause rather than the sign.

WHAT THIS ISSUE COMES DOWN TO

1. **Classify it twice, and notice when it points nowhere.** Name the pattern by what it looks like, and read the company it keeps to find the level. The gaits a psychiatrist meets most, the functional, the depressed, the catatonic, are the ones that do not localise at all, and recognising that is the move that separates a clinician's reading from a checklist.

2. **Two drug-induced gaits, opposite in everything.** Drug-induced parkinsonism is early, hypokinetic, symmetric and eased by an anticholinergic; the tardive gait is late, hyperkinetic and worsened by one. When the gait began relative to the drug, and whether the movement is too little or too much, settles which it is.

3. **The cerebellar gait is an emergency until proven otherwise.** A wide-based, unsteady walk in anyone who uses alcohol is Wernicke until excluded; a new ataxia or slurred speech on lithium is toxicity, and a therapeutic level can reassure when it should not. Treat both on suspicion.

4. **Rule functional in, treat the cause, prevent the fall.** A functional gait is genuine and diagnosed by positive signs, never by exclusion or contempt. Treat the cause rather than the readout, and deprescribe the sedatives, the falls a psychiatrist is best placed to prevent.

REMEMBER

EMERGENCY · FUNCTIONAL · DRUG · ILLNESS · LOCALISE

The order of the bedside sort: exclude the treatable emergency first, rule a functional gait in on its positive signs, then the drug-induced gait, then the gait of the illness, and refer a focal localising lesion. One safety order, and the whole issue.

BEDSIDE CHECK

A patient walks in differently

01 Is this an emergency? Fever and rigidity, confusion with eye signs and ataxia, or a new ataxia on lithium. Stabilise and treat that first.

02 Are there positive functional signs? Inconsistency, distractibility, Hoover, astasia-abasia. If so, this is a functional disorder to be named and explained, not chased.

03 Did it begin with a drug, and is it symmetric and slow? Review the antipsychotic before anything else.

04 Does it track the mood, or come with catatonic signs? Treat the state; do not escalate a drug for the slowness.

05 Does it localise, wide-based, magnetic, spastic or high-stepped? Place the level, refer what needs referring, and prevent the fall throughout.

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Every reference was verified through PubMed before publication. Antipsychotic-induced movement disorders and the extrapyramidal onset timeline are covered in Aporia 09; treatment resistance and clozapine in Aporia 08. The Caine criteria, the Hakim triad and the Bush-Francis scale are cited as primary clinical instruments; Indian service-reality detail reflects current practice and national survey data rather than a single trial.

Educational note. This clinical-reasoning essay is for clinicians and students. It is educational, not treatment advice for any individual. Care decisions belong with the treating clinician. At Weave, care is led by Dr. Niharika Reddy, Consultant Psychiatrist.

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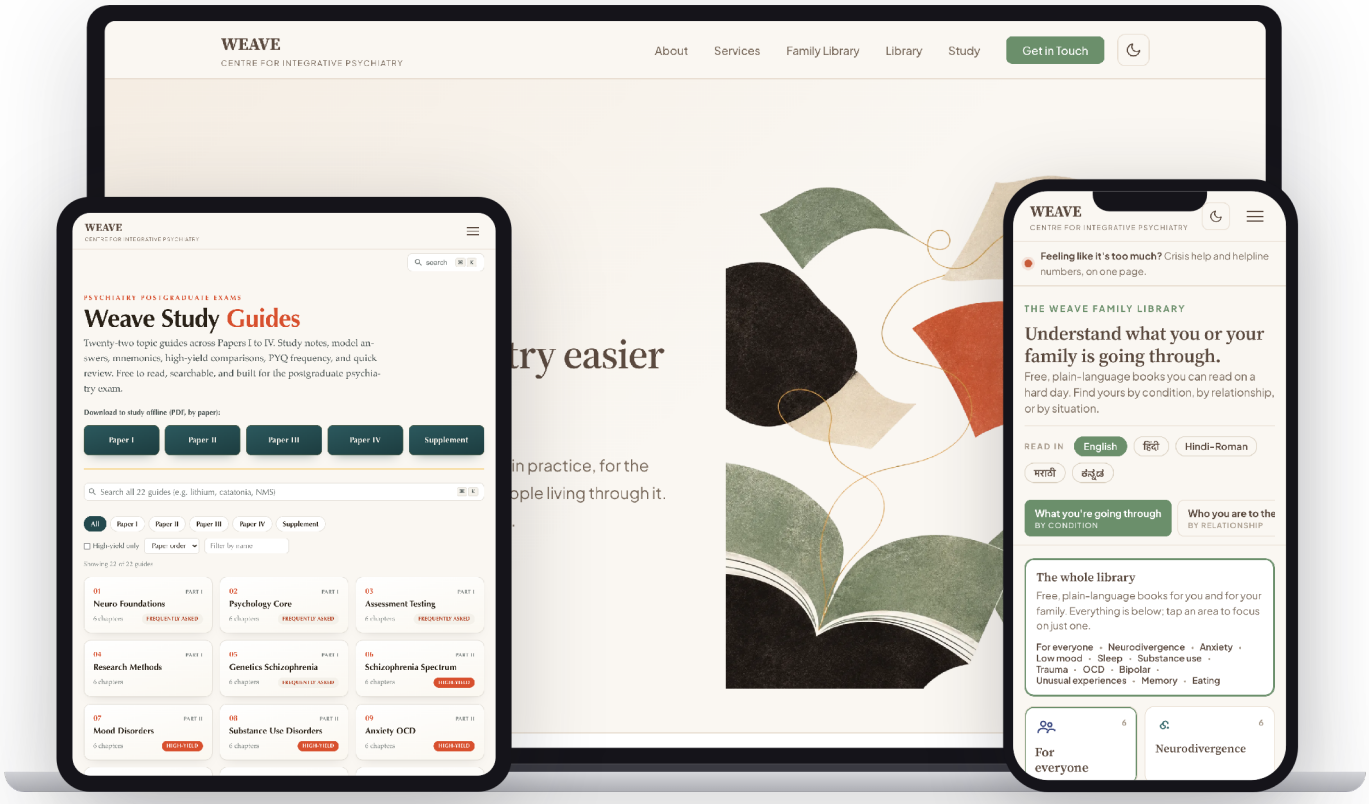

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