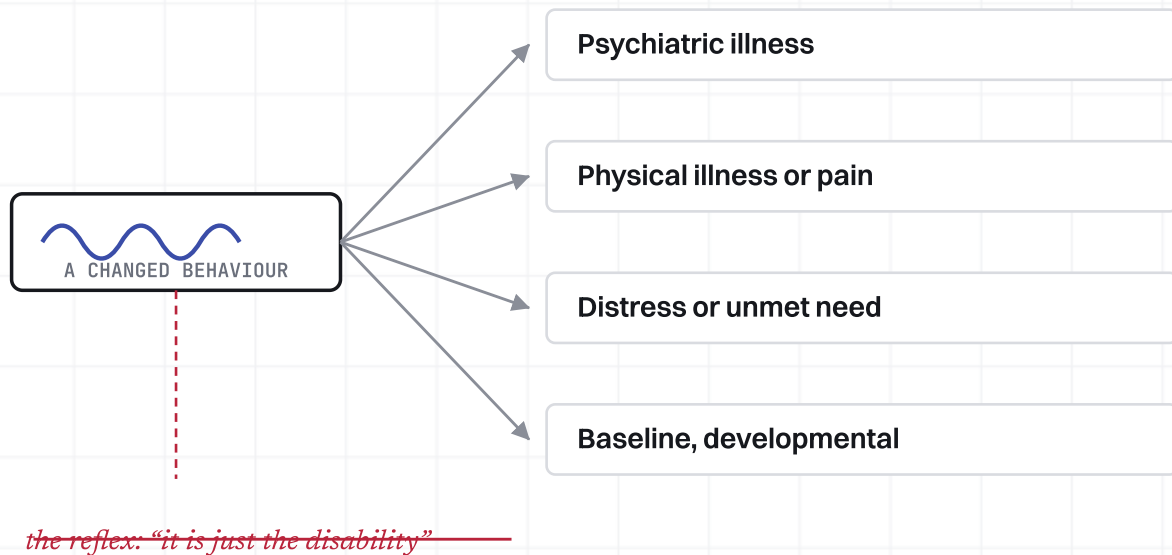




When Words Fail.

In intellectual disability, psychiatric illness is common, treatable, and routinely missed. *It is missed because it is shown rather than spoken, and because the first reflex is to blame the disability. The skill is reading the behaviour for what it is.*

A man with a moderate intellectual disability, settled for years, begins to hit himself and refuse food. The chart offers a ready explanation, the one always within reach: it is the disability. That sentence ends the assessment before it starts, and it is wrong often enough to be dangerous. A changed behaviour in a person who cannot easily say what is wrong is not a diagnosis. It is a signal with at least three readings, and the work is telling them apart.

The readings compete. The behaviour may be a new psychiatric illness, expressed as conduct because the words for it are not available. It may be a physical illness or pain, a toothache, constipation, an ear infection, reflux, a fracture, speaking through the only channel left. It may be distress or an unmet need, a change of staff, a loss, boredom, fear, or a demand that cannot be met. Or it may be near the person's baseline, a lifelong pattern now merely more visible. Psychiatric illness in intellectual disability is common, treatable, and routinely missed, and it is missed for reasons that are predictable enough to be designed around.^{1,2}

WHY STANDARD PSYCHIATRY MIS-FIRES HERE

- **The criteria assume a narrator.** Most of our diagnostic anchors, low mood reported, worthlessness, racing thoughts, delusional content, were written for patients who can describe an inner world. As intellectual disability deepens, that self-report thins and then vanishes, so the standard manuals undercount: in the landmark population study the point prevalence of mental ill-health was 40.9% by clinical judgement but only 15.7% by DSM-IV-TR, with the disability-specific criteria capturing more than twice as much.¹
- **The illness is shown, not spoken.** Limited abstraction, a sparse emotional vocabulary, and constrained communication mean distress surfaces as behaviour. The clinician reads conduct and must infer the state behind it, which is why a careful behavioural baseline, and an informant who knows the person, outrank any single interview.^{3,7}
- **The reflex is to blame the disability.** The single most studied error in this field is to attribute a symptom to the intellectual disability itself, so a coexisting and treatable illness is never sought. It has a name, diagnostic overshadowing, and the rest of this issue is built to counter it.²

THE CONCEPT CODE USED THROUGHOUT: SEVERITY BY ADAPTIVE FUNCTION

BY SEVERITY

- **Mild** most can self-report, with care
- **Moderate** mixed report and observation
- ◆ **Severe** behaviour-led, informant
- **Profound** wholly observational

Each colour is also a shape, so the code survives a greyscale photocopy. Severity is the axis that governs everything that follows: the deeper the disability, the further assessment shifts from what the person says to what they do and what those around them observe. The figure overleaf places the four bands on that single continuum.

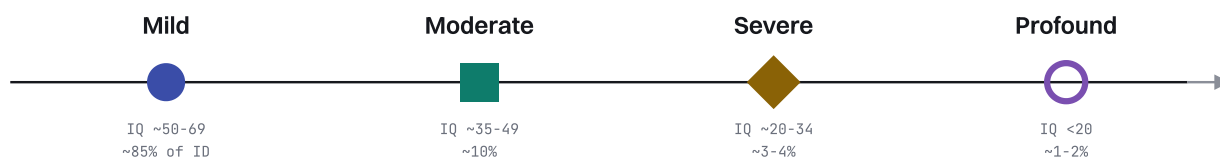
Intellectual disability is defined by three things together: deficits in intellectual functioning, deficits in adaptive functioning across the conceptual, social and practical domains, and onset in the developmental period. The decisive modern move, made explicit in DSM-5 and in ICD-11 (which renames the condition disorders of intellectual development), is away from a single IQ cut-off and toward adaptive function as the basis for grading severity. A WHO-led panel built behavioural indicators of adaptive function for exactly this purpose, to let a clinician grade severity by what a person can do where standardised testing is unavailable, and an international field study including two Indian sites found them reliable and usable across all severity bands.^{8,9} The IQ bands have not disappeared, but they now sit alongside the adaptive picture rather than defining it.¹⁰

Aetiology, in brief. Causes group into genetic and chromosomal, prenatal (infection, toxins, alcohol), perinatal (hypoxia, prematurity) and postnatal (infection, trauma, malnutrition), with a once-large idiopathic fraction now shrinking as microarray and sequencing reclassify it.¹¹ Clinical genomic testing yields a diagnosis in roughly a quarter of cases on pooled data, more with exome or genome testing, but the same review found no sequencing studies from India, Africa or Latin America, a stark reminder that aetiological work-up is a privilege of resource.¹² Intellectual disability is more prevalent in low- and middle-income countries, near 10.4 per 1000 worldwide on the best meta-analysis, which was led from an Indian group.¹³

FIGURE 1 The severity spine: one axis governs the whole assessment

GRADED BY ADAPTIVE FUNCTION, NOT IQ ALONE

SEVERITY SPINE



HOW YOU ASSESS, ACROSS THE SPINE

Self-report, cross-checked

→ Wholly behavioural and informant-based

As severity increases the population share falls and the words available fall with it.

The deeper the disability, the more a behaviour change is the only symptom you will get.

HOW TO READ THIS Walk left to right; as the band deepens the population share falls and the symptom channel slides from what the person says to what they do.

Population shares are the conventional textbook proportions; the point is the order and the gradient, not the exact percentage. Borderline intellectual functioning (IQ about 70 to 85) sits just above the threshold and carries raised psychiatric and adaptive risk without meeting criteria for the disability.¹⁰

The severity band is not bureaucracy. It predicts how much a person can tell you, and therefore how a psychiatric illness will look. Read the table down a column to picture the person; read it across a row to see how the same dimension thins from mild to profound. The clinical consequence is in the last row: the channel through which illness will reach you.

TABLE 1 **Intellectual disability, graded by adaptive function**

DIMENSION	● Mild	■ Moderate	◆ Severe	○ Profound
IQ BAND	~50 to 69	~35 to 49	~20 to 34	below ~20
CONCEPTUAL	Functional academics; difficulty with abstraction, money, time	Slow, limited academics; concrete thinking	Little academic skill; understands the here and now	Symbolic concepts largely absent
SOCIAL AND COMMUNICATION	Conversational; immature judgement, suggestible	Simple speech; relationships with support	Single words or signs; communicates needs	Non-symbolic; gesture, expression, behaviour
PRACTICAL AND DAILY LIVING	Independent with support at stress points	Self-care with prompting and time	Help with all daily activities	Dependent for all care; co-occurring sensory or motor impairment common
SYMPTOM CHANNEL	Self-report, cross-checked with an informant	Mixed: report plus observed change	Behaviour-led; informant central	Wholly observational

The deficit profile is the engine. Restricted abstraction, a thin emotional vocabulary, weak time concepts and limited theory of mind mean that an inner state cannot be packaged as a complaint. Add the high rate of co-occurring sensory and physical impairment, which constrains emotional development further, and the result is the recurring fact of this issue: distress is expressed as behaviour, and the assessment must be built to read it.¹⁴

BEHAVIOURAL PHENOTYPES WORTH KNOWING (A BRIEF NOTE)

- **Down syndrome:** near-universal Alzheimer neuropathology with age; a clinical dementia rate above 30% in adults over 36, so a loss of skills in mid-life is screened for dementia, not assumed to be behaviour.¹⁵
- **Fragile X:** the commonest inherited cause of intellectual disability and a leading genetic cause of autism, carrying anxiety, ADHD, hyperarousal and sensory hypersensitivity across the lifespan.¹⁶
- **Prader-Willi:** hyperphagia and a distinctive behavioural profile; the maternal uniparental disomy subtype carries a notably raised risk of psychosis.¹⁷

The phenotype reshapes which illness to screen for. It does not replace the assessment; it weights it.

Psychiatric comorbidity in intellectual disability is the rule, not the exception. The reported range is enormous, from 7% to 97% across studies, because everything depends on case definition and on whether behaviour that challenges is counted.¹ The defensible headline is that any co-occurring psychiatric disorder runs at roughly a third on pooled meta-analysis in adults, around 34%, and 38 to 49% in children, both well above general-population rates though with wide between-study variation.^{20,21} The child total-population work found over 40% with a significant disorder, yet fewer than one in ten with a major disorder had received specialist help.^{18,19} Rates are not flat across cause: adults with Down syndrome carry less mental ill-health than other adults with intellectual disability, a useful caution against assuming a uniform dose-response.²²

The under-count is the message. In the same population the standard manuals saw less than half of what a clinician using disability-adapted criteria saw. Three predictable errors stand between the clinician and the diagnosis, and naming them is half the cure.¹

TRAP 1: DIAGNOSTIC OVERSHADOWING

The reflex to attribute a symptom to the disability itself, so a coexisting illness is never sought. It was demonstrated experimentally: the identical phobia was judged less likely to be an emotional disorder once the person was also labelled as having an intellectual disability, and the same held for psychosis and personality-disorder vignettes.² It is not abolished by experience, which marks it as a structural bias rather than a training gap.⁴ The modern systematic review tempers the absolutism, one study in three found no overshadowing and the evidence is of low quality, so the right posture is not fatalism but vigilance: treat overshadowing as a high prior to be actively countered, not an inevitability.⁵

TRAP 2: BEHAVIOURAL EQUIVALENTS, USED WITH CARE

Because symptoms are shown not spoken, a psychiatric illness often surfaces as a change in behaviour, and the disability-specific frameworks allow such behavioural equivalents to stand in for criteria that require words. This is real and useful, but it is contested, and the caution is important: challenging behaviours are robustly associated with depression yet should not be uncritically relabelled as depressive equivalents, because the same behaviours used to make the diagnosis are then used to confirm it, a circularity that inflates everything.^{6,7,23} The safe rule: a behaviour change is a red flag that prompts assessment, never a diagnosis in itself.

TRAP 3: SOVNER'S FOUR FACTORS

Sovner named four ways the criteria distort in intellectual disability, and they remain the field's working lens.³

- **Intellectual distortion:** concrete thinking limits the reporting of abstract internal states.
- **Psychosocial masking:** limited life experience flattens symptom content, so a delusion may be simple or a manic grandiosity unsophisticated.
- **Cognitive disintegration:** stress-induced decompensation produces disorganised behaviour that can be mistaken for psychosis.
- **Baseline exaggeration:** illness amplifies the pre-existing behavioural baseline rather than adding textbook symptoms, which is why a recorded baseline is the comparator that lets you see the change.

Each row is a disorder; the columns move from the textbook picture, to how it changes when the patient cannot narrate it, to the most useful adaptation for finding it. The sections that follow expand each row and carry the citations.

TABLE 2 How each disorder presents differently in intellectual disability

Disorder	IN THE GENERAL POPULATION	HOW IT LOOKS DIFFERENT IN IDD	THE ASSESSMENT ADAPTATION
Depression MOOD	Reported low mood, anhedonia, guilt, hopelessness, suicidal thoughts	Loss of acquired skills, social withdrawal, irritability and aggression, somatic and sleep change, new or worsening challenging behaviour; low mood inferred, not reported	Observed change against baseline; informant report; GDS-LD with carer supplement; weight behavioural signs more as severity deepens
Bipolar and mania MOOD	Elated or irritable mood, grandiosity, reduced sleep, pressured speech, risk-taking	Overactivity, irritability, reduced sleep, behavioural escalation and the most pronounced challenging behaviour of any group; elation and grandiosity often unsophisticated; rapid cycling reported more	Observed overactivity and sleep loss over reported elation; track episodicity and seasonality; informant timeline
Anxiety INTERNALISING	Reported worry, fear, physical arousal, situational avoidance	Avoidance, agitation, increased vocalisation, somatic complaints and challenging behaviour, often triggered by change to routine; intolerance of uncertainty prominent	Establish behavioural baseline; rule out pain first; GAS-ID where mild, ADAMS subscale by informant otherwise
OCD INTERNALISING	Egodystonic obsessions; compulsions resisted, performed to reduce anxiety	Egodystonia and inner resistance unassessable at lower verbal levels; compulsions may lack a reported obsession; overlaps stereotypy and autistic sameness	Differentiate by function, not topography: compulsions follow visible anxiety and bring relief; stereotypy is self-soothing at baseline arousal
Psychosis PSYCHOTIC	Hallucinations, delusions, formal thought disorder, reported first-rank symptoms	Reliable diagnosis needs a verbal level above moderate ID; below it, first-rank symptoms cannot be elicited and self-talk, fantasy or fear are misread; content is simple	Longitudinal, informant-based; separate from developmentally expected phenomena; PAS-ADD interview; do not over-call psychosis NOS
ADHD NEURODEVELOPMENTAL	Inattention, hyperactivity and impulsivity excessive for chronological age	Among the commonest comorbidities in children with IDD, near 30%; the judgement is whether symptoms exceed the developmental (mental) age, not the chronological one	Benchmark against mental age and baseline; informant ratings across settings; expect smaller stimulant effect and more side effects
Autism overlap NEURODEVELOPMENTAL	Social-communication deficit and restricted, repetitive behaviour	Co-occurs with IDD very often; the question is whether social-communication impairment and repetitive behaviour are out of proportion to the global delay, not merely delayed	Compare social function to developmental level; collateral developmental history; distinguish repetitive behaviour from compulsion
Behaviour that challenges FINAL PATHWAY	Not a diagnosis; in the general population framed as a presenting problem	A description, not a disorder; the same form serves different functions; a final common pathway for illness, pain, communication failure and environment	Functional analysis (escape, attention or tangible, sensory); search for an underlying cause before any drug
Self-injury FINAL PATHWAY	Often linked to emotional regulation or personality difficulty	Can be a behavioural equivalent of untreated pain or psychiatric illness; markedly elevated in specific genetic syndromes; head-banging, biting, skin-picking, eye-poking	Treat as a signal: examine for pain and physical illness; functional analysis; consider syndromic phenotype
Dementia LATE-LIFE	Progressive memory and functional decline, typically later in life	Earlier onset, especially in Down syndrome where Alzheimer pathology is near-universal; presents as loss of established skills and new behaviour, not reported forgetfulness	Compare to a documented earlier baseline; screen Down syndrome from the late 30s; exclude reversible causes first

The first column is the trap, the second the truth, the third the move. The thread through the third column is informant history plus a behavioural baseline.

Depression is among the most common and most missable comorbidities. The prospective population data give a two-year incidence of 7.2%, broadly comparable to the general population, but the presentation diverges sharply, and the strongest single predictor of a new depressive episode in that cohort was the presence of challenging behaviour, which more than doubled the odds.²⁴ The lesson is in that statistic: in intellectual disability, depression often arrives wearing the clothes of behaviour.

How the picture changes with severity. In mild disability the textbook features survive, with low mood and anhedonia reportable if asked plainly. As disability deepens, the presentation shifts toward observed signs: a loss of acquired adaptive skills, social withdrawal, irritability and aggression, disturbed sleep and appetite, and the onset or worsening of challenging behaviour.²⁵ The most rigorous synthesis in severe-to-profound disability confirms that the core features, depressed affect and anhedonia, do still relate to depression alongside irritability, sleep disturbance, psychomotor change and appetite loss, but it cautions firmly against treating aggression, screaming and self-injury as depressive equivalents in their own right, because pain, autism and other affective disorders confound them.⁷ The clinic data make the practical point bluntly: sad mood, crying and anhedonia are what separate depression from everything else, yet most patients with intellectual disability cannot meet the required count of standard or even adapted criteria, so the diagnosis leans on observed change against a known baseline.²⁶

THE SIGNAL**What a depressive episode looks like when it cannot be described**

A person who was managing now loses skills they had. They withdraw from things they sought out. They are irritable or aggressive where they were not. They sleep and eat differently, and a settled behaviour becomes disturbed. None of these is specific alone; together, against a documented baseline and over weeks, they are how depression speaks here. The reflex to read it as the disability, or to escalate an antipsychotic for the agitation, is the error to resist.

Suicidality is reported by few patients with significant disability, but it is not absent; it must be asked about directly and in plain language, and a documented decline in self-care or new self-injury can be its only sign.^{26,23}

2×

Challenging behaviour more than doubled the odds of a new depressive episode in the prospective cohort, the strongest of the reported predictors.²⁴ In intellectual disability, depression often arrives wearing the clothes of behaviour.

Mania is the quietly remarkable finding of the incidence data. In the prospective cohort, first-episode mania occurred at a standardised incidence ratio above 40 compared with the general population, a relative excess on the order of schizophrenia, and yet, as the authors note, it has received remarkably little attention.²⁴ Part of the reason is that elated mood and grandiosity, the features clinicians watch for, are exactly the ones that the disability flattens and that overactive, irritable behaviour obscures.

What mania looks like here. Reliance shifts from reported elation to observed state: overactivity, irritability, a reduced need for sleep, increased and pressured vocalisation, and behavioural escalation. In the comparative clinic series, patients with bipolar disorder were set apart from those with depression by elevated mood, acute anger, increased speech, sexual themes and increased appetite, and they showed the most pronounced challenging behaviour of any diagnostic group.²⁶ The single most useful instrument is not a scale but a timeline: episodicity, a change from a known baseline, and any seasonality are what convert a confusing behavioural picture into a recognisable affective one.

TWO PRACTICAL CAUTIONS

- **Do not confuse mania with ADHD or with challenging behaviour.** Overactivity and impulsivity are shared; episodicity is not. A discrete change from baseline, with reduced sleep and elevated or irritable mood, points to mania, where a lifelong, non-episodic pattern points to ADHD or to behaviour with an environmental function.²⁴
- **Rapid cycling may behave differently.** On a small and dated evidence base, a systematic review suggests rapid-cycling bipolar disorder in intellectual disability may differ from the general-population form, with a male preponderance, earlier onset, and a poorer response to lithium prophylaxis, worth holding when prophylaxis is being planned.²⁷

High baseline mood-stabiliser use alongside high mania incidence in the cohort suggests enduring, under-treated illness rather than over-treatment, an unusual direction for this population and a reason to hold mania actively in mind.²⁴

SIR > 40

First-episode mania ran at a standardised incidence ratio above 40 versus the general population, a relative excess on the order of schizophrenia, yet it draws remarkably little attention.²⁴

QUICK CHECK

Overactivity and impulsivity are shared by mania, ADHD and environmentally-driven behaviour. What single feature separates mania?

- A Episodicity. A discrete change from baseline, with reduced sleep and elevated or irritable mood, points to mania; a lifelong, non-episodic pattern points to ADHD or to behaviour with an environmental function.²⁴

Anxiety. Diagnosable anxiety disorders run at around 3.8% by formal criteria in adults with intellectual disability, generalised anxiety commonest, though this almost certainly undercounts because the defining experience is internal.²⁸ The presentation is behavioural: avoidance, agitation, increased vocalisation, somatic complaints and behaviour that challenges, frequently set off by a change to routine. Carers and clinicians of minimally verbal people converge on the same heuristics, know the person, establish their behavioural baseline, and actively rule out other sources of distress, especially pain, before settling on anxiety.³⁰ Anxiety here also takes forms beyond the standard categories, notably intolerance of uncertainty, which persists across the lifespan and is easily missed.²⁹ Where mild disability permits, the Glasgow Anxiety Scale is a validated self-report; otherwise an informant measure such as the ADAMS anxiety subscale carries the assessment.³¹

OCD, and the differentiation that matters. The central task is to separate genuine obsessive-compulsive disorder from stereotypy, restricted and repetitive behaviour, and autistic sameness. The DSM anchors, egodystonia and inner resistance, are unreliable or simply unassessable at lower verbal levels. The foundational work showed compulsions occurring without any recognisable egodystonic quality, and, crucially, that trained raters could reliably distinguish compulsions from stereotypies on observable features while inner-resistance and subjective-distress items added nothing, which argues for a behavioural, externally observable approach.³² The modern review confirms substantial overlap between autistic repetitive behaviour and compulsions at the level of content and intensity, so differentiation rests on function.³³

DISCRIMINATOR**Compulsion or stereotypy, at lower verbal levels**

A compulsion is typically preceded by visible anxiety or agitation, followed by relief, interruptible only at the cost of marked distress, and tied to a feared consequence. A stereotypy or restricted-repetitive behaviour is self-stimulatory and soothing, occurs at baseline arousal, and its interruption brings frustration rather than dread. Read the antecedent and the aftermath, not the movement itself; stereotypic movement disorder is repeatedly misread as tics, autism or compulsion when topography is judged in isolation.

In Indian practice self-report tools need linguistic and cultural adaptation, so informant report plus behavioural baselining is the pragmatic default.³⁴

Psychotic disorders are markedly over-represented, but the size of the excess depends on how cases are found. The careful population study put clinical psychosis at about three times the general-population rate; whole-system register studies, which capture every diagnosed case, report relative risks in the region of eight- to twelve-fold, among the largest psychiatric excesses in this population, and highest in mild disability where the diagnosis is most feasible.^{35,36,20} Both figures are true; the discrepancy is itself the teaching point, because the lower the verbal level, the harder the diagnosis is to make at all.

The verbal floor. A reliable diagnosis of schizophrenia depends on self-reported inner experience: first-rank symptoms, formal thought disorder, the elaboration of a delusion. These require a verbal mental age roughly above that of moderate disability; below it they cannot be elicited or distinguished from developmentally expected behaviour. The consequence is over-diagnosis at the bottom of the range, where one clinical series found that careful review reclassified five of seven psychosis-not-otherwise-specified cases as non-psychotic, and where that residual label carried the worst outcomes.³⁷ Sovner's four factors are the working lens: intellectual distortion and psychosocial masking simplify and flatten any genuine content, while cognitive disintegration, stress-induced disorganisation, produces behaviour that mimics psychosis without being it.³

SEPARATING TRUE PSYCHOSIS FROM ITS MIMICS

- **Self-talk, imaginary companions and fantasy** are developmentally expected at lower mental ages and are not hallucinations or delusions.
- **Fear and odd responses to unseen stimuli** may reflect sensory impairment, pain or anxiety before they reflect psychosis; exclude those first.
- **A discrete change from baseline**, corroborated by an informant and persisting over time, is worth far more than any single cross-sectional sign.

The disability-specific criteria were built precisely because the standard manuals fail here; the DC-LD was validated against expert clinical opinion, and a tiered, parallel patient-and-informant interview such as the PAS-ADD is the structured route to a defensible diagnosis below the verbal floor.^{38,39}

AT THE BEDSIDE

A young man with severe disability, non-verbal, is labelled psychosis-NOS for fearfulness and apparent responding to unseen stimuli. Worked against baseline with an informant, the fear tracks a recent move and an unaddressed ear pain, and the label is withdrawn. In one series, careful review reclassified five of seven psychosis-NOS cases as non-psychotic, and that residual label carried the worst outcomes.³⁷

ADHD. ADHD is among the most prevalent diagnosed psychiatric disorders in children with intellectual disability, at roughly 30%, against a general-population childhood rate near 5 to 7%.^{21, 40} The whole assessment turns on one rule: symptoms must be excessive for the person's developmental, that is mental, age, not their chronological age, because slower-paced attention and higher activity can be developmentally typical for the mental age. Judged against chronological age, almost everyone with significant disability looks inattentive and overactive; judged against developmental age and a known baseline, genuine ADHD separates out.

Treatment response, and a heavier side-effect burden. Stimulants work, but less robustly and with more adverse effects than in typically developing children. The definitive double-blind trial in children with ADHD and intellectual disability found optimally titrated methylphenidate beat placebo, but with effect sizes around half those seen in the general ADHD population and clear sleep, appetite and weight effects.⁴¹ The scoping review confirms methylphenidate and atomoxetine help and are generally tolerated, though the trials are small and few.⁴² Where autism co-occurs, the response is further attenuated by lower IQ and treatment-emergent irritability and mood lability are common, so the rule is start low, titrate slowly, and monitor closely.⁴³

THE AUTISM OVERLAP (A NOTE, NOT A SECTION)

- **They travel together.** Within autism, intellectual disability co-occurs in roughly a quarter and ADHD in another quarter; in population data most children with autism also have an intellectual disability.^{44, 45} The combination is the norm, not the exception, and it raises the rate of every other comorbidity.
- **Differentiate by proportion, not by delay.** Autism is distinguished from intellectual disability alone when the social-communication impairment and the restricted, repetitive behaviour are out of proportion to the global developmental delay, rather than merely tracking it. A delayed child relates in keeping with their mental age; an autistic child does not.
- **The shared trap is overshadowing.** Once one neurodevelopmental label is fixed, new symptoms get folded into it. The systematic review tempers but does not dismiss the bias; structured, informant-based reassessment is the antidote, not assumption.⁵

~30% vs 5-7%

ADHD in children with intellectual disability, against the general-population childhood rate.^{21, 40} Yet optimally-titrated methylphenidate shows effect sizes about half those in general ADHD, with clearer sleep, appetite and weight effects.⁴¹ Judge symptoms against developmental, not chronological, age.

Behaviour that challenges is a description, not a diagnosis. Emerson's foundational work established the principle that anchors everything: there is no reliable mapping from the form of a behaviour to its function. The same aggression or self-injury serves different purposes in different people, and function is more consistent within a person across behaviours than across people who share a behaviour.⁴⁶ It is common, shown by 10 to 15% of people with intellectual disability in contact with services, more often male, clustering in adolescence and young adulthood, and strongly tied to restricted communication, which is the clue to its nature.⁴⁷ It is a final common pathway for illness, pain, an unmet need and a difficult environment, and treating it as a diagnosis to be medicated is the central error of the field.

Functional analysis is the method. Iwata's experimental work resolved the functions of self-injury into three broad classes that remain the working taxonomy: social-negative reinforcement or escape from demands, social-positive reinforcement such as attention or a tangible, and automatic or sensory reinforcement.⁴⁸ Establishing which function is operating is what turns an intractable behaviour into a treatable one. The evidence on outcomes is sobering and clarifying at once: a meta-analysis of 82 trials found only small effect sizes overall and, decisively, no significant difference between drug and non-drug approaches, which is why guidelines place psychological, behavioural and environmental work first.⁴⁹

Self-injury, and the search behind it. Self-injury, head-banging, biting, skin-picking, eye-poking, is common and can be life-threatening. It is markedly elevated in specific genetic syndromes, Cornelia de Lange, Cri du Chat, Fragile X, Prader-Willi, Lowe and Smith-Magenis among them, with Lesch-Nyhan syndrome the paradigm of compulsive self-mutilation as a defining phenotype.^{50,51,52} Crucially, it is often a behavioural equivalent of untreated pain: a documented case of severe, near-fatal self-harm resolved only when somatic pain sources were systematically treated.⁵³ People with intellectual disability carry a high physical and mental multimorbidity at younger ages than the general population, which is exactly why a new or escalating behaviour mandates a medical and psychiatric search before any behavioural attribution.^{54,55}

TABLE 3 The behavioural equivalents: the sign that stands in for the symptom

Underlying state	Common behavioural equivalent in intellectual disability
Depression	Loss of acquired skills, social withdrawal, irritability and aggression, disturbed sleep and appetite, new or worsening challenging behaviour
Anxiety	Avoidance, agitation, increased vocalisation, somatic complaints, challenging behaviour triggered by change to routine
Mania	Overactivity, reduced sleep, increased and pressured vocalisation, irritability, behavioural escalation
Psychosis	Fearfulness, responding to unseen stimuli, a discrete change in behaviour from a settled baseline
Pain or physical illness	Self-injury, aggression, agitation, refusal of food, guarding; resolves when the cause is treated

A behavioural equivalent is a prompt to assess, not a diagnosis. The same conduct can sit in any of these rows, which is why the equivalent narrows the search but the cause is confirmed by the work-up, not by the behaviour.

Assessment here reorders ordinary psychiatric practice. Collateral history and direct behavioural observation outrank self-report, and the more severe the disability the more weight shifts to the informant, until in profound disability the assessment is wholly observational. Self-rating is feasible only in mild disability, and even there it must be cross-checked. This is why almost every validated instrument in the field is built around a carer, and why the diagnostic frameworks were rewritten to accommodate how disorder actually presents.

The frameworks. The DC-LD was devised because ICD-10 and DSM-IV demonstrably miss disorder in adults with intellectual disability; in its field trial it agreed with expert clinical opinion in 96% of cases.³⁸ The DM-ID, the North American adaptation of DSM, is rated easy to use and reduces reliance on vague residual categories.⁶ Both encode the same insight: criteria written for a narrator must be adapted for a patient who communicates through behaviour.

TABLE 4 **The instruments, and when to reach for them**

Instrument	Type	What it is for, and when to use it
DC-LD	Diagnostic criteria	RCPsych multiaxial criteria for adults with ID; the default framework where standard manuals undercount
DM-ID-2	Diagnostic criteria	Adaptation of DSM for ID; specifies behavioural-equivalent criteria across the verbal range
PAS-ADD interview	Semi-structured, parallel	Interviews the person and a key informant in parallel, wording tiered to verbal level; the structured route to a defensible diagnosis
Mini PAS-ADD	Informant interview	Lets trained support staff identify likely disorder and trigger referral; good agreement with expert case recognition
PAS-ADD Checklist	Lay-language screen	A 29-item carer screen with three threshold scores; a first-pass case finder for community teams
GDS-LD	Depression, self + carer	Glasgow Depression Scale with a Carer Supplement; cut-off 13 gives high sensitivity and specificity in mild ID
GAS-ID	Anxiety, self-report	Glasgow Anxiety Scale for mild ID; validated state-anxiety self-report, cross-culturally tested
ABC	Informant, problem behaviour	Aberrant Behavior Checklist; five robust factors (irritability, lethargy, stereotypy, hyperactivity, inappropriate speech); tracks change and treatment response
DASH-II	Informant, severe-profound	Built for severe and profound ID where self-report is impossible; relies entirely on observation
Reiss Screen	Dual-diagnosis screen	Screens for co-occurring psychiatric disorder; total score validated, used in Indian samples

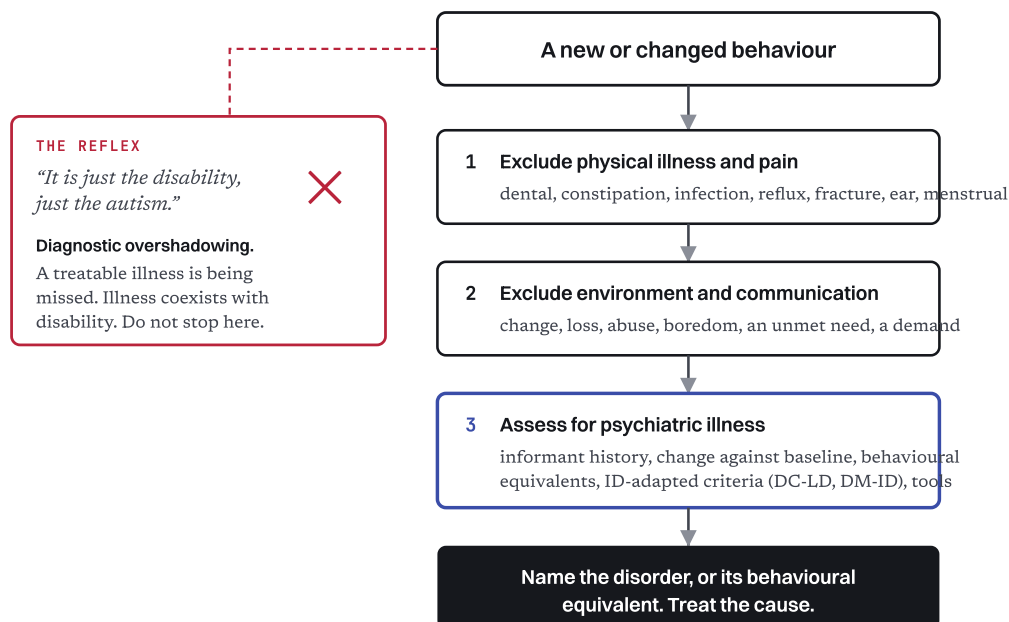
Choose by verbal level and question: a framework to diagnose (DC-LD, DM-ID-2), a structured interview to assess (PAS-ADD family), a symptom scale to measure and follow (GDS-LD, GAS-ID, ABC, DASH-II), or a screen to case-find (Checklist, Reiss).^{39,56,57,58,59,60,61,62} The instruments force the right examination and make change measurable across visits, which is how a baseline becomes a comparator.

The whole assessment can be drawn as one figure. A changed behaviour is worked through in a fixed order, physical first, environment second, psychiatric third, while the reflex that ends the assessment before it begins, blaming the disability, is named and set aside. Overshadowing is the default error; the figure exists to interrupt it.^{2,5}

FIGURE 2 **A changed behaviour: the readings, in order**

WORK THE READINGS, IN THIS ORDER

OVERSHADOWING IS THE DEFAULT ERROR



Each step is gated: treat what you find and reassess before moving on. The order is safety logic, the cheapest and most reversible causes first.

If no disorder is found, the behaviour itself is the target: functional analysis and positive behaviour support.

HOW TO READ THIS Work top to bottom in fixed order, physical first and psychiatric last, gating each step: treat what you find and reassess before moving on, and the behaviour may resolve there. The struck red box is the reflex to reject, not a step, and naming it out loud is what keeps the team from stopping at it.

WORKED THROUGH

A man with severe disability begins refusing food and hitting himself

He cannot say what is wrong, so the behaviour is the message. The reflex says it is the disability; the figure says work the order, and the examination finds a dental abscess. Treating it settles the behaviour within days, with no psychiatric label needed. The point is not the abscess; it is that the order found the cause the label would have hidden.

Management rests on a hard evidence anchor. In the landmark trial, haloperidol, risperidone and placebo were compared for aggressive challenging behaviour in adults with intellectual disability; aggression fell in all three arms but active drug was not superior to placebo, a difference that did not reach significance. Antipsychotics, the authors concluded, should no longer be a routine treatment for it.⁶³ The guidance that follows puts psychological, behavioural and environmental work first, and warns against letting challenging behaviour become a pseudo-diagnosis a drug is aimed at.⁵⁵ The over-prescription is the corollary: in a large cohort half of adults with intellectual disability had a psychotropic prescription but only a fifth a recorded mental illness, the prescribing driven by challenging behaviour, autism and dementia, not psychosis.⁶⁴

Why start low, and go slow. Adults with intellectual disability on antipsychotics have more movement side effects than matched controls and a several-fold higher rate of neuroleptic malignant syndrome, so the same dose carries more harm.⁶⁵ A drug used here should have one target, the lowest dose, a time limit and active monitoring, and it should be reviewable: long-term risperidone could be withdrawn in most patients without a rise in irritability and with metabolic gains, the evidence under STOMP, Stopping Over-Medication of People with intellectual disability, autism or both.⁶⁶ STOMP advances where teams are resourced and stalls where they are not, and behavioural care must be delivered well rather than assumed, since positive behaviour support training alone did not reduce challenging behaviour in a cluster trial.^{67, 68, 69}

TABLE 5 The management principles, and why they bind harder here

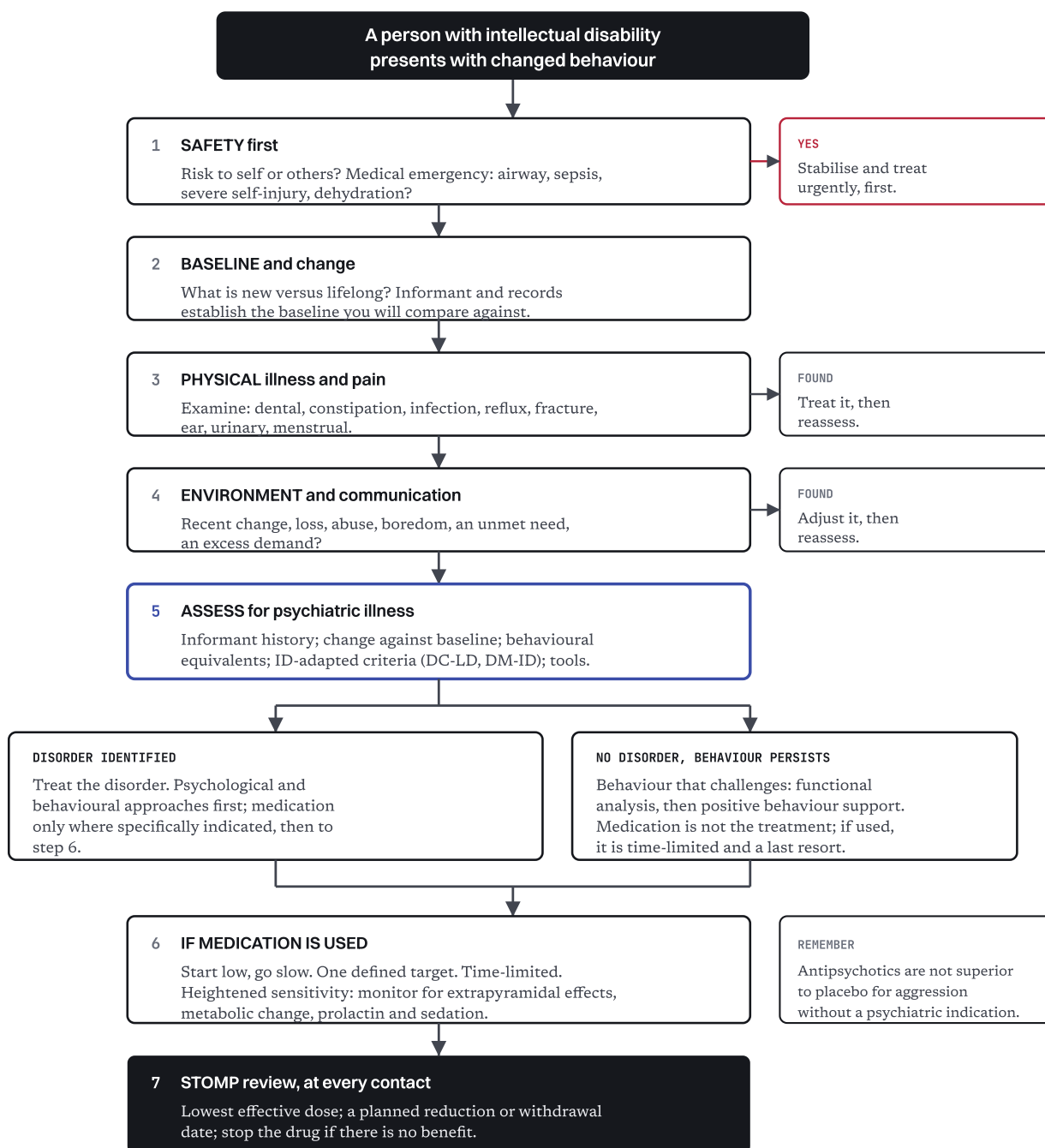
Principle	In practice	Why it binds harder in IDD
Treat the cause, not the behaviour	Work the readings; treat pain, illness, environment or the psychiatric disorder you find	Behaviour that challenges is a final common pathway, not a diagnosis
Behavioural and psychological first	Functional analysis, positive behaviour support, environmental change before any drug	Drugs are not superior to non-drug care for aggression, and carry more harm
Start low, go slow	Lowest starting dose, slow titration, one change at a time	Heightened sensitivity to EPS, sedation, metabolic and prolactin effects
One target, time-limited	Define the symptom, set a review date, stop if no benefit	Standing prescriptions accumulate; the indication is often never revisited
Monitor actively	EPS, weight and metabolic panel, prolactin, sedation, and the target itself	The patient may not report the side effect any more than the symptom
Review and deprescribe (STOMP)	At every contact reduce toward the lowest effective dose or withdrawal	Withdrawal is feasible and beneficial; over-medication is the default to undo

INDIA

What the service reality holds

Terminology has moved: the Rights of Persons with Disabilities Act 2016 adopted intellectual disability and set a 40% threshold for benchmark disability and certification, while leaving the screening instruments unclear.⁷⁰ The system is thin and family-centred, specialist services barely five decades old and city-concentrated, the family the lifelong carer and primary informant, and the treatment gap above 80%.^{71, 73} So lean on the informant and the baseline rather than absent specialist testing; use a validated India tool where one exists, the NIMHANS screener in children and ID-specific screens such as the Reiss in adults, and treat comorbid pain and physical causes first; functional, time-limited, monitored, reviewable prescribing is within reach without specialist drugs.^{72, 74}

FIGURE 3 A person with intellectual disability presents with changed behaviour



Each numbered step is gated: treat or adjust what you find and reassess before moving on. Steps 1 to 4 run cheapest-and-most-reversible first; the psychiatric assessment at step 5 is reached, not skipped to.

The reflex to read it as "just the disability" is the one move this whole sequence exists to prevent.

HOW TO READ THIS Follow the numbered spine; each step is gated, so treat what you find and reassess before descending; the side boxes are exits, not detours.

Blaming the disability. The commonest and most damaging error is diagnostic overshadowing: reading a new symptom as part of the intellectual disability, so a treatable illness is never sought. It was shown experimentally and is not cured by experience, which marks it as a structural reflex rather than a knowledge gap.^{2,4} The modern evidence says it is real but not universal, so the corrective is not despair but a habit: when a behaviour changes, ask what is new, not what is expected, and work the readings before reaching for the disability as the answer.⁵

Treating the behaviour instead of the cause. Behaviour that challenges invites a prescription, and the prescription is usually an antipsychotic. But for aggression without a psychiatric indication the antipsychotic is no better than placebo and adds real harm, and behaviour that challenges is a final common pathway, not a diagnosis.^{63,65} The error is to medicate the topography and skip the function. The fix is the work-up: a functional analysis and a search for pain, illness, distress and environment, because the same behaviour can be any of them.^{53,54}

Mistaking the behaviour for a diagnosis, or for none. The two opposite failures meet here. One is to seize on a behavioural equivalent and call it depression because the person is aggressive, when aggression has many causes and the same behaviours used to diagnose then circularly confirm it.^{7,23} The other is to dismiss a genuine, treatable illness as merely behavioural. Between them sits the discipline of this issue: a behaviour change is a prompt to assess against a baseline, neither a diagnosis to assume nor a noise to ignore.

QUICK CHECK

A non-verbal adult with intellectual disability is aggressive and is given an antipsychotic for it. What does the trial evidence say, and what was skipped?

- A For aggression without a psychiatric indication, the antipsychotic was no better than placebo and added harm; what was skipped is the work-up, the functional analysis and the search for pain, illness, distress and environment.^{63,65}

HOLD THIS**The one lesson, if you keep nothing else**

A changed behaviour in a person who cannot easily tell you what is wrong is a signal, not a diagnosis. Before you attribute it to the disability, exclude physical illness and pain, then environment and communication, then assess for psychiatric illness against a known baseline and with an informant who knows the person. Psychiatric illness coexists with intellectual disability far more often than it is found, it is treatable, and overshadowing is the reflex that keeps it hidden. Treat the cause, not the behaviour, and where a drug is used, use the least of it for the shortest time, and review it every time.

Psychiatric illness in intellectual disability is common, treatable, and missed for reasons that can be designed around. Almost all of the skill is reading a behaviour for what it is, in a fixed order, against a baseline, with the right informant.

WHAT THIS ISSUE COMES DOWN TO

- 1 **The behaviour is shown, not spoken.** As the disability deepens, self-report thins and then vanishes, so illness reaches you as a change in conduct. A documented baseline and an informant who knows the person are the instruments that read it; the deeper the disability, the more the assessment is observational.
- 2 **Work the readings in order.** Exclude physical illness and pain, then environment and communication, then assess for psychiatric illness. The cheapest and most reversible causes come first, and the psychiatric assessment is reached, not skipped to.
- 3 **Overshadowing is the default error.** Blaming the disability is the reflex that hides a treatable illness. Counter it deliberately: ask what is new, not what is expected. Each disorder presents differently here, but it presents.
- 4 **Treat the cause, prescribe sparingly, review always.** Behaviour that challenges is a final common pathway, not a diagnosis; antipsychotics are no better than placebo for aggression without a psychiatric indication and carry more harm here. Behavioural and psychological approaches come first, and any drug is one defined target, the lowest dose, time-limited, monitored, and reviewed under STOMP.

REMEMBER

**SAFETY • BASELINE • PHYSICAL • ENVIRONMENT • PSYCHIATRIC • then
PRESCRIBE**

Work a changed behaviour down the gated order: secure safety, fix the baseline comparator, exclude physical illness and pain, then environment and communication, then assess for psychiatric illness, and only then prescribe, sparingly and under STOMP review.

BEDSIDE CHECK

A person with intellectual disability is behaving differently

- 01 Is anyone unsafe, or is this a medical emergency? Stabilise and treat that first.
- 02 What is the baseline, and what exactly has changed? Ask the informant who knows the person, and the records.
- 03 Could this be pain or physical illness? Examine for dental, constipation, infection, reflux, fracture or ear before anything else.
- 04 Has something in the environment or relationships changed, or is a need unmet? Adjust it and reassess.
- 05 Only now: is there a psychiatric illness, judged against baseline, with behavioural equivalents and an ID-adapted framework? If a drug is used, what is the one target, the dose, and the review date?

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The Rights of Persons with Disabilities Act 2016, the STOMP programme (NHS England, 2016) and the DSM-5 and ICD-11 severity frameworks are cited as primary documents and statute rather than indexed evidence. Indian service-availability detail reflects current practice and national survey data, not a single trial. Antipsychotic-induced movement disorders are covered in Aporia 09; treatment resistance and clozapine in Aporia 08.

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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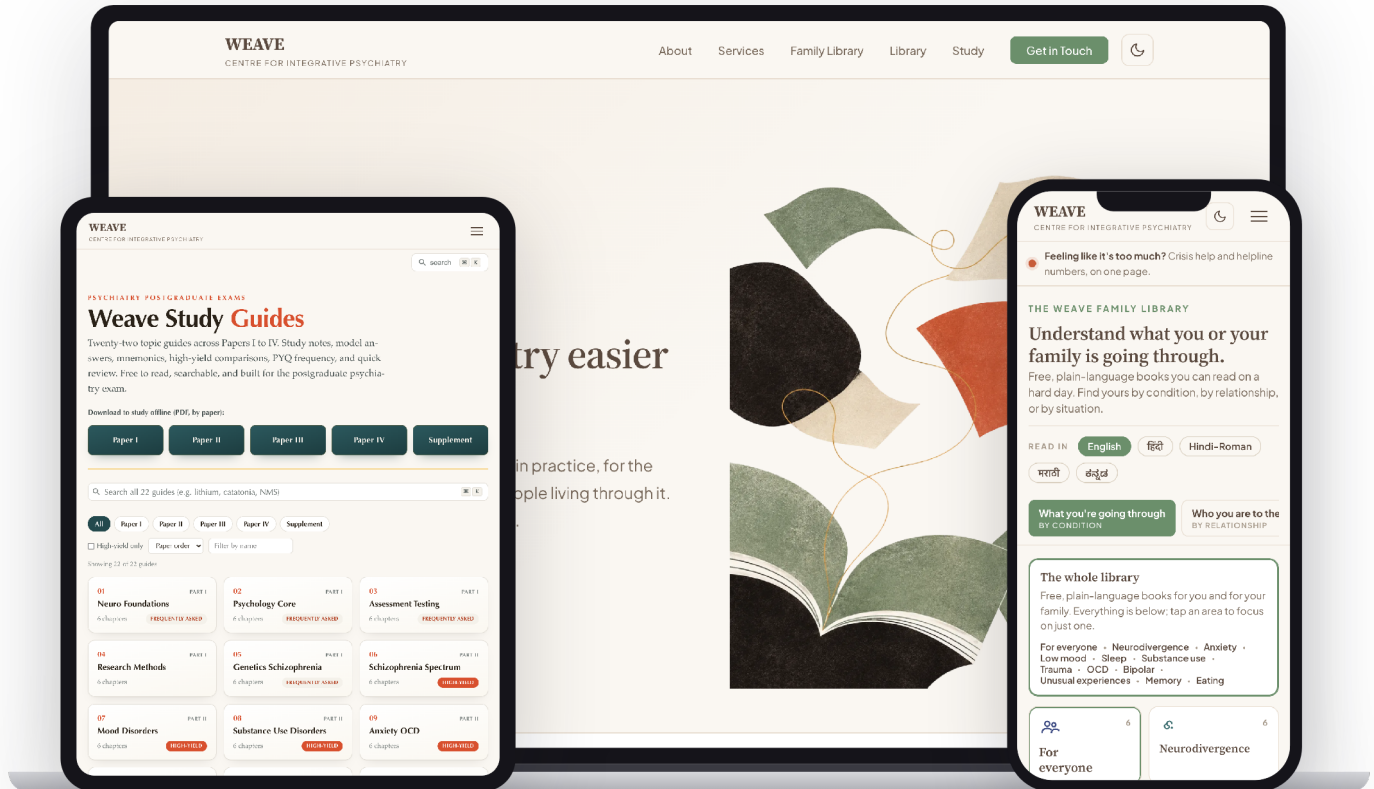
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